UNIVERSIDAD COMPLUTENSE DE MADRID

FACULTAD DE FILOSOFÍA

TESIS DOCTORAL

Variational Probabilities and Developmental Propensities: a Philosophical Study of Chance in Evolutionary Variation
Probabilidades Variacionales y Propensiones del Desarrollo: Un Estudio Filosófico del Azar en la Variación Evolutiva

MEMORIA PARA OPTAR AL GRADO DE DOCTOR

PRESENTADA POR

Cristina Villegas Cerredo

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Madrid, 2020

A Juanma y a mi abuela Antonia

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ABSTRACT

The ongoing debate over a possible extension of the explanatory corpus of evolutionary biology touches many aspects of philosophical interest, among which is the role that chance plays in its models and explanations. In particular, how evolutionary variation relates to chance seems to differ under the classical and the evo-devo perspectives. While some tools of the philosophy of probability and chance have been incorporated into important aspects of evolutionary biology, this discrepancy has not been considered from this perspective. In this dissertation, I intend to bridge part of this gap by endorsing a conception of chance in the generation of evolutionary variation that is the result of incorporating several conceptual tools from the philosophy of probability and chance into different views over the nature of evolutionary variation. My aim is to clarify the distinct roles that chance in variation plays in the field of evo-devo as compared with classical evolutionary genetics. I depart from the construction of a suitable philosophical framework about the representative role of probabilities in evolutionary disciplines and the type of explanatory causes that are responsible for them. I call this framework the causal propensity view, where probabilistic dispositions or propensities are causally responsible for structuring a sample space of possibilities to which probabilistic measures can be in principle applied in different contexts. From this view, I consider the main probabilistic notions in evo-devo and evolutionary genetics models and explanations. I argue that probabilistic notions of the classical picture, notably selection and drift, can be understood as causal propensities. However, I also defend that this picture forbids any recognition of causes at the level of the generation of evolutionary variants, namely in the production of the sample space from which extant variants can later be sampled through selection or drift. This is, I argue, primarily due to the classical conception of 'chance variation', according to which variation acts merely as the 'raw material' of evolution, and can be relatively taken for granted for explanatory purposes. By contrast, I claim that some evo-devo models of phenotypic evolution identify the probabilistic causes of

variation in development. While they generally do so without explicitly invoking formal probabilities, they certainly introduce both dispositional and probabilistic notions about evolutionary variation, which can in turn be more meaningfully understood as causal propensities. The principal result of this dissertation is the construction of a causally grounded conception of chance based on evo-devo models of phenotypic variation and grounded on the different propensities to vary of developmental types, notably their variability, robustness, modularity, plasticity, and evolvability. I conclude that these propensities invite for a reconsideration of our ways to understand chance and variation in evolutionary explanations. On the one hand, their typological nature need not be in conflict with classical population thinking, inasmuch as developmental types are the evolutionary result of both development and populational processes. Nonetheless, it does serve for overcoming the limitations of population thinking in accounting for chance in evolution and for evolutionary patterns of unity and diversity. On the other hand, this approach enables to consider development as an ultimate, and not only a proximate, cause of evolutionary transformation, inasmuch as it not only concerns the actual but the evolutionary possible.

Resumen

El actual debate sobre una posible extensión del corpus explicativo de la biología evolutiva recoge muchos aspectos de interés filosófico, entre los que se encuentra el rol del azar en sus modelos y explicaciones. En particular, la relación entre la variación evolutiva y el azar parece ser muy distinto bajo las perspectivas clásica y de la evo-devo. Mientras que algunas herramientas de la filosofía de la probabilidad y el azar han sido incorporadas en aspectos importantes de la biología evolutiva, esta disparidad no ha sido considerada desde esta perspectiva. En esta tesis, mi intención es aliviar parcialmente esta carencia defendiendo una noción de azar en la generación de la variación evolutiva que es el resultado de incorporar varias herramientas conceptuales de la filosofía de la probabilidad a distintas perspectivas sobre su naturaleza. Mi objetivo es clarificar los distintos roles que el azar en la variación juega en el campo de la evo-devo en comparación con la genética evolutiva clásica. Comienzo con la construcción de un marco filosófico que considera el rol representativo de la probabilidad en las disciplinas evolutivas y el tipo de causas explicativas que son responsables de ella. Llamo a este marco el de las propensiones causales, donde las disposiciones probabilísticas, o propensiones, son causalmente responsables de estructurar un espacio muestral de posibilidades al que pueden aplicarse medidas de probabilidad bajo distintos contextos. Desde esta perspectiva, considero las principales nociones probabilísticas de los modelos y explicaciones de la evo-devo y la genética evolutiva clásica. Argumento que las nociones probabilísticas del marco clásico, en particular la selección y la deriva, pueden entenderse como propensiones causales. Sin embargo, también defiendo que este marco prohíbe cualquier reconocimiento de causas probabilísticas al nivel de la generación de variantes evolutivas, es decir, en la producción del espacio muestral desde el cual las variantes existentes pueden ser fijadas con posterioridad por selección o deriva. Esto se debe, según defiendo, principalmente a la concepción clásica de 'azar variacional', según la cual la variación actúa meramente como la 'materia prima' de la evolución, y puede darse relativamente por supuesta a efectos

explicativos. Por contra, defiendo que algunos modelos de evolución fenotípica de la evo-devo identifican las causas probabilísticas de la variación en el desarrollo. Aunque normalmente lo hacen sin invocar probabilidades de forma explícita, estos modelos introducen afirmaciones probabilísticas y disposicionales acerca de la variación evolutiva, que por tanto pueden ser entendidas como propensiones causales. El principal resultado de esta tesis es el desarrollo de una concepción de azar causalmente fundamentada, basada en los modelos evo-devo de variación fenotípica y en las diferentes propensiones a variar de los tipos del desarrollo, en particular su variabilidad, robustez, modularidad, plasticidad y evolucionabilidad. Concluyo que estas propensiones invitan a reconsiderar nuestras formas de concebir el azar y la variación en las explicaciones evolutivas. Por un lado, su naturaleza tipológica no entra en conflicto con el pensamiento poblacional clásico, en tanto en cuanto los tipos del desarrollo son el resultado evolutivo tanto del desarrollo mismo como de procesos poblacionales. Sin embargo, esta tipología sí sirve para superar del pensamiento poblacional limitaciones en lo que concierne a dar cuenta del azar en la evolución y de patrones evolutivos de unidad y diversidad. Por otro lado, esta aproximación permite considerar al desarrollo como una causa última, y no sólo próxima, de transformación evolutiva, en tanto que no solo ocupa lo actual sino también lo evolutivamente posible.

Introduction

Nuestros historiadores, que son los más perspicaces del orbe, han inventado un método para corregir el azar; es fama que las operaciones de este método son (en general) fidedignas; aunque, naturalmente, no se divulgan sin alguna dosis de engaño.

Jorge Luis Borges

It is common to find the idea that the philosophy of science used to focus during many decades from its emergence on the theoretical aspects of fundamental physics, not only leaving aside other questions concerning scientific practice, but also every other science altogether. The situation nowadays is by far more inclusive and plural, philosophy of science encompassing modeling practices, methodological concerns and, above all, other scientific traditions. The philosophy of biology is now an important branch within the philosophy of science and is gaining more weight in recent years, making it clear that the complexity of biological systems and the different scientific practices around them are of philosophical interest in their own sake. Classical philosophy of science topics such as reduction, pluralism and realism find their particular applications to the biological realm (Rosenberg 1994, Gould 1997, Brigant & Love 2008), while genuinely biological problems such as the nature of genes or species open up new interesting debates (Beurton et al. 2000, Mayr 1995). The classical position has been that biological explanations, models and practices seem to demand a philosophical perspective that is not directly translatable into the

classical approach to the philosophy of physics (Ayala 1972, Mayr 2004). However, the influx of ideas coming from the physical sciences is also widely recognized (Sober 1984), a combination that brings about new complexities and turns some aspects of the philosophy of biology particularly interesting.

Among the topics that have been philosophically considered is what role chance plays in some biological phenomena. Not only biological models are typically probabilistic in nature, but the very characterization of chance and randomness in the processes underlying them is an important matter of discussion among biology theoreticians and philosophers. For example, considerations on the role of stochastic perturbations in development, the randomness of mutations in the genome, the probabilistic nature of natural selection and the nature of genetic drift are only but a few of the topics of interest in theoretical biology that deal with chance (Merlin 2015, Keller 1992, Otsuka 2016). Chance can be regarded as a metaphysical concern that relates to ontological indeterminism, far from scientific practices and especially from so-called non-fundamental sciences. Nonetheless, the introduction of probabilities in scientific modeling at all levels of description turns chance into a philosophy of science matter too. The philosophy of probability is the classical approximation to the role of chance in scientific domains. Indeed, it has grown as one fundamental piece of the philosophical tools for thinking about science, inasmuch as probabilities both take part in many scientific models and also seem to refer to empirical facts (Eagle 2011). However, the philosophy of probability and chance are typically associated with physical and simple gambling systems, with relatively few applications to other scientific domains despite their use of probabilistic claims and models. Certainly, the tools of philosophy of probability and chance have not been fully incorporated into the philosophical study of biology, despite the general agreement on chance playing an important role in biology. In particular, only some probabilistic notions of classical evolutionary genetics seem to have been approached from this perspective in depth. This thesis concerns the role of chance in evolutionary biology in a broader sense than this classical approach, partially filling this gap in the philosophy of biology. In particular, it intends to cast some light into

the different positions about the role that chance plays in evolutionary variation from distinct evolutionary disciplines.

Evolutionary biology is notably the core and the most precious field of the life sciences, unifying the vast diversity of living phenomena under a single idea: all living beings on earth, from amebas to coral reefs to human beings, are the products of the same unique process, with a single origin over more than three billion years ago, and following the same basic rule of reproduction with modification thereafter. The crucial role that evolution plays in the biological sciences is usually stressed by the famous quote by Theodosius Dobzhansky: "Nothing in biology makes sense except in the light of evolution" (1973). It is no surprise then that evolution has been the main focus of attention for philosophers of biology. Despite the increasing number of works devoted to other branches of the life sciences (e.g. the essays in Sarkar & Plutynski 2011), evolution remains the central piece of philosophical interest (Pradeau 2017). Not only its centrality makes evolution especial in this regard, but also the fact that evolutionary biology is undergoing in the last few decades a fascinating debate over the apparent necessity to revise its classical theoretical pillars (Pigliucci & Müller 2010, Laland et al. 2015, Huneman & Walsh 2017). While modern evolutionary biology arose during the first half of the 20th century out of a synthesis of neo-Darwinism and the genetic theory of inheritance, other approaches to evolution, such as comparative morphology and paleontology, were famously left behind in mainstream theoretical reconstructions. The growth and inclusion of these traditionally absent domains in recent years are nowadays important sources of philosophical discussions about the very explanatory structure of evolutionary biology.

The role that chance plays in evolution is one philosophical aspect touched by this ongoing discussion. The classical picture of the life sciences opposes evolutionary biology to the rest of biological fields, stressing that they are not only committed to the description and understanding of different types of phenomena, but they confer with different types of explanations altogether. Ernst Mayr (1970) famously divided the biological sciences into those dealing with the historical origin of living beings, and thus with their *ultimate* causes in a historical sense—the fields

of evolutionary biology—; and those dealing with the functioning of particular living phenomena, and thus with their *proximate* causes—the fields of 'functional biology'. He illustrated this with the behavior of migratory birds. We may wonder about the physiological and behavioral causes of a particular bird migrating in a population. In this case, we wonder about the *proximate* causes of migratory behavior, and thus about *how* the bird migrates. In contrast, we may wonder why such behavior evolved, and thus ask about its evolutionary causes. In this case, we are concerned with *ultimate* causes, and we are asking *why* it is that birds migrate rather than *how* they do so. These questions are in turn supposed to require different types of explanations.

Evolutionary questions are responded to by alluding to the three basic components of the evolutionary process, namely variation, inheritance and differential reproduction (Lewontin 1970). Crucially, these components are not equally considered in evolutionary explanations, where differential reproduction, based on natural selection, typically plays the main explanatory role. The principle of natural selection, namely the greater tendency of fitter variants to increase their relative frequency in a population, has been the central idea under the classical studies of evolution. In this classical picture, chance in evolution is associated with the lack of explanatory power of any other evolutionary factor, on the basis of their alleged randomness. In the recent incorporation of different approaches to the study of evolution, however, the randomness of some non-selective evolutionary factors seems to be questioned. In particular, evolutionary developmental biology, or evo-devo, in studying the developmental bases of phenotypic evolution, vindicates the non-random character of variation and its explanatory salience.

In this thesis, I approach this discrepancy by incorporating some tools from the philosophy of probability and chance into the views of both classical evolutionary genetics and evo-devo. This exercise will involve the philosophical study of the meanings of several dispositional and probabilistic concepts regarding variation in evolution in these distinct scientific practices. In particular, I consider how probabilistic evolutionary models and explanations refer to the causes of evolutionary variation in these two fields. My position throughout this thesis will be that, in opposition to what has been classically assumed, the potential of fitness for

accounting for the possible in evolution is strongly limited. This is because, in addition to what can be selected, the evolutionary possible needs to incorporate what can be produced in the first place. In turn, the probabilistic notions of evo-devo ought to be as well incorporated in order to have a more encompassing view of chance in evolution. As we shall see, this conception aligns with the revision of other theoretical pillars of classical evolutionary biology, such as externalism, gene centrism and population thinking, all of which are being philosophically reconsidered in recent years (Pigliucci & Müller 2010).

My proposal is that evo-devo variational tendencies, notably variability, modularity, robustness, plasticity and evolvability, shall be considered as probabilistic causes of evolutionary change, inasmuch as they are explanatory of the developmentally possible. My view is that, while the models of evo-devo variational tendencies generally don't allude to formal probabilities, the salience of evo-devo claims about evolutionary variation can be more meaningfully approached from a philosophy of chance and probability perspective. In doing this, I introduce a separation between vernacular, expected and realized variational tendencies that makes sense out of the apparent lack of consensus about the role of chance in evolutionary variation. This, as I will argue, is not only an introduction of proximate factors—i.e. development—into evolutionary explanations, as it is now generally agreed that evo-devo entails. It is also an expansion of the type of ultimate factors that can answer 'why' evolutionary questions.

I begin this dissertation developing a conceptual framework for the discussion of chance in evolutionary explanations: that of causal propensities (Chapter 1). Departing from the recognition of the probabilistic nature of evolutionary models and the philosophical interest of the notion of chance in evolution, I explore the main positions in the philosophy of probability and their consistency with some important features of probabilistic claims in evolutionary biology, namely the representative nature of probabilities, the explanatory role of dispositions, the non-entailment of indeterminism and the contrastive character of explanations. I firstly review the classical opposition between frequency and propensity views of probability, the former identifying probabilities with the

frequency of events, the latter with their generating conditions. Then I consider the complex relation that classical conceptions of probability hold with causation. This will eventually lead me to those philosophical positions that regard chance as a primitive notion embedded in the causal structure of the world, and probability as an objective measure of chancy phenomena. While these positions have traditionally been concerned with probabilities in physical systems, exemplified by gambling systems, here I consider their applicability to complex evolving biological systems. In this regard, I will argue that chance in evolution is concerned with the possible as derived from explanatory causes, and that constructing probabilistic models is the way to link chance in evolution with the random character of evolutionary patterns. Finally, leaning on some theoretical advances in the understanding of explanations and probabilistic dispositions, I conclude that a good way for analysing the explanatory structure of evolutionary biology is what I call the causal propensity view, according to which propensities are explanatory causes that structure a space of possibilities to which the mathematical tools of probability calculus can be applied in principle. Through this view, the propensities advocated for in causalist views of evolution need not represent the mathematical probabilities of evolutionary models, but rather the causal notions underpinning their application.

In the second chapter of this thesis, I present the main philosophical ideas regarding the chancy nature of variation in evolution through the conceptual tools of the causal propensity view. I begin by outlining the classical explanatory schema of evolutionary genetics, both in its historical derivation and in contemporary probabilistic models, with a special focus on how variation is conceptualized. In applying the causal propensity framework to evolutionary genetics, I argue that propensities have an explanatory role in the statistical models of population dynamics as responsible for the possible in ecological patterns of differential reproduction. In other words, I support the idea that there is a causal grounding for the probabilities of population dynamics associated with population thinking, notably fitness and genetic drift. The chapter then deepens into the so-called "problem of variation", and shows how the generation of variation in evolution, as well as the ideas about possible variations, have received a very different treatment.

In particular, I show that the notions of chance related to how variation is generated completely overlook the causal processes underlying it. I argue that the statistical treatment of variation, together with the population thinking ingrained in evolutionary explanations, is responsible for the general neglect of how variation is produced and what is its impact in contemporary debates over the probabilistic nature of evolutionary biology. This conclusion leads me to the vindication of the evo-devo approach to evolutionary variation, which, contrarily to evolutionary genetics, is interested in patterns of phenotypic variation and concerns the developmentally possible. Analogously to how the propensities of individuals, traits and populations—notably their fitness as dispositions—ground the explanatory (or creative) role of selection in population dynamics, I argue that phenotypic patterns of variation can be explained by developmental causes. I conclude that there is room for conceptualizing specific variational probabilities as based on development from the point of view of causal propensities.

The third and final chapter develops the causal propensity understanding of evo-devo variational probabilities. I begin by reviewing evo-devo models of variation and their conceptualization of the possible as based on general developmental properties. For doing this, I present the genotype-phenotype map as the core tool in evo-devo for abstracting away these properties and studying the variational tendencies they ground. Genotype-phenotype maps translate genotypic variation into phenotypic variation, thus enabling the recognition of developmental patterns of phenotypic transformation, insofar as they establish a range of possible phenotypic changes under genotypic transformations. I then deepen into the general variational dispositions these models present, namely their variability, robustness, modularity and plasticity, as well as their evolutionary origin. These capacities are distinct ways of exploring the space of possible phenotypic changes, and thus they are causally responsible for possible variations. I further argue that understanding them as causal propensities enables a conceptualization of chance in variation that differs from the received statistical view and incorporates significant recent advances in evolutionary biology into the philosophical debate over chance in evolution. Particularly, these developmental propensities ground the probabilistic nature of the very generation of

variation, enabling an important separation between vernacular, expected and realized variational tendencies. I then show that this perspective demands a revision of some of the philosophical pillars underlying the classical picture of evolution. On the one hand, this notion of chance is a direct challenge to the explanatory scope of population thinking in evolution, and indeed seems to demand the inclusion of typological explanations. On the other hand, the externalist picture favored by adaptationism reveals itself as incomplete insofar as the internal tendencies of living systems seem to determine their capacity to evolve or evolvability. I conclude with some remarks about how the ongoing expansion of the classical picture of evolution needs to incorporate not only 'how questions' in the study of evolution, but also new types of 'why questions' beyond those that can already be answered by fitness and drift.

CHAPTER 1

A Framework For Chance In Evolution: Causal Probability And Propensities

I have hitherto sometimes spoken as if the variations - so common and multiform in organic beings under domestication, and in a lesser degree in those in a state of nature - had been due to chance.

Charles Darwin

0. Introduction

As advanced in the general introduction, exploring the notion of chance in evolution demands the establishment of a conceptual framework encompassing the role of probability in explanations. Chance in evolution has been largely associated with—though rarely restricted to—probability (Gayon 2005, Millstein 2011, Ramsey & Pence 2016), and many of the discussions on its proper role have concerned either philosophical interpretations of probability—notably the propensity interpretation (e.g. Mills & Beatty 1978)—or probabilistic notions such as randomness or sampling. Additionally, the distinct notions of chance are entangled with the explanatory structure of evolutionary theory, chance being considered as an important

explanatory notion in evolution (Millstein 2006, Ramsey & Pence 2016). In this regard, it is worth to notice that not only propensities but dispositions more generally play a prominent role in biological explanations (Hüttemann & Kaiser 2019), and particularly in the evolutionary (Ramsey 2016) and evo-devo explanatory agendas (Austin & Nuño de la Rosa 2018). However, these ideas are usually considered in isolation, there lacking a general philosophy of chance perspective to the probabilistic explanations of evolution. With all this in mind, in this first chapter my aim is to present the ideas about probability, chance, dispositions and explanation that will be necessary in the remainder of this thesis for discerning an appropriate understanding of evo-devo vindications with regards to the chancy character of variation in evolution.

Philosophers have been concerned with the nature of probabilities insofar as they are both an indispensable tool of scientific inquiry and directly applicable to the behavior of rational subjects. Although a primitive notion of probability can be traced back as far as the rise of civilization goes, probability did not become a field of interest on its own until the seventeenth century (Hacking 1990), and the mathematical axiomatization of probability calculus would only take place in the twentieth century through the work of Andréi Kolmogorov. His *Foundations of the Theory of Probability* (1933) inaugurated not only a new branch of mathematics, but also a new philosophical concern: what is probability? Why does Kolmogorov's axiomatization seem to apply almost universally? The enterprise of interpreting probability and analysing it became the focus of the newborn field of philosophy of probability. The criteria usually considered for such an endeavour are *admissibility*—i.e. satisfying probability calculus—, *ascertainability*—i.e. the possibility to assign a probability value—and *applicability*—i.e. being applicable to common uses of probability[1]—(Salmon 1966). To what extent any of the existing interpretations of probability satisfies all of these criteria is debated extensively in the literature, but it

[1] What exactly involves the applicability criterion is a widely discussed topic, but it typically includes that probabilities should be non-trivial—there should be room for probabilities different from 1 and 0—and that they can be applied to frequencies of events, to the rational belief of subjects, and to ampliative reasoning (Hájek 2019).

seems that considering these restrictions as regulative principles is at least reasonable for exploring the nature of probabilities.

The main philosophical debate over probabilities in evolution is the so-called statisticalists-causalists debate (reviewed in section 2.1 of Chapter 2). In this debate, the main traditions in the philosophy of objective probability, namely the frequentist and the propensity accounts, have been represented. These traditions essentially concern the interpretation of probability calculus. That is, their focus is to establish what are the truth conditions of numerical probabilities that behave according to Kolmogorov's axioms. However, the philosophical interest in chance and possibilities in evolution relates to a primitive notion of probability that runs independently of its mathematical axiomatization. In particular, the classical, Laplacian notion of probability as ratios of possibilities, that is, as a fraction of what is considered possible, is key for understanding this broader role of chance in evolution. Although probability calculus is essential to all models in evolutionary biology, and although the philosophical discussion about the nature of evolutionary theory has been impregnated with discussions on the *interpretation* of the mathematical probabilities invoked in it, this role of chance is arguably a broader and prior topic of philosophical significance that dates back to pre-Darwinian explanations of biological diversity. What is possible and what is likely in the living world, as well as how that relates to biological causes, are classical concerns that surely involve, but are not reducible to, the probabilistic models of contemporary biology. As I will defend in this chapter, the project of interpreting probabilistic notions of evolutionary theory is inseparable from the attempt to consider this broader concern on the role of chance in evolutionary explanations. The endeavor of ascribing truth conditions to the probabilities of evolutionary models will be incomplete unless a connection between them and a broader notion of chance in the evolutionary process is made.

A major concern regarding the probabilistic nature of evolution is to what extent it can be considered as an objective feature of the evolutionary process. If evolutionary biologists use probabilities in their models and are engaged with a probabilistic modus of explaining, it is certainly not only because they necessarily lack an exhaustive comprehension of every determinant involved in evolution. It is

also, and primarily, the result of a specific means of reasoning and representing nature that is embedded in the scientific practice more generally. Our scientific enquiries and explanations of natural phenomena demand our capacity to abstract properties away from their usual context, to idealize conditions, and to imagine a range of possible though unrealized scenarios. It is these capacities that allow us to build explanatory models and theories and thus to treat a given phenomenon as a probabilistic one, namely as one in which there is a defined scope of possibile results, each of them with a certain value or strength associated. Whether or not we understand the explanatory structure of our theories as representing reality is a matter that will depend on how we interpret this representing process. My view here is that there always are causal hypotheses involved in the modelling of evolutionary probabilities, which renders this modelling a representational process. In this process, I will argue, dispositions—and particularly propensities—play an important role.

In this chapter, I provide a framework for understanding chance in evolution based on what I will call a 'causal propensity' approach. In doing so, I argue for the explanatory role of propensities both in probabilistic scientific models and in less formalized notions of chance. Although the explanatory role of dispositions is widely discussed in the metaphysical literature, the particularity of propensities explaining probabilities has been, to my knowledge, much less examined. Here I argue that considering this explanatory role helps in building a general conceptual structure that enables to regard chance in non-fundamental sciences in general, and evolutionary biology in particular. In section 1, I review the classical philosophical discussion on objective probability, specifically with regards to the frequentist and the propensionist traditions. These traditions are widely influential in current discussions on evolutionary theory—frequencies for statisticalist views, propensities for causalists. Although so far constrained to the ecological level, these discussions, as we shall see in the next chapters, will be relevant for assessing the role of development in evolution. Thus, here I present the main positions with regards to them as well as their limitations. I conclude that neither frequency views are tenable from an explanatory perspective, nor any propensity account so far satisfies the interests of causalist advocates of evolution. In section 2, I make the case for an

association between probabilities and causation beyond the received views of the philosophy of probability. After reviewing how objective probability relates to causation, I present the problem of chance in evolution more generally as part of the broader philosophical concern about the nature of chance. I argue that relating the explanatory role of chance in evolution with probabilistic models demands abandoning the task of interpreting the calculus of probability and considering instead the causes of probabilistic behaviour. In section 3, I introduce the notion of a 'causal propensity' to defend that propensities play this role of explaining probabilistic patterns acting as causal hypotheses structuring a space of possibilities. In order to do so, I explore the possible ways in which dispositions, and more precisely propensities, can be said to explain probabilistic phenomena, as well as their relation with probabilistic modeling. This general framework will allow me in the following chapters to assess the probabilistic notions of evolutionary models and the ideas about chance associated to them, both with regards to the fate of variants in evolution (Chapter 2) and their very origin (Chapters 2 and 3).

1. The Received View On Objective Probability

In many contexts, probabilities are associated with *how much* we know about the world or *how strongly* we believe in certain statements. We say that something is likely if it is coherent with our own expectations and beliefs about the world. When we assign a probability value to a coin landing heads up when tossed ($\frac{1}{2}$), we are expressing something about our own knowledge of the tossing process: namely that it has two possible outcomes—heads and tails—and that we do not know which one will turn out to be the outcome of a particular toss. Epistemic accounts of probability tend to assume that this is all there is to probability, and that whenever they are invoked we are simply referring to phenomena whose details are not sufficiently determined to be predicted with accuracy. In this line, the logical interpretation of probability (Carnap 1962) is concerned with the degree of implication or entailment between propositions, therefore with how much a description of events is supported by evidence, its development establishing the grounds for a logic of induction or

confirmation theory (Hájek & Hitchcock 2016). Meanwhile, the subjective interpretation (F Ramsey 2016/1926, de Finetti 2017/1970) is interested in degrees of belief of (rational) agents, namely in how much confidence (rational) subjects have in a particular description of events.[2]

But if there is a sense in which probabilities are closely related to epistemological considerations, many philosophers have argued that there is another sense in which they are linked not to our knowledge but to objective features of the world. This seems to be particularly relevant when it comes to the probabilities involved in scientific practice: claims about the probability of a certain organism to survive in a specific environment or the probability of a radium atom to decay within the next two hours seem to refer to how the world behaves rather than to what we know about it and how we know it. Moreover, some events seem to take place with a certain frequency that bears a relation to our probability statements, and this frequency is an empirical fact of the world. As the law of large numbers establishes, the frequency of an event will tend, in the limit, to its probability value. Objective accounts of probability, then, intend to make sense of this intuition, linking probability assignments to objective properties of reality, and situating their truth-makers in the real world. In particular, an objective view of probability will be concerned with explaining why probabilities, in addition to being an epistemological tool, seem to explain and predict empirical frequencies of events (Eagle 2011). Although entirely epistemic interpretations of the probabilities in evolution have been proposed (Graves et al. 1999), the relevance of epistemic probability in the mainstream discussions of the structure of evolution has been limited (see next chapter), which suggests a real concern among philosophers about the relationship between probabilistic models of evolution and the nature of the evolutionary process. Consequently, monist epistemic notions of probability—namely those that interpret all probability statements in epistemic terms—will not be considered here for the sake of simplicity.

[2] My interest in associating logical and subjective interpretations is based on my will to stress their epistemic nature by contrast to objective interpretations, but I don't mean that they refer to the same type of conception necessarily. See Hájek (2019) for an argument over the different nature of the concepts entailed by these traditions.

Needless to say, the relationship between objective and epistemic probability is a tricky one, to the extent that many authors claim that virtually no probability statement is fully epistemic nor objective (Ramsey 2016/1926, Gillies 2000a). Some pluralistic views on probability also recognize the hybrid nature of assigning a probability value to an event or a proposition. Notably, the language of events for objective probability can be straightforwardly translated into the language of propositions for epistemic probability (Handfield 2012). Thus, we can either talk about the probability of the coin in my hand landing heads next time I toss it, or about the probability—or degree of confidence or confirmation—of the proposition instantiated by the sentence "the coin in my hand will land heads next time I toss it." However, this is not the only reason for not drawing a neat distinction between objective and epistemic probability. As it will become apparent throughout this chapter, articulating a detailed account of objective probabilities without making reference to epistemic and pragmatic notions is not entirely possible. Nevertheless, this does not render the distinction useless. Far from it, it enables the recognition that, on the one hand, we make use of our means of knowledge to describe a phenomenon of the world as probabilistic and that, on the other hand, we make use of our ideas about the world to assess our degree of belief in a certain proposition.

As I will address in the next two sections (2 and 3), many refinements regarding the role of causation and explanation are convenient for an objective interpretation of probability to be prolific in the endeavor of understanding the probabilities of evolutionary theory. Moreover, as we shall see in section 2, the classical view of probability—understood as ratios of the possible—that inaugurated the philosophical concern on chance precedes the interpretations of Kolmogorov calculus. However, much of the discussion on probabilities in evolution has been influenced by the classical framework of objective interpretations of probability. Therefore, in this section I review the main interpretations that have been traditionally developed from the objective probability position, namely the frequency and the propensity accounts. Although other interpretations of probability can now be considered as classical as well (see Section 2), the debate on evolutionary probabilities has been focused on the relation between probabilities and empirical

frequencies of evolutionary events, and has therefore inherited the classical frame of *interpreting* the probability calculus, frequency and propensity views being referent on this matter.

1.1. The Frequency Interpretation

The interest on situating probabilities in the world starts with Venn's (1876) and Von Mises' (1957/1928) frequency interpretation, which inaugurated a frequentist tradition of thought that, despite abundant, and generally decisive criticism (Eagle 2004), is still taken as a reference in the philosophy of probability. This tradition trivially satisfies the condition of explaining the relationship between probabilities and frequencies: it identifies one with the other. The frequency theory identifies probabilities with the relative frequency of favourable cases—or of the occurrence of an attribute—in a series of events. That is, given a sequence of events—a series of coin tosses, say—, the frequentist will look for the proportion between those events with a particular attribute—landing heads up—and the total. Frequency interpretations then remain close to the classical Laplacian understanding of probability (to be reviewed in section 2) in that they make use of a ratio of favourable cases in order to interpret what probability is. But they depart from such conception to the extent that they do not make reference to the possible but to the actual instead. In this sense, this family of interpretations is framed inside an empiricist tradition: the probability of a specific kind of event is nothing more than the frequency with which it takes place in the world. For instance, the probability that the coin in my hand will land heads is just the frequency of the event type 'landing heads' when coins are actually tossed, i.e. one-half or 0.5. Similarly, a frequentist would consider the probability of a character to spread in a population as nothing but the relative frequency with which it is transmitted in that particular population.

In order for an event to have a probability it must therefore belong to a sequence or class of events in which a relative frequency of outcomes can be determined, that is, it must be part of a reference class. In the case of the event 'landing heads', the reference class may be 'types of coin tosses results.' This

establishes a class of events from which to calculate relative frequencies. What are the conditions that such a sequence must satisfy will determine a particular type of frequency interpretation. Broadly speaking, there can be said to be two different frequentist accounts. Finite frequentism was Von Mises' first proposal. Although almost abandoned by philosophers of probability, this view is still relevant for statistical theorists, insofar as it provides with an operational definition of probability that suits basic statistical practices. Finite frequentism defines the probability of a type of event as its relative frequency in a finite sequence of actual trials. That is, it defends that there is nothing but the real frequency of events, and only this frequency constitutes a probability. Needless to say, this view faces a number of problems (Eagle 2011, Hájek 1997, 2009). First of all, it attributes counterintuitive probabilities to some events, especially when they belong to a series with few or no members. Imagine that we want to establish the probability of landing heads of a (fair) coin that has never been tossed. If we consider that the event landing heads belongs to the series of events 'tossing coins'—or tossing fair coins—, we may claim that such probability is $\frac{1}{2}$ or nearly so. But we can consider instead that it belongs to the sequence of tosses of that particular coin, in which case the probability could not be assigned. Moreover, if we assign it after tossing the coin once, it will necessarily be either 1 or 0, both options far from what we intuitively believe to be the probability of landing heads for the coin.

A related problem arising from this view is that it cannot make sense of single case probabilities, that is, of the probability of an event that is not repeatable. For instance, according to finite frequentism, it does not make sense to attribute a probability to the origination of life on earth, for it is an unrepeatable event altogether. Von Mises illustrates this point through his 'probability of death' example (1957/1928, pp. 16-18). According to it, when insurance companies assign a specific probability of death to a new client, they are actually categorizing that client in a particular collective in which the relative frequency of deaths is empirically established by statistical methods. That way, if a certain client is assigned the probability of death 0.011 at age 40, it is because she belongs to a collective where 11 out of 1000 people die at age 40, but it is nonsense to claim that she has that

probability as an individual. In Von Mises' words, "[t]he phrase 'probability of death', when it refers to a single person, has no meaning at all for us." (p. 357). These difficulties, as we shall see in the next chapter (section 2), constitute the basis of the so-called 'tautology problem' in evolutionary biology, namely the circularity embedded in attributing an explanatory role to fitness—a key probabilistic term in evolution—when understood merely as a relative frequency.

In the light of these difficulties, most frequentists moved towards so-called infinite or hypothetical frequentism. Contrary to finite frequentism, this view considers infinite sequences of trials in order to establish the reference class of an event. In his *Experience and Prediction* (1938), Hans Reichenbach defines probability as "the limit of a frequency within an infinite sequence" (p. 68). Since infinite sequences are idealizations, the advocates of this approach make use of the mathematical notion of limit—from where the 'hypothetical' nature of the frequencies comes. This means that frequentists identify the probability of an event with its limiting relative frequency, that is, with the proportion to which it would tend if the sequence it belongs to were to be running *ad infinitum*. The finite collectives found in the world are merely evidence for establishing the idealised collectives that define probabilities, where trials go on infinitely and frequencies converge into a limit. Hypothetical frequentism therefore identifies the probability of an event not with the actual frequency with which it takes place, but with the frequency with which it would do so if the trials ran infinitely. The single case problem arises in different terms in this case. Thus, it could be argued that it is possible to ascribe a probability to an event even if it is not part of a real series of trials, for events must only be part of idealized series, not empirical ones. In this view, the untossed coin would have a probability of landing heads if tossed of $\frac{1}{2}$ in virtue of the toss belonging to a class of events—Von Mises calls it a collective—where the relative frequency of the outcome 'landing heads' tends to $\frac{1}{2}$ in the limit. Nevertheless, the problem of assigning probability to single events with no counterparts remains uncontested.

This move implies abandoning the empiricist approach to probability. For example, Reichenbach admits that the notion of limit lacks an operational meaning,

and defends instead that the probability calculus demands an idealization (1938, p. 348). Although still relying on properties of the real world in order to interpret what probability is, it demands referring to abstract mathematical properties as well, with little or no empirical interpretation. Still, the most salient problem that arises out of this position is one that shares with its finite counterpart: the so-called reference class problem (Hájek 1997, 2009). When it comes to establishing the relative frequency of a specific event, is the right reference class always determined? If being a relative frequency is a sufficient condition for being a probability, aren't there an infinite number of potential reference classes for each event? Getting back to our untossed coin, why should we use the set of trials of all fair coins instead of the set of trials of untossed coins for establishing its reference class? I could even consider the coin as a member of the collective of items on my desk if it was there where it was situated. Although a number of responses have been suggested (e.g. Kyburg 1983), the general consensus is that this problem is not entirely overcome by frequency advocates (Hájeck 2007). As we shall see in the next chapters, the reference class problem is at the core of some debates on the role of chance in evolution, especially with regards to the distinction between selection and genetic drift at the ecological level (Millstein 2002, Strevens 2016), and with the notion of evolvability (Love 2003). In these discussions, it is patent that the probability of a particular evolutionary event may differ greatly depending on the referent class considered, particularly on what is taken as the referent environment—e.g. a trait will have a defined probability of spreading in a population only once the referent environment of the population considered is established.

Alan Hájek (1997, 2009) famously developed up to thirty different—though interrelated—arguments against frequentism that are now considered to establish that the frequency view cannot be a full interpretation of probability. Four of them apply to both finite and infinite frequentism generally. The already mentioned reference class problem is the first of them (1). The other three also problematize the idea that probabilities depend on collectives or reference classes. On the one hand, the election of a particular collective seems to require having a notion of relevance in order to discern what similarities are required and what differences allowed in the

collective (2). On the other hand, the probability of an event—such as my coin landing heads—should not be counterfactually dependent on the occurrence of other unrelated events—like other coins landing heads or tails—regardless of their belonging to the same collective (3). Finally, no frequentist approach makes sense of single probabilities, that is, of probabilities as properties of single events (4). Following Hájek, finite frequentism faces a number of specific difficulties in addition, some regarding inconvenient ontological consequences (5, 9, 11), others intuitive ways in which finite frequencies cannot be identified with probabilities (8, 10, 12, 13, 14, 15). Moreover, he argues that the theory fails to give a satisfactory connection between probabilities and frequencies in explanatory and operational terms (6, 7). Hypothetical frequentism, on the other hand, faces its own difficulties, especially with regards to the possibility that idealized series may converge in values that do not resemble probabilities and with the already mentioned abandonment of empiricism.

The frequency tradition has barely been explicitly vindicated in discussions of evolutionary probabilities. However, as I will argue in the next chapter, it is the implicit position of those advocating a statisticalist view of them. That is, the statistical—in the sense of non-causal—approach to evolutionary models assumes that all there is to probabilities in those models is the relative frequency of the phenomena they represent. In this sense, it is my claim that there is an important line of thought in the philosophy of evolution that relies on this frequentist account, despite its patent flaws, for arguing against a causal nature in the probabilistic models of evolutionary explanations.

1.2. The Propensity Interpretation

If frequency accounts refer to classes of events and the frequencies of their properties, a different tradition in the philosophy of objective probability, the propensity interpretation, makes reference to the *generating conditions* of events. According to propensity advocates, events have a probability value in virtue of the properties of those conditions that bring them about. As we will see in the next

chapter (section 2), this tradition has a large influence in the causalist picture of evolution.

a) Peirce's 'would-be'

Although he did not coin the term 'propensity', Charles S. Peirce did inspire the propensity tradition. Peirce believed that probability is a certain *would be* that a physical system has, namely a property "quite analogous to any *habit*" that a person "might have" (1994/1910, stress in original). This proposal was developed as an amendment to his earlier ideas on probability as quantitative logic, where he supported that probability was the frequency of truthness of a means of inference (Berkovitz 2015). The novel picture acknowledged that frequencies refer to the infinite long run and thus are necessarily hypothetical, while probabilities are predicated of the physical systems that *would* bring about these frequencies. Consequently, probabilities are properties that realize frequencies of outcomes, but are not identifiable with such frequencies, similarly to how the habits of humans relate to the repeated actions they perform (Peirce 1994/1910).[3] The connection between these properties and frequencies of events is one of causation (Fetzer 1993), insofar as 'would-bes' are displayed in frequencies but not defined by means of them. Therefore, frequencies are in his view the *manifestation* of probability. Although the proposal is ambiguous when characterising these properties (see e.g. Fetzer 1993, Berkovitz 2015), it has the advantage of situating chance in the world as a separate, *explanatory* character of its manifestation in sequences of events. As we will see in the remainder of this section, this idea has been very prolific and led to the elaboration of a family of propensity interpretations of probability.

b) Popper's first introduction

The propensity theory as such was developed by Karl Popper from the idea that the probabilities involved in certain subatomic phenomena of quantum nature are real properties of singular events and can physically interact with each other under

[3] Peirce's account of chance is incorporated into his pragmatistic views on inference and induction (Peirce 1994/1878), from which the relation between 'would-bes', the probabilities of singular events and inverse probabilities can be elaborated (see Burch 2018).

specific circumstances (Popper 1957). Popper argues that the concept of propensity introduces a dispositional property of events that explains observable frequencies, analogously to how forces explain observable accelerations in Newtonian mechanics. His first argument for it is framed in the discussions regarding the conceptual foundations of quantum theory. It consists in its ability to eliminate subjective "disturbing" elements from the theory interpretation, namely the idea that the probabilities involved in quantum phenomena are merely instrumental, when the experimental results suggest that certain probabilistic distributions behave as if they were not independent from each other. In a nutshell, some of the probabilities involved in quantum mechanics demand a sample space that includes incompatible properties, such as observable position and momentum. Their incompatibility resides in the experimental nature of quantum events, that is, the events cannot be defined irrespectively of the experimental arrangement (Hughes 1989). This implies that some of these probabilities do not add obeying the Kolmogorov calculus of probability, but they do so following the 'Born rule', which is related to abstract mathematical properties of the equations describing the state of the entire system.[4] The result is that empirical frequencies are determined as if probabilities, as real forces, physically interacted depending on the experimental arrangement measuring the same system. This "physical interaction" of probabilities demands, from Popper's view, interpreting them as real properties of quantum systems, that is, as propensities (Popper 1957, 1959).

Nevertheless, and similarly to Peirce's insights, Popper found independent arguments for supporting his propensity interpretation of probabilities that did not rely on quantum phenomena. In his (1959), Popper shows that a purely statistical view of probability cannot deal with the probability of singular events unless their *generating conditions*—and not merely the class of events they belong to—are established. These generating conditions refer to the physical situation, or the experimental arrangement, that gives rise to such singular events. Propensities are then introduced as dispositional properties of physical situations, dispositions to

[4] As Eagle (2004, p. 387) points, "the additivity axiom does not in general hold: for quantum mechanical observables, $Pr(A \cup B) \neq Pr(A) + Pr(B) - Pr(A \cap B)$."

produce certain outcomes with a particular strength that can be measured by their relative frequency of occurrence in the long run. Therefore, if the same physical arrangement gives rise to a series of outcomes in which the event *a* has a relative frequency of $\frac{1}{6}$—for instance, the case of landing six when throwing a die—then this frequency *is not* the probability of such event, but a measure of it. The probability, Popper claims, is in the dispositional properties of the physical arrangement: in its physical propensities.

Popper illustrates this point with an example of a series of outcomes that, according to him, cannot be accounted for from a purely statistical view of probability (1959, pp. 29-34). Let us assume a sequence of dice throws *b* where the probability of the event 'landing six' is $\frac{1}{4}$. For producing this sequence, we use two different dice: a fair, even die with a symmetrical structure, and a loaded die whose weight distribution increases the frequency of landing six in the long run. Let us assume that we know that only three trials, which we call the sequence *c*, are done using the fair die in the larger sequence *b*. The probability of the *b* sequence of landing six is still $\frac{1}{4}$, but a subset of this sequence, the subset *c*, has a probability $\frac{1}{6}$ of landing six. Now, Popper argues, although the sequence—the reference class—*c* of fair trials belongs to the sequence—the reference class— *b*, there is no doubt that the singular probability of the throws in it is determined only by their membership of *c* and not by their membership of both *c* and *b*. Since we assumed that *c* has only three members, the relative frequency of landing six in *c* can only be either 0, $\frac{1}{3}$, $\frac{2}{3}$ or 1. Each of the trials in *c* belongs to both sequences *b* and *c*, where the relative frequency of landing six is $\frac{1}{4}$ and $\frac{1}{6}$ respectively. But *we know*, by invoking the physical properties of a fair die, that the *real* relative frequency of landing six in *c* is $\frac{1}{6}$. Moreover, we know that the true probability of landing six in *each* single trial of *c* is $\frac{1}{6}$. Thus, Popper claims, it is necessary to refer to the generating conditions of events in order to define the probabilities involved in them. In this example, Popper says, the frequentist will have to refer to the physical properties of the system that generates the series of outcomes, rather than merely to the reference class, and will therefore no longer be a frequentist but a propensity advocate. Indeed, a frequentist

would need to refer to two distinct hypothetical limits for each subsequence. However, the point for Popper is that they would do so by virtue of the physical generating conditions of each of them, which separates them from the view that probabilities are merely frequencies.

In this view, probability is then understood as a property of physical conditions, and not as a property of some given sequence. It is thus a *physical hypothesis*—perhaps a metaphysical one, adds Popper at this point—that systems generate physical propensities. These physical propensities are the truthmakers of probability statements: physical systems exhibit probabilistic behavior insofar as they have certain dispositional properties. From this perspective, frequencies are the means by which these propensities are empirically tested. If relative frequencies equal probabilities it is solely in virtue of the tendencies or dispositional properties of those conditions that generate them. This account thus identifies objective probabilities with the dispositional properties of physical systems: the probability of landing six of a fair die is its disposition to do so when certain throwing conditions are given, or the propensity of fair dice throws to produce the event landing six in the long run. This propensity to produce the event in the long run would, Popper claims, be responsible for a propensity to produce a certain outcome in single events as well. This is an extended view with regards to the probabilities of evolutionary models: they refer to the dispositions of biological systems to generate certain frequencies of outcomes. As we shall see throughout this thesis, there exist a number of matizations that need to be introduced in order to appropriately discern how dispositions are invoked in evolutionary probabilities.

Popper concludes his proposal with some remarks about how propensities relate to the classical view of probability and to Aristotelian potentialities. On the one hand, although it may be said that the main distinction between the classical and the propensity views is merely that the latter introduces 'objectionable metaphysical' properties to the classical picture (Popper 1959, p. 35), Popper argues that, on the contrary, the appeal to 'possibilities' in the former implicitly assumes dispositional properties. This is so because, according to him, measuring possibilities has always a predictive function. Whereas possibilities as such are not interpreted as tendencies to

realise themselves, probability measures on them are in fact a measure of such tendencies. That is to say, when we estimate the weight of a certain possibility, we are measuring its dispositions. On the other hand, Popper draws a distinction between propensities and potentialities in the Aristotelian sense—an important matization from the point of view of biology, given the anti-Aristotelian flavor of the modern evolutionary framework (see next chapter). Unlike potentialities, Popper warns, propensities are relational properties. The experimental arrangement that gives rise to a probabilistic series of events, namely those conditions that are constant throughout its repetition, has a certain tendency to produce an outcome. Such tendency is an abstract property of the conditions as a whole, and it therefore differs—according to Popper—from the Aristotelian notion of potentiality, which is inherent to individual things.

Generating conditions are key to understanding the probabilistic nature of evolutionary biology. They are at the core of the discussions regarding the so-called propensity interpretation of fitness (section 2.2 of next chapter), but also—and importantly—they are indispensable for discerning types of trials embedded in distinct ways of understanding possible variations in evolution, especially when comparing the classical evolutionary genetics framework with the evo-devo one (sections 3 and 4 of next chapter).

c) Further developments of the propensity theory: single-case and long-run

A number of versions of the propensity interpretation have originated after these first proposals, including a second construction from Popper himself (1990). While the interpretation can still be considered as one of the main ways of understanding probability in the philosophical literature, it has not gone through without difficulties and ramifications. Reviews tend to classify propensity theories in long-run and single-case categories, depending—in broad terms—on whether they identify propensities with dispositions to generate sequences of a certain kind or they define them as dispositions to produce single results with a given strength, respectively (Kyburg 1978, Fetzer 1988, Gillies 2000b, Berkovitz 2015). As we have seen in the previous section, in Popper's first views, propensities are dispositions to produce

events in the long run, but they are responsible for a propensity to produce a certain outcome in single events as well. This renders the "first Popper" (Runde 1996) both long-run and single-case, insofar as the long-run definition of the disposition is applicable to individual events in a derivative way (Gillies 2000b).

Therefore, even though propensities have been traditionally associated with irreducible fundamental properties, there is a line of thought that defends a long-run approach to them. Peirce's 'would bes' would find their expression in "endless" or infinite series of repetitions (Peirce 1994/1910, p. 566), and Popper's first (both single-case and) long-run vision of propensities demanded "an extremely long (perhaps infinite) sequence of events" to manifest themselves (Popper 1959, p. 29). However, the main later works on long-run propensity theories recur to finite repetitions instead.[5] Ian Hacking (1965) is one of the main authors in this line. Although he refers to Popper's propensities, he notably chooses the word 'chance' instead for referring to the dispositional character of long-run frequencies:

> This is a dispositional property of the coin: what the long run frequency is or would be or would have been. Popper calls it a propensity of the coin, device, and situation. (...) It will be convenient to have a plainly dispositional word for our property—a brief way of saying what the long run frequency is or was or would have been. 'Probability' is often so used, but I eschew it here. So I shall resurrect a good seventeenth-century word which seems sometimes to have been used in just this sense—*chance*. (pp. 9-10, stress in original)

Thus, Hacking does not adhere explicitly to the propensity tradition, but he regards chance as a dispositional property of the *chance setup* in which trials are operated, and therefore to the physical generating conditions of sequences. As regards to chance setups, he describes them as "a device or part of the world on which might be conducted one or more *trials*, experiments, or observations; each trial must have a unique *result* which is a member of a *class of possible results*"

[5] The two long-run finite propensity views that I will review here characterize the long-run indirectly, thus avoiding the problems associated with operationalism. In his (2015) review, Berkovitz explains that "in these [Hacking's and Gillies'] theories, long-run propensity is a primitive term, which is characterized by the axioms of probability theory and various postulates that relate long-run propensity to other theoretical terms and statements about long-run propensities to experimental data." (p. 639)

(Hacking 1965, p. 12). In other words, Hacking considers those systems in which the random trials of a probability space can be conducted, and whose possible results define a sample space. That is, he regards them as systems defined by *kinds* of trials. Now, the propensities or chances of those systems are what the frequencies of outcomes *would be* in the long run (p. 86).

Donald Gillies (1973, 2000a, 2000b) also develops a theory of this kind. According to this author,

> a long-run propensity theory is one in which propensities are associated with repeatable conditions, and are regarded as propensities to produce in a long series of repetitions of these conditions frequencies which are approximately equal to the probabilities (2000b, p. 822).

What is key to this account is the repeatability of conditions. Similarly to Hacking's chance setups, the conditions that generate frequencies must be repeatable (in principle) in the long-run. That is, a propensity is a capacity of certain conditions to bring about a frequency of results in virtue of being repeatable in the long run. Single events, in this view, are not considered to have probabilities if their generating conditions are not repeatable. For instance, like in Von Mises' frequentist case above, the probability of death for one particular person lacks any meaning under this approach (see section 1.1), and so does the probability of life originating on Earth.

Gillies acknowledges that his may be the closest of propensity theories to the frequentist tradition, especially to Von Mises' view (Gillies 2000a, p. 137). However, he states that his account distances itself from Von Mises' operationalism, and that probability is a theoretical concept that is characterised axiomatically rather than defined. Thus, Gillies proposes that the theory of probability explains certain empirical laws—such as the Law of Stability of Statistical Frequencies—[6] in an non-operationalist fashion, through a set of axioms of probability "which are designed to provide a mathematical theory of observed random phenomena" (p. 160). In order to do so, he first develops a "falsifying rule" for relating theoretical probabilities to observed frequencies that is in line with statistical practice, and he

[6] i.e. the stability of relative frequencies given a large enough sample.

uses them in combination with the axioms to derive the empirical laws of probability. Thus, under Gillies' view, the mathematical corpus of probability is empirically and theoretically justified through propensities and rules of reasoning—an apparent assimilation of the necessarily dual nature of probability: objective and epistemic.

On the other hand, the single-case view of propensities has been explored by a number of scholars. One of the first approaches of this kind is that of Ronald Giere. In his (1973), Giere claims that his view is concerned with the physical probabilities involved in single events, regardless of their repeatability and their belonging to a particular class. According to his view, a propensity is a tendency to produce a specific outcome with a certain strength, rather than a tendency to produce a series with a particular relative frequency of outcomes in the long run. In other words, a propensity is a property of an individual physical situation that determines how strongly it tends towards the occurrence of a phenomenon. Likewise, Giere considers such tendency as a property of the trials of chance setups independently, and not of the repeatable conditions of chance setups. For example, in his view a coin toss has a $\frac{1}{2}$ propensity to land heads solely in virtue of the properties of the specific coin toss, including not only the structure of the coin but also the particular speed and angle of throw and the entire situation determining its particular conditions.

Giere's account entails an indeterministic ontology insofar as it deals with the problem of indeterminacy of an outcome by the precedent state of affairs. He agrees with the classic Laplacian view, according to which if the world is deterministic, then single-case propensities must have value either 0 or 1 (Giere 1973, p. 503). This is the case because, under his prism, determinism implies that processes have only one possible outcome, whereas a single-case propensity, on the contrary, involves the existence of mutually exclusive possible outcomes for exactly the same process, provided that its value is somewhere between 0 and 1. In this approach, therefore, indeterminism is assumed, and natural necessity is identified with the limiting case of unit propensities (Giere 1979). In turn, the connections involved in this picture are causal relations of different strengths, that is, propensities with several values, ranging from causal impossibility to causal necessity.

In subsequent works, Popper (2013/1983, 1990) abandones the idea that the propensities of single events are grounded in the properties of repeatable conditions, and he adheres as well to a view of irreducible, single-case propensities, thus abandoning his previous 'long-run' approach (Runde 1996; Gillies 2000a). Similarly to Giere's proposal, Popper's later theory establishes that "a tendency or propensity to realize an event is, in general, *inherent in every possibility*" (1990, p. 11). He identifies propensities with *weighted possibilities* that tend to realize themselves, defending that they are as physically real as Newtonian forces. Indeed, Popper states that his theory of propensities points towards a generalization of the idea of force, where the propensity value 1 is a classical force in action, whereas a non-zero propensity of less than 1 can be taken as there being competing forces tending towards opposed directions—a zero propensity, says Popper, is just no propensity at all. These propensities are, in Popper's words, "properties *of the whole physical situation* and sometimes even of the particular way in which a situation changes" (p. 17), that is, they are predicated of the particular situation that generates an event. David Miller (1994; 1996) deepens into this position by developing a 'state of the universe' version of it. Unlike Popper's claims, he argues that propensities are not placed in particular physical situations. On the contrary, Miller proposes that propensities are to be found in the complete state of the universe that brings about a particular event. This move intends to eliminate the dependence of single-case propensities to a reference class, and he defines a propensity as "a measure of the inclination of the current state of affairs to realize [an] outcome" (1994, p. 182).

A third single-case approach to propensities is that offered by James H. Fetzer (1981, 1982). He moves from Popper's view in the opposite direction of Miller, claiming that propensities are properties of only the set of *relevant conditions* in a situation that produces an event. Fetzer writes that propensities should not be thought to depend on the complete state of the universe at a particular time, "but upon a complete set of (nomically and/or causally) relevant conditions ... which happens to be instantiated in that world at that time" instead (1982, p. 195). Note that the appeal to relevance may introduce pragmatic considerations into this account,

distancing itself from the indeterministic ontologies implied in 'state of the Universe' versions of propensities.

All these accounts share the feature of being "tendency" theories of single-case propensities, where they are identified with probabilities. However, authors such as David H. Mellor (1969, 1971) and, more recently, Mauricio Suárez (2013, 2018), have developed "dispositional" theories of propensities, in which propensities are dispositions to display probability distributions (Berkovitz 2015) but are not identifiable with probabilities.[7] In Mellor's view, dispositions are those properties that explain "conditional regularities" (1971, p. 63), namely those that are displayed when certain conditions are met. Mellor follows Hacking (1965) in ascribing propensities to chance setups, which are devices in which trials of an experiment can be carried out. In addition, he states that it is convention what makes us attribute propensities to entities or chance setups rather than to particular trials:

> The view is that the feature that I have taken to be expressed in a chance distribution ascribed to trial of some kind should be regarded as the display of a dispositional property ascribed to more permanent entities (Mellor 1971, pp. 66-67).

Thus, Mellor claims that we usually assign the propensity of landing heads to coins (chance setups) as a convention, when it is coin tossings (trials) that actually contain it. However, and unlike Hacking, he argues that propensities ought to be predicated of *particular* trials rather than of types of them. That is, an individual trial (a given coin toss) has a propensity regardless its belonging to any particular type of trials. This way, Mellor manages to neatly separate propensities (individual dispositions of trials) from the probabilities or chances that display them (1971, p. 76). That is to say, it is the dispositional properties of my coin toss that explain its 0.5 probability of landing heads, a feature shared by all fair coin tosses. In addition,

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[7] Eagle (2004) distinguished these two kinds of single-case propensities as well, but labeled the second one as 'distribution display account' (p. 381). He proposes the following terms: "The first form, which we dub a *tendency* view of propensities, maintains that propensities are fundamental non-supervening properties that govern the production of probabilistic phenomena. The second form, due to Mellor, we dub the *distribution display* view; this view maintains that propensities supervene on other properties of the trial setup and are primarily proposed to explain the observed distribution of outcomes." I prefer Berkovitz's terminology for emphasizing the distinction between disposition and probability.

Mellor stresses that, in this view, the link of a propensity with a particular result of a trial is not one of causal strength. In particular, he rejects the view that propensities are tendencies towards particular results. On the contrary, the results of trials do not display propensities, but chance distributions do. Therefore the particular result of my coin toss is not *caused* by the propensity of the trial. What is caused by the propensity is the chance distribution of heads and tails results. Thus, a propensity is manifested in "the chance distribution over the possible results of the appropriate trial" (p. 70). Interestingly, Mellor concludes that the display of a propensity entails indeterminism, insofar as only possible results, and not particular ones, are determined by the properties of chance setups.

Similarly, Suárez develops a single-case dispositional account of propensities in a series of articles. He departs from a well-known difficulty that all propensity theories face, namely Humphreys' Paradox (see Section 2.1), and argues that a neat demarcation between propensities, the chance distributions that display them and the frequencies of results helps in solving it (Suárez 2013). In building up his approach, Suárez stresses that there are well-defined probabilities that cannot be propensities as well as propensities—specially time-dependent conditional propensities (see next section)—that "lack any plausible probability representation", and that they are therefore separate things (2014, p. 222). The relation he proposes between propensities and probabilities is, as in Mellor's case, one of manifestation.

However, unlike Mellor's, the proposal exposed by Suárez does not entail indeterminism. His views reclaim the pragmatistic motivations of Peirce's 'would-bes', and he therefore develops an approach where propensities are theoretical properties that *explain* probabilistic phenomena and thus play an explanatory role in modelling practices (Suárez 2017). In this view, propensities are not "sure-fire" dispositions but probabilistic ones, and they explain and ground probability distributions of single-cases, not necessarily in a causal fashion. Thus, unlike canonical dispositions, propensities have "more than one manifestation property with some probability" (Suárez 2013, p. 87).

Given this miscellanea of accounts, philosopher Antony Eagle (2004) presents up to twenty-one arguments against propensity interpretations of

probability, in a similar fashion to Hájek's (1997, 2009) critiques of the frequency theory. In his article, Eagle considers that the proliferation of distinct propensity interpretations of probability is an indication of the flaws the tradition suffers from. The author reflects:

> Why so many? (...) In this case, the number of distinct arguments [against propensities] indicates that there are a number of features of propensity analyses that have one of these problems. I think this will make clear the potential cost of adopting a propensity analysis: that one has to make considerable adjustments to the concept one tries to explicate, and the resulting concept fits poorly into pre-existing roles for the concept of probability. (Eagle 2004, p. 383)

Eagle divides his arguments between those applying to all propensity views and those referring to specific propensity accounts. Here, I will only point at some of the general arguments, that will be decisive for abandoning the terrain of analysing probability and entering the one of explaining it in the remainder of this thesis. The first argument Eagle poses is that propensities fail to establish the axioms of probability calculus. A propensity interpretation of probability ought to justify why a physical property behaves in accordance with the mathematical calculus of probability. Thus propensities have either to "primitively satisfy the axioms, or produce empirically accessible phenomena which do" (p. 384). However, as we shall see in the next section, every propensity account in the literature faces a limitation in this regard when it comes to conditional probabilities. A related concern is the fact that the law of large numbers is a mathematical, thus necessary fact (p. 391), while nothing in the nature of propensities seems to entail necessity. Finally, propensities face their own version of the reference class problem. On the one hand, long-run propensities inherit this problem from the frequentist account directly. For, if more than one physical situation can produce a particular event, how to determine which experimental arrangement defines the *real* propensity of the event? This is just a different version of the reference class problem. On the other hand, single-case versions need to incorporate reference to theoretical description in order to relate similar events to one another. That is, if propensities are well defined in the single-case, what grounds their application to the long run? Which properties of the

single-case are relevant for a potential extrapolation to further instances of the same propensity? A reference class would be needed in order to overcome this obstacle. As Hájek (2007) pointed out, in fact every interpretation of probability faces a version of this problem. Although all of these claims have been contested in the literature, the technical details of each propensity version differ greatly, and there is no consensus at all about how much any of them can be said to go without problems (see Berkovitz 2015 for a recent review).

Regardless of these limitations, the propensity interpretation of probability has been very fruitful in philosophical discussions of evolution, as we shall see in the next chapter. It has been so both with regards to the probability of survival and reproductive success of individuals—*prima facie* single-case—and to the probabilistic trends of character types throughout generations—*prima facie* long-run. The variety of propensity positions reviewed in this section provides some background to these discussions, while it manifests the complexity of coming up with a definite interpretation of the probabilities involved in any particular evolutionary model. As a matter of fact, some authors have argued that the applicability of a propensity interpretation to evolutionary probabilities has been overestimated by philosophers of biology. For instance, Marshall Abrams (2007) argues that most evolutionary probabilities have to do with recurrent circumstances rather than with inherent tendencies, while Isabelle Drouet and Francesca Merlin (2015) forcefully defend that the literatures on the propensity interpretation of probability and the understanding of evolutionary factors such as fitness as a propensity are concerned with different explanatory issues altogether. As we shall see, these authors indeed point at relevant considerations. Nonetheless, the propensity tradition remains as one of the most significant ones in the evolutionary panorama, especially with regards to the causal and explanatory structure of evolutionary models.

2. The Problem Of Chance And Causation

As mentioned above, the ideas of the propensity interpretation of probability have been particularly relevant for the causalist view of evolutionary theory, that is, for

those advocating that the models of evolutionary biology reflect the causal structure of the process of evolution. Causalists believe that the probabilistic notions used in these models—selection, drift, mutations, etc.—refer to the causal factors responsible for evolutionary changes (e.g. Sober 1984, Millstein 2006). In evolutionary discussions, then, causalists associate the *causes* of evolution with the *generating conditions* of evolutionary probabilistic events, and in turn with a propensity interpretation of evolutionary probabilities. In this regard, I think it is important to note that contemporary debates about evolutionary probabilities mostly concern the causal structure underlying evolutionary explanations and models. The classical, Laplacian tradition of the philosophy of chance, less influential in the causalists-statisticalists debates than frequencies and propensities, makes explicit reference to causal relations and processes in a way that could be helpful for assessing evolutionary explanations, besides being less concerned with interpreting probability calculus. While its ideas have been less prolific in the mainstream discussions of the philosophy of evolutionary probability, they have a strong connection with some evolutionary conceptions of chance, as we shall see in this section. I consider that it is important, in turn, to regard how causation relates to probability and chance in a broader sense than the received dichotomy between frequentists and propensionists, in order to establish the general conceptual framework that we are seeking for in the present chapter.

In this section, I explore some ways in which causation has been related to objective chance in order to expand the framework of discussion in the philosophy of evolution to other philosophical perspectives about chance beyond the received view on objective probability. Since, as we shall see in Chapters 2 and 3, causal explanations play an elementary role in the representational nature of evolutionary probabilities, I believe that discussions on the latter would benefit from a broader understanding of how causation relates to probability. In section 2.1, I review the relationship between propensities and causation, particularly with respect to the so-called Humphreys' Paradox. In doing so, I point at some limitations that propensities face with regards to playing the causal role attributed to the notions in probabilistic models of evolution. In section 2.2, I review a different tradition that has

only recently been considered in evolutionary theory (Abrams 2017, Strevens 2013): that of 'causal probability', which has its roots in the mathematical development of probability theory initiated by Poincaré's 'method of arbitrary functions' and I defend its applicability to evolutionary phenomena. In section 2.3, I explore the metaphysics and epistemology of chance, especially with regards to the problem of chance in evolution, and their relation to philosophical accounts of probability.

2.1. Causation In Propensities

As we have seen, some propensity views explicitly refer to the connection between the propensity and its effects as causal, notably Giere's and Fetzer's, while others reject it altogether, e.g. Mellor's. Perhaps not very surprisingly, it is precisely with regards to this issue that propensities have faced their greatest complications. Indeed, the attempts to relate propensities to causation through conditional probability are typically flawed. In particular, a propensity to produce the event E cannot be understood as the probability of E *given that* its own generating conditions obtain. For example, the propensity of my coin to land heads is in the dispositions of the trial 'tossing my coin'. However, this propensity cannot be represented as the probability of the event 'landing heads' given—or conditioned on—that I toss my coin. This may seem puzzling, for it could appear at first sight that propensities are fundamentally conditional because they refer to the probability of an event *when* certain generating conditions are met. In the previous example, the propensity of my coin to land heads is defined inasmuch as its generating conditions, such as the fairness of the coin and the tossing situation, are (causally) responsible for the landing. The intuitive way of formalizing this is through conditional probability: it is the probability of landing heads (H) *if* there is a fair coin tossed (F), that is, $P(H|F)$. However, the rules of probability calculus prevent any representation of propensities in this standard conditional form, a fact that has led some to argue that propensities are not a correct interpretation of probability.

More generally, probability calculus cannot easily be applied to causal connections. There is a well-known argument, first pointed out by Paul Humphreys

(1985), that states that there is an asymmetry in causal relations, not found in probability ones, that prevents them from obeying Bayes' Theorem—a theorem that allows a reversion of conditional probability and is usually considered as part of the classical probability calculus. Humphreys' argument goes as follows. Propensities, insofar as they are a kind of dispositions, possess the asymmetry of causal relations. That is, the propensity of a fair coin to land heads up when thrown is not corresponded by a propensity of a heads up coin to have been thrown fairly, or to be fair. The physical properties and the set up conditions of the throw have a causal influence in the heads up result, whereas landing heads up, by contrast, has no causal influence at all in the physical properties of the coin or the set up conditions. Yet, if the (objective) probability for a coin of landing heads given that it is fair is defined, then typically the (objective) probability for the coin to be fair given that it landed heads up is also defined. This way, an asymmetry prevents propensities from being applicable to any objective probability statement. This not only implies that not all objective probabilities are propensities, but also that most propensities cannot be represented as probabilities either. This is so because virtually every propensity can be represented in conditional form, but for most their reverse conditional probability would lack any plausible interpretation. Formalizing a propensity as conditional probability, in turn, would in most cases violate the classical probability calculus, for whenever an (objective) conditional probability is defined, its inverse must be defined as well, as states Bayes' Theorem:

$$P(A|B) = \frac{P(B|A)\times P(A)}{P(B)}$$

The theorem describes the probability of an event A conditioned on the occurrence of a different event B—$P(A|B)$—in terms of the probability of the event B given A—$P(B|A)$—and both unconditional probabilities—$P(A)$ and $P(B)$—, provided that $P(B) > 0$. So if the probability of landing heads (H) given that the coin is fair (F) is represented as $P(H|F)$, then $P(F|H)$ must be defined as well. While the first probability could be *prima facie* understood as a propensity, its inverse could not: nothing in the event landing heads (H) *generates* a fair coin (F). In Humphreys' words, "a necessary condition for probability theory to provide the

correct answer for conditional propensities is that any influence on the propensity which is present in one direction must also be present in the other" (Humphreys 1985, p. 557). The conclusion that he draws is that "the properties of conditional propensities are not correctly represented by the properties of conditional probability" (p. 559). This argument is known as the "Humphreys paradox". As we shall see, the argument intended to be a general claim against the criterion of admissibility with regards to objective chance. That is, Humphreys believed that any account of objective chance would disobey the Kolmogorov calculus of probability in the same way and, rather than abandoning propensities or related objective chance proposals, he defended that obeying the calculus is not a reasonable criterion for an interpretation of objective chance.

However, the different propensity approaches introduced in the previous section have dealt with this paradox in distinct ways, intending to overcome it. For example, it has been argued that conditional independence, the idea that the probability of an event is independent of other conditions, should not hold in instances of Humphreys paradox. Let us define the probability of the event *A*, a die landing on 4, as $\frac{1}{6}$. This probability is not independent from the event *B*, i.e. landing on an even numbered face:

$$P(A) = \frac{1}{6} \neq P(A|B) = \frac{1}{3}$$

That is, the probability of a die landing on 4 *given that* it lands on an even face ($P(A|B) = \frac{1}{3}$) is different from the unconditional probability of landing on 4 ($P(A) = \frac{1}{6}$). Unlike Humphreys paradox examples, in this case we can calculate the reverse conditional probability by conditionalizing *B* on *A* (i.e. landing on an even face conditioned on landing on 4). This move is possible because *B* is also an event in the sample space of the die roll: the probability of landing on an even face is well defined by the same generating conditions. The unconditional probability of *B* in this example is $P(B) = \frac{1}{2}$, whereas its probability conditioned on *A* (landing on face 4) is $P(B|A) = 1$. Bayes Theorem can be applied with no restrictions to this example because *A* and *B* are events of the same sample space: there is nothing in the event

"landing on an even face" that *generates* or causes landing on 4, it is simply a different event that can result from the same generating conditions. On the contrary, when we conditionalize the event to its *generating conditions* (e.g. to the nature of the die rolled) it is hard to imagine how this symmetry of dependence can be applied at all. We end up thus with two distinct types of conditionalizing: on a different event of the same sample space or on generating conditions. Only the first kind obeys Bayes Theorem, but only the second one is a propensity. This view is what Humphreys later called a "co-production interpretation" of propensities, where conditions defining the propensity cannot be events in the sample space (Humphreys 2004, pp. 671-672).

In this line, most approaches draw a distinction between types of conditionalized probabilities in order to avoid Humphreys paradox and related problems. On the one hand, from the long-run approaches (Gillies 1973, Hacking 1965) it has been argued that propensities are not causal after all. Gillies (2000b) distinguishes "fundamental conditional probabilities" (propensities), without possibility of reverse, from "event conditional probabilities" (conditional probabilities in Kolmogorov's sense), and claims that no probability is unconditional.[8] Single-case views, on the other hand, are far from offering a unified response to this paradox. Miller (1994), following the tradition of the *second* Popper, defends that a propensity is a probability conditioned on the earlier state of the Universe, and is thus a generalization of the causal relation. Event conditional probabilities, on the contrary, do not involve any causal link for him, and thus can obey the probability calculus. Authors such as Isabelle Drouet (2011) propose similar solutions. In particular, her interpretation involves that propensities apply to non-standard conditional probability, and calls the properties determining the propensity of an event its 'determinants'. Other authors have proposed that propensities should be represented differently, such as in indexical form—i.e. with a subindex referring to the conditions—(Berkovitz 2015, Suárez 2018). For example, in Suárez's account, the relation between a propensity and its chance display is explicitly irreflexive:

[8] He thus claims that an unconditional probability $P(A)$ is actually an abbreviation for $P(A|S)$, where S represents the repeatable conditions that define the propensity.

> If *a* is the propensity and Prob (*b*) is the chance that *a* defines over the possible outcome events *b*, the indexed probability account represents this relation as $Prob_a$ (*b*) or P_a (*b*), so there can be no doubt that the attribution of the propensity itself lies outside the domain of the chance or probability function. (Suárez 2018, p. 11)

All of these solutions to the paradox show that conditionalizing to the generating conditions by standard conditional probability is not a correct way of representing propensities. A salient example is Fetzer (1981), who considers—notably with independence of Humphreys (1985) later criticism—that since propensities do not satisfy Kolmogorov axioms, a new calculus of probability must be developed. In particular, he proposes alternative axioms for probability and works out a "probabilistics causal calculus" where the strength of connection between events is non-symmetric. With respect to this, Fetzer stays:

> Perhaps this means that the propensity construction has to be classified as a *non-standard* conception of probability, which does not preclude its importance, *even as an interpretation of probability*! (Fetzer 1981, p. 285; emphasis in the original)

These attempts to overcome the difficulties raised by the rules of probability calculus and Humphreys' paradox highlight that the relation between propensities and causation stands in the way of considering propensities as an analysis of the classical calculus of probability. This may seem paradoxical if we consider that propensities were introduced precisely for considering the physical conditions giving rise to probability, and the strong relation that dispositions have with causation. Nonetheless, many authors point out that the very attempt to analyse probability calculus in a monist way—be it through propensities, frequencies or subjective degrees of belief—is doomed to failure altogether (Eagle 2004, Hájek 2008). As Eagle indicates in his critical review of propensities:

> what makes propensities an analysis of probability, rather than simply the empirical bearer of the concept of probability in this world? (...) In sum, by adverting to physical properties of systems in order to explain their probabilistic behaviour, propensity theories satisfy the intuition that probability is in some way connected to the objective situation. In doing so, they fall prey to

> an additional explanatory burden, namely giving a physical explanation for the obtaining of the mathematical facts about probability. It is difficult to see how they can satisfy this burden while remaining true to the idea that they give an analysis of probability, rather than a pseudo-scientific account of the particular facts that make probability ascriptions true of this-worldly events. (Eagle 2004, p. 285)

Despite the negative connotations of pseudo-scientificism, Eagle's critique can be interpreted as stating that those things that explain objective probabilities will not satisfy the probability calculus. After all, as pointed out above, Humphreys took his argument not as a reason for rejecting propensity views in particular, but as one for "rejecting the current theory of probability as the correct theory of chance" (1985, p. 558). As we shall see in sections 2.3 and 3, even if the 'chance role' is not entirely captured by any interpretation of the calculus of probability, it is still worth exploring how the different ideas on objective probability, and propensities especially, relate to chance more generally. As claimed before, propensities have been a major ally for those advocating for a causal view of evolutionary theory. In turn, the problems associated with the propensity interpretation of probability suggest that what seduces philosophers of biology about propensities in evolution is not their capacity to interpret the content of probabilistic models, but their potential for explaining the probabilistic patterns of evolution alluding to their causes.

2.2. Causal Probability

The connections between the 'chance role' and causation are explored in several ways in the literature. An interesting example is that causal relations have been analysed by some authors in terms of probability raising (Reichenbach 1956, Cartwright 1979), originally as a response to the limitations of a regularity-based Humean approach to causation. This position, known as 'probabilistic causation', states that *A* causes *B* if and only if *A* raises the probability of *B* taking place. Thus, the central idea of probabilistic causation is expressed in terms of conditional probability (Hitchcock 2018) and therefore faces similar problems to those raised by Humphreys paradox for propensities. Notably, Humphreys himself argued that the failure of conditional

propensities was deeply connected to the lack of a "comprehensive theory" of probabilistic causation (Humphreys 1985, p. 566). Moreover, this interpretation of causation confronts a number of further difficulties, especially in association with the problem of spurious connections (see Hitchcock 2018), that is, probabilistic relations that are not grounded on causal ones.

Probabilistic causation has been alluded to in certain views of evolutionary explanations (e.g. Sober 1984). However, this allusion has barely—to my knowledge—derived in philosophical discussions on its applicability and pertinence. That is, although some authors have assumed that one could regard evolutionary causes as probability-raisers, this identification has barely been argued for nor critically revised.[9] As a consequence, the relation of probabilistic causation to the probabilistic models of evolutionary change remains largely underdeveloped. This general lack of interest, I believe, strongly lies in the will of philosophers of biology to *explain* the probabilities of evolution by considering its causes, an endeavour that would be trivial if probabilities and causes were identified. In other words, philosophers are concerned with whether or not the probabilistic models of evolution reflect causal processes, and defining causes in terms of probabilities renders this task meaningless.

More promising, I believe, is a different tradition that relates causation and objective probability without analyzing the one in terms of the other and without relying on the use of conditional analyses. Unlike probabilistic causation, the label 'causal probability' is an umbrella term for those accounts of probability that consider complex causal relations as their basis, and entails a broader—sometimes undefined—conception of causation (see Abrams 2017 and Pietsch 2016). Marshall Abrams (2012b) defines causal probability as "a set of event types when these events usually appear in systematic patterns of frequencies, and there is a set of factors in the world that can be used to manipulate these frequencies" (p. 604). In other words, it considers those probabilistic patterns susceptible of being changed through a manipulation of their causes. Thus, this idea relates causes with frequency patterns without making explicit claims about satisfying the calculus of probability. Although

[9] A recent exception is Razeto-Barry and Frick's (2011).

Abrams himself includes some propensities as potentially causal probabilities (Abrams 2017), my aim in bringing about this notion concerns the non-interpretative nature of this view with regards to probability calculus.

The main conception of probability that has been considered 'causal' in this sense is the so-called 'natural range' conception by authors such as Jacob Rosenthal (2016) and Michael Strevens (2003). Despite having deep historical roots, this conception has been alien to the main discussions in philosophy of probability until recently, and has only been considered in the debates on evolutionary theory in the last few years. To some extent, the reasons are contingent: whereas the opposition of frequentist and propensity views of probability emerged out of the concern about giving an interpretation to probability calculus, the causal probability tradition appeared as a philosophical reflection on a different mathematical concern regarding probability, namely its fruitful application in deterministic phenomena. While the classical frequentist-propensity antithesis was primarily focused on fundamental stochasticity in nature, and notably with indeterminism in quantum mechanics, the causal probability line of thought was concerned with how a deterministic dynamic such as the ones postulated by classical mechanics can bring about apparently stochastic phenomena. Although, as we saw in section 1.2 above, not all propensity interpretations entail indeterminism, their relationship with deterministic processes remains underdeveloped. Causal probability, by contrast, explicitly arose out of the inquiry of explaining the success of statistical mechanics in a Newtonian, deterministic world. In doing so, it considers how a complex set of causal conditions dynamically interact and generate probabilistic phenomena.

The historical precedent of this tradition is Johannes von Kries' (1886) mathematical approach to the probabilities of gambling systems, Henri Poincaré's 'Method of Arbitrary Functions' (1912a/1896) and Eberhard Hopf's work on the ergodic theory (1934). The concern of these authors was to find a mathematical way to explain how a deterministic system generates objective probabilities. In particular, von Kries intended to ascertain on what grounds an equiprobability distribution of red and black can be assigned to a game of chance such as a roulette. He concluded that assuming a regular probability distribution of the initial conditions that

determine each trial of the roulette must be sufficient for explaining such regularity. Nevertheless, Poincaré is usually considered as the founder of this tradition, introducing the 'Method of Arbitrary Functions', namely "a mathematical technique for the determination of probability distributions" in dynamical systems (von Plato 1983, p. 37). Poincaré's interest was to find a way to understand chance as a real feature of the world:

> So it must well be that chance is something other than the name we give our ignorance, that among phenomena whose causes are unknown to us we must distinguish fortuitous phenomena about which the calculus of probabilities will provisionally give information, from those which are not fortuitous and of which we can say nothing so long as we shall not have determined the laws governing them. For the fortuitous phenomena themselves, it is clear that the information given us by the calculus of probabilities will not cease to be true upon the day when these phenomena shall be better known. (1912b, p. 33)

In this quotation, Poincaré suggests that, unlike those phenomena whose laws we have not yet determined, fortuitous or chancy phenomena are governed by the laws of probability not in virtue of our ignorance but because of their very causal nature. It is this nature that he intends to explore in mathematical terms: what is the mathematical structure that allows for an application of the *laws of chance* to phenomena that we assume deterministic? The author ponders that many of the events that we attribute to chance have a similar structure, namely that either small changes lead to great differences in results—e.g. a small perturbation leads an upside down cone to fall in a given direction—, or that big changes lead to small differences in results—such as changes in the speed of a trial in a roulette. Poincaré then argues that the structure of the causes involved in each chancy phenomenon may not say enough to determine each particular outcome of it, but it does so to determine what the outcome will be "*on the average*" or in the long term (1912b, p. 41), that is, if the phenomenon is repeated enough times. In the case of a roulette, the causes underlying trials do not determine the particular results of every trial, but they determine that black and red results will be distributed equitably *on average*.

Poincaré introduces the method of arbitrary functions as a means for representing this fact mathematically. In doing so, he applies a function—i.e. a dependence relation between two variables—mapping initial states to end-states in a given chance setup. In the case of the roulette, we may imagine a mathematical function from all the possible initial positions and velocities of a trial—initial states—to the sample space of results, black and red—end-states. If we apply the probability distribution of the initial states—which must fulfill a few restrictions—to this function, we obtain the probability distribution of results, regardless of any consideration of the actual causal pathways linking the ones to the others. The only restriction to this function is that it must be continuous, that is, it must not contain "jumps" in its domain. In the case of the roulette, a normal distribution of initial velocities and positions would determine the equal, i.e. $\frac{1}{2}$, probability of red and of black. The reason for this is that very similar initial velocities and positions lead to red and black indistinctly, that is, regardless of the probability of those particular initial states obtaining. The function assigning each initial position to a particular result is continuous insofar as there are no "forbidden" values within a range, meaning that every initial position within that range is possible. Thus, if we assume that there is a normal probability distribution of initial states—i.e. that the speed that the croupier impinges to the roulette oscillates around a mean value—, through the function we obtain an equitable probability distribution of red and black. In turn, by defining an *arbitrary function* from initial states to end-states, the final probability distribution of results is mathematically explained, without a consideration of the underlying dynamics, provided that the function is continuous.[10]

The method of arbitrary functions was criticised by Hopf (1934), who argued that assigning a mathematical function can explain long-run frequencies, but not single events. He claimed that the status of probability distributions was problematic, insofar as only specific causal trajectories are realized in the real world, though not

[10] Actually, Poincaré's assumption was that the arbitrary function had a derivative whose value stays in a given range, that is, that the function draws a curve whose degree of inclination states within a given interval. Indeed, this is required by some recent accounts as well (e.g. Strevens 2003, see section 2.2.c below). Subsequent developments of the method by Emile Borel and Maurice Frechét, however, introduced the idea that the distribution only needs to be continuous (von Plato 1983).

all the possible ones (von Plato, 1983). This criticism can be applied to the present discussion in this way: what does the structure relating initial conditions to results tell us about what probabilities are, provided that only a limited subset of possible trials will obtain? Hopf's work on ergodic theory, the study of dynamical systems, led to his derivation of probability distributions out of explicit equations of motion.[11]

Despite differences, the two mathematical traditions share the same concern and inspired the work of those interested in how are objective probabilities possible even at those levels of description where indeterminism can play no role. In opposition to the propensity interpretation of probability, this tradition does not concern *how much* or *how frequently* a cause or a generating condition entails a particular event. Rather, it concerns *how possibly* the causes of a deterministic process give rise to a given probabilistic pattern. In this sense, causal probability can be understood as implying that certain causes produce phenomena that, under certain circumstances, satisfy the calculus of probability. However, it cannot be taken to mean that it is those causes that satisfy the calculus, since the causes, as we have seen, merely relate an initial situation with the chancy phenomenon dynamically, but are not the chancy phenomenon *per se*. This puts the tradition at a much better starting position than propensities for the purposes of this chapter.

a) The range conception: probability out of determinism

From these mathematical branches concerned with the relation between dynamical systems and the laws of chance, a philosophical school of interpretation of probability has emerged in the last few decades. Its main idea is that probabilities are embedded in the causal structure relating initial and termination conditions of dynamical processes. Jacob Rosenthal has named this philosophical position the 'natural-range conception' of probability (2010, 2016), in allusion to the idea that probabilities are derived from ranges in—abstract—spaces of initial states. Rosenthal argues that the conception is inheritor of the classical Laplacian view of probability, according to which the same probability must be ascribed to events *equally possible*

[11] This line has been mathematically explored only moderately, due to the complexity of even simple dynamical systems. For example, Joseph B. Keller (1986) derives the equations of a coin's motion when tossed only under a number of idealizations, such as its lack of thickness.

in application of a "principle of indifference".[12] Although the allusion to indifference suggests an epistemic nature for such principle—and indeed this has been the most widespread view about Laplacian probability—, Rosenthal points out that the weighting of possibilities was always acknowledged as containing a physical aspect, even though "what this aspect could be was largely underdeveloped" (2016, p. 152). The development of this aspect in the works of von Kries, Poincaré and Hopf showed that for the principle of indifference to hold it suffices to assume that the possible initial conditions determining a chance setup are distributed indifferently with respect to the possible outcomes. Rosenthal suggests that we can identify the probability of an event "on a chance trial" as "the proportion of initial states that lead to the event in question within the space of all possible initial states associated with this type of experiment, provided that the proportion is approximately the same in any not too small subregion of the space" (2012, p. 217). He thus defines the range conception as follows:

> Let *E* be a random experiment with an associated continuous state space *S*. Let *A* be a possible outcome of *E*. If *A* is represented in each bounded and not-too-small interval within *S* with roughly the same proportion *p*, then there is an objective probability of *A* upon a trial of *E*, and its value is *p*. (Rosenthal 2016, p. 157)

In other words, in this conception, the objective probability of a given outcome or event is the proportion—or range—of possible initial states in a given setup leading to the outcome. For instance, the probability of a die landing 4 is the proportion of possible initial velocities and positions that lead to landing on that face given certain throwing conditions. This proportion is roughly the same in any—not too small—region of the space of possible velocities and positions in a die roll. That is, any small change in these initial conditions will lead to landing on 4 in the same proportion, $\frac{1}{6}$, regardless of the region of the space of initial conditions—e.g. the region of very slow rolls.

[12] Although this term is associated to Laplace's position, it was actually introduced later in (Keynes 1978/1921), see (Laplace 1995/1825).

Similarly, in his *Bigger than Chaos. Understanding Complexity through Probability* (2003), Michael Strevens defends an account of probability in terms of ratios of initial states and, in addition, he intends to explain them in terms of the causal dynamical properties of complex systems. He too relies on the Method of Arbitrary Functions for his understanding of physical probabilities, claiming that a mathematical—and causal—simplicity underlies the dynamical complexity of chance setups. For Strevens, the patterns of simplicity that we encounter in natural systems are regularities of complexity. But these regularities, he suggests, are found at a higher-level of description of the phenomena. That is, the functions relating initial conditions and outcomes of a chance setup can be applied in relating *microconditions* to higher-level phenomena. Statistical mechanics is based upon this view: probability measures are associated with the ratio of microstates that realize a given macrostate. This fact is explained, Strevens defends, by the properties of microstates. The microstates, just like the initial conditions of a complex causal process, must possess the property of *microequiprobability*, that is, they must be such that they are not biased with respect to a specific outcome. This means that the probability of a microstate is not biased with respect to the macrostate it leads to: all microstates are equally probable. Similarly, the function relating such initial conditions to a distribution of outcomes must be mathematically smooth, that is, it must have continuous derivatives in its domain, meaning that the function must draw a curve whose degree of inclination states within a given interval. Were these conditions not met, the final probability distribution would be altered (Figure 1.1).

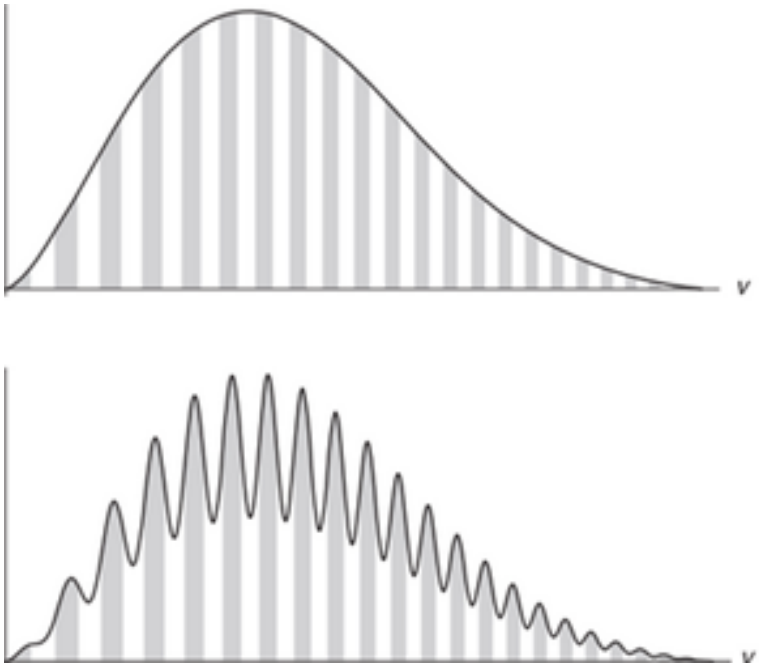

Figure 1.1. Smooth (top) and non-smooth (bottom) functions from initial conditions to end-states. In the first, smooth distribution, the possible initial conditions are distributed irrespectively from the result—white or gray. In the second, non-smooth distribution, the function is biased so that there are more initial conditions leading to gray results than to white. Reproduced with permission from Strevens (2013).

The idea is that the dynamics of a system are microconstant with respect to some outcome if the space of initial conditions can be divided into small portions, each of them having the same ratio of conditions producing that outcome. Philosopher Carl Hoefer provides the following definition of this idea:

> [I]f the dynamics of a system is such that small differences in initial conditions lead to very big differences in the "output" state, then an enormously wide variety of distributions of such initial conditions (over the relevant space of possible initial conditions for the type of system) will all lead to approximately the same stable probabilistic regularities in the outputs of the system. (Hoefer 2016, p. 38)

This tradition is thus close to the classical view of chance insofar as it identifies it with ratios of possible states realizing a particular event. A number of scholars have developed their own version of this approach (see, e.g. Hoefer 2007, Pietsch 2016, Abrams 2012b), but it suffices for present purposes to note the

connection between this perspective and causation. The causal structure of these chance setups make initial conditions map into termination conditions in this specific way. Indeed, in developing his own perspective, Wolfgang Pietsch (2016) labels the tradition as the "causal probability" view, a name that Marshall Abrams (2017) uses to encompass as well, as mentioned above, other notions of objective probability that take causes into consideration, such as some versions of propensities. In the remainder, I keep the label 'range conception' for this specific tradition, while I leave the idea of causal probability as introduced above, that is, as an umbrella term *à la* Abrams.

The relevance of the range conception for evolutionary probabilities can be exemplified by the general consensus among philosophers about indeterminism not playing a significant role in evolutionary probabilities (e.g. Millstein 2010, Weber 2001). This moves the propensity inclination of causalists even further away from the classical discussions on propensities as interpretation of the probability calculus in the previous section. However, despite its promising nature, it is important to note that the range conception faces, in principle, the same problem about reverse probabilities than propensities do, if understood as *an interpretation* of probability. If we intend that dynamical processes obey the probability calculus, we must prove that Bayes' Theorem can be applied to them, just like in the case of propensities. In order to avoid this concern, Hoefer (2016) acknowledges the need of combining the range conception with epistemic notions of probability, implying that reverse conditional probabilities are not objective but subjectively derived. This introduction is in the pluralist line already advocated for in this thesis (e.g. Eagle 2004), according to which a monist approach to probability, favouring only one interpretation of it, is necessarily flawed. Still, this should not be considered problematic since we have already abandoned the endeavour of interpreting the calculus of probability in a monist fashion. In any case, bringing the causalist position about evolution close to the range conception and causal probability—as indeed some authors are doing (Strevens 2013, Abrams 2017)—enables a recognition of the broader role that causation plays in the probabilistic models of evolution. That is, it permits to

consider the causes of probabilities rather than probabilities *as causes* and, as such, it deserves to be treated as part of a causal conception of probability (cf. Abrams 2017).

b) The causal probabilities of complex systems

The ideas of causal probability and the range conception have been recently considered from the point of view of mechanisms and complex systems. These views have a special interest for the philosophy of biology, since biological systems qualify as complex systems, and because biological explanations often involve the recognition of mechanisms (Craver & Tabery 2015). As we have seen, Strevens' conception actually relies on the properties of complex systems. As he states,

> The phenomenon is quite general: systems of many parts, no matter what those parts are made of or how they interact, often behave in simple ways. It is almost as if there is something about low-level complexity and chaos itself that is responsible for high-level simplicity. (...) The key to understanding the simplicity of the behavior of many, perhaps all, complex systems, I will propose, is probability (2003, p. 2).

In this quote, Strevens seems to defend that generalities emerge from complexity at lower levels, and that probability can play a role in illuminating this emergence. Indeed, his view throughout the book is that the properties relating initial and termination conditions as exposed in the previous section are responsible for connecting this lower-level complexity with simple patterns at the higher level. This simplicity needs not preclude this view from being applied to biological systems, which obviously show a high level of complexity. This is because the point Strevens intends to make is relational in nature: the same system tends to show more complexity at lower levels of organization. For instance, the complex behavior of a cell is 'simple' if we compare it with the molecular complexity underlying it at a lower level. In his *Tychomancy* (2013), Strevens applies his probabilistic reasoning to the complex systems of evolutionary biology, notably to the case of ecological interactions and the probabilities they show through the notion of fitness (chapters 9 and 10 of his book). The author claims that, despite the complexity of ecological interactions, there are stable reproductive tendencies that overcome short-term

changes. He argues that the complex relations to be found in such systems are very sensitive to initial conditions, in the same way that the outcomes of stirring a roulette are. However, even if an immense number of conditions can apply to the same system, they are probabilistically distributed so that the reproductive outcomes of individuals present stable tendencies in the long run. In this sense, the many aspects that compose an ecosystem affect reproduction in the same way that the many possible initial velocities in a roulette affect the outcome: in the aggregate, the results will show stable probabilities precisely because of the many microstates that realize each higher-level outcome. The consequences of this view for evolutionary probabilities will be assessed in more detail in the next chapter, once the specific debates on evolutionary theory have been introduced (section 2.3).

More generally, the case has been made in favor of a mechanistic understanding of evolutionary biology (Skipper & Millstein 2005, Abrams 2015, DesAutels 2015), and therefore an application of these mechanistic ideas to evolution seems at least promising. Marshall Abrams (2015) departs from a "difference maker" sense of cause in order to assess this type of probability. His view is that complex mechanisms can be seen as chance setups that realize probabilities in virtue of their causal properties. In turn, it should be possible to alter the values of these probabilities "by altering properties of the chance setup that realizes them" (p. 529). Abrams describes a probabilistic or stochastic mechanism following Peter Machamer and coworkers' (2000) influential definition of mechanisms:

> Mechanisms are entities and activities organized such that they are productive of regular changes from star or setup conditions to finish or termination conditions (Machamer et al. 2000, p. 3).

Abrams points out that, in stochastic mechanisms—such as cell polarization (Bressloff 2014)—, the regular changes must present a probability distribution. More specifically, these mechanisms define "activity probabilities", that is, the probabilities of "an activity producing certain changes in entities" (2016, p. 171). Again, some of these probabilities are such that small changes in initial states lead to great differences in outcome, while on average complex initial conditions lead to stable

patterns of outcomes, explaining thus the probabilistic patterns arising out of underlying complexity. The author advocates for the application of this view of mechanisms to complex biological systems, arguing that these exhibit a probabilistic behavior based on the complexity of its interacting components.

Interestingly, biological mechanisms are typically defined in terms of their function, a notion that is historically charged with teleological connotations (Craver & Tabery 2015), and which has been associated with dispositions. To this respect, some proponents of the mechanistic approach to biological systems have also defended a dispositional approach to biological functions, the activities involved in mechanisms being referred to in dispositional terms. In this view, the functions of mechanisms are seen as the contributions of certain components to some behavior or *capacity* of the system they belong to, understood as a probabilistic disposition or a propensity (DesAutels 2015). This way, the complexity embedded in biological stochastic mechanisms presented in this section can be approached as well from the point of view of dispositions, and therefore from an understanding of propensities that distances itself from interpreting the probability calculus. As we shall see, this is precisely the framework advocated for in the last section of this chapter.

2.3. The Philosophy Of Chance

The complex relationship between causation and objective probability so far outlined in this section, suggests that, even if probability cannot be analysed in terms of propensities or complex causal connections, there is a strong sense in which the probabilities invoked in complex systems such as biological ones depend on the structure of the causal processes they undergo. As pointed out previously, despite the fact that the discussions on the nature of probability have proliferated independently of the traditional frame of the philosophy of chance, many of the problems that arise out of the attempt to analyse the concept of probability result precisely from such a decoupling. Therefore, now that a revision has been made on the main senses in which objective probability relates to causation, it will be useful to examine the main

philosophical views about chance. In so doing, it will be worth pointing out certain connections with how the notion of chance has been considered in evolution.

Recall Poincaré's view that chance is whatever physical phenomenon that is governed by the laws of probability. Poincaré wonders the following: "Has chance thus defined, in so far as this is possible, objectivity?" (1912b, p. 47). The classical view is that objective chance is incompatible with a deterministic world. For example, Jonathan Schaffer (2007) argued that the only function that can play the role of chance in a deterministic ontology is that of assigning probabilities with either 0 or 1 value. The idea is that if the present state of the world entirely determines the future, then the objective chance of any event will be either 1 or 0. In other words, if the world is deterministic, every description regarding the future can only be impossible or necessary. If determinism holds, objective probabilities are not real: probabilities are always subjective. This conception of chance as indeterminism appears in some discussions of evolution, especially with regards to the nature of mutations (Brandon & Carson 1996). Mutations, namely changes in the genotypic material, are believed to have a complexity of stochastic components, some of them related to the lack of knowledge about the mechanisms responsible for them (see Houle and Kondrashov 2006). The idea that there can be an indeterministic aspect in these components derives from some interpretations of quantum subatomic effects, which could arguably play a role in the chemical reactions causing genetic changes (e.g. Cannon et al. 1995, Brovarets & Hovorun 2014). However, the fact that such indeterminacy could play a prominent role in evolutionary probabilities is highly problematic (Graves et al. 1998), there being certain consensus about the lack of relevance of quantum indeterminism for the probabilities of evolution (Richardson 2006, Lenormand et al. 2009, Millstein 2011). On the contrary, as we shall see in the next chapters, evolutionary probabilities are typically based on patterns arising from higher-level deterministic causes.

Thus, this classical picture is not very appealing from the point of view of non-fundamental sciences. Whether the world is deterministic or not, the probabilities of sciences such as biology refer to a higher level of explanation where chance seems to emerge. The precedent section on probability out of (deterministic)

causation is a good example of this. Nevertheless, chance has not been neglected in the philosophical tradition. Independently of providing or not a correct analysis of the calculus of probability, the existence of a meaningful notion of chance has been traditionally acknowledged. However, given its assumed incompatibility with determinism, traditional accounts have usually intended to reduce chance to other features.

In *A Philosophical Guide to Chance* (2012), Toby Handfield distinguishes two main approaches in the philosophy of chance: actualist and possibilist accounts. Actualist accounts of chance follow the Humean tradition of considering all features of reality as supervening on categorical properties. More recently, it has been developed from the philosophy of probability perspective by David Lewis' (1994), and it is known as the 'best systems interpretation' of probability (Lewis 1994, Loewer 2012, Hoefer 2007). The Humean tradition poses that modal phenomena, such as the possible or the probable, are derivative from more fundamental elements of reality that are not modal themselves. From the eyes of a Humean, only the actual is real, the modal supervening on features of reality such as its regularities or the frequencies of its events. More specifically, Humeanism can be seen as "the view that all truths supervene on the distribution of fundamental categorical properties over points or regions of spacetime" (Schwarz 2016, p. 424). Following this tradition, in the 'best system' interpretation of probability,

> chances are identified with probabilities in ideal physical theories whose aim is to provide a kind of summary statistic of actual outcomes; getting close to the frequencies is one virtue of probabilistic theories, but it trades off against other virtues such as comprehensiveness and simplicity (*ídem*)

Lewis (1994) thus argued that the laws of chance supervene on the properties of the best description of the world available. That is, it is a certain structure of the actual regularities of the world that we perceive what makes us describe its laws in a probabilistic way. From this analysis, it follows that the categorical properties of the world need to be governed by laws of nature that are approximated in our best possible picture of their nature.

The possibilist tradition, on the other hand, envisions chance as measures of possibility (Handfield 2012). Pierre-Simon Laplace is considered the initiator of the classical, pre-theoretical picture of probability, that stays that the probability of an event is the ratio of positive cases in all possible cases (Laplace's rule):

$$P(A) = \frac{favorable\ cases\ to\ A}{possible\ cases}$$

This rule, however, is only applicable when cases are "equally possible". Following our example of a die, we can estimate that there are six possible faces the die can land on, and that only one of those is favorable to the event 'landing 6'. Nevertheless, the classical view was not clear with regards to how to establish the *correct* division of possibilities. As Handfield points out, "why not say that there are two possibilities: (1) it lands on six or (2) it doesn't?" (2012, p. 90). Laplace's "Principle of Indifference" intends to overcome this difficulty. According to the Principle, whenever there is no sufficient reason for doing otherwise, possibilities must be weighted equally. In the case of the die, there are solid grounds for weighting "not landing on six" heavier than "landing on six". Laplace summarizes his view like this:

> The theory of chances consists in reducing all events of the same kind to a certain number of equally possible cases, that is to say, to cases whose existence we are equally uncertain of, and in determining the number of cases favourable to the event whose probability is sought. The ratio of this number to that of all possible cases is the measure of this probability, which is thus only a fraction whose numerator is the number of favourable cases, and whose denominator is the number of all possible cases. (Laplace 1995/1825, p. 4)

Needless to say, this tradition has a clear counterpart in the field of analyzing probability calculus, namely the range conception, which understands probability in terms of ratios of initial states leading to end-states. However, the classical view cannot entirely be identified with the newer range conception enterprise. On the one hand, the classical picture assumed that the relevant characterization of possibilities could be established *a priori*. On the other hand, Laplace's position had no direct empirical application (Handfield 2012). What is interesting to note is that, unlike the

actualist, Humean view of chance, the classical Laplacian picture is one where the possible is considered as a foundation of the actual and not the other way round. Problems arise, of course, with regards to how the correct possibilities can be established. If this is not an empirical task, where frequencies of events can guide our estimation, how do we justify our knowledge of the possible? Notably, the epistemology of modal thought is an important philosophical concern with no easy consensus. As we will see in the next section and throughout this thesis, conceiving the possible is key to understanding the role of dispositions and chance in evolutionary theory.

Chance in evolution has been discussed in different contexts and consequently several meanings of the notion have been identified by some philosophers. Jean Gayon (2005) recognized three distinct conceptions of chance in evolutionary debates, namely as "luck", as "random event" and as "contingency with respect to a theoretical system" (pp. 40-41). What all these notions share, Gayon states, is a sense of unpredictability that nonetheless may rely on the objective structure of the world. Roberta Millstein (2006) argues that the notion of chance may play different roles, depending on theoretical and pragmatic aspects of the context. For instance, chance can be *explanatory* of some evolutionary phenomena, such as non-adaptive divergence among close species; or it can be *instrumental* in the building of stochastic models. Chance can also be *representational* in the sense that it is sometimes used to describe a particular probabilistic phenomenon. Finally, Millstein proposes that chance sometimes plays a *justificatory* role inasmuch as it serves as contrast with other, seemingly less chancy phenomena—especially as contrast with natural selection. Moreover, in a similar fashion to Gayon's review, she identifies up to seven different meanings for chance in evolutionary contexts: "indeterministic chance", "chance as ignorance of the real underlying causes", "chance as not designed", "chance as sampling", "chance as coincidence", "evolutionary chance" and "chance as contingency" (Millstein 2011, p. 426). As we shall see in the next chapter, all of these notions have played a particular role in the configuration of the current understanding of variation in evolution, their functions being manifold. Nonetheless, Millstein provides a "Unified Chance Concept" for subsuming the

common generalities of the notions of chance in evolutionary discussions that may serve as a guide on the relationship between chance in evolution and the philosophy of chance more generally. Her view is that what all these diverse notions share is that "Given a specified subset of causes, more than one future state is possible" (2011, p. 428).

Thus the possible is an important component of the role that chance plays in evolution. We can rephrase Millstein's unified concept in the terms of this chapter like this: some causes are responsible for a space of (more than one) possible effects. Identifying these causes, establishing the possible effects, and elucidating their relationship with probabilistic models is in turn the enterprise of causalists about evolutionary theory. The relationship with probability is indeed proposed by Millstein (2011) herself, who suggests that chance like this understood can be translated into conditional probability directly. That is, she states that her notion of chance can be formalized as the probability of an evolutionary event—e.g. the appearance of a new trait in a species—conditioned on the causes considered—e.g. mutational, developmental and ecological factors—. However, since we have already reviewed the flaws of conditional probability for representing causal relations (section 2.1 above), this is a position that we shall not take. Other authors have stressed the probabilistic component embedded in the broad notion of chance in evolution, especially with regards to randomness—namely the relative lack of pattern in a series of events—and random variables—or the variable result of a trail in a chance setup— (Gayon 2005, Plutynski et al. 2016). Thus philosophers have acknowledged a relation between the notion of chance in evolution and modeling practices that make use of probability. In turn, the development of evolutionary biology seems to have brought a diffuse notion of chance that has both a "central explanatory role" in different contexts and a deep connection to probabilistic models (Ramsey & Pence 2016, p. 2).

From the general philosophy of chance, philosopher Antony Eagle (2019) has analysed the connection between chance and the randomness of events. According to the author, there is a "commonplace thesis" relating both notions that states that "something is random iff it happens by chance". In order to elucidate the meaning

and correctness of this thesis, Eagle makes the useful distinction between chance as applied to processes and randomness as applied to events. Whereas he identifies chance with objective probability roughly in the terms so far developed in this chapter, he considers randomness to be a characterization of a series of events or outcomes without order of pattern. For example, a series of outcomes of dice rolls such as {3, 5, 3, 1, 4, 2, 6, 4} can be considered random only insofar as an equiprobability distribution is assumed for each outcome. With the same assumption in mind, the series {1, 6, 1, 6, 1, 6, 1, 6} would be considered non-random. As Eagle himself shows in his interesting review, determining the absolute randomness of a series is a tough mathematical task. Randomness is usually, thus, relative to some referent pattern—i.e. a null statistical model. Chance, on the other hand, cannot be established in virtue of the nature of an outcome or event but in consideration of "the properties of the process leading up to it [and] the causal situation in which it occurs".

After a number of counterexamples, Eagle concludes that the commonplace thesis establishing a necessary connection between chance and randomness cannot hold. However, what is of interest here is that, while this connection may not be necessary, it may serve as a regulative principle for inquiring into the possible and its probabilistic modelling. As he stays,

> This is not to say that there is no link between (...) randomness and physical chance. The observation of a random sequence of outcomes is a defeasible incentive to inquire into the physical basis of the outcome sequence, and it provides at least a prima facie reason to think that a process is chancy (...). Moreover, if we knew that a process is chancy, we should expect (eventually, with high and increasing probability) a random sequence of outcomes. (Eagle 2019)

Similarly to its unavoidable relations to frequencies, causation, possibility and the calculus of probability, chance seems to have an important connection with randomness as well. The randomness of events—given a null statistical model—provides grounds for examining what causes can be responsible for such a pattern. In doing so, identifying frequencies and the probabilistic models that may account for them is but one aspect—though a necessary one—of the more complex

task of explaining why a particular phenomenon obeys the laws of chance. As we shall see in the next chapter, the randomness of variation in evolution is a disputed issue, there being several null hypotheses against which it could be tested (e.g. Lenormand et al. 2009, Merlin 2010, A. Wagner 2012a). The relationship between these hypotheses and distinct notions of chance will be addressed there in some detail (section 3).

3. Propensities As Explanatory Causes

It has been pointed out that the term 'propensity' has become a kind of synonym of objective non-frequency interpretation of probability (Gillies 2000a). That is, regardless of any commitments to dispositions, propensity talk usually involves the acknowledgement that probability refers to a certain extent to a feature of the world responsible for but different from the frequencies associated with them. Following this intuition, in this section I will defend that dispositions, and specifically propensities, are a good ally for causal approaches to probability in a general sense. In particular, I will defend that propensities play an important explanatory role in the causal representation of the probabilistic phenomena involved in the models of evolutionary biology. I will thus argue that, regardless of any analysis of probability ascriptions, propensities serve as means for establishing a range of possible results, as well as for pointing at the underlying mechanisms responsible for probabilistic behavior, in turn playing the 'chance role' demanded in the philosophy of evolution.

Despite its centrality, the explanatory virtues of propensities with regards to probabilistic patterns are relatively unexplored from the point of view of how dispositions explain phenomena. The explanatory role of dispositions is widely discussed in the literature, but the particularity of propensities explaining probabilities has been, to my knowledge, much less examined. Moreover, it remains unclear what is the relationship between propensities and the probabilities that complex systems show. In this section, I will attempt to fill a small fraction of this gap through a causal approach to propensities. First, I will defend the explanatory potential for dispositions, especially with regards to evolutionary phenomena (3.1).

Then, I will introduce and develop the idea of causal propensities (3.2). This will serve as a means for assessing some particular roles of chance and probability in evolutionary theory in the following chapters.

3.1. Dispositions And Explanation

How do dispositions explain? Does saying that a glass is fragile explain its breaking? And does saying that a coin has a $\frac{1}{2}$ propensity of landing heads when tossed explain any particular landing or series of it? These are matters that have been largely debated in philosophy, usually in the frame of discussing modality and its reduction to the actual more generally.

One of the main arguments against the explanatory relevance of dispositions is usually taken to its extreme form by alluding to the "dormitive virtue", a property attributed to the opium poppy in Molière's 1935 play *Le Malade Imaginaire* in order to explain why it puts people to sleep. Jennifer McKitrick (2004, 2005) calls this the 'analyticity argument' against the causal and explanatory relevance of dispositions. This argument states that a disposition is necessarily connected to its effect (like "dormitive virtue" to sleeping), while causes are typically contingently connected to their effects. Because the meaning of, say, 'fragile' is being disposed to break, there is a conceptual necessitation between fragility and breaking. A ball hitting the glass can be easily considered causally relevant for its breaking, but it is, on the contrary, contingently connected to it. Following this argument, conceptual entailment is neither a causal nor an explanatory type of relation.

A different argument against the—causal and—explanatory relevance of dispositions is that they are redundant and thus superfluous. This is the 'exclusion argument' (McKitrick 2005, Choi & Fara 2018), according to which the causal basis of a disposition, namely the properties that realize it, are sufficient for explaining the effect of the disposition. So, in the case of a fragile glass, one may say that the physical structure of the glass realizes its fragility, and that it is this physical structure that constitutes a sufficient causal explanation for its breaking. This would make the

fragility realized in it superfluous for the explanation, insofar as it overdetermines the glass' physical structure, and thus non-explanatory of the breaking.

However, these arguments are not conclusive. As McKitrick (2005) has shown, the main philosophical accounts of causal relevance give no grounds for rejecting dispositions as causally relevant. On the one hand, the independence between causes and effects assumed in the analyticity argument would rule out as non-causal other relations that are intuitively causal. For example, this assumption would imply that sunlight is not causally relevant for sunburn, since there is not a possible world in which sunburn is not caused by sunlight (McKitrick 2005, p. 363). While it is reasonable to demand that something must not be identical to what it explains, it is not so clear that something must not entail conceptually what it explains. On the other hand, the assumption made by the exclusion argument, namely that there cannot be overdetermination of causes, is often not met in everyday examples of causal relevance since causes are usually decomposable (Schaffer 2003). For instance, the ball hitting the window and causing it to break can be divided into parts that could also be sufficient for explaining the breaking and would therefore render the ball as an overdetermination of the cause. As a consequence, our everyday reasoning about causes is filled with overdetermination, insofar as we are constantly alluding to causes that can in principle be divided up into other causes that would be sufficient for explaining the same event. What is needed, thus, is some criteria for how much specification is reasonably required in different contexts.

Let these remarks suffice for claiming that the case can be made for dispositional explanations. In the remainder of this section, I will not attempt to revise in depth the philosophical arguments regarding explanation and dispositions. Instead, I will try to establish some minimal requirements for the explanatory role of dispositions in accounting for chance and probabilistic phenomena. This will allow me to develop a causal explanatory approach to propensities in the sections that follow, which will be the basis for understanding the claims of causalists with respect to selection and drift (Chapter 2) and, more importantly, the variational probabilities of evo-devo (Chapter 3).

a) The minimals of dispositions

We have yet to make explicit what dispositions are. Only after this is done, can the explanatory role of propensities be characterised more properly. In the following, I sketch the main features and distinctions characterizing dispositional properties.

Functional individuation

Dispositions are properties characterized by a specific type of manifestation under certain conditions. For instance, fragility is characterized by the manifestation 'breaking' under conditions such as being hit or thrown. Dispositions are opposed to categorical properties insofar as they can be attributed to entities or states of affairs even if they are not manifested. Properties that are presumably categorical, such as height or position, are always manifested if possessed. Dispositional properties such as fragility or solubility, by contrast, are only manifested under certain conditions, even if possessed in a latent form when these conditions are not met. The conditions that dispositional properties must encounter in order to find their manifestation are generally referred to as triggering or stimulus conditions. Being hit by a rock is typically a triggering condition for fragility to be revealed, while being put into water is the stimulus condition for manifesting solubility. Dispositions are thus defined functionally, as a relation between stimulus or triggering conditions to manifestation conditions.

Manifestation/effect distinction

When a disposition is manifested, a specific effect of some kind is met. However, the manifestation of a disposition should not be confused with its effect (Molnar 2003, Mumford 2009, though see McKitrick 2010 for a critique of the distinction). While the manifestation is the condition that defines a particular disposition, an effect is the event that results from a particular manifestation of it. In this sense, it is said that the manifestation *contributes* to a particular effect. For instance, when the fragility of a glass manifests in its breaking, a possible effect is that it will break into a thousand different pieces. However, the same fragility can have the effect of breaking into five

pieces instead. In general, the effect of a disposition is a particular, actual way of being manifested.

Modal nature

Metaphysicians have been concerned with the possibility of reducing dispositional properties to categorical ones and *vice versa*. In this regard, there are dual views of properties, where both categorical and dispositional properties are considered ontologically real, as well as monist positions, according to which only one of these two types of properties is real. Categoricalism (see e.g. Carnap 1936), namely the view that only categorical properties are real, regard dispositions as non-actual properties. The categorical view of the world is influenced by empiricism and logical positivism, and thus has intended to analyse dispositions in conditional terms so that having a disposition towards *M* is nothing over and above than manifesting *M* when certain conditions are met. Given the complexity and plurality of indicative and counterfactual analyses of conditionals, a substantial variety of accounts—or reductions—of dispositions is subsumed under this view, each of them showing different virtues and problems (see Choi & Fara 2018 for a review). Moreover, a difficulty that categoricalism faces generally is how to ascribe dispositions that are not manifested.[13]

Dispositionalism, on the other hand, is the view that only dispositional properties are fundamental (e.g. Bird 2007) or, more radically, that only dispositions are real (e.g. Mumford & Anjum 2011). Dispositionalists consider that the essence of a property "is wholly constituted by the nomic or causal roles [it] plays", meaning

[13] Categoricalism faces other difficulties that are interesting from a metaphysical point of view. On the one hand, individuating categorical properties on a strictly empiricist criteria may be problematic. If categorical properties are individuated based on what they actually *are*, rather than what they do, their nature may be empirically inaccessible. On the other hand, and perhaps more importantly, such characterization of categorical properties is detached from the causal processes properties take part into, implying that nothing in the nature of the property relates to the laws of nature. In other words, if a categorical property is characterized without a reference to its causal relations, does that involve that there are possible worlds in which the laws of nature are different but the same properties hold for all objects? Such a situation would imply that two *identical* objects could be subject to different laws of nature. If it was the case, then one should admit that categorical properties are only contingently related to the laws of nature, rather than necessarily so, which seems *prima facie* not plausible (see Bird 2007).

that two properties are identical when they play the same *theoretical role* (Choi & Fara 2018). This view intends to provide an account of modality where laws of nature follow from the causal powers of properties, thus opposed to the classical Humean supervenience framework. From a metaphysical perspective, dispositionalism can be argued to face the same problem as categoricalism, namely that it fails to give solid criteria for ascribing dispositions that are not manifested. However, from an epistemological point of view, individuating properties in terms of their causal role is an interesting position, insofar as it allows for a characterization of properties in terms of what they—dynamically—do rather than what they—essentially—are. In this sense, dispositions are a natural way of explaining and describing the functional complexity of living beings. As mentioned in the previous section, dispositions are typically associated with the functions of biological mechanisms: stability, plasticity, catalysability, etc., are but a small number of functions that organismic components perform. Thus the properties of biological systems can be more easily accounted for from the point of view of their dynamical activities.

Multiple realizability

Usually, one may distinguish between fundamental and non-fundamental properties. A fundamental property is a property that cannot be reduced to others. It is typical to list properties such as shape, mass and spin as fundamental: their existence does not depend on the existence of any other lower-level property. Higher-level properties such as color or molecular structure are said to depend on lower-level properties and thus to have a certain "causal basis" distinct from themselves. Thus, most dispositions, notably non-fundamental ones, have a causal basis, meaning that their causal efficacy has a base different from the disposition itself.[14] The following definition accounts for the causal basis of a disposition:

> A causal basis for disposition *D* is the property or property-complex that, together with the characteristic stimulus of *D*, is a causally operative sufficient condition for

[14] Although the case has been made for dispositions being their own causal basis (see McKitrick 2003b), the dispositions that concern this thesis typically have a causal basis distinct from the disposition itself.

> the characteristic manifestation of *D* (...) (Prior et al. 1982, p. 251. Cited in Choi & Fara 2018)

Although there is no consensus on the nature of the relation between a disposition and its causal basis, it seems clear that at least some dispositions have a causal basis. For example, the fragility of a glass can be causally based on its physical structure. In this case, it is said that the physical structure of the glass *realizes* the disposition. This brings us to an important feature of dispositions: they are typically *multiply realizable*, meaning that they can be instantiated in a variety of causal bases (Prior et al. 1982, McKitrick 2003b). Fragility, for example, can also be instantiated in the physical structure of porcelain and of eggshells. Biological dispositions, moreover, are typically multiply realizable as well. For example, 'fitness' is the capacity to survive and reproduce realized in every kind of living being, while 'divisibility' is a capacity of cells independently of the cell type (Hüttemann & Kaiser 2018). This multiple realizability will be fundamental, as we shall see, for characterizing evo-devo variational dispositions—such as modularity or robustness—in Chapter 3 of this thesis.

Intrinsicness/extrinsicness

A different concern that many academics share with regards to dispositions is whether they are intrinsic properties of objects. Since they are defined with respect to both triggering and manifestation conditions, their nature seems to be relational. However, it is usually held that objects having exactly the same intrinsic properties will be disposed alike, regardless of any extrinsic factors (Lewis 1997). This has been challenged by Sydney Shoemaker's (1980) example of a key being disposed to open a certain door: if we changed the lock to the door, then the key would lose its disposition without suffering any intrinsic change. To overcome this problem, it has been argued that the *real* disposition that a key has is the disposition to open locks of a certain *type* (Molnar 2003). Moreover, McKitrick (2003a) argues that, in fact, dispositions can have extrinsic causal bases. This concern is particularly relevant for evolutionary dispositions, for evolutionary changes can only be explained with regards to environmental conditions. Are those conditions part of the triggering or of

the disposition? We shall see in Chapter 3 that this is indeed an important matter of concern for evo-devo dispositions, as illustrate contemporary debates over the nature of evolvability (Love 2003). In the context of overcoming this type of difficulty, Andreas Hüttemann and Marie Kaiser (2018) argue that, in addition to stimulus or triggering conditions, it is important to identify sustaining—necessary conditions through the manifestation process—and background conditions—namely those assumed under normal circumstances—for biological dispositions. This point will be relevant in the next chapters when discussing the problem of the relevant environment—that is, of which environmental conditions are taken as reference for defining the dispositions and, in turn, the probabilistic behavior, of biological systems.

Surefire/probabilistic distinction

Finally, most authors distinguish between *surefire* and *probabilistic* dispositions. Surefire dispositions are those that, when triggered, necessarily manifest in a particular type of effect. Probabilistic dispositions, on the other hand, are those that manifest only probabilistically, namely with a certain probability. Probabilistic dispositions, in turn, are identifiable with propensities. As specified in section 1 of this chapter, propensities can be understood as tending towards a specific manifestation with a certain probability (tendency views of propensity), as dispositions whose manifestation is a probability distribution (distribution display views), or as a disposition to manifest a pattern in the long run. However, all of these views of propensities differ from surefire dispositions in that they do not define a single necessary effect.

b) The minimals of explanation

Besides mere description, explaining phenomena is an essential scientific task, explanation arguably being the most central topic in the philosophy of science. The question is, then, do dispositions explain their manifestations? And, in addition, do they explain their particular effects? The literature on scientific explanation can be overwhelmingly ample, especially considering that most of the big debates in the

general philosophy of science, such as the nature of scientific theories and the role of laws of nature, are entangled with the problem of explanation. However, I believe that a few minimals can be established for the purposes of this chapter without getting involved in the ramifications of these debates.

Explananda in evolutionary biology are the diversity and adaptation of living forms, but also their commonalities and complexity. Finding a good explanation for these phenomena typically does not imply a fine-grained causal chain leading to a particular evolutionary event from a defined state in the past. Rather, it usually means showing that, under the consideration of certain factors, "the event to be explained could have resulted in a number of possible ways" (Sober 1984, p. 142). Although more will be said on the explanatory structure of evolutionary theory in the next two chapters, the present task is merely to account for a minimal explanatory role of propensities under these considerations. The question is, therefore, do propensities explain chancy evolutionary changes? Do they show that certain evolutionary events are possible in a number of ways? As we saw in the introduction to this section, dispositions have been accused of being causally—and therefore explanatorily—irrelevant on the basis of trivially entailing the phenomenon they are supposed to explain and of being superfluous for such explanation if their causal bases are known.

From the classical *deductive-nomological* (DN) model of logical positivism to contemporary pragmatic theories, the reference to nomical and causal relevance seems to play a decisive role in explanation.[15] Regardless of the particular position taken about causation, explaining a phenomenon seems to be entrenched with finding its causes. Carl Hempel's DN model (1965), for example, considers causal explanations to be involved in DN-type explanations, so that whenever a cause is invoked there is an implicit commitment to a lawful relationship between such cause and the *explanandum*. Similarly, Philip Kitcher's (1989) unificationist account regards causation as a derivative from unification. According to this view, when we explain phenomena we are typically unifying them under the same argument pattern from which specific causal claims can be derived. More explicitly, Wesley Salmon's

[15] See Suárez and Villegas (2018) for a recent review of causation in science.

two distinct models of explanation were motivated by the idea that explanation *must* refer to causal relationships (reviewed in Woodward 2014). On the one hand, Salmon's early model of statistical relevance (1971) relied on conditional dependence for this matter. That is, explanatory relevance was considered as causal relevance from a statistical view of causation under this account. This point will be key for the criticism of the statisticalist position about evolution to develop in the next chapter (section 2.1): the statistical paradigm of explanation is flawed because it fails to give an account of statistical relevance without making reference to causal relevance. On the other hand, Salmon's later causal mechanical model of explanation (1984) is committed to a processual view of causation, according to which a cause physically transmits a certain structure to its effect.

Needless to say, pointing at a cause is not the only criteria considered for something to be explanatory. Even if for the main classical accounts mentioned in the previous paragraph causal relations are important, the relevance of the cause must fulfill some requirements. As the philosopher Nancy Cartwright (2002) has pointed out, causal relevance has taken the field of scientific explanation. A way to assess explanatory power without engaging with any of these classical accounts of causation in particular is to approach explanation from a pragmatic point of view. There exist a number of approaches to explanation that consider both cognitive and contextual factors to be necessary in order to hold that something is explanatory (Woodward 2014). The main idea behind these accounts is that scientific theories, concepts, models and the like are only context-dependently explanatory. That is, matters about the interests and backgrounds of those involved in explaining or receiving an explanation of a phenomenon are determinant as criteria of explanation. In practice, this means that the validity of a scientific explanation can change with respect to things such as the epistemic goals of a given discipline or research agenda, as well as with regards to time.

For example, in *The Scientific Image* (1980), Bas van Fraassen considers that, although the only purely scientific aim is empirical adequacy, explanation is a virtue that emerges in the context of scientific practice. When a "why" question is posed in the practice of science, the explanatory answer will come as "a three-term relation

between theory, fact *and context*" (Van Fraassen 1980, p. 156. Stress added). So an explanation of why a window breaks will not only consist of relating the window-breaking event with theoretical—maybe dispositional—factors. Importantly, it will also refer to the matters concerning the explanatory situation. For example, it may be explanatory of a particular window-breaking to point out that the house where it is placed suffered from a soft earthquake, in the context of not knowing this particular fact. However, for someone who knows about that earthquake but still wants to know why the window, but not the chimney, broke, pointing at the window's fragility can be a good explanation. As we shall see, the introduction of pragmatic considerations into explanation is central in current philosophy of biology, as illustrate recent revisions of the notion of evolutionary novelty (Brigandt & Love 2012) and of the use of typological notions (Brigandt 2007, Love 2009).

In connection to these pragmatic considerations, there is an important literature in the philosophy of science that argues for the *contrastive* nature of explanation (Lipton 1990). According to this tradition, an explanation is always, even if implicitly, contrastive, meaning that it contrasts the phenomenon to be explained with a dispare situation that would not require an explanation in a given background. Thus, in the window examples above, not only the second explanation would be contrastive (why did the window *rather than* the chimney break?), but also the first one (why did the window break *instead of not breaking*?). What this idea suggests is the apparent fact that things that demand an explanation are to some extent unexpected with regards to our background knowledge. For example, an evolutionary *explanandum* could be 'why did this lineage evolve rather than go extinct?', or 'why did this trait change in this way rather than in another?.' Thus even if a number of causes can be involved in a given phenomenon, we usually want to make reference to those causes that *make a difference*. In this regard, there is an increasing literature in the philosophy of science about difference-making accounts of causal explanation (e.g. Waters 2007). More particularly, the idea that evolutionary explanations have a contrastive nature is gaining weight among philosophers (Witteveen 2019). As a consequence, questions such as whether or not a cause that entails its effect conceptually—such as in the case of dispositions—is explanatory have no unique

answer, for it may be the case that pointing at this type of analytic cause will be explanatory in certain contexts. For example, it seems that if we are explaining some *kind* of regularity, it is fair to refer to analytic arguments of the type: "*As* show behaviour *B* because they belong to the class of things that typically show *B* under conditions *C*". This explanation could well be expressed in a lawful form. For example, a lawful generalization of the type "If *A* and *C* then *B*" could be part of the explanation of *B*. Lawful generalizations are not typically regarded as irrelevant despite their analyticity because they are assumed to be explanatory *under certain circumstances*.

All this implies the well-known fact that there are many—perhaps infinite—possible explanations for the same phenomenon, meaning that the phenomenon *underdetermines* its explanation. Indeed the key seems to be finding *better* or *preferred* explanations. Peter Lipton (1991) famously developed the idea that scientific explanations are typically 'inferences to the best explanation' (IBE). That is, they are always the result of a contrast between rival hypotheses for explaining the same state of events, and picking up the one that explains it better in a certain context. This idea derives from Peirce's theory of abduction. Peirce was concerned with ampliative reasoning, namely the process by which new explanatory hypotheses are inferred. An abduction *à la* Peirce has the following form:

> The surprising fact, C, is observed;
> But if A were true, C would be a matter of course.
> Hence, there is reason to suspect that A is true. (Peirce 1994, p. 1744)

Through a reasoning of this form, explanations are inferred for "surprising" or unexplained phenomena. Lipton's IBE, however, refers to a slightly different process in which different possible explanations are already conceived for the same fact and a choice must be made among them. Here not only causal—i.e contextually dependent—relevance must be considered, but also other so-called "epistemic virtues", such as unification, heuristic role in inferences, depth, precision or robustness (Lipton 2001, Ylikoski & Kuorikoski 2010).

The process of inferring good explanations is not an entirely theoretical one. As an empirical science, biology has, in addition to cautious observation of living phenomena, an important empirical source in experimentation. This circumstance has made manipulation-based approaches influential in biology (see Weber 2018) and, more specifically, it has turned James Woodward's so-called 'interventionism' (Woodward 2003, 2015, see also Pearl 2009) a paradigm of causal inference in the philosophy of biology of the last years (e.g. Mitchell 2008, Baedke 2012, Weber 2018). According to the interventionist view of causation,

> *X* causes *Y* if and only if there are background circumstances *B* such that if some (single) intervention that changes the value of *X* (and no other variable) were to occur in *B*, then *Y* or the probability distribution of *Y* would change. (Woodward 2010, p. 290)

Thus a cause is anything to which an intervention can be made that changes the value of its effect under certain conditions. Notice that this notion is not merely manipulabilist but counterfactual, that is, it refers to those things that can undergo an intervention *in principle*, without any restrictions to the viability of such an intervention. Thus, this view not only serves for detecting causes through manipulation in experimental arrangements. Importantly, it serves as well for inferring *possible causes* under hypothetical interventions (Woodward 2003). It is also important to notice that the quote above refers to changes in particular effects as well as to changes in probability distributions. This is important from the point of view of generality and pattern explanations: interventions not only refer to changing specific outcomes, but also probabilistic patterns. From the point of view of dispositions, we can conceive of interventions affecting a manifestation—i.e. a pattern or event type—or an effect—i.e. a particular outcome of a disposition. Both questions are different in nature and, as we shall see in Chapter 3 of this thesis, distinguishing among them is key to understanding the probabilistic nature of evo-devo notions of variation.

We can conclude from these few remarks that explaining something can be understood as pointing at its difference-making causes with respect to a specific level of description, and doing so in *the best possible way* among the availables with

respect to pragmatic considerations and epistemic virtues. Some important components of this process are, as we have seen, theoretical background, empirical knowledge and the conception of potential interventions. While this is surely a very wide notion of explanation, there is no need to constrain our views further for the purposes of this thesis. Thus the problem of explaining variation in evolution to be addressed in the following chapters can be framed into these broad considerations: in order for explaining a particular evolutionary pattern, it will suffice to find among the causes that may be relevant under a particular theoretical context and background knowledge, the ones that explain the better in a pragmatic sense.

c) Dispositional explanations

Let us now make the case for dispositional explanations. First of all, we can distinguish different types of phenomena in need for explanation. For instance, Cartwright (2002) argues that explanatory relevance is always applied to general cases, but that singular events demand a different approach in terms of single-case causation. In her *Nature's Capacities and their Measurement* (1994/1989), she exposes that, unlike directly testable causal claims, general claims refer to *capacities*, namely to "facts about what things can do" (Cartwright 1995, p. 156). Her view is that most scientific laws cannot be interpreted in terms of regularities and categorical properties, but in terms of capacities instead. Although Cartwright claims that not all 'capacities' are dispositions (2007), her characterization of them is somewhat similar to the one presented above.[16] Just like in the case of dispositions we can distinguish between the disposition, its manifestation and its effects, Cartwright indicates that her notion of capacity requires a threefold distinction between (1) "the obtaining of the capacity", (2) "its exercise" and (3) "the manifest ('occurrent') results" (2007, p. 24), similarly to the latent form of a disposition, its manifesting and its effects. In

[16] Cartwright acknowledges the similarity between a capacity and a disposition, but she points that capacities are typically not associated with a conditional, for "there need be no set of conditionals that connects a capacity with specific manifestations" (2007, p. 53). However, dispositions do not necessarily have their set of conditionals specified in order to be classified as such. For example, the fragility of the window can be triggered in infinite ways: different ways of being hit, different ways of suffering from a structural deficiency in the building, different ways of vibrating in response to sound waves, etc. Therefore, I believe that Cartwright's criteria for using capacity-like explanations for general regularities can hold for dispositions as well.

sum, the author refers to a type of system that "always tries to do the same thing even if the results differ" (p. 49). She illustrates this through the following example:

> Gravity [is] the capacity to make heavy objects fall. The attraction of a heavy body constitutes the exercise of the capacity [i.e. the manifestation]; the motion of the heavy body is the actually manifested result when the capacity is exercised [i.e. the particular effect] (*ibid.* p. 25)

Thus dispositions causally explain their manifestation, but not the particular effects that may obtain. In the case of gravity, such capacity or disposition explains the attraction of heavy bodies—i.e. of the manifestation—, but it does not explain, at least in isolation, particular movements of those objects—i.e. particular effects. In turn, each of these distinct facts demands different types of explanation and can be engaged in the explanation of events in distinct ways. Fetzer (1974) has a similar point regarding the explanatory role of dispositions in lawful generalizations:

> It then becomes possible to distinguish between lawful and accidental generalizations of either universal or statistical form on the basis of theoretical grounds, namely that lawlike generalizations are those for which we possess a theoretical warrant in the sense of attributing to the conditions they describe a dispositional property that is lacking in the case of mere correlations (p. 196)

Thus the functional nature of dispositions seems to play an explanatory role for patterns and generalizations. In a similar line, recall Mellor's view about dispositions, which he considered as those properties that explain "conditional regularities" (1971, p. 63, see section 1.2.c above). In turn, it seems like we postulate dispositions for alluding to the causes responsible for certain empirical patterns—manifestations—as well as for subsuming the behavior of objects under specific *types*.

In addition, some authors have considered that ascribing dispositions is an instance of abductive reasoning, meaning that dispositions are often—if not always—the result of an inference to the best explanation (Biggs & Wilson 2019). Let us illustrate this through an example. Consider the competing theories for explaining that windows break more easily than walls or chimneys. On the one hand, we have

the brute regularity that, so far, windows have broken more frequently than walls and chimneys and that their facility to break supervenes on that fact and the system of laws of nature that best describes our world—i.e. the Humean, actualist position. On the other hand, we have the ascription of a property to glass that consists of tending towards breaking when given conditions are met, and that these conditions are more regularly met than the conditions under which both walls and chimneys break—dispositional position. In what sense would the second option explain better than the first one? It would do so in the sense that it renders the breaking of windows a matter of fact—that is, that follows from the properties of the world—rather than an unexplained fact—i.e. a regular fact that we incorporate into a descriptive law.[17] Moreover, notice that a regularity-based account would render counterfactual explanations meaningless. While a dispositionalist can say 'if crystal wasn't fragile, this window would not have broken when hit', a Humean could not make sense of such kind of explanation.

Furthermore, as William Rozeboom (1984) argues, dispositional properties serve as a guide of scientific reasoning in virtue of pointing at the set of properties that could explain a particular manifestation in the inquiry for a finer-grained explanatory theory of the phenomena. I consider that we can see this in everyday reasoning. For example, knowing that a glass is fragile allows us to inquire into the relations between breaking manifestations and structural properties of glass. Since it also allows us to investigate how breaking relates to the laws of nature, our understanding of fragility serves as a heuristic bridge between knowledge of the particulars of a certain class and general knowledge of breaking phenomena. This implies that a finer-grained explanation in terms of specific mechanisms and laws is *in principle* possible. Its possibility, however, does not justify its explanatory

[17] A categoricalist may reply to this that the law is not merely descriptive but nomic. Thus consider now a slightly different competing explanation for windows breaking more easily than walls and chimneys, namely that the categorical properties of glass show a breaking behavior under the laws of nature more regularly than the categorical properties of bricks. Laws of nature would explain the differences between the ones and the others. However, this implies that properties are only contingently related to the laws of nature—i.e. the same object could be subject to different laws of nature in different possible worlds—, and that nothing in the categorical properties of objects explains their behavior, which seems implausible (Bird 2007, see footnote 13 above).

superiority with respect to dispositions under all circumstances. It may be that a particular mechanism is explanatory of a given outcome, but it is not explanatory of general regularities. Or it may be that we want to explain why both windows and eggshells tend to break when dropped. In this case, pointing at separate mechanisms for each will not be helpful, whereas a dispositional explanation can guide towards an inquiry of what are the features shared by both mechanisms.

In sum, causal-mechanical explanations are different from regularity explanations. When we want to explain why a certain window broke rather than not breaking, it seems more explanatory to point at the triggering conditions of the breaking, namely that it was hit by a rock. However, if we want to explain why windows *typically* break, it would not be explanatory to say that they are sometimes hit by rocks, since many things that don't break are sometimes hit by rocks as well. What is more explanatory, in this case, is to point at the fragility of the window. Finally, if we want to explain why the window broke *in a certain way*, pointing at the fragility merely serves as an explanation of its possibility, but not as a fine-grained causal explanation of it. Dispositions are thus explanatory of certain patterns that we find in the world. Our modal reasoning seems to be committed to the existence of dispositional properties in virtue of the explanatory role they play.

Moreover, it is plausible that in some important cases, dispositional explanations are simpler than explanations that don't make reference to dispositions. For example, regularity-based and lawful explanations might be inconvenient to specify and examine in complex enough systems. In this regard, dispositions play the role of labeling a type of phenomenon in a simple, comprehensive way. On the other hand, as pointed out above, referring to dispositions can unify different phenomena under the same type of manifestation, therefore helping in identifying commonalities that may serve for supporting counterfactuals and laws. Furthermore, dispositional explanations might provide more depth insofar as they act at a level of abstraction that allows for non-fundamental explanations (see Weslake 2010). Dispositional explanations are also productive insofar as they can play a heuristic role by pointing at the lower-level properties that realize them—their causal bases.

With all this in mind, let us finally argue for the non superfluity of dispositional explanations. In *Getting Causes from Powers* (2011), Stephen Mumford and Rani Lill Anjum present an account of causation and explanation based on a *powers ontology*, namely the idea that all properties are causally active dispositions.[18] The metaphysical details of their account need not concern us here,[19] but their representation of dispositional causation is helpful for visualizing the ideas considered in this section. The authors model causal relations in a vector space where each vector represents a disposition and thus a causal power. The vectors point towards—or away—the realization of a certain manifestation. For example, we may model the disposition of a given window to break as in Figure 1.2.a.

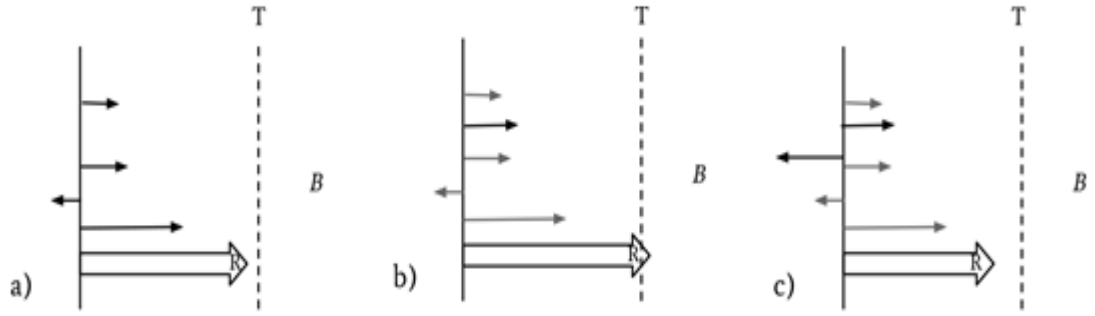

Figure 1.2. Three different vector compositions of the dispositions of a window tending towards and against breaking (*B*). *T* indicates the threshold from which the breaking manifests. Only in b) the threshold is reached. See text for details. Modified from Mumford and Anjum (2011).

The arrows pointing towards breaking (*B*) are those properties that dispose the window to break, such as its physical properties. The resultant *R* is the combination of all the dispositions represented in the same vector space at a given time. Although these dispositions are always tending towards their manifestation in the same way, breaking will only take place once a given threshold is reached (*T*). If a new disposition was added to this vector space that tended strongly enough towards breaking, the threshold would be reached and the manifestation would take place, meaning that the window would break at time t_1 (Figure 1.2b). If, instead, a different vector was added that tended away from the manifestation—such as if a protective

[18] See e.g. Marmodoro (2010) for a defense of a powers ontology that differs from Mumford and Anjum's.

[19] In particular, by no means I want to imply that what follows is only tenable if their metaphysical view of the world is assumed. See McKitrick et al. (2013) for a discussion.

packaging was put around the window—, the resultant at time t_1 would be such that adding the same disposition could not lead to reaching the threshold (Figure 1.2c).

The point of bringing about this mode of representing causal relations is that it makes it possible to visualize what different sorts of things dispositions explain. Fragility explains the fact that small vectors tending towards breaking—that may represent a hitting rock—reach the breaking threshold. It is thus better represented as one large vector tending towards such threshold, rather than as a fine-grained composition of several factors of less strength—e.g. the location, composition and thickness of the window glass. Although all of these factors are part of the causal basis of fragility, they only explain breaking insofar as they combine together to constitute fragility. In the figure introduced above, fragility is the resultant of all the properties disposing towards breaking, and thus has to be identified with the resultant vector rather than with its constituents.

3.2. Causal Propensities

Now that it has been defended that dispositions do explain, we turn to specify what sort of explanation propensities provide. Note that probabilities cannot be argued to explain the way dispositions have been claimed to do so. Probabilities are a type of mathematical modeling that follows Kolmogorov calculus. Dispositions, on the contrary, are a type of property with causal efficacy. A probability measure can be argued to provide a statistical kind of representation, but surely not a nomical, causal explanation. Thus once we engage with the idea that dispositions are explanatory, the possibility that they are identified with probabilities is ruled out. This is not a disadvantage considering that we had already abandoned the task of analysing probability. We are, on the contrary, in search of a way to link chance with the probabilistic models of evolutionary theory—recall that, for a number of authors, probability calculus does not exhaust the "chance role" (e.g. Humphreys 1985, Eagle 2011, Handfield 2012). The present task is rather to connect different roles of probabilities and chance in evolution with the explanatory role that has now been acknowledged for dispositions. What follows, in turn, is a vindication that theoretical

propensities are directly explanatory of probabilistic phenomena, and responsible for frequency patterns in turn.

The theoretical nature of propensities is acknowledged by propensity proponents such as Giere (1973), in opposition to the empirical quality of frequencies. Thus their role as explanatory is not entirely disentangled from the original aims of propensity advocates. However, in line with the remarks about dispositions causally explaining their manifestations but not their effects, the efficacy of propensities for the single case may not be as clear as for frequencies of events or, more accurately, for probabilistic patterns. As Giere states,

> A central question concerning the relation between single-case propensities and frequencies is whether it is possible to deduce values of one from values of the other. The answer, as one would expect since propensities are theoretical, is negative. (Giere 1973 pp. 504)

The apparent lag between propensities explaining patterns and the realization of particular events is an instantiation of the already mentioned explanatory role of generalizations for their manifestations, by contrast to their specific effects. Thus this understanding of propensity may be problematic for assessing the single-case in causal explanatory terms. However, it will prove virtuous in deriving a non-causal explanation of the single case from the long-run, or from the probabilistic patterns that they causally explain.

Since propensities are theoretical properties, one central question is how they are inferred. As Giere continues in his work, "in the absence of a well-developed theoretical background, observed relative frequencies may provide the only evidence for propensity statements" (Giere 1973, p. 504). However, it is worth wondering what a "well-developed theoretical background" may be that could serve as a basis for inferring the causes of a probabilistic phenomenon beyond a mere induction from relative frequencies. In this final section, first I present the notion of causal propensity, and then I introduce its relationship with probabilistic modelling.

a) Propensities as structuring causes

According to the definitions introduced in section 3.1.1, the only requisite for a disposition to be a propensity is that it is not surefire. That is, a propensity is a disposition to manifest *probabilistically* or, perhaps more correctly, in a chancy way. This is to say that propensities are causally responsible for probabilistic patterns, which raises the question of how to regard them as causally efficacious in the single case. A probabilistic pattern is something that manifests either in the long term or in an ensemble of dispositions. Therefore, it is worth questioning what is the contribution of such a type of manifestation to particular, single effects in which the propensity is causally involved.

Let us turn back to the fair coin example. When tossing a fair coin, we may observe a pattern of results where heads and tails are approximately equally instantiated. For explaining such a pattern, we infer that the coin has a propensity to land both heads and tails with equal strength. This propensity acts as a theoretical term invoked in explaining a phenomenon to which we assign a probability distribution of $\frac{1}{2}$ probability of heads and $\frac{1}{2}$ probability of tails. Now imagine that we encounter a piece of wood with the shape of a coin. We may infer, without any need of tossing it, that the piece has the same propensity as the coin has. The same propensity would explain this *expected* pattern even if the piece of wood is never actually tossed. In turn, frequencies may serve as guidance in assigning probabilities, but it is dispositions that we employ for causally explaining them. Propensities are explanatory of these patterns of behavior because they are the cause that makes the difference with respect to them. The propensity instantiated in both the coin and the piece of wood explains why they behave probabilistically in a particular manner *when*—or *if*—tossed in contrast to things such as balls or pens.

However, propensities are typically not difference-makers of their particular effects. If we want to explain why our coin landed tails *in a specific toss*, its propensity to land tails may not be as good an explanation as the triggering conditions of this particular toss. The probabilistic manifestation of the propensity surely contributed causally to the effect 'landing heads'. Nevertheless, the explanatory

relevance of the propensity in such context is shared with the specific triggering conditions that lead to this effect. In this regard, the propensity is only explanatory of the possibility of the effect as subsumed into the possible instantiations of the probabilistic manifestation it causally explains.

Following Grant Ramsey (2016), I believe that propensities are better understood as *structuring causes* of probabilistic behavior. If a triggering condition of a propensity makes a difference for a particular effect of the disposition, the propensity itself is a difference-maker of a particular causal structure relating triggering to manifesting conditions. In other words, it is a difference-maker of why things *tend to* behave in such and such way. The distinction between structuring and triggering causes can be found in Fred Drestke's (1988, 2004) work on the explanation of psychological behaviour. Although this distinction is analogous to the distinction between a disposition and a triggering cause, it is helpful in suggesting that what propensities do is *to structure* a space of possibilities. So a propensity structures the possible ways in which a triggering condition can affect the system that yields it. For example, the propensity of a die to land on its 6 different faces structures how velocity and angle affect the result of a trial. So imagine that we roll the same die ten thousand times and that we end up with roughly $\frac{1}{6}$ of each of the possible results. The propensity of the die manifests itself in this pattern, and it is the *structural* (but notably not the difference-maker) cause of each of the results. In other words, the initial conditions of each roll could only lead to these 6 different outcomes, and exactly with the same probability, because of the causal structure relating those conditions to outcomes.

Ramsey (2016) illustrates this idea with the example of a fruit falling into a river that bifurcates into two branches. The process leading from the fall of the fruit (initial conditions of triggering cause) to the arrival of the fruit to one of the possible destinies set by the structure of the river (effect) is a probabilistic process in the same sense that rolling a die is (Figure 1.3).

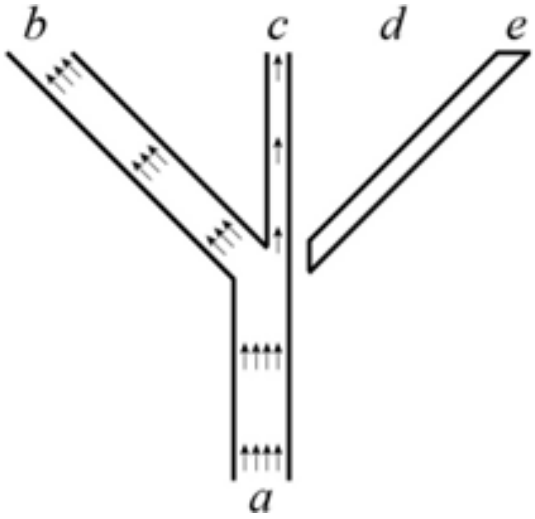

Figure 1.3. A river ramifying into three different branches, whose structure leads fruits from *a* to either *b* or *c*. End-states *d* and *e*, on the contrary, are forbidden by the structure of the river. Reproduced with permission from Ramsey (2016).

Thus Ramsey (2016) writes:

> While the triggering cause of the fruit's destiny is its plunge into the river, the structuring cause of the fruit ending up at *b* is the erosion, deposition, and other forces having set the river's shape. Given this shape and the flowing water, we have a causal explanation for why the fruit ended up at *b* instead of, say, *d* or *e*. The riverbed morphology prevents the fruit from ending up on dry land, *d*, or in the isolated lake, *e*. Considering the question of why the fruit ended up at *b* instead of *c*, the structuring cause results in intermediate probabilities for each outcome, but does not say with certainty which one will occur for each fruit in question. (p. 7)

In this case, the causal structure of the river structures the set of possible results, namely arriving at *b* or *c*. Similarly, the causal structure of a die determines the set of possible results of a roll. It is this structural cause that grounds the propensity. Ramsey (2016) takes this precisely as an example of how dispositions can structure the process of evolution, by considering both fitness and drift in this way. I will elaborate further on his account about evolution in section 2 of the next chapter. In any case, thinking of propensities as the structuring causes of a space of possibilities fits into the requirements posed for causal explanations of evolutionary change. Propensities to survive, reproduce, and produce variants of a certain kind, are

the causal and explanatory ground of evolutionary possibilities. They are, nonetheless, only part of a complete causal explanation of particular evolutionary events.

A further question is to consider other epistemic virtues that propensities may have with respect to explaining probabilistic phenomena. A propensity can have the virtue of unifying different phenomena under the same capacity, namely all the phenomena that behaves probabilistically in the same way. But also, and perhaps more importantly, it is a heuristic tool for deepening into mechanisms underlying it. As Mellor highlights with regards to the explanatory virtue of propensities:

> We require (...) of at least the explanatory and explainable dispositions introduced by the sciences that they be linked to other properties and relations of the entity. (...) The links between dispositional properties that make them nontrivially usable in explanation are the laws into which they enter, however loosely these may be formulated. What laws a disposition enters into is of course contingent. It is no part of the meaning of 'soluble' that solubility is connected specifically to chemical composition. (1971, pp. 65-6)

But this is exactly what a propensity amounts to inquire. While propensities remain at a level of abstraction that allows for unification, they also point at the specific behaviours of a probabilistic system that may need further explanation, be it in dispositional terms or not, and thus they serve as a means for scientific development.

With all this in mind, we may define a *causal propensity*[20] as follows:

> A causal propensity is *a probabilistic disposition that structures a space of possibilities in such a way that it is possible—in principle—to derive a probability measure from it in different contexts*.

That is, it is the capacity of a chance setup to manifest a probabilistic pattern, susceptible of being represented by probabilistic models. This implies that the probabilistic representation is *in principle* possible but need not be well-developed in

[20] The term 'causal propensity' was used in (Good 1984) in the rather different context of Bayesian probability, a subjective, inference-based account of probability. Nonetheless, to my knowledge this is not a widespread use in the literature.

mathematical terms. That is, a causal propensity is a property explaining patterns to which probability measures *could be applied*, but its existence does not depend on there being a well-defined probabilistic model of the pattern. The possible outcomes of the chance setup trials will be both limited and enabled—i.e. structured—by the causal propensity, while the particular effects resulting from each trial will highly depend on its triggering or initial conditions.

b) Propensities and probability

This discussion has moved far away from the inquiry of interpreting probability, notably the reason why propensities were introduced in the philosophical literature in the first place. Recall that the philosophy of probability is mostly concerned with the truth-makers of probability calculus, namely those things that satisfy it and ground their application to the world or our reasoning about it. Using causes for explaining probabilistic patterns is a different task. However, these probabilistic patterns are typically modeled through probability calculus. A few remarks on the relation between explanatory dispositions and the propensity interpretation of probability will therefore be helpful in order to situate the present approach.

When Popper introduced propensities in his (1959), what relation holds between probabilities and propensities was left ambiguous. On the one hand, he would regard propensities as an interpretation of probability. In fact, he developed a different probability calculus in order to reconcile the identity between probabilities and propensities. On the other hand, sentences such as the following seem to require that both notions refer to different things:

> But this means that we have to visualize the conditions as endowed with a tendency, or disposition, or *propensity*, to produce sequences *whose frequencies are equal to the probabilities*; which is precisely what the propensity interpretation asserts (1959, p. 35. Stress added)

Formulations of the type can be found among propensity advocates. Recall that Gillies, for example, claims that propensities are tendencies "to produce in a long series of repetitions (...) frequencies which are approximately equal to the probabilities" (2000b, p. 822). How exactly propensities and probabilities relate to

each other is not always addressed, however, in the propensity literature. A distinction between propensities and probabilities has nonetheless been actively defended (as we saw in section 1.2c) by Suárez (2018), who criticises that virtually every propensity approach falls into what he calls the "identity thesis", namely the assumption that propensities are probabilities:

> [C]ontemporary theories tend to identify propensities and probabilities, which means that to be coherent they must ascribe propensities to the elements of a sigma field, i.e. events, or propositions (2013, p. 67)

The details of this criticism need not concern us here, but the author's proposed solution points at some of the considerations about explanation and dispositions of the previous sections, and thus can be helpful for the present discussion. The proposal, presented in (Suárez 2017), consists in fitting a threefold division between frequencies, probabilities and propensities into the general tripartite distinction between observed data, inferred phenomena, and explanatory theory that James Bogen and James Woodward (1988) introduced in the philosophy discussion on scientific modeling. Bogen and Woodward defend that, unlike phenomena, data are specific to a particular experimental arrangement and do not necessarily show regularity. Phenomena, on the other hand, are real, stable features of the world that do not depend on data. Data serves as evidence of a particular phenomenon, giving grounds for its inference. Finally, theoretical frames and notions are embedded in the explanations of phenomena, but not of data.

Following Suárez (2017), we can therefore understand the frequencies of a certain probabilistic event as data that serves as evidence for probabilistic phenomena, while propensities serve as an explanation of such phenomena. This is in line with the metaphysical distinction between a disposition, its manifestation and its effects introduced above. Under this view, a probabilistic phenomenon—i.e. a manifestation condition—is thus inferred by the consideration of both frequencies—i.e. particular effects—and propensities—i.e. inferred dispositions. Through this inferential process, a statistical model for representing the probabilistic phenomenon is established, where probabilities will have a representational role.

Similarly, recall Giere's claim that "relative frequencies may provide evidence for propensity hypotheses" (1973, p. 504). According to his view, frequencies might be the only evidence available for assigning propensities when the theoretical background is not sufficiently developed. This implies, however, that developing a theoretical frame for inferring propensities in the absence (or scarcity) of relative frequencies is indeed possible. At the end of the day, that is just what we ordinarily do when we ascribe the same dispositional properties to similar objects regardless of them being manifested (see previous section).

As for the relation between propensities and other causal accounts, particularly the range conception, an argument can be made for the better explanatory role of propensities. Let us say that, from the range conception view, the probability of landing on 4 of a die is the ratio of initial angles and velocities of a roll leading to the result 4. This ratio is an abstract, mathematical relation, and indeed probabilities are. An explanation of such ratio is, however, not fully provided by the range conception. A propensity, on the other hand, can be involved in the explanation of such ratio. Even if the specific causal paths can be depicted linking initial stages to manifestation events, the type of properties that explain the probabilistic pattern represented in the ratio relation—such as the symmetry of the die with respect to potential results—can be argued to be of dispositional nature. Let us recall that evolutionary explanations tend to stress why some evolutionary state of affairs is likely to be the case, rather than pointing to the specific causal pathway leading to it. For instance, in the evolution of a trait, it suffices to point that the trait was likely to obtain considering reproductive and selective factors. On the contrary, lower-level details of the evolution of the trait—such as the actual depiction of all genetic transformations that led to the trait's appearance in a population—are not explanatorily relevant. A natural range conception of probability would explain this by saying that a significant ratio of possible initial states led to the event under consideration by virtue of the dynamics relating the two. In addition to overcoming the problems derived from identifying ranges with probabilities—which could be ruled out by alluding to an explanatory version of the range conception—, a causal

propensity view would add to this explanation something fundamental: the event was just one possible effect of a tendency that has been manifested.

Finally, it is worth pointing at two matters regarding the nature of probability. On the one hand, there must be a connection between propensities and subjective probabilities. An important principle of chance not explicitly mentioned so far is that it must show a connection with subjective probabilities (Eagle 2019). Lewis' Principal Principle, usually taken as a basic way to formulate this relation, states that subjective probabilities or credences must approximate objective probabilities. Hoefer defines this principle in the following way:

> Let *Cr(_|_)* be a rational *subjective* probability function (credence function), *A* be a proposition in the domain of an objective chance function *P(__)*, *E* be the rest of the agent's background knowledge, assumed to be "admissible" with respect to *A*, and *X* be the proposition stating that *P(A) = x*. Then:
>
> *(PP) Cr(A|XE) = x*
>
> (2016, pp. 35-36)

The Principal Principle follows naturally from an account of probability of this form, insofar as all probabilities are actually inferred. However, this is not to say that, under this approach to propensities, all probabilities are subjective. On the contrary, it merely means that, since agents are able to infer *objective* probabilities when they model probabilistic phenomena, it is reasonable to expect that their degrees of belief will follow from them. As Giere (1976) points out,

> [I]t is correct to say that the propensities we attribute to macroscopic systems are in some sense relative to our knowledge. Yet these instrumentally assigned values are in no direct way a measure of our knowledge or our ignorance of the systems in question. (p. 346)

On the other hand, conditionalizing should not be problematic through this understanding of probability. This is so because the mathematical body of probability represents specific, context dependent features of the manifestation of propensities—the probabilistic behavior—, rather than modeling the propensities themselves. If probabilities are part of mathematical models of phenomena, they are

not actually representing the causes that explain such phenomena. So the asymmetry embedded in causal relations that rendered propensities inadequate for analysing probability does not hinder them from explaining the phenomena that probabilities represent. Hence probabilities can obey Kolmogorov's calculus without propensities falling into Humphreys' Paradox.

Evolutionary models invoke probabilities in a number of ways: the probability of the fixation of an allele, the probability of an evolutionary change taking place, the probability of a particular type to survive and reproduce. The explanatory scope of dispositional terms in evolution is, nonetheless, much wider and nuanced. As we shall see in the next chapters, a causal propensity view such as the one presented there, can be fruitful in bridging the gap between the numerical treatment of probabilistic models of evolution and the complexity of evolutionary explanations, where chance tends to play an important role.

4. Concluding Remarks

In this chapter, I have discussed the main views on probability and chance, especially with regards to those ideas that I believe are important for the philosophical debates over evolutionary biology, in order to establish a suitable framework for analysing them in the following chapters. In the first section, I have reviewed the received approach to objective probability, namely the opposed traditions of frequentists and propensionists. I have shown the main features as well as limitations of these traditions, and I have related their views to the main positions about evolutionary probabilities. On the one hand, statisticalist positions about evolution have aligned with the arguments of frequency advocates. As we have seen, the main flaw of the frequentist interpretation of probability is the so-called reference class problem, an ingredient that will prove fatal for the alleged statistical means of explanation of statisticalists. On the other hand, evolutionary causalists have typically vindicated the propensity interpretation of probability. In this regard, by introducing generating conditions and dispositions into the probabilistic puzzle, propensities have proven to be fruitful in evolutionary explanations. Fulfilling the expectations of causalists,

however, remains problematic for propensities given the complexity and plurality of positions about them.

In the second section of this chapter, I argued for an abandonment of an interpretation of probability calculus as the paradigm for understanding the role of probabilities in evolutionary models. Instead, I advocated for their close connection to chance as an explanatory notion in evolutionary biology. In this regard, I reviewed how propensities fail to assess the role of causation in probabilistic relations, and I stressed that evolutionary explanations are indeed committed to a broader notion of chance. Moreover, through the notion of causal probability, I argued for the possibility of explaining probabilistic patterns even if determinism is assumed and if we engage with a causal explanation of them. This broader understanding of chance demands conceptualising the possible by means of what different scenarios a given causal arrangement can bring about. In turn, a connection of chance in evolution with a notion of randomness in evolutionary patterns is possible through the construction of probabilistic models, associating the causal explanatory nature of chance with the mathematical models of probability.

In the last section of this chapter, I have argued for the explanatory role of propensities in this understanding of chance, specifically through the notion of a causal propensity. I have defined a causal propensity as a probabilistic disposition that structures a space of possibilities in a way that probabilities can be applied to them in principle. The usage of a causal propensity, as we have seen, is justified by several features of dispositions, such as their functional and dynamical individuation, the distinction between their manifestation and effect that they allow for, as well as their multiple realizability. In this regard, I have defended that propensities have a theoretical, explanatory role that, under some contrastive contexts, account for those phenomena that we model through the tools of probability calculus. I have further argued that propensities can be considered as explanatory of probabilistic phenomena independently of a variety of factors, notably the existence of indeterminism and the level of description at which the probabilistic phenomenon is modeled. In doing this, propensities must be distinguished from probabilities, which

are mathematical models of probabilistic phenomena, and which therefore play a representative but non-explanatory role.

In turn, I believe that this general framework will be fruitful in its application to the probabilistic models of evolutionary biology, where complex systems and dispositional explanations are usually involved. We shall see in the next chapters of this thesis how this understanding of chance and probability can be applied to these models, helping as well in finding the causal hypotheses embedded in them. In the following, we shall see how this view helps in discerning what are the causal components considered in classical models of evolutionary genetics (Chapter 2), and to construct a causal notion of chance for the probabilistic notions of evo-devo (Chapter 3).

CHAPTER 2

Variational Probabilities And The Sample Space Of Evolution

In short, variation is an endless source of challenging questions

Ernst Mayr

0. Introduction

Now that we have established a conceptual framework for assessing chance in probabilistic models of evolution, let us deepen into the puzzle that the chancy nature of variation poses to the explanatory structure of evolutionary biology. Chance has been a matter of concern particularly for two aspects of the evolutionary process, namely for the spread, fixation and disappearance of variants in populations, responsible for changes in their composition and structure; and for the very origin of those variants, responsible for the possibility of those changes in the first place. A very important ingredient of my exposition here will be the different treatment—ironically based on their characterization as similar—that these two aspects of evolution have undergone historically, which in turn has influenced the practices and theoretical approaches of biologists, and so the probabilistic models

used for representing them. The so-called "problem of variation" (cf. Stern 2000) refers to the latter of these aspects—i.e. the origin of variation—, and it is the core of evo-devo vindications about the role of development in evolution and the main concern in the remainder of this dissertation.

The problem of variation has many different aspects and connotations, and understanding its genealogy is as important as discerning its historical role from the role that it plays in contemporary debates. The world of living beings is characterized by a wide diversity of forms, but also by the striking unity of characters and structures across species, a situation that has always been a source of bewilderment for naturalists and biologists. The fact that, in contemporary philosophy of biology, variation can be seen as a random phenomenon for some (e.g. Merlin 2010), while considered as not random in any defined expectation by others (e.g. A. Wagner 2012a) is the consequence of a discrepancy about variation and chance whose origin can be traced back at least to the very origin of evolutionary thought. However, understanding this discrepancy in contemporary biology requires, in addition, to consider current practices and models of evolution. My view is that assessing these ideas from a causal propensity approach can help in understanding the importance of variation in evolutionary explanations, as well as in situating evo-devo claims into a causal perspective of evolution. As briefly mentioned in the previous chapter (section 3.1), the historical explanations of evolutionary biology involve showing the plausibility of an evolutionary *explananda* given the particular space of possibilities derived from postulated causes. Thus considering the framework developed in Chapter 1, explaining evolutionary facts demands, as we shall see in this chapter, pointing at the propensities responsible for such space of possibilities, which in turn will give grounds for the refinement of probabilistic models describing and predicting the phenomenon they pertain to. Consequently, I will defend that the problem of variation can be understood for the present purposes as the problem of conceptualizing the possible in evolution by virtue of the very causes of variation, that in turn should be interpreted as propensities.

The conception of the possible in evolution has been strongly influenced by the Darwinian paradigm of "population thinking". According to the most classical

and influential view of evolution (e.g. Mayr 1959, 1972, Sober 1980), Darwinism provided a systematic approach that entailed the end of 'typological thinking' in biology—a position that had presumably prevailed all over the course of natural history since its origins in Aristotle's writings—and replaced it with "population thinking" (cf. Mayr 1959). For instance, Sober (1980) argues that, before the rise of Darwinian thought, naturalists advocated what he calls Aristotle's "Natural State Model", where variation, understood as diversity, is a deviation from the "natural state of things". Thus the causes of variation for pre-Darwinians were, according to this commonplace story, disturbance forces with the capacity to influence the "natural state" only insofar as this state is contingently perturbable. The population kind of thinking endorsed by Darwinian evolutionary theory, by contrast, involved that only individual variations are real, and that characterizing unity—e.g. characterizing a group of individuals as one species—is a matter of abstracting away the statistical properties of populations. We shall see in this chapter that typological and population thinking—both pre- and post-Darwinian—are way more complex than this story tells. However, the exaltation of population thinking and its virtues in the modern understanding of evolution has crucial consequences for how the possible is taken into consideration in it. In broad terms, population thinking entails that only differences among individuals are perceived as fundamental for evolutionary purposes, and that these differences ground the tendencies of variants to spread and reproduce. As a consequence, the possible gets conceptualized as the conceivable differences in survival and reproductive capacity—as conceivable differences in fitness and adaptation.

In connection to this is the traditional separation between the 'proximate' causes of biological phenomena, namely those that explain their synchronic functioning; and their 'ultimate' causes, or the causes that explain their remote origin in an evolutionary sense (Mayr 1963). Only the latter are typically conceived of as pertaining to the scope of evolutionary biology, the former belonging to the domain of functional branches of the life sciences—such as physiology, molecular biology, genetics or ethology. This separation conceptually hinders any consideration of how the proximate causes of diversity and unity relate to what is possible in an

evolutionary sense. The exceptions in the contemporary paradigm are genetics and ecology, the former articulating how traits are inherited, the latter showing how traits ground differential survival and reproduction in natural populations. In this context, the already mentioned notion of the possible as the conceivable differences in survival and reproductive capacity is by large represented in the ecological notion of (heritable) fitness understood as a propensity. As mentioned in the previous chapter, this understanding of fitness is the core of the causalist position about evolution. However, as we shall see, the potential of fitness for accounting for the possible in evolution is strongly limited. Recognizing this will come at hands with the vindication of a role for other seemingly 'proximate' causes of biology—notably development—into the ultimate explanations of evolution.

In this chapter, I argue that the origin of variants on the one hand, and their fate in populations on the other, are considered differently in the theoretical framework and the classical models of evolutionary genetics, and that, accordingly, the philosophical considerations about chance and dispositions have equally been substantially different in each of them. I will be critical with this state of affairs, aligning with evo-devo vindications about the role of development in causing evolutionary changes. Leaning on the framework developed in the previous chapter, I consider here the causal propensities responsible for the conceptions of the possible embedded in evolutionary probabilistic models, both at the level of the origination of variants and of their fixation in a population. In doing this, I reconstruct the explanatory structure of both mainstream evolutionary biology and evo-devo from the point of view of causal probability and propensities. In turn, this will lead me to advocate for an explicitly developmental notion of chance in evolution. Such a conception will be the starting position of the analysis of evo-devo probabilistic notions in Chapter 3.

The chapter is divided as follows. In Section 1, I present how variation and chance are conceptualized in modern evolutionary biology by showing their Darwinian historical roots and by exposing the probabilistic models of evolutionary genetics in their philosophical consideration. In Section 2, I deal with how chance is understood in these models with regards to population dynamics, namely to the

spread and fixation of variants in evolution, and I argue that causal propensities can be fruitfully applied to explanations at this level. Section 3 argues that the origin and generation of variation have been approached through a different prism in these models, derived from the primacy of selection as an agent in evolution. I will claim that this perspective, in contrast to the causal arguments about population dynamics, has prevented any causal understanding of the production of variation in similar terms. Finally, in Section 4 I argue that the evo-devo understanding of development as producer of variation serves in providing evolution with a suitable sample space, or space of the possible, that in turn fills the causal gap in evolution about the origin of variation.

1. Variation in the evolutionary tradition

Darwin has been pictured as the "Newton of the grassblade" (Haeckel 1868, Grene 1974), in allusion to Kant's asseveration that even apparently simple living forms such as a blade of grass could never be explained without referring to purposiveness (Cornell 1986, Nuño de la Rosa & Etxeberria 2010). The Newtonian worldview is one where purpose is an epiphenomenon and only passive reactions to external forces explain the existence of movement. Although mechanicism as such had intended to colonize the living world way before Darwin (see Nicholson 2012), the origin of the complexity, divergence and adaptation of living beings could arguably not be completely accounted for in mechanistic terms until the Principle of Natural Selection was introduced into the picture. In this sense, purpose was released not from organisms themselves and their life cycles, but from their very diverse and adapted nature: the fact that there are complex living beings is explainable in the same terms than the movements of planets are. In turn, only passiveness towards external forces such as natural selection was needed to explain the diversity of life.[21]

[21] However, notice that explaining evolutionary changes in mechanical terms is by no means an exclusion of purposiveness in evolution. For, how to explain the origin and maintenance of self-organization and reproduction? Darwin's "struggle for existence", intended to be an *explanans*, can be problematized if turned into an *explanandum* by raising the question of why individuals are supposed to persist and, even more strangely, reproduce no matter what (Richard Dawkins' (1989) *selfish gene* idea reduces the later to the former, but the problem of self-preservation still persists). As John F. Cornell (1986) wrote: "Hence Darwin's famed triumph

In this anti-teleological perspective of the living world, variation has to play the role of 'raw material' of evolutionary changes without itself breaking the Newtonian picture of passiveness. On the one hand, it has to be susceptible to changing the composition of populations merely by the differential reproduction of variants—i.e. it has to enable population thinking. On the other hand, it has to originate without responding to any purpose of organisms. These requirements deeply determined the conceptualization of variation and chance in evolutionary thought, influencing as well the probabilistic models of mainstream evolutionary biology of the last century, and in turn the philosophical ideas about them.

In this first section, I first review the Darwinian notion of chance variation as the 'raw material' of evolution and its instrumental role in the theoretical building that led to the Modern Synthesis of evolution in the last century (section 1.1). Then, I present the philosophical understanding of the modern models of evolutionary genetics that arose from the Synthesis and the probabilities embedded in them, especially with regards to the analogies raised with physical systems (section 1.2). This will serve me for assessing in subsequent sections the notions of chance and the possible in connection to these models.

1.1. Chance variation: the 'raw material' of evolution

The world of the life sciences before the rise of Darwinian thought was somewhat pattched, but a vast number of fields in it involved a certain conceptualization of diversity: morphologists, embryologists, naturalists, breeders and taxonomists engaged in discussions on what diversity and unity among organisms and species represent. The work of Darwin is situated into the discussion of variation with regards to the taxonomic 'problem of species', and his theory of evolution by natural selection aligned with the trend in this tradition towards a statistical view of

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over teleology must remain difficult to measure. Our assessment of the extent to which he explained the teleological properties of organisms -and became Kant's "Newton of the grassblade"- depends on how much we believe such an explanation is possible if it fails to confront fully the mystery of purposive, self-organizing matter" (p. 421). There is an important philosophical literature devoted to this problem (see the recent, comprehensive treatment of the core issues dealt with in this tradition by Moreno & Mossio 2015). Fascinating as it is, however, it will not be addressed in this dissertation.

variation. As we shall soon see, the diversity of organismal forms was approached by morphologists in a different way, and remained relatively independent from the influence of Darwin's theory. In the following reconstruction I leave aside the morphological tradition, which will be addressed in the last section of this chapter (section 4), to focus on the scientific atmosphere where Darwin elaborated his theory. Any extensive discussion of Darwin's ideas, which have been the object of uncountable historical, philosophical, scientific and even theological studies since their publication, is beyond the scope of this thesis. Instead, in what follows I lean mostly on the works of the historian Peter Bowler (1990, 2005) and the philosopher John Beatty (2006a, 2008), since their work reflects in enough depth the philosophical aspects of Darwin's views about variation and chance.

Naming and classifying the diversity of organisms into different categories was the task of systematics and, from the 18th century, of taxonomists. From the point of view of classifying organisms, taxonomists, naturalists and breeders were interested in extant differences among closely related individuals. Which characters showed variation within populations of the same species, and which differences between populations indicated the existence of distinct varieties or species were their main epistemic targets, in their pursuit of criteria for coherently labelling specimens into categories. For example, Linnaeus' first systematization of a classification of the natural world (1735) established an ordered hierarchy of classes, orders, genera, species and varieties that aimed to apply to all plants, intending to provide an exhaustive and mutually exclusive way to segregate specimens into logical classes based on their traits.

This tradition cohabitated with a variety of positions with regards to the origin of variation, reproduction and inheritance of organismal characters. However, among naturalists of Darwin's time, the most extended view was that the determinant—controversially often referred to as "essential" (see Winsor 2006)—characters of species are faithfully inherited and reproduced from parents to offspring, while differences among individuals are not: they are accidentally recreated in each ontogeny (Bowler 2006a, Müller-Wille & Rheinberger 2012). Thus variable traits in a population were considered the accidental consequences of disturbing

forces, and variant individuals as the result of deviations in ontogeny from the species 'norm', due to either internal or external forces acting upon inheritance (Sober 1980, Bowler 2005). Consequently, this view opposed variation to the very idea of heredity. While the latter was conceptualized as the copy from parent characters to offspring, the former was conceived of as the deviation from exactness in this process, limiting the scope of heredity in a population. A key component of this view was the intrinsic disposition of species to vary in specific ways. As Mayr (1970) put it, under such a view,

> Every group of animals is predisposed to vary in certain of its structures, and to be amazingly stable in others (p. 365)

The historian of evolutionary thought Peter Bowler has called this tradition the 'developmental viewpoint' of variation (2005), where variation was understood as the product of additions or disturbances to the process of ontogeny. Accordingly, deviations in the hereditary process were also conceived of as changes in the development of new individuals. Indeed, many naturalists considered, before and during quite some time after Darwin, that the forces causing variations were situated in the ontogenetic drive of individuals:

> Even if the variant were triggered by an external factor disturbing heredity, the disturbance was likely to generate a change that was to some extent latent within the processes of ontogeny. (Bowler 2005, p. 10).

Moreover, since the distinction between soma and germ lines was not introduced before the end of the 19th century, naturalists typically contemplated the transmission of acquired characters—which, as it is well known, was accepted by Darwin himself. That is, it was common belief that changes acquired throughout lifespan—perhaps through the use and disuse of parts—could be transmitted to offspring just like any other type of variation.

However, the extent to which variations were able to produce changes in a species was a matter of discussion in the 18th and 19th centuries. Whereas some taxonomists were inclined to believe that variations were capable of inaugurating new species, others saw variants as strongly marked members of the same species. In

this regard, it was usual to distinguish between small variations, often considered of a superficial character, and greater variations or 'sports' of nature. The role of each of these was distinct for many naturalists, who often believed that small variations were trivial, whereas larger-scale, abnormal variants could originate new separated inbreeding populations—although not necessarily a new species. Even though the taxonomic place of variants was oftentimes a source of disagreement among naturalists, the existence of well-differentiated varieties and subspecies derived from the same species in the wild was acknowledged by most of them. The work of breeders in originating and perpetuating new varieties was also well known, even if the nature of those varieties was a matter of dispute. Within these debates, some transformationist ideas in the 18th century contemplated the origin of a new species from changes undergone by a previous one. Worth of mention are the well-known evolutionary theory of Jean-Baptiste de Lamarck, who believed that use and disuse of parts could transform species; and the saltationism of morphologist Étienne Geoffroy Saint-Hilaire—about whom more will be said in section 4—, who considered that some abrupt changes taken place in development could instantaneously initiate a new species.

a) Darwin's notion of variation

Darwin owed much both to the treatment of the 'species problem' of the taxonomical tradition and to the plurality of views about inheritance and reproduction of his time. In this sense, he was framed into the traditional view that considered variation as a "disturbing force" in the conception and development of an individual that made it deviate from a species "norm" (Bowler 2005). However, unlike many of his contemporaries, in *On the Origin of Species* (2009/1859) Darwin regarded the results of these deviations as "chancy" in the sense of undirected, and he considered that such deviations accumulated indefinitely, which supposed a significant departure from the developmental views of other naturalists. Darwin argued that organisms within the same species compete with one another in their environments—they struggle for their existence—, some reaching resources and reproducing with a better degree of success than others. This process preserves and

accumulates some variants, of "chancy" origin but fitter than others to their environment, while discards others, producing tendencies in populations that can originate new species in the long run.[22] Even if evolutionary ideas were already present at the time, his forceful, "long" argument (cf. Mayr 1991) about the mechanism of natural selection, and the abundance of details in his descriptions of sources and empirical observations made his work a unique piece for the science of his time with an unquestionable impact in the community (Browne 2002).

Darwin's writings were highly influenced by the view of breeders, who stressed the importance of individual variants. He devoted much of his work to accumulated empirical data showing that there are always small differences among individuals in natural populations, and that the differential survival of those differences can, in principle, explain adaptations. The English naturalist took adaptation to environmental conditions as the main phenomenon to be explained in solving the problem of species, stressing the importance of ecological opportunity in their transformation: it is not the direction of variation what determines evolution, but the fact that small variations can make a difference in the reproductive success of organisms, and thus produce, in the long term, specific divergences. Moreover, in deriving his analogy with artificial selection, he interpreted that the production of variation was incentivated by selection conditions, and argued that domesticated plants and animals usually presented not only a higher level of diversity within groups, but also a higher number of "sports" or big deviations of character (Darwin 1868). In other words, Darwin considered that strong ecological selection of variants would enhance the production of new random variants that could be selected, because of an alteration of the reproductive conditions. This influence in the production of new variants, as we shall see in section 3 of this chapter, is crucial for the contemporary idea that selection is responsible for the production of variation.

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[22] This "natural means of selection" of variants leading to the diversification of species had been introduced in conjunction with Alfred R. Wallace in a communication to the Linnean Society the year before the *Origin* was published (Darwin & Wallace 1858). However, the developments of Darwin's and Wallace's ideas were independent, and Darwin's 1859 book not only had a much greater impact in the scientific community of the time, but included many aspects now associated with natural selection that were absent in the work of Wallace, such as the importance of individual variants, the possibility of a common ancestor to all living beings, and the analogy with artificial selection (Bowler 1990, Beatty 2016).

Darwin saw these random variations as the "raw material" for natural selection (Bowler 2005), that would provide evolutionary changes with directionality, while he would "leave details to chance" (Beatty 2008). That is, while natural selection brings the adaptive directionality to the process of evolution, non-directional differences may take place among lineages with the same selective pressures simply because the material selection acts upon may differ by chance. These chancy differences, nonetheless, are considered irrelevant for explaining trends in the long term. For Darwin, the survival of fitter characters is what leads to "directional change, divergence of species and speciation" (Beatty 2016, p. 662). Darwin acknowledged that his characterization of variation as chancy was partially due to the unknown nature of its causes, pointing to an epistemic notion of chance. However, he provided another, ontological sense of chancy variation with important implications for the Darwinian tradition: he stressed that those unknown causes have no relation with environmental needs (Beatty 2008) and thus no impact in the directionality of evolutionary change. In other words, Darwin argued against the traditional view of there being a tendency to vary in certain directions determined by either individual ontogenies or by the species "norm". His position was that evolutionary tendencies are exclusively due to the natural selection of variants whose appearance had no relation with such tendencies.

As it is well known, Darwin engaged in several discussions with opponents and was much criticised by many of his contemporaries. But, perhaps more interestingly, he was also criticised by supporters for exaggerating the importance of natural selection. According to Beatty (2010, 2016), Darwin initiated a "subordination of chance variation to selection" that would prevail until the days of the Modern Synthesis, discarding any role for variation in evolutionary trends. Although Darwin certainly knew that which variants arise by chance do make a difference in the final result (as he stressed in his works on orchids, see Beatty 2006a), he considered that they did not play a role in what, according to his view, was the real product of evolution, namely adaptations. He illustrated this point through the analogy of an architect:

> [Evolution by natural selection] absolutely depends on what we in our ignorance call spontaneous or accidental variability. Let an architect be compelled to build an edifice with uncut stones, fallen from a precipice. The shape of each fragment may be called accidental. Yet the shape of each has been determined … by events and circumstances, all of which depend on natural laws; but there is no relation between these laws and the purpose for which each fragment is used by the builder. In the same manner the variations of each creature are determined by fixed and immutable laws; but these bear no relation to the living structure which is slowly built up through the power of selection. (Darwin 1887, Vol. 2, p. 236. Cited in Beatty 1984)

Similarly, influenced by his belief in blending inheritance (Beatty 2008)—which would prevent the spread of new variants if rare—, Darwin also stressed the importance of variations being small and abundant. According to his view—*natura non facit saltum*—, natural selection acts on the accumulation of small differences inside a population, rather than with the appearance of new, larger ones, constituting a gradual process:

> Natural selection can act only by the preservation and accumulation of infinitesimally small inherited modifications, each profitable to the preserved being; [and without any] great and sudden modification (Darwin 2009/1859, p. 93).

This way, Darwin's position relied on the availability of abundant variants in order for selection to bring directionality to evolutionary changes. His key contribution was the seed to many current notions of chance in evolution, especially the ideas of chance as not designed and chance as not aligned with the directionality of evolution—evolutionary chance—(cf. Millstein 2011). In turn, we can conclude that the possible for the Darwinian view of evolution refers to ecological opportunity: what possible adaptations can selection bring about given that there is always abundant 'raw material' for constructing them.

b) Variation in the Modern Synthesis of Evolution

Although both evolution and the mechanism of natural selection were relatively well accepted within the decades that followed Darwin's writings, both cohabitated for

quite some time with diverse ideas about the origin of variation and the mechanisms of inheritance, which led to an interesting variety of positions towards the end of the 19th and early 20th centuries. Moreover, the general acceptance of natural selection as a mechanism of evolutionary change implied a heterogeneity of opinions with respect to its relative importance in accounting for both speciation and adaptation; although most scholars accepted its existence, many of them considered its impact on evolution as minor as compared to other factors. In this panorama, the idea of chance variation was particularly influential for the neo-Darwinian position that led to the so-called Modern Synthesis of Evolution. In the first decades after Darwin, the chance variation view rivalized with schools such as the defenders of orthogenetic evolution—which maintained the traditional developmental view of variation in the new evolutionist picture—or the neo-Lamarckian movement—which stressed the importance of inherited acquired characters in adaptation.

The turn of the century brought the configuration of a theoretical space of heredity, marked by the distinction between soma and germ lines, the rediscovery of Mendel's laws of inheritance and the new notion of 'gene'. It is well known that these ideas provided a new framework for the saltationist tradition, and generally for those who stressed the importance of variation in evolution, that crystallized in the mutationist movement. The controversy between mutationists and neo-Darwinians has been analysed in detail by historians of science, and I lean in what follows on Peter Bowler's traditional account (2005), Arlin Stoltzfus and Kele Cable's revision (2014), and John Beatty's recent exposition (2016, 2019). Liderized by geneticists such as Hugo de Vries, William Bateson and Thomas Morgan, the Mendelian view of definite and particulate heredity directly opposed Darwin's conception of blending inheritance. This mutationist tradition confronted the Darwinian idea that selection always acts on gradual, abundant variation, pointing out instead that mutations, or undirected changes in the germ line, can initiate and direct the evolutionary process. In fact, one of the main points of mutationists was that new variants arisen by mutations have the potential to form new populations and start a process of evolutionary divergence.

Neo-Darwinians, on the other hand, influenced by the work of breeders, considered that individual variations were constantly preserved by blending inheritance and chance variation, enabling selection to initiate and direct evolution whenever necessary under ecological opportunity. Note that this idea contrasts with the classical, pre-Darwinian view that opposed variation to heredity: rather than being a deviation from the inherited material, variation was precisely what heredity preserved throughout the course of generations. The main idea underlying the neo-Darwinian school of thought was that populations are always provided with abundant, small variants reproduced by inheritance that can be the target of selection under changing conditions. This was the origin of what Mayr would later call "population thinking" (1959), namely the thought that only individual variations inside populations are real constituents of evolutionary change. In this new approach, evolution was always considered possible if the right selective pressures were present, variant characters being always part of a wider—in principle unlimited—range of possible variation (Bowler 2005). This view was specially patent in the work of biometricians of the early 20th century such as Francis Galton and Karl Pearson. The biometrical school applied a statistical approach to variation in populations, associating the distribution of trait variants to a 'normal' or 'error' distribution around a particular mean value susceptible to being shifted by selection (Figure 2.1).

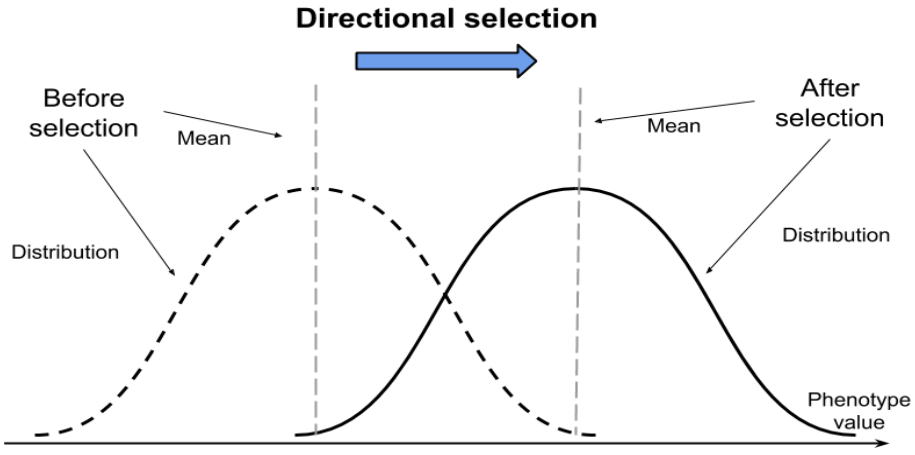

Figure 2.1. Two normal distributions of variants around different mean values for the same trait, each corresponding to a different time for the same population. The first one represents the distribution of the original population, while the second one represents the distribution after selection has acted to increase the mean of the trait.

So the main point of divergence between mutationists and neo-Darwinians was the relative importance of selection and variation in directing the course of evolution, as based on the distinct roles that inheritance played for each tradition—as a producer of divergence for mutationists or as a maintainer of small, individual and chancy variants for selectionists. The discrepancy mostly derived from neo-Darwinians' theory of blending inheritance being incompatible with the new, particulate view of heredity brought by Mendelism (Stoltzfus & Cable 2014). These confronted views were progressively reconciled by some scholars, who worked on the building of a mathematical frame for basing the gradual variation of quantitative traits on segregating Mendelian factors (e.g. Bateson 1909, Fisher 1918). A major achievement in this regard was the discovery of the Hardy-Weinberg principle, which relates allele frequencies from parent to offspring generations, and allowed the recognition that sexual reproduction did not blend variation strictly speaking, and thus "has no inherent tendency to destroy the genotypic variation present in the population" (Okasha 2016). On the contrary, genotypic frequencies remain constant in reproduction when no external factors are involved. The years that succeeded this conciliation up until the mid 20th century were witnesses of the progressive incorporation of the neo-Darwinian view of selection of chance variation into the fields of comparative biology, in what came to be known as the Modern Synthesis of Evolution (cf. Huxley 1942), intended to encompass all branches of natural history.[23]

Separating the notions of genotype and phenotype was an important component of this process, that based its explanatory agenda on the idea that natural selection acts on genes. The genotype refers to the hereditary material of organisms, a conjunction of Mendelian factors, or alleles, determining the formation of the traits of organisms. The phenotype, on the other hand, makes reference to the organism as resulting from the developmental process, whose characterization only affects evolution insofar as it determines the selective advantage of the genes that "code" for

[23] Although it has been claimed that the idea of the Modern Synthesis as a unit should be abandoned (Cain 2009), it seems clear that the research goals and explanatory strategies of the new statistical approach to genetics largely eclipsed other research agendas. Crucially from the point of view of this thesis, there seems to have been a common understanding of chance throughout the authors of the 'synthesis' (Plutynski et al. 2016).

its—phenotypic, physiological, behavioural—traits. The "breakdown" of the classic, developmental view of variation was culminated (Bowler 2005): variations were considered as random copying errors of the genotype, rather than additions to ontogeny; they had gradual phenotypic effects, they accumulated in reproduction, and they were not affected by the developmental process.

One of the highest achievements of the Modern Synthesis (hereafter M.S.) was that, variation like this conceived, natural selection was considered the *explanans* of any evolutionary change. The classical work of Roland Fisher, *Genetical Theory of Natural Selection* (1930), which inaugurated the field of population genetics, is paradigmatic of this view:

> The whole group of theories which ascribe to hypothetical physiological mechanisms, controlling the occurrence of mutations, a power of directing the course of evolution, must be set aside once the blending theory of inheritance is abandoned. The sole surviving theory is that of natural selection and it would appear impossible to avoid the conclusion that if any evolutionary phenomenon appears to be inexplicable on this theory it must be accepted at present merely as one of the facts which in the present state of knowledge seems inexplicable. (Fisher 1930. Quoted in Wright's 1930 review)

In other words, once the well-known gradual changes that breeders produced in selecting variants could be accounted for in terms of non-blending, segregating alleles, no mechanism other than selection was considered necessary for explaining evolutionary changes. The neo-Darwinian position of relegating chance variation to selection encountered in the newborn field of genetics a powerful ally. This conceptual transition was possible through the M.S. idea of the "gene pool" (Stoltzfus 2006). For the M.S. view, populations are pools of genetic variation that can provide any combination of variants by recombination, constantly fueled by random mutations. In this frame, evolution could be conceptualized as a change in the relative frequency of genes or alleles in the genetic pool of a population, a frequency that is determined by the fitness values of genes. The following quote from Sober illustrates the importance of the gene pool:

> For species like *Homo sapiens* and *Drosophila melanogaster*, the number of loci has been estimated to be about 10,000 or more. What this means is that the number of genotypes that can be generated by recombination is greater than the number of atoms in the visible universe ... For species with this number of loci, even a single male and a single female can themselves reproduce a significant fraction of the variation found in a population from which they are drawn (Sober 1980, pp. 376-7).

Perhaps the most striking component of the new synthesis was that it intended to assimilate all the patterns of unity and diversity that characterized the comparative branches of natural history into the same frame that explains the gradual changes that were known to breeders. For example, paleontologists of the early 20th century were very much seduced by the idea of orthogenetic evolution, for they interpreted the morphological patterns of the fossil record as ontogenetic tendencies to change (Ulett 2014). However, the M.S. incorporated macroevolutionary patterns into its *explanda*, insofar as it considered that all speciation processes could be explained in terms of changes in gene frequencies. As Richard Lewontin (1965) put it, through the M.S. evolution was redefined for most purposes as "the conversion of intra-population variation into inter-population variation *in space and time*" (cited in Stoltzfus 2006, stress added). Moreover, as we shall see in section 4 of this chapter, notions that were familiar to anatomists, morphologists, embryologists, and generally to the comparative naturalists, such as "body plan" or character "type" were relegated to mere consequences of common ancestry, with no explanatory relevance in the evolutionary process (Amundson 2005). In sum, these achievements famously left out of their umbrella many of the traditional fields of natural history, while vindicated explanatory exclusiveness for all evolutionary phenomena. One of the most important of such neglects, and surely the crucial one from the point of view of this thesis, is the so-called blackboxing of development. I will get back to this point later in this chapter (section 4).

The M.S. brought an immense and complex field of evolutionary biology headed by the mathematical models of gene frequency changes of population genetics, and—based on the former—by the models of phenotypic change of quantitative genetics. The implementation of this apparatus to natural populations

required, nevertheless, that selection not only fed from, but cohabited with an old friend: chance. Sewall Wright, one of the founders of the M.S., is well known for having vindicated the importance of genetic drift in evolution. Wright had observed that in small, isolated populations variations were likely to be spread by chance, and not only by natural selection, which led him to postulate drift as an important, size-dependent source of evolutionary change (Beatty 1984). Wright identified drift with "the effects of random sampling in a breeding population of limited size", whose composition is expected to change "merely by chance" (Wright 1931, p. 205). With this introduction, the explanatory corpus of the M.S. models of evolutionary genetics turned explicitly probabilistic in nature: fitter variants *tend* to reproduce more *on average*, and this will lead to a change in the composition *of large enough populations in the long run*. Although Darwinian natural selection was never intended to be deterministic, the introduction of genetic drift enabled the construction of the whole field with the mathematical tools of probability theory (see Hansen 2017). Moreover, it established a twofold role for chance in the process of evolution: in providing the 'raw material' of natural selection—Darwin's chance variation—and in contributing to the fixation or disappearance of variants—the new notion of genetic drift. As Mayr put it,

> Let us remember that evolutionary change is a two-factor process. One stage consists in the generation of genetic variation. It is on this level that chance reigns supreme. The second stage is concerned in the choosing of genotypes that will produce the next generation. On this level natural selection reigns supreme and chance plays a far less important (although not negligible) role. (Mayr, 1963, p. 214)

This way, the process of evolution was established as a two-step process, only one of which is responsible for directionality, either adaptive or chancy. The first step, where "chance reigns supreme", concerns mutations as "the ultimate source of variation" in the M.S. (Plutynski et al. 2016, p. 98).

1.2. The fate of variation: probabilistic models of evolution

The mathematical models derived in the M.S. are the basic corpus of modern evolutionary genetics, notably the models of theoretical population genetics and quantitative genetics, including the empirical research of ecological genetics and the use of so-called optimization models, as well as the fruitful application of all of them to dispare levels of organization, such as molecular evolution or speciation processes. Although their scope of application has enlarged throughout the years, their basic structure remains stable. On the one hand, the population genetics approach studies selection on specific alleles or gene loci, ignoring the phenotype and thus focussing on changes in allele frequencies in populations. They describe and predict these changes on the basis of the fitness values associated with particular genes. On the other hand, quantitative genetics typically use infinitesimal models derived from the mathematical treatment of quantitative traits, thus focusing on one particular phenotypic character and idealizing its genetic basis as composed of infinite many loci (Csilléry et al. 2018). They therefore describe and predict changes in the mean value of a particular trait in a population on the basis of its fitness value and its heritability. Thus evolutionary genetics either maps directly genes to fitness values ignoring phenotypes, or maps the quantitative value of a phenotype to fitness ignoring the genotype and assuming that certain changes in gene frequencies produce phenotypic changes in a particular manner. The evolutionary factors that these models incorporate are natural selection, migration, mutation and genetic drift. Let us now see how this statistical treatment together with the Darwinian view of chance variation enabled an influx in the philosophical discussions of evolution from ideas of the physical sciences.

In this characterization, the roles that chance played were many. Philosopher Anya Plutynski and her colleagues (2016) have identified at least five different roles that chance had in the M.S., in a similar fashion to Gayon's (2005) and Millstein's (2011) general classification of chance in contemporary evolution (see section 2.3 of Chapter 1). In addition to the common notions of chance as indeterminism and as

contingency, they underline how M.S. theoreticians used the notion of chance as random—in the sense introduced in the previous chapter—as well as chance as a "proxy for probability"—as in 'the chance of this coin landing heads up is $\frac{1}{2}$'—(Plutynski et al. 2016, p. 80), as a reflection of the probabilistic nature of their explanations and models. Finally, and in accordance with the Darwinian characterization of variation and drift, M.S. advocates adopted a notion of chance as opposing the direction of selection, aligned with the new general anti-teleological picture of nature: those factors not included in natural selection—notably drift and mutations—are 'chancy' in the sense of not bringing directionality to the process of evolution.

It is in this context that the newborn field of philosophy of biology was conferred with a reasoning mode imported from the physical sciences, influenced as well by the initial restriction to physics in philosophical discussions of science as separated from natural philosophy (see Mayr 1963 and Sober 1984 for some classical criticism of this fact). In this regard, the idea of bringing evolution near Newtonian mechanics enabled a penetration of philosophical thought inspired in the world of physics into evolutionary thinking. It is not surprising then to find that much of the philosophical discussion on evolution—and specifically on selection and drift—has incorporated analogies with the physical world for clarifying purposes. Here I will first present two important aspects of population dynamics that have been discussed through the prism of an analogy with physical systems: the probabilities of evolutionary models understood as representing sampling processes (section a) and their causal aspect as analogous to a theory of forces (section b). Then, I will address how this classical view, similarly to the classical framework of physics, does not entail any commitment with indeterminism (section c).

a) Evolution as a sampling process

Evolution has often been compared to a sampling process where individuals in a population get sampled in order to take part in the next generation (Wright 1931, Roughgarden 1979, Beatty 1984, Millstein 2003, 2006). Typically, individuals survive and reproduce *differentially*, meaning that they will do so with distinct degrees of

success. Under certain assumptions, notably that there is heritable phenotypic variation that covaries with the number of offspring (see Godfrey-Smith 2007), differential reproduction will make the composition of the next generation differ from the previous one in certain aspects. Given a population, a sampling process can be regarded as a determination of "which organisms of one generation will be parents of the next, and how many offspring each parent will have" (Beatty 1984, p. 188). The analogy of an urn from which balls are picked and introduced into a second urn is intended to capture this sampling process (e.g. Beatty 1984, Millstein 2003, Nuño de la Rosa & Villegas 2019). In doing so, it also provides some insights on the probabilistic nature of evolution. In this analogy, an urn with balls of different colors represents the individuals (or alleles) in a population at a given time (see Figure 2.2). Each color can represent a different trait or allele type, so the proportion of, say, red balls is the proportion of individuals with trait *A* in the population. A sampling process takes place in this population pool simulating the selection of those alleles or individual types that will be part of the next generation. It is important to note that, even if it is illuminating to think of evolution as a sampling process in this way, the hands of the urn analogy never represent selecting *agents*. The sampling process is a result of birth and death rates and, even if they highly depend on environmental conditions, these conditions only 'select' in a metaphorical sense.

Natural selection results from the higher tendency to survival and reproduction of the individuals in a population that are better adapted to its environment, that is, the individuals that are *fitter* than other individuals. Thus, it is the sampling of those parents (and their offspring) that are better adapted. In the urn analogy, a hand picking balls on the basis of their color corresponds to natural selection: if the trait *A* (being red) is fitter than traits *B* (being green) and *C* (being yellow), then the hand will tend to sample more red balls than balls of the other two colors. The hand will deposit the selected balls into a new urn representing the gene pool of the next generation. Thus, in our example, red balls will be more frequent in the second urn than in the first one, because balls were selected by virtue of their colors.

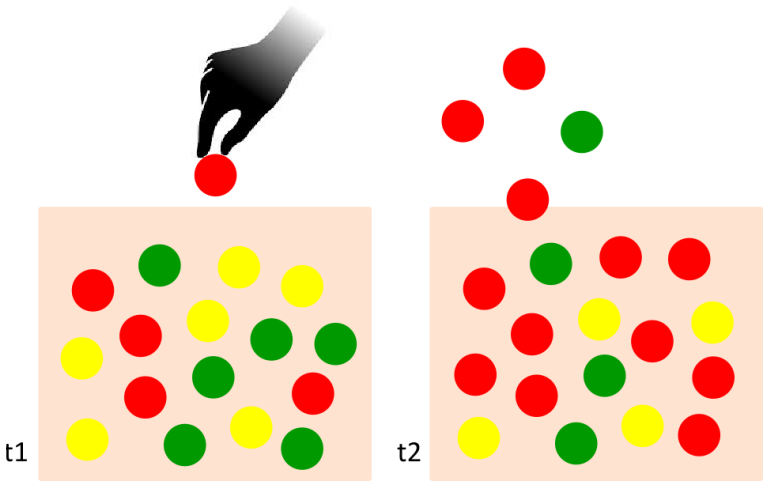

Figure 2.2. Two urns representing the gene pool of a population at t_1 (before selection) and t_2 (after selection). In the first urn, red, green and yellow balls are equally represented, while in the second urn, red balls are more frequent due to selection.

But changes in the composition of populations are not only determined by natural selection. Populations undergo genetic drift, namely changes that result from the chancy aspect of differential reproduction. Darwin already noted that variations can become fixed in a population even if they do not make organisms better adapted to their environment (2009/1859, e.g. p. 63). Some variations may be neutral with respect to fitness (for example, having or not a spot in one knee), and thus each variant will contribute to the next generation with equal probability. Others may be deleterious in such a small proportion that they may never be eliminated by natural selection. Recall that Wright had introduced drift as the effect of random sampling in small populations, where changes in composition can occur "merely by chance" (Wright 1931, p. 205). This sense of randomness entails that the sampling from one generation to the next one is produced entirely at random, namely with each member of the population having the same probability of being sampled, regardless of their physical properties (Beatty 1984). This *random*—or indiscriminate—sampling contrasts with the discriminate sampling of natural selection. Modern population dynamics models also consider drift as the effect of random sampling in this sense, which will be greater the smaller the population is (reviewed in Millstein 2017). In this sense, size limitation is key to the concept of drift: if populations were infinite in

size—an idealization present in some classical models of evolution—there would not be chancy effects of sampling.

Therefore, in our analogy, changes in urn composition do not only depend on discriminating hands but also on *indiscriminate sampling* (Millstein 2002). Let us illustrate this with a modification of the same example.[24] Suppose that, instead of having red (fitter), green and yellow balls, the first urn only had green and yellow ones, which have no differences in terms of fitness. That is, the hand is *colorblind* in this case in the sense that traits *B* (being green) and *C* (being yellow) are not relevant for the chances of being selected. In this case, sampling indiscriminately from an urn composed of 20 green balls and 20 yellow balls could result in a change in the relative frequency of each color—e.g. 25 green balls and 15 yellow ones—*merely by chance* (Figure 2.3). Notice that this result will be more likely the smaller the sample is. If only three balls were sampled, the ratio between the traits would change dramatically regardless of which balls are picked—at most, we would go from a 1:1 ratio to a 1:2 one. However, notice as well that the same situation can hold even if the hand is discriminating colors, that is, even if we consider natural selection only, provided that its sampling from the urn is not deterministic but probabilistic instead. If we consider that natural selection samples fitter parents differentially with a given probability, then there is always a chance that unlikely results will obtain from time to time, without any need for introducing non-selective—colorblind—factors. This has been acknowledged as the problem of discerning between drift and the unlikely results of selection (Beatty 1984), and there is much literature on whether this implies that drift is empirically indiscernible from selection (Millstein 2008). Interestingly, this can be seen as a problematization of the notion of chance as a random event (Gayon 2005, Plutynski et al. 2016), insofar as the event can only be considered random with respect to a particular defined expectation (see section 2.3 of the previous chapter). As we shall see later in this chapter (section 2), this concern will be used by statisticalists for arguing against the causal nature of evolutionary models.

[24] What follows is inspired by Millstein's "indiscriminate" urn analogy (2002, p. 36), to which I add the probabilistic discriminating hand as a contrast with the "unlikely results" of natural selection (cf. Beatty 1984).

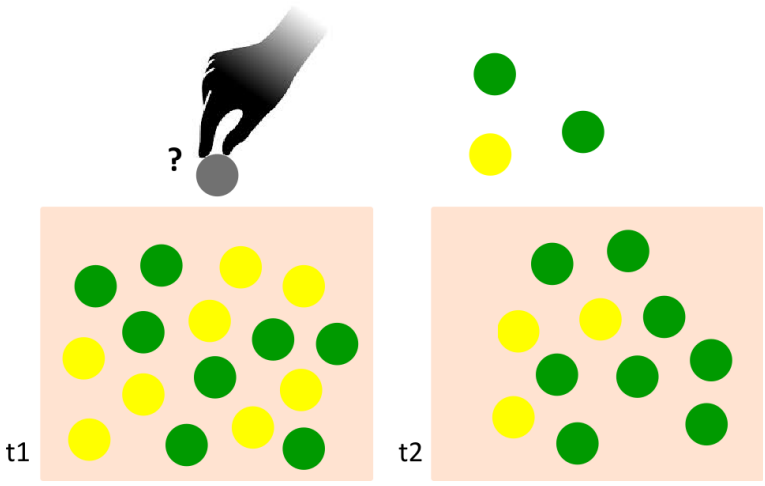

Figure 2.3. Two urns representing the gene pool of a population at t_1 and t_2. In the first urn, green and yellow balls are equally represented, while in the second urn, green balls are more frequent. This time, the hand picking up the balls is colorblind, thus the change in urn composition is due to drift.

Other components typically included as determinants of evolution are mutations, genome recombinations and migrations. Mutations are stochastic changes in the genetic material inherited from parents to offspring.[25] In order to consider them in our analogy it would be necessary to allow changes in the sampled balls once the process is iterated. Similarly, genome recombinations, which refer to the interchange of genetic material between DNA strands in the cellular processes involved in reproduction, could only be incorporated through some mechanism affecting the characteristics of sampled balls. Finally, migrations refer to the exchange of characters and genetic material between different populations of the same species. Although migrations can be an important cause of evolutionary change—and although it points to the interesting topic of the relevant reference population—, their role does not affect the argument of this thesis, and thus migrations will not be addressed here for the sake of simplicity. For any of the other two factors to be considered, the balls in the urn need to change themselves in some sense in addition to being part of the sample space of a sampling process.

Even if these factors can be relatively easily incorporated into this influential urn analogy, to my knowledge none of them has been so. The analogy has merely

[25] The sense of this stochasticity will be addressed in section 3 of this chapter.

served for analyzing the nature of populational and external factors in determining the course of evolution, leaving aside the aspects relative to chance variation. This externalist approach aligns with the M.S. population thinking, exalting extant variants as responsible for possible evolutionary changes. Consequently, it has served, on the one hand, for discussing chance at the level of the spread and fixation of variants, particularly with regards to what Millstein (2011) labeled chance as sampling and Plutynski and colleagues (2016) called proxy for probability. On the other hand, it has served as well for the problematization of chance as random event (Gayon 2005, Plutynski et al. 2016)—in the difficult task of separating selection from drift—mentioned above.

b) The dynamical picture of evolution

In *The Nature of Selection* (1984), Elliott Sober draws a well-known analogy between evolutionary drives and physical forces. Sober argues that evolutionary theory can be understood as a theory of forces just like Newtonian mechanics, a position that has come to be known as the dynamical view of evolution (Hitchcock & Velasco 2014). Similarly to how forces such as gravity, electro-magnetism or friction are genuine causes of motion in physical systems, the factors acting upon biological populations, changing their composition—natural selection, migration, mutations and genetic drift—, are forces driving them towards a particular evolutionary direction. Under the dynamical picture, these factors are described as the "source laws" of evolution, namely those that "describe the physical circumstances that generate particular kinds of forces" (Sober 1984, p. 50). These forces would be causally responsible for evolutionary changes in biological systems, understood as changes in the frequencies of alleles in a population. Sober writes:

> *All* possible causes of evolution may be characterized in terms of their 'biasing effects.' Selection may transform gene frequencies, but so may mutation and migration. And just as each possible evolutionary force may be described in terms of its impact on gene frequencies, so it is possible for a cause of evolution to be present without producing changes in gene frequencies. ... All this is to locate evolutionary theory in familiar territory: it is a theory of forces. (Sober 1984, p. 31)

According to Sober, source laws of evolution, or their origin explanations (Godfrey-Smith 2009), are to be found mostly in theoretical ecology. The principle of natural selection, as described by Darwin, represents one of such forces, for it "names a wide set of processes whereby valuable variants are generated, maintained and refined in a population of organisms" (Lewens 2018, p. 5). Notice that the source laws corresponding to drift and migrations are also to be found in theoretical ecology. However, the source laws corresponding to mutations must be encountered in the field of genetics. Sober does not develop this in much detail, but he does characterize a notion of chance in mutations that is in line with the anti-teleological ideas of the M.S., particularly with chance as opposing selection (Plutynski et al. 2016) or 'evolutionary chance' (Millstein 2011): "mutations do not occur *because* they would be beneficial" (Sober 1984, p. 105). The only representation of mutations in this framework are descriptions of mutation rates, which are empirically based but lack theoretical treatment. In this context, mutation rates have been considered relevant in particular as a source of the maintenance of polymorphisms—i.e. of more than one variant for the same trait—even when selection tends to reduce genetic variation (Okasha 2016). In line with the general tendency to population thinking, source laws were considered important insofar as they provided grounds for the ecological aspects of evolutionary change, as based on differences in fitness values. As we shall see in detail in section 3 of this chapter, this situation can be seen as a lack of source laws not only for mutations but for variation more generally in the context of the M.S. framework (Stoltzfus 2006).

Additionally to source laws, a theory of forces is one where laws describe a system when forces are applied to it—consequence laws—, as well as its behavior in the absence of forces—so-called "zero force laws". These laws would provide distribution explanations (Godfrey-Smith 2009), in contrast to the origin explanations provided by source laws. What exactly means for a population not to experience the action of forces depends on the particular view of the forces analogy in consideration. Most philosophers tend to agree that this *no-forces* state is an equilibrium—described by the Hardy-Weinberg principle—where the properties of

the population do not change along evolutionary time.[26] In this view, it is considered that the lack (or cancelling out) of selection, drift, mutation and migration will maintain the population without changes throughout generations.

With regards to *consequence laws*, these have been placed in the theorems of population genetics. They describe the dynamics of populations once ecological and heritable factors have been introduced. That is, once the frequency, mutation rates, fitness values and heritability of traits are posed, the models of population genetics predict the evolutionary course of populations under certain idealizations. It is classically considered that Roland Fisher's Fundamental Theorem of Natural Selection is the primordial consequence law of evolution:

> The rate of increase in fitness of any [type of] organism at any time is equal to its genetic variance in fitness at that time. (Fisher 1930, p. 35)

Although some authors have vindicated other frames as consequence laws,[27] they all similarly predict a change in the frequency of alleles in a population that is proportional to the variance of those alleles in fitness, as derived from this fundamental theorem. For the models of phenotypic change of quantitative genetics, the consequence laws are typically formulated in the Breeder's equation (Lush 1937, Falconer 1960) and its multivariate version (Lande 1979, Lande & Arnold 1983). The relationship between source and consequence laws is a pillar of the influential debate between statisticalists and causalists to be reviewed in the next section, for it determines how causal knowledge—source laws—influences the probabilistic models of evolutionary change—consequence laws.

A number of critiques have been raised to the dynamical view of evolution, ranging from the disanalogy between evolutionary and Newtonian forces and their

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[26] Some philosophers consider that a biological system without the action of forces will tend to diversity, thus assuming that genetic drift, in lacking a specific direction, is not really a force but the natural state of a system when forces do not act upon it (e.g. Brandon 2006). However, there is a certain consensus among the advocates of the force analogy that the zero force law of evolutionary biology is described by the Hardy-Weinberg equation, which describes a population in equilibrium that does not undergo changes throughout generations, analogously to how the law of inertia describes a physical system without the intervention of forces.

[27] Such as Li's theorem (Matthen & Ariew 2002), Richard Lewontin's Conditions for Evolution by Natural Selection (Lewontin 1970, Godfrey-Smith 2007) or the Price Equation (Price 1970, Luque 2017).

respective roles in models and theory, to the rejection of evolutionary factors as causes whatsoever. On the one hand, evolutionary forces have been charged on the basis of their non-decomposability into vectorial factors, preventing them from being treated linearly and thus lacking the advantages that an analysis in terms of forces presumably has (Matthen & Ariew 2002).[28] On the other hand, and closely related to the former, this picture has been challenged on the basis that the models of population dynamics do not provide causal explanations—as theories of forces do—but statistical explanations instead. The so-called statisticalist view of evolutionary theory posits that our explanations of populational dynamics do not make reference to the real causes of the evolutionary process (Matthen & Ariew 2002, Walsh et al. 2002). A central component of this critique is the idea that the process of Darwinian evolution is not one where different factors can be mathematically traced and measured. As the statisticalist Tim Lewens argues,

> Darwin did not approach evolution in a way that demanded a quantified decomposition of different evolutionary 'forces,' hence he was not driven to define evolutionary processes in a way that would permit sharp differentiation between selection, drift, mutation, and migration. His strong conceptual linkage between natural selection and the explanation of adaptation meant that he sometimes omitted to distinguish between what we would now think of as mutation, on the one hand, and selection, on the other. (Lewens 2018, p. 6)

However, at this point the discussion moves from the correctness of an analogy with Newtonian forces to a consideration of the causal nature of evolutionary factors more generally. Christopher Stephens (2004) points in his influential defense of the forces analogy that "it makes perfect sense to think of selection, mutation, migration, and drift as causes since they are factors that *make a difference*" (p. 568). But such a difference-maker role may not necessitate an interpretation of evolutionary factors in terms of forces. As it has been pointed out by several authors (Lewens 2018, Otsuka 2016), the correctness of the analogy with

[28] This argument relies on the fact that there is no homogeneous metric that could serve for comparing evolutionary forces under the same frame, partially due to their interdependent nature. However, see Hitchcock and Velasco (2014) for a reply arguing that Newtonian forces indeed face the same problem.

Newtonian forces and the causal nature of evolutionary theory are two distinct aspects that can be addressed independently. In fact, even if the statisticalist position emerged as a critique to the Newtonian forces analogy, it initiated an autonomous controversy on the role of causal explanations in evolution known as the causalists-statisticalists debate, to which I shall refer to later in this chapter (section 2.1).

c) Evolutionary probabilities and indeterminism

A pervasive topic in the origins of the contemporary philosophical discussion on the role of chance in evolution was whether the underlying ontology of the evolutionary process was deterministic or not—i.e. chance as indeterminism (Millstein 2011, Plutynski et al. 2016). Getting back to the urn analogy, the discussion concerned whether the probabilistic nature of sampling balls implied that the picking balls process was itself indeterministic. The probabilistic models of population dynamics—together with the forces interpretation of evolution—were perceived as entailing indeterminism insofar as population factors, when combined, only allow for probabilistic predictions: how likely it is to be sampled for the next generation, how probable it is that a particular trait will spread or disappear in the next generations, etc. Interpreting population dynamics as resulting from causal probabilistic processes at the population level—that is, insofar as they refer to sampling from a population—was thus often perceived as entailing an indeterministic ontology. Moreover, a drifting *force* (or a blind hand), in its lack of directionality, was seen as a stochastic mode of causation acting at the population level.

In a classical paper, Robert Brandon and Scott Carson (1996) argued that, if one is a realist about science, then one must admit that evolutionary theory is indeterministic. On the one hand, they believed that the indeterminism supposedly embedded in quantum mechanical phenomena could "percolate up" from spontaneous mutations in the genome and have population effects.[29] Thus they

[29] This supposition of indeterminism is not really entailed by every interpretation of quantum mechanics. Brandon and Carlson's critique is based on Bell's inequalities, which show that no *local* hidden variables (i.e. undiscovered variables that would explain the phenomena by local causal chains) could save the determinism of the theory. However, non local deterministic hidden

defended that fundamentally stochastic effects take place in the genetic material, affecting in turn stochasticity at the level of population dynamics. On the other hand, they defended that, when modelling evolution, there is a fundamental indeterminacy assumption at the level of births and deaths in a population. In other words, they consider that the probabilistic nature of evolutionary models is based on the idea that the models represent a phenomenon that is truly indeterministic. More importantly, their view is that any theory including deterministic "hidden variables" about the lives and deaths of individuals would serve no theoretical purpose at all at the population level. Their conclusion is that the only way to preserve the determinism of evolution is to be an instrumentalist about evolutionary theory, a position that they do not favour. To this position, Leslie Graves, Barbara Horan and Alex Rosenberg (1999) responded that, on the contrary, "hidden variables" are posited in theories in order to be discovered, and therefore that realism and determinism are not opposed. In the case of evolutionary theory, their view is that the indeterministic propensities of populations supervene on the deterministic properties of individuals and that, consequently, probabilism about evolution is only epistemically motivated, and it would ideally be replaced by deterministic finer-grained models.

As we saw in the previous chapter, however, probabilistic causes at higher levels are not incompatible with determinism at lower levels of organization, nor do they demand any type of anti-realism. It is possible, as we saw in the case of the range conception of probability (section 2.2) and the causal propensity (section 3.2) views, to talk about the probabilistic causes of populations as real features of the world without making any commitment to indeterminism. This is precisely what Marcel Weber (2001) pointed out in his response to this discussion, and what other authors, notably Millstein (2003), have kept vindicating afterwards. Weber (2001) noted that, instead of being a dispute over indeterminism, the interesting discerning points of the determinism/indeterminism positions were about the realism of evolutionary theory. In other words, whether or not the probabilistic theory of evolution is

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variables are at the core of some interpretations of quantum phenomena, notably Bohmian mechanics.

instrumental in the sense of not representing any real feature of the world despite its pragmatic value, or whether it makes reference to real properties of the world. One can thus wonder if the theory is dispensable had we more fine-grained descriptions of evolutionary processes:

> A theory may be dispensable in the sense that an omniscient being would be able to understand the phenomena in question at a deeper level, but it is still possible that this theory correctly represents some aspects of reality. [...] The fact that a theory falls short of giving us a *complete* account of some complex causal processes does not imply that this theory has no representational content whatsoever (Weber 2001, p. S217)

From this point of view, whether or not the sampling processes in populations are causes of evolution becomes detached from the idea that natural selection and drift imply any fundamental indeterminism. Thus the notions of chance involved in evolutionary thinking typically don't concern fundamental indeterminism but the randomness of certain outcomes with respect to the possibilities a particular cause allows, as reviewed in the previous chapter. That is, they concern probabilistic patterns as derived from higher-level causes that manifest in an array of possibilities rather than in a determinate outcome, but which entail nothing with regards to the ontology—deterministic or not—underlying the manifestation process.

With respect to mutations, on the other hand, it is unclear whether or not indeterminism could play any role in principle. What seems clear, however, is that the notions of chance as ignorance of causes, as not designed, as random and as opposing selection (Millstein 2011, Plutynski et al. 2016) typically apply to variation in general and mutations in particular without any commitments to an indeterministic ontology. As we shall see in the next chapter, this detachment of chance from indeterminism is important in turn for understanding the probabilistic nature of the origin of variation. In the remainder of this chapter, I will review the discussions on the probabilistic nature of population dynamics and variation without considering any assumptions on the deterministic or indeterministic ontology

underlying it, therefore only referring to the representational nature of probabilistic causes.

2. Chance as sampling: ecological causes and propensities

Now that the Darwinian understanding of variation and the philosophical concerns about the M.S. probabilistic models have been introduced, let us make sense of a causal view of population dynamics from the point of view of causal propensities as introduced in the previous chapter. For doing so, I will now address chance as sampling (Millstein 2011, see also Plutynski et al. 2016), a notion derived from the population thinking endorsed by the M.S., with the conceptual tools developed in this thesis. Chance as sampling includes the probabilistic processes of natural selection and genetic drift inasmuch as they constitute the differential reproduction of variants. It therefore represents the main probabilistic aspect specifically present in the models of evolutionary genetics.

In this section, I first review the debate that emerged in the last decades between statisticalists, who claim that evolutionary models are only statistical tools and do not consider the causes of evolution, and causalists, who believe that, by contrast, evolutionary models provide causal explanations of evolution (section 2.1). In that section I will align with causalist advocates, and I will argue that the statisticalist position is not tenable for several reasons, particularly for endorsing a frequency view of probability, for leaning on a non-causal understanding of explanation, and for privileging causation at the level of individuals. In section 2.2 I review the main consensus around the propensity interpretation of fitness, namely that it refers to a disposition of individuals, and relate that to the probabilistic models of evolutionary genetics from a causal propensity view. Finally, in section 2.3 I argue that this causalist picture enables recognizing population level propensities, the process of drift, and types of individuals or traits as causally relevant for the process of evolution. All of these considerations will be significant for further discussions with regards to the probabilistic character of variation in the remainder of this thesis.

2.1. Statisticalists and causalists

Departing from the recognition that evolution is, at least in epistemic terms, a probabilistic process, the so-called 'causalists/statisticalists' debate discusses whether the ecological components of evolutionary models make reference to the causes of evolutionary changes. Causalists defend that natural selection—as understood in those models—is a cause of such changes, whereas statisticalists argue that selection is better understood as an explanatory statistical aggregate of causes that act at a more fundamental level, namely the level of particular individuals in their ecological interactions with the environment. I believe that it is useful to regard this controversy as an instantiation of the contraposition between frequentists and propensionists (see section 1 of previous chapter) or, more generally, advocates of causal probability as reviewed in Chapter 1 (section 2). In exposing this debate, thus, I will endorse a causalist view and criticize the statisticalist position further on the basis of its consideration of causation and explanation.

The statistical interpretation of evolutionary dynamics was inaugurated by two seminal articles, one by Mohan Matthen and André Ariew (2002), and another by Denis Walsh, Tim Lewens and Ariew (2002).[30] In these two papers, statistical advocates argue that the models of population dynamics do not represent the causal processes that Darwin described in his works, since the notions involved in dynamical explanations are way too abstract to qualify for real causal factors. For instance, when predicting that the frequency of a certain trait will increase in the next generation, a population dynamics model will consider the fitness mean and variance of that trait in the population, both statistical measures that, according to these authors, do not demand any causal knowledge about the composition and dynamics of the population under consideration. In their view, these models represent probabilistic trends that emerge at the population level, where no real causes are acting, and merely describe the consequences of a statistical property, namely variation in fitness. Similarly to the frequency interpretation of probability,

[30] Early precedents can be found in Rosenberg (1988) and Horan (1994).

statisticalists see the probabilities of evolutionary models as derived from the properties of collectives, rather than from their causal or generating conditions. As a result, they suggest that a better analogy than the theory of forces for the role of population dynamics models is that of the statistical models of thermodynamics, an analogy that Fisher (1930) had already pointed to in his inaugurating work of the field of population genetics:

> It will be noted that the fundamental theorem [of natural selection] bears some remarkable resemblances to the second law of thermodynamics. Both are properties of populations, or aggregates, true irrespective of the nature of the units which compose them (Fisher 1930, p. 36)

Thus the statisticalist view is actually a position with regards to the models of population genetics as predictors and explanatory of evolutionary changes: they do not refer to the causes of such changes. Matthen and Ariew (2002) argue that, in fact, when evolutionary models make use of fitness values, they are not referring to fitness in the real Darwinian sense. Instead, they are referring to an abstract property of *types* of individuals—what they call "predictive fitness"—, while Darwin alluded to a causally efficacious property of *particular individuals*. They call this latter, ecological notion "vernacular fitness". The way in which these two different notions of fitness relate to each other, they argue, involves considering non-selective and non-causal populational factors, implying that predictive fitness is not strictly speaking derived from vernacular fitness. This situation hinders any derivation of the statistical 'consequence laws' of evolutionary models from the 'source laws' of theoretical ecology (cf. Sober 1984, see section 1.2.2 above). Matthen and Ariew consider that evolutionary models actually state "general truths about growing ratios" in aggregates, regardless of the complexity of causal processes underlying them (p. 74), that is, regardless of the ecological 'source laws' or factors causing changes. They conclude by distinguishing a "fundamental" type of causation acting at the level of ecological interactions and a "probabilistic" or stochastic one acting at the level of populations. Similarly, Walsh, Lewens and Ariew (2002) argue that, in evolutionary models, both selection and drift are "statistical properties of an assemblage of 'trial' events: births, deaths and reproduction", while "[t]he only genuine forces going on in

evolution are those taking place at the level of individuals (or lower)" (p. 453). Their view is that describing the properties of aggregates of trials—i.e. of populations—does not demand a causal understanding of each of the individual trials.

To my view, this statisticalist approach is in line with the frequency interpretation of probability presented in the previous chapter, according to which probabilities are assigned through the establishment of a reference class of trials with a defined frequency of outcome events. The statisticalist position resembles the frequentist tradition insofar as it considers the probability of fixation of an allele in a population as implied by its frequency and relative fitness value, namely as derived from the statistical properties of the aggregate they belong to, without referring to the causes underlying such fixation. Statisticalists thus define a particular ensemble of individuals, a population, similarly to how frequentists would define a reference class of trials, and define probabilities out of their statistical properties. As a consequence, statisticalists discard the view of selection and genetic drift as distinct processes or forces acting upon populations. Rather, they consider that the only way to make sense out of drift is as sampling statistical error (Walsh et al. 2002), that is to say, as a deviation from the mathematical expectations in the models applied to the process—similarly to how finite empirical frequencies may deviate from hypothetical frequencies (see section 1.1 of the previous chapter). In this view, what is considered drift and what is considered natural selection will depend on the statistical model applied in each particular case, and not on any distinct type of causal process—such as discriminate and indiscriminate sampling (see previous section)—being modeled.

In this situation, the statisticalist tradition inherits the difficulties associated with a frequentist account of probability (see section 1.1 of previous chapter). The most streaking of them is the reference class problem, especially affecting the distinction between selection and drift mentioned above. The failure to make this distinction is more important than it may seem at first sight, for it is not merely a problem about not knowing which factors are present in an evolutionary transformation—i.e. selection or drift. Importantly, it is also a problem about defining what is the fitness of a particular trait of allele. Recall that we saw in the previous chapter that defining the probability of an event through its frequency could be

especially problematic when the reference class is small. Thus imagine that we want to establish the fitness value of an allele in a population—i.e. its probability of fixation—after an environmental change affecting such value. How many generations do we need to measure—thus how many trials of the random experiment do we need to consider—in order to have a meaningful change in the relative frequency of the allele? And how many individuals must the population contain for one measurement to be meaningful? These are questions that, as we saw, demand the introduction of the notion of mathematical limit through hypothetical frequentism (Reichenbach 1938). That is, they demand that we consider an idealized infinite sequence of trials for establishing any probability value. But in order to construct such an idealized sequence, we require that the properties of our reference class, including its long-term tendencies, are well defined in the first place (Hájeck 1997, 2009). For, how do we postulate a mathematical limit without the introduction of non-statistical properties when the number of trials—or the sample size—is small? In the case of fitness above, doing so could demand considering which are the *generating conditions* responsible for fitness values, namely the causes underlying them. Only this way could the pertinent extrapolations enabling the construction of a mathematical model be made in the absence of an extensive enough record of generational trials.

In addition, statisticalists cannot provide a satisfactory relation between probability and explanation (cf. Hájeck 1997). On the one hand, the frequentism embedded in their view prevents them from giving any explanation of frequencies in terms of probability. That is, from the statisticalist position, probabilities *are* frequencies, thus a brute fact without an explanatory connection with theory. On the other hand, they implicitly endorse a notion of explanation of population changes as statistical relevance (Salmon 1971). This is the case because they consider that probabilistic models provide explanations of evolutionary phenomena without making reference to their real causes, thus arguing that, for instance, the notion of natural selection as a statistical trend can explain changes in the composition of populations without causing them. This is particularly clear in Matthen and Ariew's (2002) idea that population genetics models provide a "stochastic" or probabilistic

type of *causation*, which they consider non-fundamental. In their view, a probabilistic cause is not a cause "in the sense appropriate to fundamental processes" (Matthen & Ariew 2002, p. 81), which would only take place at the level of individual interactions. Consequently, probabilistic causes would only explain statistical trends of populations, but not the real causal processes underlying them. However, recall that, as we saw in the previous chapter, there is typically no distinction between statistical relevance and causal relevance in the probabilistic causation tradition (see Woodward 2014 for a review). Indeed, as reviewed in the "minimals of explanation" developed in the previous chapter (section 3.1.c), causal relevance is the contemporary paradigm for scientific explanation in the general philosophy of science literature, which leaves purely statistical notions in a difficult situation from the explanatory point of view. This situation hinders a neat distinction between the probabilistic causes of statistical trends and the alleged fundamental causes of "real" processes, as statisticalists demand. In turn, the distinction between the way natural selection explains, on the one hand, and how individuals cause changes, on the other, gets lost when we consider the flaws of a purely statistical, non causal view of explanation.

Moreover, statisticalism has been contested on the basis of how causation is in fact considered in evolutionary models. As we saw in the previous chapter (section 3.1), explanations tend to make reference to causal relations, thus most attempts to defend the explanatory role of evolutionary models point to the causal hypotheses ingrained in their use. On the one hand, Roberta Millstein, Robert Skipper and Michael Dietrich (2009) have argued that it is not the mathematical apparatus of the theory what determines its causal or non-causal nature but its *interpretation*: it is the pragmatic context of a mathematical construction that assigns any meaning to it. The position of Millstein and colleagues is that mathematical models do not involve interpretations nor ontological consequences on their own; rather, the original purpose for which they are constructed must be taken into account in order to discern the nature of the notions involved in them. Consequently, they claim, there is a pragmatic relation between vernacular (ecological) and predictive (statistical) fitness inasmuch as the models of population dynamics intend to explain changes

caused by ecological interactions. This relation implies, in the view of these authors, that predictive fitness inherits the causal nature of vernacular fitness.

In a similar fashion, Alex Rosenberg and Frédéric Bouchard (2005) defend that mathematical notions of population genetics models are causal in nature by virtue of their derivation. According to these authors, the mathematical apparatus of population dynamics is derived from the Principle of Natural Selection as described by Darwin and stated by Fisher's fundamental theorem. They observe that "when we consider the Darwinian assumptions, about selection for ecological fitness, from which [Fisher's] theorem is derived, its derivative status as a theorem becomes evident" (Rosenberg & Bouchard 2005, p. 346). Thus, in their view, no pragmatic interpretation is needed in order to establish that the statistical apparatus of population genetics is indeed making reference to the causes of evolution: the very formulation of such apparatus implies it.

Both lines of criticism reflect Sober's initial idea when introducing the analogy of forces, namely that

> The words 'causality' and 'cause' are not to be found in the algebra or diffusion equations of quantitative models but in the conceptual framework that motivates them and makes them intelligible (Sober 1984, p. 8).

Statisticalists have replied to these arguments clarifying their position. For them, the models of population genetics are not intended to represent evolutionary phenomena in the first place. On the contrary, their role is to serve as a tool for predicting and explaining evolutionary trends by abstracting away the causal nature of the process (Ariew & Ernst 2009, Ariew et al. 2015). Their approach relies on the decoupling of population genetics models from the causal structure of evolution: it deems the components of statistical models as predictive tools in virtue of their mathematical, but not representative, nature. However, as Jun Otsuka (2016) forcefully argues in his recent review of the controversy, no particular explanation or prediction can be derived from the statistical models of evolution without introducing causal assumptions about the populations to which they apply. In particular, these models could not work without assuming that they are considering

the ecological causes of population changes: no predictive value could be attributed to the models if the statistical measures they employed contained no causal information.

Moreover, as mentioned above, in denying the causal content of evolutionary models statisticalists engage with a notion of explanation completely detached from causation. It has been claimed from the statisticalist position that considering causes at the level of population dynamics is "superfluous" (Walsh 2007), in the sense that lower-level causes—i.e. individual causes—are sufficient for explaining populational processes. However, one may wonder in what sense the models of evolutionary genetics refer to individual causes at all and, more importantly, how they explain population dynamics without referring to the causes acting at the level of population changes. Indeed, a recent defense of statisticalism by Walsh, Ariew and Matthen (2017) argues that in evolutionary models, cause and explanation are decoupled:

> The plausibility of statisticalism rests on its ability to demonstrate that while change in trait distribution is caused by the capacities of individual organisms to survive and reproduce, it is nevertheless explained (and *not* caused) by the statistical properties (fitnesses) of abstract trait types. (Walsh *et al.* 2017, p. 6)

But how can a statistical property explain without making reference to causes? As reviewed in section 3.1.b of the previous chapter, the idea embedded in statistical explanation is that something is explanatory of a phenomenon if it is statistically relevant to it. In other words, *A* explains *B* statistically if *A* is statistically correlated with *B* in the sense of raising its probability (e.g. Salmon 1971). However, as pointed out there, *explanantia* of this type, just like other types of *explanans*, are better understood as causally relevant. Indeed, the model of explanation as statistical relevance precisely intended to capture relations of causal relevance by statistical means. It is generally well agreed, however, that this tradition is flawed for failing to do so (Woodward 2014. See section 3.1 of previous chapter). In any case, what would it mean for trait fitness to be statistically relevant to changes in trait frequencies if it does not mean being causally relevant to it? Can the fitness mean of a trait explain a particular evolutionary result without making reference to any causal property? To

my view, if statisticalists were right in their criticism, evolutionary models could not be explanatory at all—something a statisticalist would not assume. But this seems untenable considering their predictive and empirical applications to microevolutionary processes (see Otsuka 2016).

What lies at the core of the statisticalist position, I believe, is a very rigid and unjustified attitude towards what can be considered as a real cause. Statisticalists privilege the level of individuals as causally efficacious, and render higher-level explanatory terms as causally vacuous. However, notice that this privilege could be problematized by alluding to chemical or micro-physical causes rather than the causes acting at the level of individual organisms. Why benefiting one level of causes over the other if it is not precisely by pointing at explanatory relevance? Actually, the contextual character of both causation and explanation stressed in Chapter 1 (section 3) makes any assumption on the primacy of individual-level causes untenable. As we have seen, the causes of a phenomenon have to be posed with a reference to that phenomenon in particular. It is populations, species and clades that evolve, that is, that change in the characters that constitute them. If we want to acknowledge this fact as a real process with a causal structure—rather than a pseudo-process that supervenes on individual births and deaths—, then we have to explain it by pointing at the causes that make a difference to it. Indeed, notice that Darwin's introduction of natural selection did not only refer to individual causes but to a probabilistic mode of causation in populations of diverse organisms (see Hodge 2016). Let us recall that Darwinian and neo-Darwinian population thinking precisely enabled the recognition that individual *differences* in a population are causally relevant for evolutionary changes. That is, natural selection depends on there being differences among the individuals in a population, a fact that cannot be reduced to the sum of individual properties. As it has been pointed out now by a number of authors (Millstein 2006, Otsuka 2016), what is at stake in the causalists-statisticalists discussion is the relevance of causes at higher levels than the level of individuals: variation in heritable fitness; mutation, recombination and reproduction rates; population size; etc. From a causalist position, then, one needs to argue that these factors represent causes of

evolution. This has been defended, as we shall next see, through a propensity understanding of fitness.

2.2. The propensity interpretation of fitness

Propensities were first introduced into the evolutionary dynamics discussion at the level of individuals, in order to solve the problems associated with an exclusively empirical understanding of fitness. In biological practice, 'fitness' is considered as an attribution of individuals, of their traits, or of types of them—and sometimes of populations or even species—, which grounds their probability of survival and reproduction.[31] Hence fitness is, in our urn analogy, whatever grounds the probability of red balls to be picked by the discriminating hand. Let us recall that natural selection, however understood ontologically, is the phenomenon of individuals that are 'fitter' with respect to their environment having better chances of surviving and therefore tending to reproduce more, or to leave more offspring than others. Defining fitness is thus a major focus for philosophers of biology interested in the causal and explanatory structure of evolutionary theory and its models. Now, the explanatory scope of natural selection leans on this property being a *capacity*, a dispositional property (or perhaps a set of such properties) that manifests itself only given certain circumstances and with a certain probability. This is because identifying fitness with *actual* survival and reproductive success, rather than with a capacity, would render the 'survival of the fittest', and thus the theory of evolution by natural selection, unexplanatory. The classical, empirical understanding of fitness, in identifying it with a measure of actual survival and reproductive success of types (e.g. Lerner 1958, Dobzhansky 1970; see Beatty 1984 for a classical discussion), indeed faces this problem. Imagine that we want to determine whether red balls are fitter than green balls in our urn analogy. If we are actualist about fitness, we must establish this by examining the rate of sampling of the distinct types of balls throughout iterations of

[31] Some authors have vindicated a more inclusive notion of fitness, and proposed the idea of differential expansion (Van Valen 1989) or differential persistence (Bouchard 2008), instead of reproduction, so the notion could apply to living beings other than metazoans. Although this point is certainly important for a potential generalization of these debates to all kinds of living beings, it does not affect the core aspects of the present discussion.

the sampling process. We observe the rates of sampling red and green balls, obtaining an empirical measure of their fitness values, and conclude that red balls are fitter than green ones based on the sampling rates. Now let us suppose that we want to *explain* the distinct rates of sampling, that is, the evolutionary trend in our urns. For doing so, what we need to explain is why red balls are being sampled more often than green ones. In turn, we need to make reference to the higher probability of red balls being picked. But this is just alluding to their fitness values, which we established by measuring the same rates that we want to explain. We have fallen into a circularity insofar as fitness values are defined through rates of balls sampling and vice versa, rendereding the concept of fitness completely unexplanatory.

To overcome this paradoxical situation of the concept—the so-called circularity problem (e.g. Popper 1974)—, Susan Mills and John Beatty (1979) famously proposed a propensity interpretation of fitness (hereafter PIF), in a similar fashion to Robert Brandon's (1978) account of adaptedness. This interpretation states that fitness (or adaptedness) refers to a disposition of individuals to survive and reproduce—rather than a frequency—that is distinct from their actual survival and reproductive success. This is usually illustrated by the example of two twin organisms living in the same environment, where one suffers from a sudden lightning shock while the other lives a long and successful life (Scriven 1959). The actualist picture would force us to attribute a higher fitness level to the surviving twin when, as we know *ex hypothesis*, it was as fit to its environment as its unlucky twin. This illustration serves to make a more general point about the models of evolutionary dynamics: while actual rates of reproductive success are used for establishing fitness values, they do not define the fitness of organisms. Consequently, the *expected* fitness of individuals needs to be different from their *realized* fitness.

From the statisticalist point of view, the distinction between 'expected' and 'realized' fitness only applies to the ecological notion of vernacular fitness (e.g. Walsh *et al.* 2017), clarifying its causal and explanatory role in the ecological interactions of individuals. However, it does not apply to the predictive sense of fitness of population genetics models, since the models make no reference to causal factors at all. From the causalist position, by contrast, this distinction has a strong connection

with the representative role of evolutionary models. As a matter of fact, the PIF was born not only for solving the tautology problem conceptually. It arose as a way to consider fitness values as mathematical expectations, that is, as probability measures distinct from relative frequencies. Mills and Beatty (1979) originally understood fitness as the mean value of expected number of offspring of an individual in the short term. However, this first approximation soon proved to be limited, and the authors later developed a refinement of their previous work in (Beatty & Finsen 1987). According to this revision, the PIF should incorporate—among other things—populational as well as trans-generational features such as the variance of rates of reproduction. In this view, the distinction between 'expected' and 'realized' fitness—between fitness as a propensity and actual number of offspring—intends to apply to the complex structure of evolutionary probabilistic models: it applies to the predictive sense of fitness in addition to the vernacular, ecological one.

The PIF has undergone many further developments that reinforce this idea of complex relational properties being intrinsic to fitness as a propensity. Indeed, the number of specific statistical factors to include in the estimation of fitness is "probably infinitely large" (Rosenberg & Bouchard 2020). A salient example is the recent account developed by Charles Pence and Grant Ramsey (2013). Their proposal reformulates the PIF, arguing that fitness is a scalar magnitude derived from the complex set of dispositions to survive and reproduce of individuals, as represented by a sample space of "possible daughter populations", namely the possible sets of descendents of a particular individual at a given time in the future. According to Pence and Ramsey, the fitness of an individual can be established as the limit in the long run of the averaged sizes of all possible daughter populations of this individual, that is, as the average value to which the sizes of its possible daughter populations tend in the limit. On the basis of some simplifying assumptions (see Pence & Ramsey 2013 for details), this derivation is reducible to the traditional PIF.[32]

In a very influential chapter, Sober infers from the complexity of non-intrinsic factors in the PIF that mathematical fitness is not a propensity *of*

[32] There are possible cases in which the limit does not converge into a particular scalar quantity, such as extinction and chaotic dynamics, but it is reasonable to assume that it will be convergent under normal circumstances (see Pence & Ramsey 2013, pp. 862-3 for details).

individuals. Instead, it is "a more 'holistic' quantity; it reflects properties of the organism's relation to its environment ... [as well as] a property of the containing population" (Sober 2001, p. 36). An important element is that fitness is typically used in probabilistic models to assess not only the number of offspring a particular type may have, but also its relative frequency in the overall population. Sober thus recognizes that fitness has "two faces", namely a twofold role in *explaining* and in *predicting* evolution. The propensities of individuals explain, while the 'propensities' of probabilistic models predict evolutionary events. Notice that Sober's distinction is not a complete detachment between ecological fitness of individuals and probabilistic models—a position a statisticalist would take. Sober is not accusing evolutionary models of not considering the causal dispositions of individuals. Instead, he is acknowledging that the mathematical expectation they assimilate does not only depend on intrinsic factors, but on the setup conditions for these factors to be manifested. Indeed, Sober's position is that the PIF has an explanatory *and* a predictive side as mathematical expectation. From the point of view of this thesis, we may consider his point to be that the predictive side of fitness needs to integrate factors of the entire experimental arrangement, and not just intrinsic properties of individuals, as it is indeed the view about propensities more generally for most propensity advocates, notably Popper (1959, see Chapter 1).

I take the distinction that Sober draws between an explanatory and a mathematical sense of fitness as a reflection of the more general distinction between a propensity and the probability measures that can be derived from it (see section 3.2 of previous chapter). Propensities are, as we have seen, the difference-maker cause of a particular stochastic pattern, which in turn must consider the dynamical properties of complex systems. Deriving specific probabilistic measures is a context-dependent representational problem that applies to every propensity to a larger or lesser degree, and it does not preclude probabilities from reflecting the manifestation of real causal factors. In turn, the PIF makes use of dispositions as grounds for deriving the mathematical apparatus of expected fitness, and this derivation will necessarily

include different relevant causal factors depending on the types of trials considered—hence depending on the types of patterns to be explained.[33]

In this regard, it is important to notice that separating causal propensities from probabilities helps in clarifying the situation for causalists. Ecological fitness has been defined as the correspondence of an organism's traits to the "various aspects of the environment the organism is living in" (Rosenberg & Bouchard 2020). Arguably, there is a mismatch between a definition such as this and the presumably measurable nature of fitness. But this is just the mismatch found in every propensity and the distinct context-dependent probability measures that can be derived from them: under different experimental arrangements—different chance setups—, the same (sub-)system can show different probabilistic behaviors. The propensities of individuals manifest in a probabilistic pattern and , in turn, account for the possible evolutionary scenarios that are considered in practice for deriving probability measures. Indeed, some recent accounts explicitly differentiate between fitness as a probability measure and the dispositions that ground it. For example, Pence and Ramsey's (2013) account can be framed into Ramsey's general causal picture of evolution (2006, 2013, 2016), where the dispositions of individuals are responsible for a set of possible lives they can experience, whose structural properties ground the probabilistic notions of evolutionary models. In similar lines, a causal dispositionalist account (Mumford & Anjum 2011) of fitness has been recently defended in the literature. According to this view, "expected [mathematical] fitness might be interpreted as the manifestation of fitness [as a disposition] in an ideal world" (Triviño & Nuño de la Rosa 2016, p. 12). This approach explicitly relates the causal, dispositional properties of individuals to the mathematical expectation used in models of evolutionary change without reducing the former to the latter.

I consider that this situation where vernacular fitness grounds mathematical fitness follows from Darwinian population thinking. Ecological or vernacular fitness—Darwin's notion—is not an intrinsic value of an organism but a relational one: its nature is comparative. Darwin was concerned with the *differential*

[33] In a different context, Abrams (2012a) makes a similar distinction between causal and modeled conceptions of fitness.

reproductive capacity of individuals, namely with the extant differences in survival and reproduction among individuals in a population. That is the essence of population thinking. It therefore makes no sense to consider ecological fitness as an intrinsic property different from what the models of population dynamics represent. The notion of fitness must give grounds for the application of the principle of natural selection (Rosenberg & Bouchard 2020), which, as stated by Fisher (1930), relates changes in the frequency of a trait in a population with the variance in fitness inside the population. Consequently, it is a concept that in all cases must make reference to the population and environment of organisms in some way or another. If ecological fitness is a propensity, then, it is so in the same sense that predictive fitness—of individuals, of traits, of populations, etc.—is: it is a dispositional feature that plays a causal role in ecological dynamics from which particular context-dependent probabilistic models can be derived. In the remaining of this section, I consider how a causal propensity understanding of population dynamics makes sense of important debates taking place among causalists, enabling considering the propensities of populations and the process of genetic drift as causal determinants of evolutionary change. As we shall see, this position can make room for a kind of typological explanation as complementary to the traditional "population thinking" of evolutionary theory.

2.3. Causal propensities of populations, types and drift

Embedded in the received population thinking is the idea that the properties of populations—by large their fitness differences—are causally relevant to evolutionary change. To this regard, philosopher Roberta Millstein argues that population-level causes are the "distinctively evolutionary way" of depicting causes (2003, p. 1326),[34] insofar as population factors are difference-makers in the long run from an evolutionary point of view. Her view is that, even if we may know of lower-level

[34] Following Weber's (2001) idea on the representational role of probabilistic properties (see section 3.3 above), Millstein moved from an agnostic view of probability in evolutionary theory (2000) to a realism about population level propensities as causally responsible for population dynamics (2003).

causal details—including individual interactions—these are not the factors that make a difference in the evolutionary sense. As she claims,

> we choose to ignore these causal factors, rather than being ignorant of them. In fact, even if we knew all the causal factors, we still might choose to use probabilities, as Elliott Sober has argued, because they allow us to compare similar populations to one another, ignoring the "nitty-gritty" causal details of individual populations (Millstein 2003, p. 1321)

So from this point of view, regardless of the nature of properties at the micro-level, we point at population properties because they are the ones that are relevant for explaining population dynamics in a causal way. Millstein develops a propensity view of population properties as the evolutionary version of long-run propensity interpretations of probability, claiming that the evolutionary meaningful way of talking about repetitions of experiments in the long run is to talk about ensembles of populations instead. Interestingly, she suggests that the single-case/long-run distinction is better conceptualized in evolutionary theory as a single-case/kind one, analogous to the token/type distinction (2003, p. 1324, footnote 4). In her view, the different long-run propensity interpretations of probability, such as the ones reviewed in section 2.2.3 of the previous chapter (i.e. Gillies' and Hacking's), can be applied to the probabilistic properties of populations.[35] Following Millstein, we can state that population level probabilities are real causes of the world even if they are "relative to a particular specification" (p. 1323). The particular specification refers here to all the relevant background conditions that make these properties causally explanatory. For instance, the fact that we want to explain that white fur increased its frequency in a population of rabbits in the snow, rather than remain in the same proportion, makes the fact that this trait increases the relative fitness of their beares in this particular population causally relevant for explaining it.

[35] Interestingly, Millstein reviews Fetzer's and Giere's propensity interpretations, both single-case, and applies them to evolutionary populations, which she explicitly associates with the long run. This apparent inconsistency is, I believe, a strength of her account insofar as she considers populations as particular entities rather than as collections of individual experiments. Her long run or "kind" propensity view actually depends on considering *ensembles* of populations as kinds, and not really the populations *per se* as ensembles of individuals, a distinction that she makes at certain points (p. 1322) but gets lost throughout the paper.

Other factors such as mutation rates or population size can also be considered causally determinant of this increase of frequency, for instance if mutations from the allele associated to brown fur to the one associated to white fur was very common. The size of the population, on the other hand, affects the efficacy of selection and in turn the scope of genetic drift in this process.

In sum, fitness differences, rates of mutations and recombinations, and population size are all populational properties that are causally relevant factors for explaining evolutionary dynamics. If we wanted to explain population dynamics by simply pointing at the "nitty-gritty" details of the dynamics underlying this process, then we would end up with a non-explanatory description of an evolutionary process. Recall that, similarly, we could depict the details of the causal chain that leads a particular die to land on 4 in a particular roll (see section 2 of Chapter 1). For sure specific triggering conditions and causal paths leading to this result could be described in principle. However, as discussed in the previous chapter, such a depiction would be of no explanatory power for explaining general trends. If we assume that evolutionary processes can be explained at all by the properties of populations—as evolutionary models do—then we need to acknowledge the causal relevance of populational properties.

I believe that considering Millstein's distinction between type and token propensities in evolution from a causal propensity view has interesting consequences for the alleged "population thinking" introduced through neo-Darwinism. As seen in section 1 of this chapter, population thinking refers to the idea that only individuals—as opposed to *types*—are real and that it is the differences among them that may (causally) explain evolutionary changes. However, there is a sense in which a type propensity of populations can be considered not merely as the propensity of a collection of tokens but as *kinds* of populations instead (Millstein 2003, p. 1324).[36] It is interesting to note Millstein's reference to the comparison between *similar populations* in the quotation above. For some evolutionary explanations, it will not only be interesting to point at abstract properties of a particular population such as

[36] Interestingly, philosopher Jun Otsuka argues that the gene-view of selection—namely that selection acts on genes—is a kind of typological rather than population thinking, insofar as it is not individual genes but *types of genes* that matter (Otsuka 2019).

their variance in trait mean or fitness, but perhaps to idealized properties of population *types* such as similarities in migratory trends, mutational rates or trait response to selection in different populations.

This relates to another interesting debate in the philosophy of evolutionary biology, namely whether trait or type fitness is derived or not from the fitness of individuals. The controversy underlying this debate is whether, when biologists refer to the fitness of a particular trait—such as the fitness of white fur—, they are talking about the mean fitness value of individuals bearing the trait (e.g. Mills & Beatty 1979); or if they refer to some populational property such as trait variation (e.g. Sober 2013). The debate clearly overlaps with the causalists-statisticalists controversy in its treatment of populational properties.[37] However, for the purposes of the present discussion, it suffices to point out that, under a causal propensity view, it is possible to make sense of type propensities as distinct from abstractions of individual or token propensities. This is not just an alignment with causalists but a further vindication of idealized higher-level dispositions as explanatory (see section 3.1 of previous chapter). As suggested in Millstein's quotation above, considering the propensities of higher-level realities is not exclusively an exercise of abstraction from details. It is also an exercise of idealization that enables comparison among tokens pertaining to the same type, e.g. among traits that ground survival in a certain way, among populations that grow in a particular manner, etc. Let us remember that causal propensities can be instantiated in a variety of ways that need not retain anything in common except for a particular way of relating triggering to manifestation conditions (previous chapter). From this point of view, propensities may serve not only for explaining in terms of individual differences in a population—i.e. population thinking—but also for explaining in terms of *types* of individuals, populations, or even species and higher taxa as contributing to differential reproduction in a particular way—typological thinking. As we shall see later in section 4 and in the next chapter, this consideration is crucial for explaining the origin and generation of variation in evolution.

[37] In particular, the debate has been important due to the association between trait fitness and population properties. However, the inclusion of the topic here concerns only the particular understanding of 'type' that I relate in the main text.

From this position, we can now acknowledge another interesting population-level component of a causalist picture: its understanding of drift as a cause of evolutionary change. This issue is at stake because it affects one of the most prominent debates in the recent history of evolutionary biology, namely the neutralist-selectionist debate (Hey 1999, Plutynski 2007). In a nutshell, this debate is an argument about the relative importance of selection and drift in evolution, namely a revival of the controversies that brought about the M.S. (see section 1.1.b above). Neutralists are aligned with advocates of mutation-driven evolution, their main claim being that mutational rates can affect the fixation of alleles with independence of natural selection—thus by drift only—(Svensson & Berger 2019). Selectionists consider that the possibility of allele fixation by drift is negligible for explanatory purposes given its unimportance as compared to natural selection. As mentioned in the previous section, drift has been considered by some causalists as a process distinct from natural selection, where changes in the composition in a population are due to a sampling process in which physical differences among individuals are not causally relevant (Beatty 1984, Millstein 2002, 2003, 2005). In other words, whether or not an individual will be sampled by drift for the next generation is not influenced by the particular properties of that individual as compared to others. An evolutionary change due to the process of drift is thus part of a different sample space than the one that corresponds to a change due to selection: it is an event in the sample space displayed by a chance setup where the reference environment does not imply differences in fitness—e.g. an environmental random event such as a lighting shock. With regards to the relative importance of this process in evolution—i.e. the neutralism debate—, Millstein argues:

> To a large extent, the debate between neutralists and selectionists is couched in terms of the prevalence of drift vs. the prevalence of selection ... the concern is whether variants are 'neutral' or not (i.e., causally irrelevant to differences in reproductive success or not), not over the extent of deviation from expectation (Millstein 2005, pp. 172-173).

In other words, when we ask whether a trait evolved by natural selection or drift, we are concerned with whether or not its fitness value made a difference in the

causal process that brought about such trait. Needless to say, the same event can be part of different sample spaces, and thus can result from different chance setups. For example, the same trait distribution can be the result of natural selection or drift—or a combination of both (Beatty 1984). But this is exactly the case for all probability spaces. Landing on 4 is an event of the sample space generated by trials of a fair die as well as of the sample space generated by trials of a loaded die. The difficulty lies in that evolutionary events are unique—rather than members of a well-defined collective—and thus postulating different generating conditions will lead to different explanations of the same event. Thus, idealizations are needed in order to provide evolutionary explanations. A causal propensity view captures this insofar as it regards evolutionary probabilistic models as derived not only from frequencies but also, and importantly, from our conceptualization of the possible as based in the causes we consider.

Other ways of understanding drift causally are also in line with a causal propensity view of it. For instance, Rosenberg and Bouchard (2005) regard drift as "the departure of [the] initial conditions" of a selective process from an equal distribution (p. 352), that is, as a bias in the conditions generating an evolutionary change. While fitness distribution in a population will set a particular sample space of possible evolutionary changes—given a probability distribution over possible triggering conditions—, a process of drift is one where the trials involved were triggered only by low-probability triggering conditions. Recall that according to the range conception of probability (section 2.2 of Chapter 1), the probability of an event is the ratio of initial conditions in a chance setup leading to that event. As we saw, the initial conditions must fulfill a series of requisites in order for this to be the case, such as being smoothly or equally distributed. The position taken by Rosenberg and Bouchard (2005) is thus that selection fails to obtain in the sense that a bias in the initial conditions of a selective process may influence the frequency of results, rendering fitness differences irrelevant. So under this view, drift can indeed be considered a deviation from expectation, but only on the basis of a specific kind of causal process—and not because a given result was not predicted by a given model. The causal process is the realization of a chance setup under specific, biased,

triggering conditions. Under the causal propensity view, drift like this considered should be regarded as biases in the conditions triggering the propensity of a population to change in composition. Notice that in this approach the final result or event is part both of the sample space of natural selection and of drift. The difference from the process account above is that the sample space of drift is a subset of selection's sample space—one that has specific, unequally distributed initial conditions. Again, we face the problem of evolutionary events being unique and there being more than one possible set of generating conditions for a specific result.

Philosopher Marshall Abrams' position is interesting in this regard. As we saw in the previous chapter, Abrams is an advocate of causal probability and, specifically, of the range conception. In his essays over the probabilistic nature of selection and drift, Abrams introduces the notion of "organism circumstance probability" (2007), namely the probability that an individual encounters particular environmental circumstances, and stresses the importance of considering a reference environment in evolutionary probabilistic models (2007, 2009). In his view, the reference class problem is essential for understanding many discussions among causalists, including the discerning between natural selection and drift. A consideration of the reference environment in such a way allows for a refinement of the complexity of the mechanisms involved in evolutionary processes, as well as a recognition of the strong context-dependency of probability ascriptions in them. In his (2007), Abrams emphasized that the strong context-dependency of probabilities in evolutionary theory may drastically limit the scope of the propensity interpretation of fitness. However, following his later works on mechanisms and causal probability in evolution (Abrams 2015, 2017)—where he seems to endorse a less strict view of propensities that falls into the category of causal probability—, I believe that the context-dependency of evolutionary probabilities can be approached through the causal propensity view of this thesis.

The general picture of the causal structure of evolution developed by Ramsey (2013, 2016) indeed enables a consideration of this context-dependency and fits nicely into the causal propensity framework here advocated for. As introduced in the previous chapter, Ramsey considers that the dispositions of individuals are structural

causes of evolutionary change, while the particular—environmental, populational, developmental—circumstances they face trigger a specific evolutionary outcome (Ramsey 2016). Thus Ramsey argues that fitness and drift are structuring causes of evolutionary outcomes. Under his view, there is a set of possible lives a particular individual can live determined by its intrinsic, genetically-based, properties. Each possible life represents a determined course of events with a given reproductive result (e.g. having three offspring). Now, even if there may be infinite possible such lives, their reproductive results will be constrained in particular ways, the possible lives constituting a structured set. In this view, fitness can be seen as a measure of the reproductive success of the set of possible lives of the individual. This account considers the dispositional properties of individuals as causally responsible for evolutionary changes, in connection with—but not by reduction to—the mathematical models of expected fitness. Fitness like this understood is a structural property of the possible lives of an individual, and can be seen as a structural cause of evolution (Ramsey 2016). There are, additionally, triggering causes that determine which particular possible life of the individual is actually lived—such as encountering a predator before reproductive age—and which will be determinant of the realized fitness of the individual. Moreover, Ramsey (2013) introduces the concept of *driftability* for drift as an evolutionary propensity. He argues that, since the outcomes of an organism's possible lives constitute a heterogeneous set, we can establish a particular degree of heterogeneity in such a set: the organism's driftability. Driftability like this understood is thus intra-organismic heterogeneity in the set of possible lives an individual can live, since "not all ways an organism can live its life have the same outcomes" (Ramsey 2013, p. 3915). In turn, this capacity to drift of individuals—namely to live their lives in heterogeneous ways—affects the driftability of populations, especially if their size is small. We can now extend such a view for considering higher levels of structuring causes. The possible is structured by individual capacities, but also by higher-level dispositions such as fitness differences. Only through considering higher-level factors can we conceive what is *evolutionarily* possible and in turn construct a probabilistic model of evolution.

We have seen that there are different ways of understanding the causal structure of evolution at higher levels than individual births and deaths. As I argued, these views can be accommodated into a causal propensity understanding of probability, where the dynamical properties of populations ground different probabilistic dispositions structuring sets of possible evolutionary changes. The derivation of particular probabilistic measures will be a context-dependent procedure that will also depend on the availability of specific frequencies—always relative to the relevant reference class specified by the explanatory propensity. In sum, I believe that the causalist picture of evolution could benefit from a causal propensity view, which makes sense of the explanatory character that ecological interactions play in evolutionary biology. In particular, it provides a causal view of 'chance as sampling' (cf. Millstein 2011), namely of the probabilistic process of differential reproduction of extant variants, at the core of the selectionist view of evolution endorsed by the advocates of the M.S.

3. Chance in the sample space of evolution

If differential survival and reproduction can be understood as a sampling process where ecological interactions ground the probabilities of being sampled, one may wonder about the origin of this sample space. That is, one could consider what is the origin of the variants that selection and drift sample for being the parents of the next generation. Trivially, they are the result of the reproduction of certain variants, precisely through selection and drift sampling processes. Nevertheless, this doesn't tell us anything about *how* the variants are generated. This generation is something more than mere sampling: it is the origination of an entirely new sample space from the variants sampled in the previous generation. Unless we reduce the diversity of all natural populations to mere differences in the combination and frequencies of the same extant variants, the causalist view of evolution needs to incorporate this generation into its picture—particularly the appearance of new variants, or of new combinations of pre-existing ones.

As mentioned in the introduction to this chapter, chance has also been a matter of concern for philosophers at this level of how variation is generated. Recall that Mayr characterized the origin of variation as a level where "chance reigns supreme" (1963, p. 214, see section 1.1.b above), an idea that was captured in the Darwinian notion of chance variation. This notion meant that variation was supposed to be always abundant and unbiased, waiting for its 'ecological opportunity', and thus lacking any impact in the course of evolution. Indeed, an essential component of the philosophical discussion reviewed in the previous section is that the variants considered are always already present in the analysis. However we decide to understand the role of a given trait in evolutionary changes, or of a given organism—always defined as a consistent set of traits—the origin of those traits remains unexplored. The population thinking embedded in the M.S., in basing changes exclusively in extant differences, stimulates this neglect.

This situation is in accordance with Sober's (1984) asseveration that most 'source laws' of evolution are situated in theoretical ecology, namely in the causes explaining ecological interactions and in turn grounding natural selection, drift and migrations. However, let us recall that the source laws of the fourth factor of evolution considered in these models—i.e. mutations—are to be found in the field of genetics instead (see section 1.2.b above). Mutations are supposed to be the ultimate source of variation. However, there is a lack of connection between the origin and nature of mutations and the consequence laws of evolution, that is, the models of evolutionary genetics. Chance in mutations can be in some contexts characterized as indeterminism or as ignorance of causes (Millstein 2011), but the significance of these 'source laws' for the models of evolution is very anecdotic. As mentioned previously in this chapter, mutation rates are the only mutational factors considered in evolutionary genetics models, namely in the consequence laws of evolution, specially as maintainers of the extant polymorphisms in a population (Okasha 2016).

This scenario gets even more complicated when we consider that variants at the ecological level are phenotypic. Notice that the causal components explaining population dynamics act at the level of phenotypes while models of population genetics directly map genotypes to fitness values. This entails a limitation insofar as

the explanatory scope of these models is constrained to those phenomena where the fitness values of genes remain constant. This fact is not merely a problem of extrapolating fitness values to different ecological environments. It is primarily a problem about the intrinsic context-dependency of genotypic effects, which are highly sensitive to the genetic and developmental environment. Thus any change in a genotypic or developmental aspect of an organism can affect the effects of genes, something that is not reflected in their statistical treatment.

Indeed, evolutionary models have been accused of statisticalism about the causes of variation at the phenotypic level. This has been particularly striking with respect to the phenotypic models of quantitative genetics, which do refer to phenotypes but do so without considering the genotype. Evolutionary biologist and philosopher Massimo Pigliucci (Pigliucci 2006, Pigliucci & Kaplan 2006) argues against these models on the basis that they do not consider the processes underlying the statistical properties they incorporate. In these models, the evolutionary response of a population to selection is given in the form of a change in the mean value of a quantitative character—such as an increase in the average height of a population through the iterative selection of higher individuals—, which is modeled through statistical properties of the genes and their associated phenotypic values. But not only that: the models are based on assumptions about these properties. Pigliucci argues that, since phenotypic responses are calculated through the correlation of genotypic variants with phenotypic values, they do not rely on any causal knowledge about how genes influence the construction of phenotypes. Despite similarities with Matten and Ariew's (2002) and Walsh and coworkers' (2002) statements about population dynamics in evolutionary models, Pigliucci's concern is not the decoupling between explanation and causal factors. Rather, it is the total lack of explanation of variation in these models, which are based—according to him—on statistical assumptions rather than measures.

Unlike the case of ecological factors reviewed in the previous section, this statisticalist criticism cannot be contested from a causalist point of view. The considerations of chance at the level of variation, as we shall see, are not based on causal knowledge about its origin. Rather, chance at this level is characterized against

the directionality of natural selection, which only acts at the ecological level of evolution—i.e. which is only relevant for chance as sampling. This is one of the most salient consequences of the adaptationist inclination of the M.S., namely of the prevalence of selection in explaining evolutionary processes. In this sense, chance has been associated with an opposition to directionality in similar terms to how drift is considered at the ecological level (Eble 1999, Millstein 2011). However, since the causes of variation are distinct from the causes of ecological interactions, this association has stood in the way of achieving a causal understanding of the generation of variation whatsoever.

In this section, I explore how chance is understood in the generation of the sample space of evolution, namely in the origination of variants that will be sampled by selection and drift in ecological interactions. First, I review two ideas that complemented Darwin's chance variation and that were instrumental in the adaptationist tone of mainstream evolutionary biology, namely evolutionary chance mutation and the idea that phenotypic changes are random (section 3.1). Then, I introduce the culmination of what I will call *the chance variation view*: the perception of selection as a creative force, responsible not only for preserving fitter variants but crucially for generating new evolutionary possibilities (section 3.2).

3.1. The received notions of chance in variation

Let us briefly recall that the Darwinian notion of chance variation rivalized in the early 20th century with other views that considered variation to be an important driver of evolutionary change. In section 1.1.b of this chapter, I reviewed the main discussion leading towards the models of the M.S., namely the discrepancy between early geneticists—or mutationists—and neo-Darwinians. But as mentioned there, the panorama at the time was more complex than that. Two other influential positions of the time are key for understanding the historical development of how chance is perceived in evolution with respect to variation. On the one hand, neo-Lamarckians believed that the capacities acquired through the life of individuals affected positively the adaptiveness of new variants. On the other hand, orthogeneticists argued for the

intrinsic potential of development in guiding evolution. Both schools of thought challenged Darwin's chance variation by ascribing directionality to variation to the detriment of selection. Moreover, with the rise of the new geneticist paradigm, both developmentally-inspired schools underwent an important reconceptualization that shaped the view of the M.S. about variation.

Here I review the notions that have stood in the way of an acknowledgement of the causal role of variation in modern, post-synthesis evolutionary biology, and that have promoted the view of a Newtonian-like, passive nature of evolution. First, I introduce the most widespread and explicitly recognized characterization of chance variation in modern terms: the notion of 'evolutionary chance mutation', which was introduced to oppose neo-Lamarckian ideas (section a). Then, I review the less acknowledged but still central to the classical picture idea of the randomness of phenotypic changes, which rejects any connection between the origin and the effect of variation (section b).

a) Evolutionary chance mutation

Inspired in the evolutionary theory endorsed by Lamarck almost a century before, so-called neo-Lamarckians of the early 20th century emphasized the transmission of acquired characters as an important causal factor in the evolutionary process of adaptation: environmental demands on individuals influence which variants they tend to generate through reproduction (see Gliboff 2011). However, as mentioned in section 1.1 above, the experimental approach of early geneticists soon established the separation of germ and soma lines as well as the particulate nature of inheritance. This led to an assimilation, through the first half of the 20th century, of the notions of gene and mutation, and to the rejection of any effect of somatic factors on the genetic material. The new framework specially affected the core *explanans* of neo-Lamarckism, which was reconceptualized after the M.S. in the movement known as "mutational" Lamarckism (Merlin 2010, Razeto-Barry & Vecchi 2016). Mutational Lamarckism has been defined as

> [the] hypothesis according to which the environment can induce mutations directed towards producing phenotypes that increase the fitness of the organism in that particular

> environment (Razeto-Barry & Vecchi 2016, p. 2; see also Keller 1992, Merlin 2010, Millstein 1997).

In other words, it is the view that the effects of mutations can be influenced by environmental factors, facilitating the appearance of adaptive variants in reproduction. This hypothesis was influenced by experimental observations in populations of bacteria (e.g. Luria & Delbruck 1943; reviewed in Keller 1992), whose fast adaptation to some extreme environmental changes revitalized the thought that environmental demands influence the production of variation in adaptive ways. Mutational Lamarckists put the focus on the mechanisms producing new mutational variants, and they postulated an adaptive potential for them. Because of the complexity of the phenomenon and the variety of possible mechanisms accounting for the experimental results that succeeded, this controversy is far from being solved in contemporary debates of chance. Empirical research on "mutator mechanisms" is wide (Chen *et al.* 2012), there being a consensus about the environmental induction of some mutations. One of the most salient examples of the last years is the change in mutation rate in populations of bacteria *E. coli* under thermal stress. The stressful environmental condition increases the rate of mutations in these bacteria, and in particular it increases the probability of traits conferring thermotolerance to arise (Jablonka & Lamb 2005, Koonin & Wolf 2009).

Against this background, philosophical advocates of the M.S. perspective have preserved the pointview of chance variation through the idea that the effects of mutations are random regardless of their origin (Sober 1984, Millstein 1997, Merlin 2010). This idea of random mutational effects is widespread, although the exact meaning of this randomness is not always defined with precision (Razeto-Barry & Vecchi 2016). In any case, the randomness of mutations—sometimes referred to as 'spontaneity'—is emphasized as a natural fit for Darwin's idea of chance variation being the 'raw material' for natural selection (Keller 1992). Recall that this notion rested on the fact that the causes of variation were unknown at Darwin's time, and the assumption that those causes are unrelated to adaptiveness (Beatty 2008). The inclusion of mutator mechanisms implies thus rejecting any relation between them

and adaptations. Douglas Futuyma's formulation is representative of mutational randomness:

> Mutation is random in [the sense] that the chance that a specific mutation will occur is not affected by how useful that mutation would be (Futuyma 1986, p. 78)

And more recently:

> Mutations are random with respect to what will improve survival and reproduction. New conditions do not increase the frequency of mutations that are beneficial in those conditions (Futuyma & Kirkpatrick 2018, p. 95).

In this context, philosophical advocates of the M.S. view have offered several explicit definitions of mutational randomness by invoking the lack of a cause-effect relation between environmental conditions and the adaptiveness of mutational effects (reviewed in Razeto-Barry & Vecchi 2016). This idea of randomness has been called 'evolutionary chance mutation' in the literature (Merlin 2010), a label that synthesizes the idea that mutations contrast with selection as the source of adaptive directionality, and that I shall use in what follows to refer to it. Francesca Merlin's (2010) definition summarizes the central claim of this position, stating that mutations are chancy if and only if

> there is no specific causal connection between the probability of a mutation being beneficial in a given environment and the probability of it occurring in this environment (Merlin 2010, p. 6)

This particular position is a matization of a general view that mutations are simply 'random'. Merlin herself specifies in a later work (Merlin 2016) that this is a "weak" notion of randomness, in opposition to a stronger claim about the total lack of patterns in the production of genetic variants. To this regard, it is now well known that there exist important biases in mutations. For instance, the genetic context of a particular gene affects its probability of mutating. Moreover, genomes are known to have 'hotspots' with high mutation rates while they are relatively conservative in other areas. These areas vary widely among species, suggesting that they may be the products of evolution. On the other hand, biases in the effects of mutations are also

well established, such as the salient transition-transversion bias: transitions—either purine-to-purine or pyrimidine-to pyrimidine changes—generally obtain way more often than transversions—purine-to-pyrimidine or pyrimidine-to-purine changes—(Houle & Kondrashov 2006; A. Wagner 2012a). Therefore, rather than being a general, abstract idea of random variation, Merlin's (2010) formulation takes the null hypothesis to be that the probability distribution of genetic effects is not biased with respect to fitness, as based on George G. Simpson's (1953) statistical notion:

> Mutations are not random in the full and usual sense of the word or in the way some early Darwinists unrealistically considered as fully random the variation available for natural selection. ... [T]he term 'randomness' as applied to mutation often refers to the lack of correspondence of phenotypic effect with the stimulus and with the actual or the adaptive direction of evolution. (Simpson 1953, pp. 86-87. Cited in Merlin 2010, p. 6)

This idea of randomness aligns with what Millstein (2006) called a justificatory sense of chance. As mentioned in the previous chapter, notions of chance in evolution are sometimes invoked to justify the explanatory relevance of other, seemingly less chancy phenomena, notably natural selection. Thus the "weak" randomness implied in the 'evolutionary chance mutation' concept serves as contrast with selection inasmuch as it underlines the directedness or non-randomness of the latter.

According to some biologists, this "weak" notion of randomness is still seriously challenged by the evidence on mutator mechanisms, for they are known to provide abundant variation that typically increases the probability of adaptive traits arising (e.g. Jablonka & Lamb 2005, Koonin & Wolf 2009). Against this, defenders of the M.S. view have argued that the notion of evolutionary chance mutation remains unchallenged insofar as it only refers to the adaptive directedness of variation, and not to its availability. Under this view, in the *E. Coli* thermotolerance example, what increases under stress is the absolute number of mutations, without necessarily implying a change in the relative frequency of fitter variants among those mutations. Thus, for the M.S. advocates, the increase of thermotolerant traits would not be

caused by a bias towards thermotolerance in the generation of variants. Rather, it would be caused by the general augmentation of the mutational rate under thermal stress, which proportionally increases the probability of some mutation being fitter—and rapidly spread through selection. So, even if a bigger number of fit variants arises under specific environmental conditions, the position of chance mutations advocates is that the relative frequency of fitter variants remains unchanged.

In a thoughtful, recent review of the debate, philosophers Pablo Razeto-Barry and Davide Vecchi (2016) conclude that notions of mutational randomness tend to either be too broad, to be too narrow, or to not provide formal standards to empirically assess this controversy. I consider that their analysis and their proposed definition of mutational randomness cast some light into this debate. The authors define mutational randomness as the independence between the probability of mutations arising in a particular environment and the average fitness effect of those mutations in that environment. If this independence condition is violated—they argue—, then there is a legitimate case of mutational Lamarckism. Thus whether or not a mechanism increasing mutation rates—such as the mutator mechanisms involved in the *E. coli* example—is Lamarckian will depend on the net fitness effect of the mutations it produces. They consider that their definition gives empirical grounds for solving this issue in principle, while they acknowledge the difficulty of its application insofar as it depends on reliable means for measuring fitness effects in environmental changes by contrast to other environmental conditions. In turn, their view is that this discussion "remains an open empirical problem" (Razeto-Barry & Vecchi 2016, p. 9). In any case, not only the existence of 'Lamarckian' mutational mechanisms is an empirically open question, but, interestingly, there seems to be nothing in the Darwinian view of the M.S. that forbids accommodating these mechanisms conceptually. As Sober acknowledged in his classical work (1984), mechanisms of this kind are possible in principle—although highly unlikely—, as long as they are the result of an evolutionary process.[38] It is not impossible, for

[38] Sober refers to this possibility as a "Rube Goldberg arrangement" (Sober 1984, p. 109), in allusion to the American cartoonist, whose works usually represented absurdly complex designs performing very simple tasks.

instance, that natural selection has favored mutator mechanisms that increase the probability of thermotolerance-related traits under thermal stress—indeed mutational Lamarckism is compatible with the selective origin of such possible mechanisms (e.g. Jablonka and Lamb 2005). This considered, the debate over mutational Lamarckism turns into a discussion over the relative importance this type of mechanisms might have as compared to other factors of evolutionary change (Sober 1984)—similarly to the neutralism debate about the relative importance of drift.

Finally, let us recall the contrastive nature of causal explanations pointed out in section 3 of the previous chapter. In accounting for the possibility of a Lamarckian—adaptively biased—nature for mutator mechanisms, we must have a reference point for contrastive purposes. A focus on the environmental optima in the moment of reproduction—e.g. thermal stress 'demanding' thermotolerance—is already complex enough to assess. How to additionally consider whether or not regular mutations, without sharp environmental changes to make comparisons with, are biased? What should we contrast the fitness effect of those regular mutations with? A more general consideration of mutational Lamarckism of this kind could not be empirically assessed at all unless a default fitness expectation is established.[39] Thus, in the worst scenario for advocates of chance mutations, the focus on environmental optima renders their view as a mere rejection of teleology, in the sense that it only serves for preventing the effect of a mutation from affecting its causes—e.g. the production of thermotolerance cannot affect the mutations causing it. This rejection is already implicated in the genetic conception of heredity insofar as the genetic content is supposed to not be affected by the developmental process it is responsible for. More promising, however, is the idea that the rejection of mutational Lamarckism is a claim about the relative importance in guiding evolution with respect to other factors, notably selection. But, as we have seen, this claim is not only empirically open in some—perhaps few—specific cases, but it may be empty of content when not restricted to specific environmental optimas. As in the case with natural selection and drift discussed in the previous section, a conceptualization of

[39] This point will be relevant for similar debates regarding evolvability (section 4 in Chapter 3).

the environment is key to considering which dispositions causally explain which probabilistic patterns. However, the prominence of natural selection—and thus of ecological opportunity—in guiding evolution has stood in the way of philosophically exploring how the mechanisms responsible for variation relate to adaptation.

The take-home message of this debate is that the M.S. notion of evolutionary chance mutation entails a rejection of any adaptive potential for mutator mechanisms that is not grounded on conceptual or empirical bases, but may have been influenced by the historical controversy over the relative importance of natural selection in evolution. A consequence of this, to my view, is an unjustified lack of philosophical analyses of the mechanisms of variation and their role in evolution in similar terms to those employed in debates on population dynamics. Regardless of the alignment of mutations with selection, it would be interesting to explore how the origin of variation could influence the consequence laws for evolution, namely how they can affect the probabilistic understanding of evolutionary changes.

b) Random phenotypes

A different tradition opposed to both neo-Darwinism and geneticists in the rise of the M.S. was the theory of orthogenetic evolution (see Ulett 2014). Following the classical developmental viewpoint (Bowler 2005), orthogeneticists believed that new variants were additions to the process of ontogeny that were guided by the intrinsic drives of the developmental process itself. The idea behind it was that there is an innate tendency to complexity in organisms, and that the directionality of evolution was influenced by such tendency. It is not hard to see how this idea clashes with the prominence of natural selection in guiding evolution of neo-Darwinians: a key aspect of the M.S. narrative was the sufficiency of natural selection of chancy and abundant variants to account for diversity, adaptation and complexity in the living world.

Similarly to neo-Lamarckism, orthogenetic evolution was severely challenged by the new geneticist paradigm of the 20th century. The conceptual separation between genotype and phenotype introduced by Wilhelm Johannsen (1911) was a turning point in their developmental view. If hereditary changes are changes in the genotype—as the new theory suggested—; why suppose that there is a bias towards

complex *phenotypes* in the production of variation? Aligned with the anti-Lamarckian notion of evolutionary chance mutation, the M.S. view of variation accentuated a lack of causal connection between mutations and specific phenotypic effects, predicating the randomness of phenotypic changes. This idea had its highest impact after the so-called molecular revolution that ran parallel in the days of the M.S. The rise of molecular biology brought—among other things—the idea that the functioning of organisms can be explained in terms of their molecular interactions—so-called molecular reductionism (e.g. Rosenberg 2008; see Brigandt & Love 2017 for a review). Subsequently, the Darwinian chance variation notion was associated with the idea that genotypic changes have random molecular effects in organisms, whose fate will only be determined by their effect on fitness.

Jacques Monod and his classical *Chance and Necessity* (1972) is one of the main representatives of this received view. According to Monod, mutations and inheritance—what he calls 'invariance'—are the only responsible for variety. But, while inheritance reproduces with high fidelity the phenotypes that have been preserved through selection, mutations produce only *random* effects on them. These random effects may be passed to subsequent generations only on the basis of their impact on the fitness of their beares, namely individuals. Monod's view is that this randomness-and-selection mechanism can account for all novelty and variety in the living world:

> We call these [mutational] events accidental: we say that they are random occurrences. And since they constitute the *only* possible source of modifications in the genetic text, itself the *sole* repository of the organism's hereditary structures, it necessarily follows that chance *alone* is at the source of every innovation, of all creation in the biosphere. Pure chance, absolutely free but blind, at the very root of the stupendous edifice of evolution: this central concept of modern biology is no longer one among other possible or even conceivable hypotheses. It is today the *sole* conceivable hypothesis, the only one that squares with observed and tested fact. And nothing warrants the supposition—or the hope—that on this score our position is likely ever to be revised. (Monod 1972, pp. 112-3).

Monod here refers to the apparent lack of correlation between the molecular structures of genes and the proteins they code for, suggesting that even the genetic code is the result of an evolutionary—and thus for him, selective—process. This point is further stressed when he mentions the "absolute coincidence" between a mutation and its effect:

> the initial elementary events which open the way to evolution in the intensely conservative systems called living beings are microscopic, fortuitous, and utterly without relation to whatever may be their effects upon teleonomic functioning ... In effect natural selection operates upon the products of chance and can feed nowhere else (Monod 1972, p. 118)

This influential view aligned with anti-Lamarckism in the non-adaptive production of mutations, but added to it a further sense of chance: regardless of any consideration about fitness, mutations are random with respect to their phenotypic effects. Notice that, as we have seen, mutations have been shown not to be random in the sense of lacking any probabilistic pattern. However, under the M.S. view their effects can still be seen as random in the sense that there is no causal connection between the origin of variation and its effect. This idea radically opposes the views of orthogeneticists (Ulett 2014), who considered that variation can only be conceptualized as a change in the developmental process—which connects the origin and phenotypic result of variations—determined by its own dynamics. Through the rejection of this idea, however, the M.S. could eliminate another competitor for selection—namely, development—in the explanation of complexity.

This general sense of random mutational effects, while generally accepted, has also been problematized recently. In particular, the cellular context of genetic changes seems to be an active agent in the generation of mutations, a phenomenon that may result in a non-random relation between the molecular causes and effects of mutations (Shapiro 2013). In any case, and as we shall see in the last section of this chapter, the randomness of phenotypic effects can be assessed at different levels than the molecular one. However, the idea of random phenotypic effects has also been influential in the lack of philosophical analyses in the causal terms of population dynamics: if variation is completely random and only natural selection—together

with genetic drift—are responsible for evolutionary tendencies, the study of the generation of variation has no impact in the causal structure of evolution whatsoever, and thus can be ignored.

3.2. The creativity of selection

The alleged randomness of mutations with respect to adaptation and to phenotypic changes of the M.S. were thus subordinated to the exclusiveness of natural selection in providing directionality to evolution. In turn, this rendered superfluous any consideration of the way biases in variation could affect the evolutionary process. Recall that the occurrence of mutations is random only in a 'weak' sense of the word (Merlin 2016). However, as we have seen throughout this chapter, in the M.S. there are no source laws of variation to be found—namely the causes of variation—, and in turn variation is not represented in consequence laws—i.e. in the mathematical apparatus of its models. But how to justify that only chance and selection as regarded in these models are responsible for the complexity and diversity of living beings? The answer lays in what Stephen Jay Gould considers the "essence of Darwinism", namely the creativity of natural selection (Gould 2002, see also Beatty 2016, 2019):

> Natural selection obviously lies at the center of Darwin's theory, but we must recognize, as Darwin's second key postulate, the claim that natural selection acts as the creative force of evolutionary change. The essence of Darwinism cannot reside in the mere observation that natural selection operates—for everyone had long accepted a negative role for natural selection in eliminating the unfit and preserving the type. (Gould 2002, p. 139)

According to Gould, the neo-Darwinian flavor of the M.S. demands that variation is always abundant, small in extent and undirected or isotropic (Gould 2002, pp. 141-145). In other words, for selection to be able to create new phenotypes, mutations were considered copiously available in all directions, affecting phenotypes in small degrees and with no biases in their effects:

> What else but natural selection could be called "creative," or direction-giving, in such a process? As long as variation only supplies raw material; as long as change accretes in an

> insensibly gradual manner; and as long as the reproductive advantages of certain individuals provide the statistical source of change; then natural selection must be construed as the directional cause of evolutionary modification. (Gould 2002, pp. 140-141)

Even if these restrictions are not always entirely or explicitly demanded (see Beatty 2016 for discussion), the conceptual tool of the gene pool indeed enabled the idea that selection can create the variation it acts upon through the recombination of the selected alleles. In reconciling the models of allele frequency of mutationists with the idea that it is selection that initiates and guides evolution, the gene pool metaphor debilitated the role that mutations had for geneticists as a source of "*discontinuity, initiative, creativity* and *directionality* in evolution" (Stoltzfus 2006, p. 306). By establishing the abundance and copiousness of extant variants, the gene pool provides enough variation for selection to create the new combinations demanded by environmental circumstances. What the M.S. eliminated was the geneticists view (e.g. de Vries 1905) of selection as a mere "sieve" that discards or preserves variation (Stoltzfus & Cable 2014). To the contrary, the M.S. gave selection the capacity to combine the alleles of the gene pool to create new variants almost with no restrictions. The main exponent of this view in contemporary evolutionary genetics is the so-called "infinitesimal model", based on Fisher's work, which "assumes that the genetic component of a trait is determined by an infinite number of unlinked genes that each has an infinitesimally small, additive effect" (Gienapp et al. 2017).

The gene pool and its potential for recombination is a step further in the neo-Darwinian idea that selection can shift the mean of a trait: it can also shift the *range* of available variants (Beatty 2019). Interestingly, Beatty suggests that this idea is at odds with the neo-Darwinian notion of chance variation (Beatty 2016, 2019). In his careful study of the history of this controversy, Beatty points out that a key aspect of the M.S. is that variation is not entirely random but *biased by selection itself*. Let us remind that Darwin envisioned the very production of variation as stimulated by selective conditions—e.g. as in domesticated species facing artificial selection. According to Beatty, this goes against the very spirit of the idea of chance variation.

As a consequence, the received picture of evolution as consisting of a two-steps process—the production and the sampling of variation—is flawed in the M.S. conceptual apparatus. This is the case because in the M.S. conceptual framework selection is responsible for a "new center for the variations occurring in the following generation" (Weismann 1902/1896, p. 34. Cited in Beatty 2016), and thus selects for the mechanisms responsible for new variation. In other words, selection determines which individuals will provide small, gradual and isotropic variations—i.e. offspring minimally, gradually and isotropically deviating from their own character values—, therefore biasing the production of variation precisely in the direction of the selective regime. In sum, by sampling individuals, selection biases which new variants can arise in future generations, which are always considered available in the direction of selection itself. Thus the creative potential for selection of the M.S. has two crucial components. On the one hand, the alleged abundance, graduality and isotropy of variation provides selection with exclusiveness in directing evolution. On the other hand, new variation can be introduced in the 'gene pool' precisely because selection biases which variants reproduce and thus vary abundantly, gradually and isotropically.

What this implies is that selection and drift are responsible not only for the space of possible daughter generations of a particular population—as we have seen in the models of population dynamics of the previous sections. They are considered responsible, in addition, for the space of *new possible variants* that can be generated in those daughter populations throughout evolutionary time. Let us see this more clearly through the analogy of the urn. Although it has been claimed that the urn analogy faces a limitation when it comes to represent reproduction and mutations (Millstein 2017), I believe it is the focus on population dynamics that pervaded classical evolutionary biology which has precluded the analogy from being extended this way. As we saw in section 1.2 of this chapter, mutations can correspond to changes in the colors of balls when they are introduced in the second urn. Thus it is the randomness of color changes—whether or not changes in color are biased—what is at stake. On the other hand, an important additional feature of the picking hand is that it can multiply the balls it selects before introducing them into the new urn,

illustrating the reproduction of distinct types in the new generation. The selecting hand has thus the capacity not only to decide which balls will take part in the next generation, but also how many copies of them can be generated. In our example of red balls being fitter than green ones, the hand can multiply red balls by three each time it selects one, but multiply blue and green balls by two or introduce them without multiplying them (see Figure 2.4).

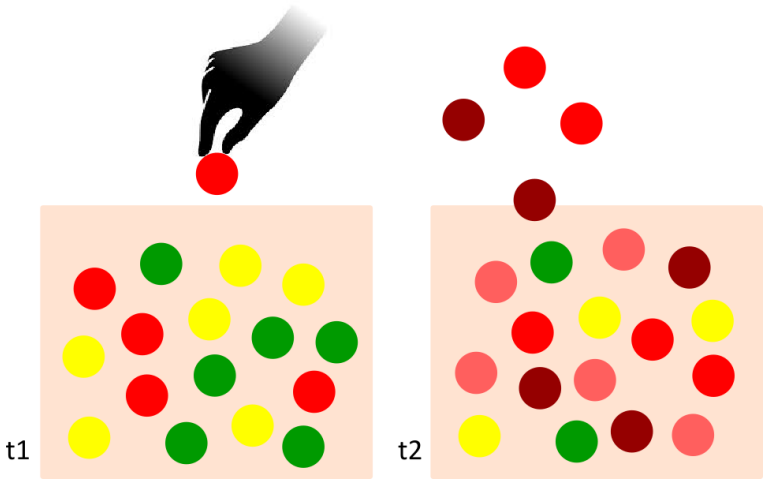

Figure 2.4. The creativity of selection. In this example, the red balls picked by the selecting hand at t_1 multiply themselves with gradual changes in color in the second urn at t_2 producing new variation of red tones, namely dark red and light red. The graduality implied in all deviations from the parent generation enables that the generation of new variants is only biased by the selecting hand.

With these changes, we can wonder whether variation is biased with questions such as: Is the probability of a mutational change from green to red larger than the probability from red to green? Is it always more likely to produce a darker color than a lighter one? How much can the color of a particular ball change when reinserted and multiplied in the second urn? In order to answer this sort of questions, we need to know about how ball colors are produced in the first place, that is, how the multiplication of a ball derives into a change in ball color. However, the creativity of selection view answers that the color of balls changes gradually and in all possible directions without biases. As a consequence, if many red balls are picked—because they are fitter—many gradual variants of red will arise in all

directions: slightly lighter, slightly darker, slightly oranger, etc. This enables the creation of *new variants* precisely in the range of selection.

However, if we consider that the randomness of mutations is 'weak' (Merlin 2016) and that their effect on phenotypes is evolutionarily contingent (Monod 1972), why suppose that mutations produce gradual, isotropic variation? Moreover, why is it even possible that they do so considering the overwhelming complexity relating genes to phenotypes? It is time to consider a different chance setup for the space of possible variations in evolution. This chance setup needs to incorporate how *weakly* random genotypic changes produce changes in the target of selection: organisms and their phenotypes.

4. Evo-devo and variational probabilities

So far, in this chapter I have defended that the focus on population dynamics of evolutionary genetics has enabled a causal propensity understanding of evolutionary models as related to the ecological dispositions of individuals, populations and types. Thus the probabilistic notions embedded in evolutionary genetics explanations refer to the causes of evolution at the ecological level, where fitness and drift as propensities structure the ecological possibilities to which probabilistic models of evolution are applied. Moreover, I have argued that the conceptualization of Darwin's chance variation in the M.S. and afterwards prevented any causal understanding of variation and how it is generated in similar terms to the ones associated with ecological factors. To the contrary, it enabled the postulation of natural selection as the main causal factor in the construction of variational possibilities. However, since natural selection is not strictly speaking a cause of the production of variation, no causal propensity understanding is possible for how variation is generated in this classical view. In other words, the aspects that selection can explain—such as population structure, the fixation of certain characters or the recombination of alleles—do not refer to the very generation of variation, i.e. to the way in which phenotypic variants are constructed in living systems. For overcoming this limitation, in this final section my aim is to introduce the notion of variational probabilities and

to argue that a causal propensity conception of them is possible only if development is considered as the cause of variation in evolution. For doing so, I lean on the variational notions used in the explanatory framework of evolutionary developmental biology or evo-devo.

Evo-devo is a discipline that emerged in the 1980s and keeps growing since. It combines developmental and evolutionary biology for assessing both the evolution of developmental processes and the influence of development in evolutionary phenomena. Since for all multicellular organisms the hereditary material and the selected phenotype are mediated through an ontogenetic process that involves cell differentiation and morphogenesis, the properties of this process arguably influence the course of evolution. One of the aims of evo-devo is to understand how they do so and to assess their importance for evolutionary explanations beyond the classical framework so far presented in this chapter. The causal, generative component of variation vindicated in the previous section is but one of the philosophical counterparts of evo-devo claims with respect to the explanatory scope of classical evolutionary biology. Their consideration of developmental processes is an explicit inclusion of the causal pathways responsible for phenotypic variation. In turn, it is through the work of evo-devoists and the philosophical reflections they have raised that the ideas concerning the generation of the sample space of evolution as regarded in this thesis have to be considered.

In this final section of the chapter, I first review the genealogy of evo-devo vindications as derived from a typological and developmental pre-Darwinian conception of variation (section 4.1). Secondly, I assess evo-devo's perspective and argue for the causal role of development in creating phenotypes, and in turn giving directionality to evolution (section 4.2). Finally, I consider the views of the possible embedded both in the adaptationist and in the evo-devo understanding of evolution, in turn arguing for the inclusion of development in the notion of chance at the level of variation (section 4.3). For doing so, I first review how the adaptive conception of the possible embedded in the creativity of selection view is limited precisely for neglecting development (section a). Then, I introduce the notion of variational probabilities (section b), intended to capture that developmental properties

determine what evolutionary paths are possible and, more importantly, which ones are more developmentally probable. This will enable me to provide a genuinely developmental notion of chance in variation and to develop a causal propensity view of it in the next and final chapter of this thesis.

4.1. The typological view of variation

Considering the origination of phenotypic variation in evolution is at odds with Darwinian 'population thinking'. The generative component vindicated in the last section—that is, the introduction of how variation is generated—contrasts with the idea that it is extant individual differences within a population all that matter for evolutionary change. In demanding the causal role of development in building up variation, evo-devo stresses the fact that the internal properties of developmental systems have the *capacity* to vary in certain ways. In this sense, the evo-devo agenda is sometimes perceived as a vindication of the 'typological thinking' that had been sentenced to irrelevance and pseudo-scientificism by the Darwinian convention (Amundson 2005, Brigandt 2007, Lewens 2009b, Love 2009). In fact, much of the explanatory focus of evo-devo studies is typologically-leant as a result of its historical roots: development has traditionally been a focal point for those interested in organismal form, its study being entangled within the typological thinking of morphological disciplines. In this section, I sketch the morphological tradition leading to the evo-devo research agenda, especially with regards to its typological means of understanding ontogeny and variation. This will provide us with a perspective of the epistemic goals of evo-devo necessary for introducing the dispositions of development into the probabilistic explanations of evolutionary biology.

Organismal form can be defined as the figure or shape of organisms or as the structural or topological relations between their components (Nuño de la Rosa 2012). Broadly considered, morphology refers to three different aspects of organismal form (Amundson 2005). On the one hand, it applies to the forms within one organism: how the parts composing an individual living being are arranged and relate to each

other. On the other hand, it regards the similarities and divergences in shape and structural relations among different organisms or among different species or higher taxa. Finally, it concerns how form arises in ontogeny, namely with the formation and differentiation of parts in the development of organisms from the egg to the adult stage. Among morphologists of the 18th and 19th centuries, typological notions were familiar for referring to commonalities in form: Type, Archetype, unity of type, and unity of plan (Hall 1999). This is mostly because this tradition is interested in unity rather than diversity: those structural aspects shared by apparently unrelated organisms. In this sense, the notion of body plan or *Bauplan*, one of the main focuses of morphology, refers to the basic common organizational plan shared by organisms belonging to a major taxon, such as a phylum. For example, all vertebrates share the same *Bauplan*, namely similarities in the structure and the parts composing disparate vertebrate species, such as the vertebral column and the spinal cord.

In section 1.2 of this chapter, we saw that the taxonomists of the 18th and 19th century were traditionally concerned with intraspecific variation. The morphological tradition, in contrast, gave biological diversity a very different meaning. Their task was to evaluate the similarity and correspondence of forms within individual organisms and among organisms of distinct species, and thus they approached variation as a measure of divergence of form between body parts and structures. Comparative morphology was in turn interested in inter-specific variation and stability with regards to organic forms. For instance, morphologists were concerned with the differences and similarities of limbs across vertebrate species, such as the wings of birds and the forelimbs of horses. With this epistemic goal in mind, the limb can be considered as an abstract or idealized 'type' whose instantiations differ across vertebrates (Figure 2.5). The view of so-called transcendental or idealistic morphologists envisioned the plurality of species as deriving from a small number of types. Their tradition was one of "logical dispositionalism" insofar as it concerned the possible forms that a logical type could generate, without that generation being causal in nature. That is, it referred to the conceivable forms in which a certain archetype—e.g. the vertebrate limb—could be instantiated (Nuño de la Rosa 2012).

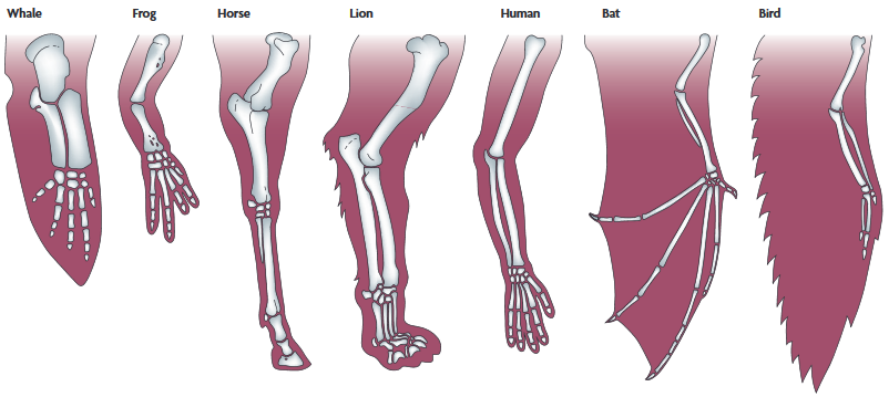

Figure 2.5. The tetrapod limb in different species. Reproduced with permission from (Wagner 2007b).

In the turn to the 19th century, morphologists Étienne Geoffroy Saint-Hilaire and Georges Cuvier engaged in a well-known debate (reviewed in Appel 1987) over the relative importance of form and function. Geoffroy was a structuralist, advocating the primacy of form over function, that is, of the conditions of *possibility* of morphological characters over their actual functionality. He conceived of the unity of type as a means of subsumming those animal types composed by the same parts in the same relative positions, introducing the concept of analogous structures—homologous in contemporary terms—for referring to the same entities in seemingly unrelated species, such as the digestive tract in vertebrate and mollusca species. In contrast, Cuvier represented the functionalist tradition, defending the primacy of function over form, and regarded similarities among animal types—among the four *embranchements*: *vertebrata*, *articulata*, *mollusca* and *radiata*—, as based on the similar conditions of existence of animals. The debate was highly influential in the works of naturalists of the time and the years that followed, and its endurance got lengthened by a lack of unified terminology about the phenomena to be explained. In this sense, the work of Richard Owen synthesized the ideas of both traditions, conceptually separating the similarities due to unity of plan and those due to similar functionality (Hall 1999). Owen is well known for being the most influential morphologist of Darwin's time and for establishing some of the main concepts of modern comparative biology. He introduced the contemporary distinction between homology—i.e. the same organ in different species—and

analogy—i.e. different organs performing the same function in different species—, as well as the notion of serial homology, namely one structure repeated several times in an organism, such as the vertebra in vertebrate animals.

Within the morphological tradition, the field of comparative embryology emerged, interpreting morphological connections as embryological in nature. Although some morphologists had already vinculated development with the maintenance of morphological types, the main figure in pre-Darwinian 19th century comparative embryology was Karl Ernst von Baer, who envisioned the embryo as the archetype. Von Baer postulated that the stages of differentiation in embryogenesis reflected the acquisition of typological identity for the individual, that is, the belonging to types ranging from the most general to the particular (Hall 1999):

> The scheme of development is nothing but the becoming type, and the type is the result of the scheme of formation. For that reason, the Type can only be wholly understood by learning the mode of development. (von Baer 1828, cited in Amundson 2005, p. 60)

Thus the comparative embryologists understood development as the acquisition of form, an idea that later came to be known as morphogenesis (cf. Turing 1952). Morphogenesis refers to the mechanisms responsible for "the emergence of the parts that make up an individual organism's form", conforming the form of its species (Maienschein 2007, p. 111). In turn, the typological conception of morphologists aligned with the classical 'developmental viewpoint' of variation (cf. Bowler 2005), which envisioned variation as changes in the developmental processes of individuals, and against which Darwin and his advocates endorsed chance variation and population thinking (see section 1.1 above).

After the rise of evolutionary thought, the newborn field of evolutionary morphology focused on providing a phylogenetic explanation of form that could conciliate the morphological unity of type with the Darwinian idea of descent with modification. Evolutionary morphologists intended to infer phylogenetic patterns from morphological similarities. In the new evolutionary paradigm, unity was linked to common descent, while differences remained associated with conditions of existence on which selection acted. Within this scientific atmosphere, the first

evolutionary embryologists reinforced the idea of a parallelism between ontogenetic and evolutionary stages. For instance, in the frame of his theory of recapitulation, Ernst Haeckel is well known for having associated the gastrula stage of early embryogenesis with the origin of multicellularity, postulating the 'Gastræa' as a hypothetical universal ancestor of all multicellular organisms (Hall 1999).

However, the rise of the M.S. during the first half of the 20th century relegated evolutionary morphology to a conceptual truism insofar as for synthetic biologists structural identity was identified with—rather than explained by—genealogical identity (Amundson 2005, Nuño de la Rosa 2012). Hence, in the case of homology, two parts or organs in different species were considered to be the same if and only if they came from the same ancestral structure. In this sense, the *explananda* of evolutionary morphology—the unity of form—was rendered superfluous, considered as the natural state of things (Caponi 2012), and its agenda was displaced by population thinking and the adaptationist program of the M.S.

By no means this entailed the closure of evolutionary morphology, but merely its decline. For example, after the rise of the M.S., the field of evolutionary embryology kept showing its concern with "uncovering developmental mechanisms underlying evolution" (Love & Raff 2003, p. 329). One of the most prominent phenomena studied in this regard was heterochrony, namely the change in the relative timing of a developmental process affecting the phenotypic result. Gavin de Beer (1958) studied heterochrony in developmental phenomena, noting that differences in rates of gene expression led to differences in the timing of formation of certain structures, affecting in turn their adult form and size (Richmond 2007). Another key research program of late evolutionary morphology was the embryological studies of the origin of Bauplans (Hall 1999). For instance, the establishment of the antero-posterior axis in most groups in the animal kingdom, in being a common basic embryological stage shared by most animals, was considered as having a common evolutionary origin. Finally, the field of functional morphology was concerned with the origin of evolutionary innovations, providing a bridge between the morphological tradition and the study of adaptations (Love 2003a).

Although the relevance of this morphological tradition was minimized by the M.S. historiography (Love 2003a, Amundson 2005), today it is recognized by many historians as one important precursor of the evo-devo agenda (e.g. the essays composing Laubichler & Maienschein 2007). In this regard, the interest in incorporating development into evolutionary biology by modern evo-devo advocates is directly inherited from the morphological tradition. However, regardless of the similarities in explanatory focus, modern evo-devo distances itself from its precursors the morphologists. As we shall see, contemporary evo-devo is a very heterogeneous research field with many ramifications (Love 2015). In broad terms, the evo-devo agenda can be divided into those domains intended to bring comparative and evolutionary tools to developmental biology and those whose aim is to inquire how development influences the course of evolution (Müller 2007). The former, which we may call comparative evo-devo, is inheritor of the morphological tradition. The latter has been called "developmental evolution" or 'devo-evo' by Günter Wagner, one of its most influential contributors (Wagner 2000, 2007a, 2014). As he states about it:

> Devo-evo is starting from a stronger position than evolutionary morphology. Nowadays the study of phylogenetic relationships and character evolution are somewhat separated problems. Evolutionary morphology had the enormous task of resolving both simultaneously while including hypotheses regarding the process of evolution. In contrast, the objective of devo-evo is to explain the developmental basis for evolutionary changes of the phenotype, not to reconstruct phylogenetic relationships or patterns of character transformation. (Wagner 2007a, p. 528)

In this quote, Wagner is manifesting the synthetic character of devo-evo: it intends to cast light into evolutionary biology by introducing development as the source of variation, but it does not have the pretension to reduce the study of evolution to that of evolutionary morphology. On the contrary, it concerns phenotypic—or character—evolution as a separated research question within the broader field of evolutionary biology. In turn, general evo-devo, in encompassing both comparative evo-devo and devo-evo, inherits the explanatory agenda of

morphology without turning its back on the vast corpus of phylogenetic knowledge arisen out of the M.S. and its models of evolutionary genetics.

From the evo-devo perspective, the questions concerning the origin and transformation of phenotypes demand development—a seemingly proximate cause—as a causal factor in evolution. In considering this, the evo-devo agenda inherits the typological and dispositional explanatory schema of the morphological tradition as just reviewed, complementing it with the evolutionary framework and with empirically-based research. This understanding of possible variations within a type, and of development as the acquisition of form, brings as a consequence a reinsertion of typological thinking into the explanatory framework of evolutionary biology, insofar as it is not individual differences in a population what is considered to explain change (i.e. population thinking), but rather the potentialities of a—relatively stable—morphological type.

4.2. Development as a cause of evolution

We have seen that the core *explananda* of evo-devo, inherited from the research tradition of evolutionary morphology, is organismal form and its evolution. Thus this discipline is concerned with the origination of phenotypic changes in reproduction, a phenomenon that lacks a causal explanation other than adaptation or contingency in the classical evolutionary framework, as we have seen throughout this chapter. Let us recall that the phenomena that classical evolutionary genetics can explain are changes in the relative frequency of variants in a population as modelled by natural selection and chancy ecological factors. Only the creativity of natural selection and the chanciness of mutations account for the appearance of phenotypic transformations under this view. By contrast, the evo-devo perspective is that developmental processes play a key causal role in phenotypic changes.

In fact, the relevance of evo-devo research arose as a result of manifest limitations in the evolutionary genetics framework derived from the M.S., especially with regards to the evolution of phenotypes. As evo-devo biologist Gerd Müller (2007) claims, the underlying motivation in the formation of evo-devo was the

recognition of severe limitations in the explanatory scope of the adaptationist paradigm inherited from the M.S. The phenomena out of the reach of the adaptationist program, Müller states,

> included the biases in the variation of morphological traits, rapid changes of form evident from the fossil record, the origin of nonadaptive traits, apparent dissociations between genetic and phenotypic evolution, and the origination of higher-level morphological organization such as homology, body plans, and novelty—to name but some of the major open questions. (Müller 2007, p. 500).

Early evo-devo is situated in the last decades of the 20th century with the advent of a mechanistic approach to the interactions between evolution and development. Both theoretical biologists and experimental embryologists started at this time to relate the mechanisms of development with evolutionary changes, an endeavour to which comparative developmental genetics joined soon after that. The late 1970s and early 1980s saw the arrival of several important works that were critical with mainstream evolutionary theory and its limitations, and which pointed at development as a fundamental cause of evolution, such as Gould's *Ontogeny and Phylogeny* (1977), John T. Bonner's *Evolution and Development* (1982) and Rudolf A. Raff and Thomas C. Kaufman's *Embryos, Genes and Evolution* (1983). The new evo-devo agenda that followed used comparative, experimental and later computational tools to assess these development-related phenomena that were not accounted for in the frame of the M.S. In the turn of the century, evo-devo consolidated its institutional presence and its research agenda gained relevance in the scientific community. However, the way in which it fits with overall evolutionary theory remains problematic, as manifests the diversity of accounts about its history, scope, epistemic goals and theoretical importance (Müller 2007).

The field of developmental genetics was the first to get the general attention in the 1980s (Love & Raff 2003, Müller 2007). The discovery of the highly conserved 'homeobox' genes (McGinnis *et al.* 1984, Scott & Weiner 1984) was a turning point in this regard, insofar as it inaugurated the importation of tools from experimental genetics into the comparative agenda of evo-devo. The homeobox is a genetic

sequence of about 180 base pairs present in the genome of every animal species that plays a key role in their developmental organization despite the manifest differences among taxa (Arthur 2011). Afterwards, the field of comparative developmental genetics has been very prolific in the discovery of differences and similarities among the sequences and the patterns of gene expression present across species, assimilating the morphological patterns of unity and diversity to similarities and differences in the expression of genes. Nonetheless, although this geneticist approach to evo-devo is relatively recognized in evolutionary genetics, the models of the latter, in their statistical treatment of variation, overwhelmingly still ignore developmental mechanisms. To the moment, the genetic approach to evo-devo and the evolutionary genetics frame largely work in isolation from each other.

Developmental genetics provided the evo-devo vindications with certain relevance in the mainstream evolutionary frame because of the direct translation from the well-established genetic picture into the molecular bases of gene expression. However, the experimental success of developmental genetics has been overemphasized as the main contribution of evo-devo to the classical evolutionary frame, while in fact evo-devo's demands are more complex and have larger historical roots. As we have seen, the explanatory agenda of evo-devo is independent from the experimental approach of developmental genetics, and emerged from the influence of morphological and embryological traditions and their combination with the new evolutionary picture (Love 2003, Love & Raff 2003). To this regard, it is worth stressing that, while much of the evo-devo field is concerned with the evolution of gene expression patterns, development can be understood in many different ways, some approaches within evo-devo situating their explanations at the level of ontogenetic processes that are not straightforwardly translatable to genetic language (Müller 2020).

The reason for this lies in the nature of development itself: genes do not specify phenotypes. Rather, they are one component of a complex system whose dynamical rules determine the possible phenotypic outcomes. Individual ontogenies unfold in the complex interaction of numerous genetic, epigenetic, cellular, physiological and environmental components. Over the course of this process, every

cell in the organism shares the same genetic material, but each of these cells will only express a small subset of it according to contextual factors, showing properties that are specific to their cell type. This process generates a differentiation of tissues and body parts whose physico-chemical properties affect the expression patterns of their cells and thus the resulting phenotype. Given this complexity, morphogenetic properties—"such as extracellular matrix composition, cell adhesion, mitotic rate, diffusion constant, kinetic activity, etc." (Alberch 1991, p. 6)—are causally responsible for the building of phenotypes, while genes constitute one important causal factor of such process at a lower level of organization. This enables the recognition of the "problem of development" as one of "spatial differentiation and growth" (Laubichler & Wagner 2001, p. 57), where genes are only part of the story (see also Forgacs & Newman 2005 and Newman *et al.* 2003).

Moreover, developmental modifications are relatively decoupled from genetic changes. On the one hand, many evolutionary transformations are alterations in the temporal, spatial and quantitative regulation of gene expression, rather than adjustments in coding regions. On the other hand, the context-dependency of gene effects is determinant. This means that the same genetic alteration can have radically different phenotypic outcomes depending on the genetic and developmental context of the mutation. In their debate with Alex Rosenberg (1997) on the reducibility of development to the molecular aspects of gene expression, Manfred Laubichler and Günter P. Wagner argue that development is not reducible to molecular genetics insofar as developmental processes tend to vary "more profoundly" than the phenotypes they construct (2001, p. 65). Their illustration is one of the most salient examples that came from early developmental genetics comparative studies: the fact that all dipteran—i.e. fly—species undergo an early phase of body axis determination even if this phase is not determined by the same genes. In particular, the gene *bicoid* is necessary for such a process in the species of *Drosophila*, but it is absent in other dipteran species undergoing the same process. In sum, a consideration of the creative role of development in evolution demands going beyond the gene-centric view of classical evolutionary genetics, and considering instead the morphogenetic processes responsible for the construction of phenotypes.

In addition to this, it is interesting to note that the notions of gene in evolutionary genetics and evo-devo differ in significant ways. As Scott Gilbert (2000) has put it, the gene in classical evolutionary genetics is a mathematical abstraction of the average phenotypic effects of alleles in a population. That is, it represents a statistical effect without any consideration of the processes underlying gene expression. Because genes like this understood serve for explaining selection, these abstractions represent the differences a specific allele may cause in phenotypic effects. From a developmental perspective, nonetheless, a gene is a specific DNA region with a mechanistic function in development. Developmental genes are "manifest by their similarities" (Gilbert 2000, p. 180), in the sense of their belonging to similar developmental pathways, and their differences are studied in a mechanistic way. The association of genes to developmental pathways enables the explanation not only of the generation of form but also, and importantly, of the phylogenetic relations among different developmental systems sharing similar genes. In turn, the evo-devo interest in genes implies a focus on the developmental pathways and mechanisms that relate them to phenotypes, rather than on statistical abstractions of population effects.

Evo-devo thus studies the interface of development and evolution not only at the level of gene expression but also, and importantly, at higher levels, like developmental pathways, cell differentiation and tissue and organ formation. Conceptually, it incorporates to the classical picture of evolution the generative mechanisms responsible for variation at the phenotypic level, and thus concerns, besides how variants are retained and sorted, the developmental construction of phenotypes and phenotypic changes. As argued by critics of the M.S. view, the microevolutionary models of evolutionary genetics cannot account for morphological phenomena such as the stability of organismal form (i.e. homologies and body plans) and the appearance of phenotypic novelties (Müller & Newman 2005), thus face the so-called "problem of variation" (cf. Stern 2000). As Mary Jane West-Eberhard (2003) argues, "all novel adaptive phenotypes must originate before they can be modeled by selection" (p. 35). For example, if we understand evolutionary genetics models as modeling the modification and populational distribution of a biological

character, the origin of the character identity remains out of the scope of these models (Wagner 2014). In turn, the morphological phenomena that were left outside the range of the M.S. models of evolution are the main subject of study in evo-devo. Indeed, macroevolutionary patterns tend to oppose the gradualistic assumptions of the adaptationist view, stasis (Levinton 1983, Hunt 2007) and punctuated equilibrium (Eldredge & Gould 1972) being common in evolutionary history. The inclusion of development and morphological *explananda* has the potential to explain these macroevolutionary patterns, and challenges the alleged reducibility of all evolutionary phenomena to microevolutionary models favored by the M.S.

The recognition of these limitations and the growth of new research areas combining evolutionary biology with other disciplines in the life sciences encouraged a debate over the current state of the M.S. and its epistemic goals. In particular, the criticism of the adaptationist program—boosted by Stephen J. Gould and Richard Lewontin's (1978) seminal paper—vitalized a discussion over the pertinence of extending the evolutionary synthesis (Pigliucci & Müller 2010). Not only development raised as the neglected component that ought to be reincorporated into evolutionary thinking: phenomena such as nongenetic, inclusive inheritance (Jablonka & Lamb 2005) and the influence of organisms in their own selective pressures—so-called niche construction (Laland et al. 2015)—, conform the other pillars of the vindication of an extension for the received neo Darwinian view of evolution. From an epistemological point of view, the criticism to adaptationism has crystalized in a pluralistic approach to evolutionary causes, development being considered one causal factor of evolutionary change.[40]

In turn, in studying the evolutionary transformation of developmental systems and the role of development in those phenomena outside the scope of evolutionary genetics, evo-devo vindicates the causal and creative nature of development in evolution. The study of developmental mechanisms, their stability across species and their versatility in constructing phenotypes enables a recognition of development as

[40] Other causes include nonadaptive trade-offs—sometimes called 'spandrels', cf. Gould and Lewontin 1978—, punctuated equilibrium and the role of contingency (Gould 1989, 1997, Orzack & Forber 2010).

the builder of phenotypic variation. In the last, final section of the chapter we shall see how this study impacts the way chance is considered in evolution.

4.3. The sample space of possible variants

Let us now see how the pluralism ingrained in considering development as a cause of evolutionary transformation facilitates a view of possible variations as what can be generated through development. First of all, we must notice that whatever variants selection and drift may filter and recombine, they will only generate other developmentally possible variants, an idea that is completely absent from the idealizations of the possible merely in adaptive terms. With all due caution, this vindication aligns with the traditions that opposed the Darwinian notion of chance variation, notably the position taken by mutationists in the early stages of the M.S. as well as the classical 'developmental viewpoint' of variation (see section 1.1). Indeed, the theoretical biologist Arlin Stoltzfus (2006) has defended that early mutationism and developmentalism "provided an alternative perspective on evolutionary causation as necessary today as it was a century ago" (p. 304). Such perspective consists in acknowledging a dual nature of causation in evolution: both selection *and variation* should be considered as creative causes of the evolutionary process. In such a dual causation approach, both internal components and ecological factors are causally responsible for phenotypic variation. This is a consequence of considering phenotypic evolution, rather than adaptation, as the main *explananda* of evolutionary theory:

> On a one-dimensional scale of fitness or adaptedness, every change is either "up" (beneficial) or "down" (deleterious), but in a multi-dimensional space of phenotypes, every change has a distinctive direction. (Stoltzfus 2006, p. 307)

In other words, if we are interested in phenotypic change rather than only adaptation, the movement along a space of evolutionary possibilities cannot be determined by selection only. On the contrary, the creativity of selection is silent about how one phenotype is transformed into another regardless of their adaptive advantage. But does this conception really affect the evolutionary understanding of

the possible? If mainstream evolutionary biology and its philosophical picture have so far gone through without development, do we really need to incorporate it to our notions of chance in evolution? In the remainder of this section, I argue that developmental causation enables a conception of the possible that is relevant for a more inclusive and encompassing notion of chance in the evolutionary generation of variation.

a) Evolutionary spaces of the possible

In the non-causal understanding of variation endorsed by the founders of the classical view of evolution, the space of evolutionary possibilities is both generated and explored by selection. As we have seen, by recombining alleles selection is considered to create new evolutionary phenomena. But what is the structure and composition of this space of possibilities where alleles recombine typically considered? The creativity of selection as articulated in the previous section enables the construction of idealized spaces of possible variations extrapolated merely from knowledge of genotypic and phenotypic traits. These idealizations consider thus adaptive possibilities in either spaces of possible genotypes or possible phenotypes, where selection can explore new areas as long as they are the—genotypic or phenotypic— 'neighbors' of previously examined ones.

Sewall Wright's fitness landscape (1932) is one such idealization and one of the most influential ones in evolutionary thought. The fitness landscape is a genotypic space where either genotypic combinations or allele frequencies—individual and population versions, respectively, which nonetheless are relatively easy to transform into each other—map into a fitness value represented by a height level. The mapping function famously constitutes a fitness landscape with peaks and valleys, and enables to visualize the "uphill" movements driven by selection. Although limited in its empirical applicability (Pigliucci 2012), the fitness landscape plays an important conceptual role in the understanding of population genetics models: it provides a means for representing how selection can change the

genetic composition of a population and move it towards a fitness peak in an idealized space of possible adaptations.[41]

Philosopher Daniel Dennett metaphorically speaks of the "Library of Mendel" (1996) as a way to understand the exploratory nature of selection over an idealized space of all possible genotypes. In the Library of Mendel, all possible genotypic combinations are present, arranged by means of sequence similarity. Natural selection can explore this space without any *a priori* restriction, favoring the exploration of those areas with a higher fitness value. Considering the factors introduced in the previous section—such as evolutionary chance mutation and the creativity of selection—, selection thus not only will change relative frequencies of preexisting genotypes, but it will be the one determining which *new* genotypic areas will be explored. However, Dennett is aware of the possibility that some areas of the Library of Mendel may not be reached for reasons that have nothing to do with their low fitness value, but rather by their non-accessibility through mutations. In fact, he regards that some genotypes will be "more possible" than others, which brings up the question of the accessibility of the genotypic space. Less metaphorically, the computational field of genetic algorithms (Mitchell et al. 1996) indeed explores this issue. In genetic algorithms, selection "explores" the genotypic space throughout iterations of algorithmic rules. In these algorithms, the genotypic space is conceived of as a space of "possible solutions" to an environmental problem, that is, as a space of possible genetic instantiations of a fitness value. This leads to the recognition of the possibility of mutational constraints to this exploration, namely limitations on the possible mutations that can take place in a genotypic population. In fact, Dennett introduces his notion of biological possibility as a means to acknowledge the limitations of natural selection to explore any conceivable genotypic combination:

> *x* is biologically possible if and only if *x* is an instantiation of an accessible genome or a feature of its phenotypic products. (Dennett 1996, p. 118)

[41] Nevertheless, Wright had to postulate his shifting balance theory (Wright 1982) in order to overcome the difficulty of selection moving a population away from a local optima (Pigliucci 2012). More recently, some developments on the fitness landscape have introduced the notion of a "holey landscape" for this situation in multi-dimensional landscapes (Gavrilets 2003).

The accessibility of an idealized mutational space has two important limitations that have nothing to do with low fitness values. On the one hand, the location of a species or population in such space has been driven not only by selection but also by drift, which brings about a contingency factor in the exploration of the genotypic space: which areas are immediately accessible for a population will be restricted by its current position. On the other hand, mutational biases may turn certain conceivable sequences unlikely or simply inaccessible—recall that mutations are only random in a weak sense of the word (Merlin 2016, A. Wagner 2012a). Nonetheless, advocates of adaptationism regard these limitations as "local" constraints, in the sense that there is nothing *in principle* that would prevent selection from overcoming them provided that there is some 'uphill movement', no matter how complex, leading to the locally restricted area.

Another related interesting limitation of this metaphor, explicitly acknowledged by Dennett and crucial from the point of view of this thesis, is that "there may be fundamental differences between the space of genomes and the space of 'possible' organisms" that develop from them (Dennett 1996, p. 117). Dennett recognizes that the genotypic sequence does not provide a phenotype without a context enabling its expression, just like—in his metaphor—a book in a regular library does not supply a story without someone who reads it. And just like different readers may interpret the same book under different views, each sequence in the Library of Mendel demands its specific reader. However, Dennett considers the diversity among readers—among the expression contexts of genotypes—to be non important for his genotypic-based metaphor:

> We might say that every species that has ever existed on this planet has had its own dialect of DNA-reading. Still, these dialects have had a lot in common with each other. The principles of DNA reading are apparently uniform across all species, after all (Dennett 1996, p. 114)

What this position entails is that genetic content is enough for determining phenotypes regardless of the varieties of gene-expression context. Following Dennett thus the properties of the genotypic space—fitness values included—must account for the properties of the possible phenotypes, despite the recognized possibility of

"fundamental differences" between them. However, we have seen that the differences between genotypes and phenotypes are profound and strongly context-dependent, rendering this metaphor insufficient for establishing a space of possible evolutionary variants. In turn, the direct mapping function from genotypes to fitness values cannot fully account for phenotypic evolution and thus for how evolutionary variation is generated. On the one hand, such function presumes an unrealistic 1:1 correlation between genotypes and phenotypes, that is, it extrapolates genotypic differences to phenotypic ones. On the other hand, the fitness values of unexplored genotypes cannot be known *a priori*.

A different type of widespread idealization in evolutionary biology is Simpson's (1944) adaptive landscape, which takes the phenotype instead of the genotype as the reference measure mapping to fitness. The adaptive landscape of Simpson was introduced as a metaphor for speciation as a result of ecological isolation. It is thus a conceptual illustration of macroevolutionary events, but again its empirical application has been very limited. However, a microevolutionary version of it has been very prolific in models of quantitative genetics. Through fitness surfaces (Lande & Arnold 1983), the statistical treatment of quantitative traits can be seen as the exploration by selection of a phenotypic space of quantitative values for a particular character or a set of them. Richard Dawkins' (1996) metaphor of the "Museum of All Possible Animals", or its simplified cousin the "Museum of All Possible Shells" (pp. 198-223), can serve to illustrate the selective exploration of the phenotypic space. Inspired by David Raup's 'cube' (1966), Dawkins invites us to imagine an enormous museum where all possible shell morphologies are present, arranged in such a way that any two different shapes will be connected by "a continuous series of intermediate shells on the way" (1996, p. 210) (see figure 2.6).

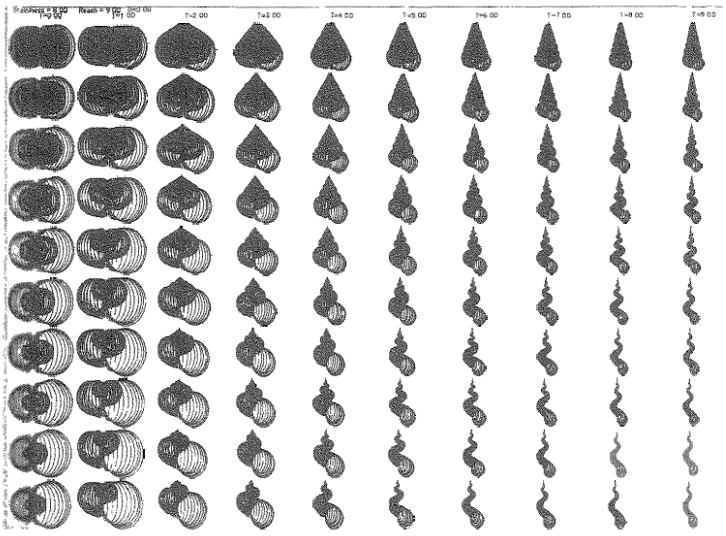

Figure 2.6. A continuous phenotypic space. Dawkins' computer simulation of a simplified Museum of All Possible Shells. Reproduced from Dawkins (1996, p. 209).

Thus for Dawkins, it should be possible to arrange phenotypes so that they are always surrounded by neighbors with the smallest possible difference between them. Needless to say, such a museum must be multidimensional, the specific number of dimensions depending on the number of characteristics composing a particular trait—such as its haight and wideness.

It is interesting to note that, through the museum metaphor, Dawkins differentiates between those biologists who "take genetic variation more or less for granted" and those who consider variation an important component of the evolutionary process:

> Some biologists feel that as you walk the long corridors of the museum what you will find is smooth gradations in all directions. ... A different set of biologists ... feel that the large portions of the museum are forever barred to natural selection. (Dawkins 1996, p. 222)

The first set of biologists would correspond to those aligned with the idealizations of evolutionary genetics models that lay at the core of the M.S. view, while the second would be those vindicating the causal role of variation—and thus of

mutations and development involved in the generation of such variation.[42] Interestingly, Dawkins nuances the latter position, claiming that, for those critical with the M.S. view, "[t]rue neighbours [in the museum] are those forms which, as a matter of fact, can be reached in a single mutational" step (*ídem.*, pp. 222-3), regardless of their degree of morphological similarity. That is to say, for those opposing the classical view, not all conceivable forms are possible, but only those available through mutations, which may represent morphological discontinuities at the phenotypic level.

This matization is similar to Dennett's recognition of mutational limitations: not all phenotypes may be reachable through mutation. However, when applied to the phenotypic case we deal with morphological discontinuities that nonetheless may follow from a single step in the genotypic space. This is so not only because the genotypic limitation of what is mutationally possible, but also because—as we have seen—the genotypic space says virtually nothing about the structure of the phenotypic space, which in fact is the only one that matters to selection: it is the one in which fitness differences among individuals are grounded. Thus similarly to the space of genotypic possibilities, the phenotypic representation of the possible does not account for such limitation either. It recognizes phenotypic limits as a possibility or—in the best case scenario—as a brute fact. The limitation is due, again, to the fact that the generation of phenotypes is not really considered: in mapping phenotypes to fitness directly, no consideration of how they can possibly—and not merely conceivably—change is present. As a matter of fact, this lack is the other face of the same coin that limits the genotype-fitness mapping function as a tool for understanding possible variations: it ignores how organisms develop.

The main metaphors for variation in mainstream evolutionary biology—genotypic and phenotypic spaces—assume that variation is possible in every direction and that only selection and drift will account for their exploration across evolutionary time. This is why the situation embedded in the conceptual arena of evolutionary genetics has been labeled by evo-devo advocates as a "blackboxing" of

[42] Dawkins aligns with the former, although he recognizes to have "an open mind" about the controversy (p. 223).

development (Amundson 2001, Müller 2005). For instance, philosopher Ron Amundson (2005) argues that while the tradition since Darwin initially recognized evolution as "an interplay of heredity and adaptation," the later establishment of Mendelian genetics in the M.S. rendered development irrelevant for evolution insofar as it was detached from inheritance (p. 159). As a result, the explanations of evolutionary genetics, in associating genes or phenotypes directly with their fitness values, not only lack a causal view of how phenotypic variants are constructed. Crucially, they also lack causal—i.e. not merely statistical—hypotheses about how they can change.

In the advent of evo-devo, a major progression towards the inclusion of development into evolutionary biology was the recognition that there are evident limits to the exploration of genotypic and phenotypic spaces. However, these limits are typically conceptualized by the adaptationist tradition as *constraints* to selection's potential for exploration, which has negative connotations that suggest a non-causal role for them (see Orzack & Forber 2010). Constraints can be mutational, as derived from the mutational biases already mentioned in the previous section, and which are one of the focuses of so-called neomutationism (Nei 2013); or they can be developmental, as derived from the very nature of phenotypic construction, and which are a major focus for evo-devo. In a classical consensus paper, a developmental constraint is defined as "a bias on the production of variant phenotypes or a limitation on phenotypic variability caused by the structure, character, composition or dynamics of the developmental system" (Maynard Smith et al. 1985, p. 266).

In the optimization models of adaptationism, a widespread way of labelling this phenomenon is as "constraints on adaptation" (Stephens & Krebs 1986, reviewed in Amundson 1994), implying that development is a limitation of the creativity of natural selection. However, from the point of view of evo-devo, developmental constraints can be conceptualized as *constraints on form* (Amundson 1994), which concern how morphological variation is generated rather than how adaptation is possible (Brigandt 2007). Moreover, in opposition to the limiting connotations of *constraints*, positive terms such as developmental drive (Arthur 2001) or developmental bias (Brigandt 2019) have been proposed to vindicate the causal,

creative role that development has in shaping the structure of the morphospace. The key is that, for evo-devo, the space of possible phenotypes is *developmentally* constructed. Raup's original 'cube' (1966)—Dawkins' inspiration for his Museum—indeed represented a limited morphospace of possible shells, intending to capture the idea that only certain forms are producible through development. In this space, large areas are completely empty, corresponding to the lack of species showing specific morphological patterns (Figure 2.7). The particularity of Raup's proposal was that the possible forms were not an extrapolation of extant phenotypes—less of extant genotypes—but the result of a *generative rule* simulating different ways in which a shell shape can *develop*.

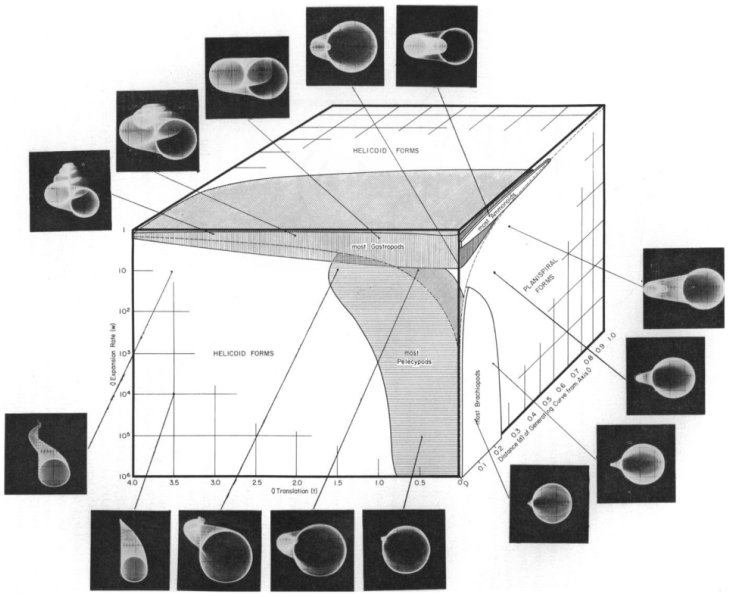

Figure 2.7. A developmental morphospace: Raup's cube. Reproduced with permission from Raup (1966, p. 1184).

In this regard, evo-devo explains the fact the the morphospace is irregularly occupied (Nuño de la Rosa 2014), that is, that forms are not randomly generated but clustered in certain regions of the space of possible phenotypes—only a limited number of body plans exist, of which only a limited number of variants is possible. It explains so in terms of what can be generated and what is more likely to be preserved and to undergo transformations, with independence of the adaptive character of the resultant phenotypes. In turn, we may identify the evo-devo spaces of the possible as

developmental morphospaces (cf. Eble 2003). In these spaces, fitness values only have an *a posteriori* relevance: once a phenotype is originated through changes in development, natural selection can act on it and modulate its presence. By contrast, those fitness values do not serve for conceiving the possible, which is primarily grounded on developmental properties.

b) Variational probabilities

The vindication of a causal, creative role for development in evolution entails a rupture of the proximate-ultimate causes distinction as introduced by Mayr (1963). In Mayr's influential view, development is the proximate cause of phenotypic construction: the ontogenetic building up of particular phenotypes, and as such is widely considered to pertain to the domain of *how* rather than *why* questions (see the introduction to this thesis). However, from the perspective here defended development can help solve why-questions—that is, evolutionary questions—inasmuch as it reveals itself as the main cause of phenotypic transformation: developmental properties are responsible for possible phenotypic changes. For the purposes of this thesis, it is worth wondering how the developmental generation of variants in evolution can result in a biologically meaningful developmental notion of chance. That is, whether the fact that development is responsible for a non-random space of phenotypic possibilities can bring about a non-statistical conception of chance in the generation of variation. More particularly, we must consider if development provides any "source laws" for patterns of variation, analogously to how ecological properties ground the source laws of the probabilistic models of evolutionary genetics, thus enabling a causal understanding of chance at the level of variation.

Much of the evo-devo research has been devoted to depicting developmental mechanisms responsible for particular evolutionary changes (Brigandt 2015a, Baedke 2020), such as changes in the patterns of gene expression and cell proliferation that underpinned the fin-to-limb transition (Wagner 2014, Arthur 2011). A consideration of particular developmental mechanisms into evolution in this way can be seen as the redemption of the "causal completeness" criticism made by developmentalists

(Amundson 2005). According to Amundson, one of the main problems of the M.S. perceived by developmental biologists was the lack of a complete causal picture relating one phenotype to another:

> In order to achieve a modification in adult form, evolution must modify the embryological processes responsible for that form. Therefore an understanding of evolution requires an understanding of development. (Amundson 2005, p. 176)

There is a severe limitation, nonetheless, in considering particular developmental mechanisms as proximate causes of evolution. Although they serve for causally depicting particular evolutionary transformations, their general properties are harder to grasp. In his reconstruction of the causal components of evolutionary models through causal graph theory, Jun Otsuka (2014) expresses this limitation in this way:

> phenotypic organizations obviously differ from organism to organism in such a way that it is meaningless to speak of the phenotypic causal structure. We thus need to empirically build a model case-by-case. (Otsuka 2014, p. 64)

According to this author, the complexity of developmental interactions can be introduced in the mathematical structure of evolutionary models, but only with the cost of turning evolution "a local process" (Otsuka 2015, p. 31). Despite its prominent role in contemporary evolutionary biology, the mechanistic view of development embedded in this evo-devo approach is only relevant from a causalist point of view for the explanation of particular evolutionary changes, rather than as a component of the probabilistic nature of evolutionary explanations. As a matter of fact, probabilities are not invoked in the mechanistic approach of evo-devo, which is interested in actual evolutionary changes and how they took place, such as the origin of particular phenotypic novelties or the transformation of certain developmental process, rather than in possible changes and their probabilities. In this regard, philosopher Brett Calcott speaks of "lineage explanations" in evo-devo, namely explanations concerning the details of a specific developmental mechanism "and how it changed over time" in evolution (Calcott 2009, p. 52). The particularities of developmental mechanisms indeed connect the genotypic space to the phenotypic

one in a generative manner. However, they do not allow for constructing a sample space of possible phenotypic changes, and thus are futile for the purpose of considering the general *variational tendencies* (cf. Wagner 2014) that explain patterns of phenotypic variation with independence of selection. Just like a mechanistic understanding of ecological interactions may serve the purposes of ecology but not of general tendencies in the models of population dynamics, a mechanistic understanding of development serves the purposes of comparative evo-devo but not of the general variational tendencies of developmental evolution. Analogously to how possibilities are understood in sampling, then, one needs to consider the general dispositions structuring the possible in evolution from a developmental point of view.

Philosophers have indeed pointed to this limitation of a mechanistic understanding of evo-devo (Brigandt 2015a, Austin & Nuño de la Rosa 2018), and in turn have considered that it is the general dispositions of developmental systems that play an important explanatory role in accounting for possible evolutionary changes. Within the view of evo-devo, then, it is the *capacities* of development to generate organismal form that matter for explaining evolutionary change. This consideration illuminates the significance of evo-devo for the problem posited in this section, namely the establishment of a space of phenotypic possibilities that is logically previous to and independent from the space of possible adaptations and changes in gene frequencies in a population. In other words, if we want to establish a sample space of possible phenotypes that may be produced through reproduction, we need to incorporate the dispositions of development to produce phenotypic variation in reproduction. In regarding this space of possible changes, the probabilities of different phenotypic variations could be incorporated to evolutionary explanations.

With this purpose in mind, it becomes necessary to distinguish between extant variation and variational possibilities. That is, it is important to consider variation beyond extant variants and differences among them—as regarded in the M.S. population thinking—, and to contemplate what is possible from the point of view of development—e.g. 'which phenotypic changes are possible given certain properties of development?'. In particular, considering variability as a distinct

phenomenon is key for assessing the general directionality that development imposes to the process of evolution:

> it is essential to clearly distinguish between "variation" and "variability," even though these words are often used synonymously in the literature. The term variation refers to the actually present differences among the individuals in a population or a sample, or between the species in a clade. Variation can be directly observed as a property of a collection of items. In contrast, variability is a term that describes the potential or the propensity to vary. Variability thus belongs to the group of 'dispositional' concepts (Wagner & Altenberg 1996, p. 969)

In this quote, Günter Wagner and Lee Altenberg stress the conceptual distinction between extant variation and the potential to vary. The pretension of variability is to conceptualize possible variations as based on the developmental causes of phenotypes, specially regarding their dispositions and with independence of adaptive possibilities—as both logically independent and temporarily previous to them. Thus variability has been defined as a tendency to generate differences or, importantly, as the "propensity to vary", and it refers to the process that generates variation rather than to any set of extant variants (see also Hallgrímsson & Hall 2005). Considering variability in its own sake, as a capacity of organisms, rather than as "what is absent from an idealized distribution" (Wagner & Draghi 2010, p. 395), can provide a causal understanding of chance at the level of variation.

In turn, and considering the concerns of this thesis, variational tendencies can be regarded as those general capacities of development that structure a sample space of possible phenotypic changes. As we saw in Chapter 1, dispositions involved in probabilistic modeling are better understood as causal propensities, in the sense that they manifest themselves in patterns susceptible of having a probability measure. Thus variational tendencies can be pictured as structuring causes of a space of phenotypic possibilities in reproduction, responsible in turn for the variation that will take part in the sampling process of the next generation. Let us briefly see what this entails in terms of our urn analogy. Natural selection and drift are analogous to hands sampling those balls that will generate in turn the next urn for the next

sampling process. These hands can merely reinsert the balls in the second urn—classical population thinking—, or they can multiply them, recombine them and allow mutational changes in them. I mentioned previously (section 1.2 a) that this second option has not been incorporated into the analogy, and I explored in section 3.2 how it could be considered from the point of view of the creativity of selection. In a nutshell, the creativity of selection entailed that the possible new variants—new ball colors—arising in the generation of a new urn were aligned in the color range of selection. However, as we saw, this was merely a supposition of the adaptationist framework. From the point of view of evo-devo variational possibilities, the possible changes in color of the balls in the urn will be structured by how balls themselves are generated. Thus we are interested in the probabilistic tendencies of the process of *reinsertion* and *multiplication* of the balls into the second urn as responsible for the construction of balls in the first place. Let us imagine that, rather than reinserting balls, a different hand had to build up new balls which are similar to the former ones. Instead of providing merely 'random deviations', this process will only produce those deviations that are possible considering how the construction itself works. For instance, if balls are first assembled and then painted, it is likely that changes in color will be uniform in the surface of balls. If, on the contrary, they are first painted and then assembled, it is possible that multicolor balls arise. In turn, we want to deal with how the construction process determines a sample space of possible ball colors upon which selection and drift can later act. This relates to the typological character of variation received from the morphological tradition in this way: how do certain *types* of balls tend to vary by contrast to others, as based on the way in which they are constructed? For instance, we could talk about the type of balls that are potentially multicolor.

In his recent book, Günter Wagner has referred to the typological view embedded in the evo-devo research agenda as "variational structuralism", which makes reference to the general "variational tendencies" of developmental systems to produce phenotypic changes (Wagner 2014). In his words, "the structuralist intuition" is that "complex systems … play a causal role in determining their evolutionary fate" (Wagner 2014 p. 18), in the sense that their internal

properties—such as the way they develop—influence how they can change. For instance, some developmental systems will be more prone to changes than others, while some will produce more discontinuous changes than others (Salazar-Ciudad 2007). In turn, developmental systems are structured in distinct "pathways" guiding possible phenotypic changes. With these tendencies in mind, we can talk about the 'variational probabilities' of a certain developmental system determining the chances of phenotypic changes (Nuño de la Rosa & Villegas 2019). We can define variational probabilities in this way:

> Variational probabilities are *the probabilities of generating different types of variations from a generation to another in the process of reproduction.*

Variational probabilities determine a space of possible variants that may arise, and only those actually arising will have a probability of being sampled by selection or drift. When referring to variational probabilities, we can regard reproduction as the trial of developmental chance setups, whose possible outcomes are phenotypic changes. Mutations will play an important role in these trials but, significantly, their properties do not structure the space of possible variants, but developmental properties do. Importantly, the variational properties of evo-devo concern the probability of generation of phenotypic variants, independently of their ecological role and fixation in a population.

Although some recent studies consider that evo-devo has the potential to provide probabilistic predictions of phenotypic variation (e.g. Jaeger et al. 2015), we shall see in the next chapter that the establishment of probabilistic models for evo-devo variation is a complex task far from being completely achieved. However, the probabilistic means of explanation embedded in evo-devo studies of variational tendencies enable us to talk about the "propensities of variation" (Stoltzfus 2006, p. 310) more generally, as well as their capacity to explain why "not all variants in a population arise with equal probabilities" (Müller 2020, p. 4). The developmental view of variation presented here is motivated by the will to assess chance in the generation of the sample space of evolution—in the construction of the variants that will be sampled discriminately or indiscriminately—, which is distinct from chance as

understood in sampling but also, and importantly, distinct from the negative, statistical notions of chance in variation reviewed in section 3.1 of this chapter. Particularly, it is a vindication of the causes of the generation of phenotypes as causes structuring what is possible in variation, as opposed to their classical understanding as factors merely limiting the possible for selection. In sum, through variational probabilities we can finally explore the causal bases of chance at the level of variation. To this we shall turn to in the next and final chapter of this thesis.

5. Concluding remarks

In this chapter, I have presented the main philosophical ideas regarding probability and chance in evolutionary biology, especially with regards to a potential causal understanding of variation in evolution. In the first section, I have reviewed the Darwinian notion of chance variation, as well as its implementation in contemporary models of evolutionary genetics. In doing so, I have argued that the 'population thinking' ingrained in Darwin's conception and in mainstream philosophical views of evolution has enabled the canalization of ideas coming from the world of physics, in turn precluding the consideration of internal factors in evolutionary changes and stimulating an externalist picture where passiveness against external forces are all there is to explanation. This has served not only for pointing at the contingent, historical origin of the Darwinian notion of chance variation and for stressing the adaptationist flavor embedded in modern evolutionary models. In addition, it has been instrumental in characterizing evolutionary probabilities as understood in these models, and in introducing some important ideas about them. Among these ideas, the understanding of ecological probabilities as sampling processes; the relation between causes or 'source laws' and evolutionary models; and the independence of evolutionary probabilities from indeterminism stand out.

In the second section, I have argued for a causal propensity view of chance as sampling, particularly with regards to how ecological propensities ground the probabilities found in evolutionary models of population dynamics. I have shown that, contrary to what the statisticalist position defends, these models are explanatory

insofar as they relate to ecological factors through pragmatic, theoretical and experimental considerations. Moreover, we have seen that statisticalism is flawed by virtue of its rigid, unjustified consideration of causation in evolution. A broader notion of causal explanation, as presented in Chapter 1, facilitates regarding the causes of evolution in a more inclusive way. From a causalist position, I have then argued for the strong relationship that the classical propensity interpretation of fitness has with a causal understanding of higher-level properties in evolution, in turn advocating for a causal propensity approach to ecological components as responsible for possible populational changes. This conception of the possible grounds the construction of evolutionary probabilistic models with regards to population dynamics, therefore enabling a causal propensity view of the notion of chance as sampling.

In the third section of this chapter, I have argued that the probabilistic models of evolution don't allow for an analogous understanding of chance at the level of how variation—i.e. the sample space of chance as a sampling process—is generated. I have shown that, in contrast to the consideration of causal ecological factors in deriving these models, no examination of the processes underlying the generation of variation is present in them. Instead, the treatment of variation is merely statistical, the prevalent notions of chance at this level being the independence of mutations with regards to adaptive needs and the randomness of phenotypic changes. As we have seen, this situation is the result of the adaptationist view endorsed by advocates of the M.S., which resulted in a picture where selection acts as a creative force even for the generation of variation. In exposing this framework, I have been critical with this uneven condition for the sampling and the generation of variants, stressing that the creative view of natural selection is based on assumptions about the nature of variation rather than on causal knowledge about its generation.

In the last section of this chapter, I have shown evo-devo's criticism with this adaptationist program and its view of the possible, consequently arguing for a developmentally grounded notion of chance in variation. Through the characterization of the historical background of evo-devo vindications, we have seen

that a renaissance of typological conceptions of the possible is embedded in them. My position has been that it is in this revival that a convenient sample space of variation in evolution can be established. I have then reviewed the main limitations of classical spaces of evolutionary possibilities, particularly with regards to their neglect of development in bridging genotypic and phenotypic variation. I have also defended that development plays a causal role in determining evolutionary variation beyond the received view of developmental constraints. Understanding variability, namely the potential for variation, as a developmental property distinct from extant variation, therefore challenging 'population thinking', is key to this position. In turn, the causal structure of development, in its capacity for building up phenotypes, can provide grounds for conceptualizing the possible in a causal way, therefore allowing for the construction of probabilistic models of variation. The notion of variational probabilities as introduced in this last section intends to capture this idea.

CHAPTER 3

Developmental Propensities: Understanding Evo-devo's Variational Probabilities

This is the problem not of genetic stability but of developmental stability—of the conspicuous robustness of developmental processes and their capacity to stay on track despite inevitable environmental, cellular, and even genetic vicissitudes.

Evelyn Fox Keller

0. Introduction

The complexity ingrained in conceptualizing chance in evolution and the different roles it plays in evolutionary explanations have revealed themselves in the preceding chapters. Let us remind that the central task of this dissertation is to explore chance in evolution from a causal probability and propensity view, especially with regards to the generation of evolutionary variation. While in Chapter 1 I developed a philosophical framework for approaching this issue, Chapter 2 was devoted to identifying the notions of chance that can be meaningfully assessed from this framework, both in their historical perspective and in contemporary models of evolution. In doing this, I have argued that the models of population dynamics only

enable a causal understanding of chance at the level of ecological interactions, while the causal grounds for chance at the level of how variation is generated are better situated in evo-devo models of variation. This final chapter develops this latter idea, in turn concerning the proposal of a *developmentally grounded conception of chance* in evolutionary variation, as based on the results obtained in the previous chapters. More specifically, we saw that the probabilistic models of evolutionary genetics allowed for a conception of natural selection and drift as sampling processes based on ecological causes: the probabilities of being sampled for being parents of the next generation are grounded in the ecological propensities of individuals, populations and types. Analogously, we shall now explore how evo-devo models facilitate a consideration of variational probabilities—the distinct probabilities of generating different sample spaces from which parents may be sampled—as based on developmental propensities. In turn, the present task is to deepen into how evo-devo studies, in their consideration of development as a fundamental causal component of evolutionary transformation, can influence our notions of chance in the generation of a sample space for evolution (cf. previous chapter).

If the classical philosophical conception of evolution was constructed, as we have seen, out of the neo-Darwinian worldview of the M.S., the incursion of development into our understanding of chance in evolutionary variation can be perceived as pushing the limits of this classical picture. Not only the idea of 'chance variation' as the raw material of evolution seems to be challenged by this endeavour. In addition, we shall see that the conceptual pillars of population thinking, the separation between ultimate and proximate causes, and externalism, also demand a reconfiguration. Indeed, the ongoing discussion about extending the theoretical structure of evolutionary biology beyond the limits of evolutionary genetics entails a challenge to these very philosophical conceptions (Amundson, 2005, Pigliucci & Müller 2010). While the consensus around these issues is currently far from reach, it is worth noting the potential consequences that can be drawn from the discussion of chance from an evo-devo perspective. On the one hand, introducing developmental causes into evolutionary explanations seems to entail, as we have seen, a rupture of the proximate/ultimate dichotomy in biological thinking. But the precise meaning of

this remains relatively unexplored when it comes to analyse variational evolutionary tendencies of developmental systems, rather than particular developmental mechanisms. Considering the probabilistic potential of evo-devo models for evolutionary variation could help in opening up new philosophical discussions about what kind of causation is involved in phenotypic evolution. On the other hand, the typological thinking ingrained in evo-devo's approach to evolutionary variation, as we have seen, exceeds the boundaries of population dynamics and population thinking. Reconciling the typological approach of evo-devo with the evolutionary consensus and contemporary standards on causation is a philosophical task that is currently being explored in some depth (Amundson 2005, Brigandt 2007, Love 2009, Lewens 2009, Wagner 2014). This matter too can benefit from a consideration of how development can ground our notion of chance in evolutionary variation, inasmuch as types can play a causal role in our conceptions of the phenotypically possible. Finally, the integration of developmental causes in evolution directly opposes the externalist, Newtonian-like picture of evolution inherited from the M.S. To what extent the existence of internal properties affecting the directionality of the process of evolution challenges any current consensus is a largely developed and unsolved debate—as we saw in the previous chapter with regards to 'chance mutations' (section 3). However, I believe it is worth wondering how this 'chance role' (cf. Chapter 1) played by development can affect other notions of chance directly related to the internalist/externalist opposition, such as the role of necessity and contingency in evolution.

Interestingly, evo-devo models of variation make use of several dispositional notions that refer to the phenotypic potential of developmental systems. We saw in the previous chapter that variability, in referring to possible phenotypic variations, is one of such notions. Others include modularity, robustness, plasticity and evolvability. The dispositionality ingrained in evo-devo has been perceived by philosophers as a manifestation of its interest in the phenotypically possible (Austin & Nuño de la Rosa 2018). The question now is, in what sense can these dispositions generate a sample space for evolution? The notion of variational probabilities is key for conceptualizing the generation of this sample space. As we saw in the previous

chapter, building up this sample space requires that we consider the causes underlying the production of phenotypic variants, namely the "raw material" of evolution. In our analogy of the urn, we saw that how balls are constructed is relevant from the point of view of which variations are possible in evolution. Importantly, the hand constructing the balls is not the same hand that picks them up: the building up of new balls is independent from the sampling process. In other words, developmental properties are largely independent from the local selective environment. Regardless the diversity ingrained in evo-devo research, models of developmental variation reflect precisely this idea: how organisms develop influences the way in which they can provide phenotypic variation, an issue that is prior and relatively independent from the local selective environment. One of my aims here is to show how variational probabilities are instantiated in these models. In achieving this, the problem of variation as depicted in the previous chapter turns into the problem of how development structures a sample space of possible variations. Far from being trivial, the position here defended is that this notion casts some light on the above considered current philosophical concerns regarding explanation in evolutionary biology.

In this chapter, I unfold the developmentally grounded notion of chance in evolutionary variation and consider the philosophical consequences that may follow from it. The chapter is divided as follows. In Section 1, I present evo-devo models of phenotypic variation and I argue that they entail a causal understanding of the developmental relation between genetic and phenotypic variation. There I show that in abstracting the causal structure relating genotypes and phenotypes, developmental encoding enables the statement of a space of phenotypic possibilities that in turn can ground variational probabilities. Section 2 deepens into the general properties of development grounding phenotypic patterns of variation. There I review the variability, robustness, modularity and plasticity of genotype-phenotype relations, arguing for their presence in most developmental systems and for their dispositional nature. In Section 3, I apply the causal propensity framework developed in Chapter 1 of this thesis to the variational dispositions of genotype-phenotype maps. In doing this, I argue that developmental types or characters are idealized chance setups

responsible for structuring a space of possible phenotypic changes. This enables me to distinguish a vernacular, an expected and a realized sense of variational chance analogous to the distinctions found in the philosophical literature about fitness reviewed in section 2 of the previous chapter. Finally, in Section 4 I briefly explore some connections of this developmental notion of chance with the general explanatory structure of evolutionary biology. In doing this, I relate the present approach of developmental propensities to one of the most salient recent notions in evolutionary biology: evolvability. I conclude with some general reflections of the consequences of this approach for the foundational pillars of the classical philosophical picture of evolution, notably the consideration of types and the understanding of development as an ultimate, rather than a proximate, evolutionary cause.

1. Genotype-Phenotype Maps

Once established, as we have seen in the previous chapter, that it makes sense to talk about variational probabilities, the way to proceed for providing a causal propensity understanding of them is to explore developmental models of variation. Recall that such an exercise differs from identifying variational probability measures—in the sense of mathematically well-defined—as propensities. Rather, as we saw in Chapter 1, this endeavour entails the recognition of those causes that structure the relevant sample space of possibilities. As a consequence, the present task demands that we consider an adequate space of evolutionary possibilities and the causes accounting for it. As we have seen, neither the genotypic or phenotypic space—nor the alleged 1-to-1 correlation between them of the M.S.—provide good starting points for studying patterns of variation due to the neglected complexity of the properties of development relating the two. On the other hand, developmental morphospaces, such as Raup's cube (Raup 1966, see previous chapter), enable us to conceptualize phenotypic possibilities in terms of developmental accessibility: the possibility of a phenotypic transformation is determined by its developmental viability. This considered, the way to explore variability in a developmental morphospace is to

study patterns of genotype-phenotype relations. The vindications made in the field of evo-devo with regards to the inclusion of development derived into the conceptual tool of the genotype-phenotype map, an abstraction of developmental pathways that assigns sets of phenotypes to sets of genotypes (Alberch 1991). As we shall see, this tool is used in a number of ways and for a variety of purposes, inside and outside the field of evo-devo. Besides, it constitutes the abstract representation of those properties that are relevant for developmental patterns of variation, and thus for constructing developmental morphospaces, in the senses introduced in the previous chapter.

A remarkable disappointment in the recent history of biology was the realization that some of the promises of genomic sequencing cannot be fulfilled (Keller 2002, Pigliucci 2010). Despite the unquestionable success in providing highly valuable comparative and statistical data, the capacity for such data to explain how phenotypes are built is virtually nonexistent: knowing genetic sequences does not improve our understanding of phenotypes. This is so because phenotypes are not a simple function of genotypes; instead, their relation constitutes what is known as "a nonlinear mapping problem" (Kell 2002). As we have seen in Chapter 2, the effect of genes is context-dependent to the extent that it makes no sense to attribute phenotypic properties to them unless enough of their genetic and developmental environment is specified (Gilbert 2000). A G-P map serves as a means for overcoming this difficulty ingrained in the gene-centrism of mainstream evolutionary biology, since it represents an abstraction of how *genotypic differences* give rise to *phenotypic differences*.

The basic idea of a G-P map is that we can define a mapping function from specific genotypic variants to specific phenotypes (Figure 3.1).

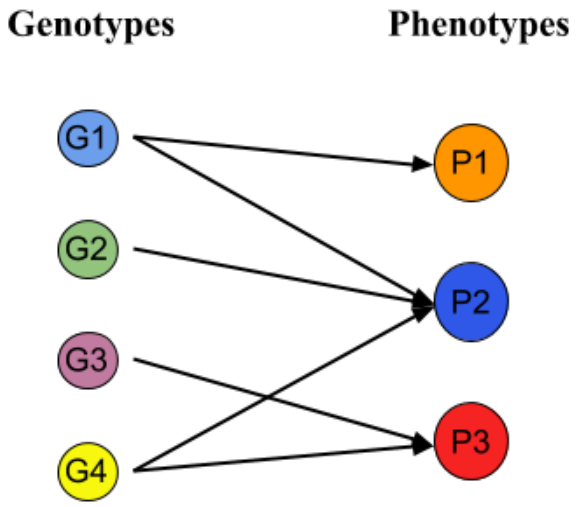

Figure 3.1. A G-P map function where four different genotypes (G1, G2, G3 and G4) map to three different phenotypes (P1, P2 and P3).

The parameters defining such a function—which in some simplified cases can be mathematized—represent developmental pathways that remain constant in a variety of developmental systems and environmental contexts but whose values depend on variable genotypic states or inputs. Thus different genetic variants in the domain of the function will map into a different point in the parameter space. For example, a G-P map can take as parameters the folding, transcription and translation rules that relate different genetic sequences to the proteins they code for, establishing a mapping relationship that will be shared by a large subset of living organisms. The mapping relation enables the building up of a parameter space of possible proteins, and each considered genetic sequence will map into a point in this space. Thus the same mapping function assigns a protein—a phenotype—to each possible genetic sequence in its domain. Another G-P map can relate human genotypes to eye color, taking as parameters the developmental rules building the human eye; and yet another G-P map can represent the relationship between those same genotypes and height or handedness type. Therefore, a G-P map does not necessarily take an entire genome and its phenotype, but rather establishes a relationship between genetic and phenotypic variation at any defined level, and in turn "it specifies which *genetic differences* give rise to which *phenotypic differences*" in a given context (Wagner & Mezey 2004, p. 341, stress added). As we shall see throughout this chapter, the

structure of a G-P map captures, in addition, general properties of how phenotypes change under genetic perturbations, such as how robust or plastic changes are, or whether they take place in an integrated or modular way.

It is important to note that the phenotype under interest can be defined at many different levels, which enables the development of a G-P map for homologous traits shared across species or even higher taxa. Thus the patterns of morphological and developmental unity under interest for the morphological tradition (see section 4 of the previous chapter) can be explored through developmental patterns with G-P maps. This core tool serves several purposes in different fields. For instance, one can generate a G-P map from statistical data of quantitative genetic analyses, or from comparative or experimental developmental genetic studies. Similarly, the G-P map can serve as a means for studying statistical properties of development that may be of interest for understanding variational patterns, as well as they can be a good ally for discovering interesting causal paths in development. Observational data, interventions and computational approaches are the bases of the causal hypotheses modeled in G-P maps. This variety of usages is a symptom of its versatility and scope: managing a mathematical abstraction of developmental properties is instrumental in the study of patterns of variation in a general sense. Although they have been proven useful for scrutinizing patterns of extant variation (see Hansen 2008) and specific developmental mechanisms responsible for them, G-P maps have also been very influential—as we shall see—in the conceptualization of general variational tendencies as based in developmental properties. For instance, a G-P map will typically contain different phenotypes that the system can develop into—such as brown, blue and green for eye color—and will typically represent many-to-many correlations, which contrasts with the 1-to-1 correlations between genotypes and phenotypes assumed in the very simple models of genotypic and phenotypic spaces of the M.S.

The concept of a G-P map was introduced in evo-devo by Pere Alberch in 1991. Alberch argued that any phenotype can be understood as the product of spatial and temporal developmental interactions whose regulation depends on what he calls

"morphogenetic parameters" (p. 7), namely genetically controlled properties of the developmental system. In his words:

> Morphological diversity is generated by perturbations (regulation) in parameter values -- such as rates of diffusion, cell adhesion, etc. -- or initial conditions. The structure of the interactions among the components, however, remains constant. Given this assumption even if the parameters of the system are randomly perturbed, by either genetic mutation, or experimental manipulation during development, the system will generate a limited and discrete subset of phenotypes. (Alberch 1991, p. 7)

Thus the idea underlying this representation of the genotype-phenotype relationship is that it models the possible changes a developmental system can undergo through the establishment of a developmental parameter space. This will, in turn, facilitate the construction of a developmental morphospace. Of course, since the parameter space itself depends greatly on genetic properties, it is prone to changes. However, it is useful for modeling the possible effects of those mutational perturbations that do not affect the parameter space itself. Specifying the right parameter space is thus crucial for the evolutionary significance of G-P maps.

Many of the research goals of the diverse field of evo-devo that we have revised in the previous chapter make use of the G-P map. As a consequence, its usages inside the field are heterogeneous. In this section I review some paradigmatic models of G-P mapping in evo-devo (section 1.1); then I develop the idea that these maps represent a 'developmental encoding' approach to evolutionary possibilities (section 1.2); and finally I present two minimal models of a G-P map coming from molecular evolution that serve as models for evo-devo variational probabilities: the RNA and the minimal cell models (section 1.3). This will enable me to approach the variational properties of G-P maps from a dispositional point of view and to develop a causal propensity understanding of them in the sections that will follow.

1.1. Evo-devo models of G-P maps

Regardless of its instrumental role in the population dynamics view of evolution, the statistical treatment of evolutionary genetics is not satisfactory for the evo-devo

research agenda. In this context, the model that Alberch introduced was a vindication of the evo-devo goal of explaining patterns of phenotypic variation in terms of the properties of development. Because of its historical relevance and its connection with current evo-devo models, it is worth reproducing the main features of this model here.

Before introducing the G-P map in 1991, Alberch had worked with Emily Gale in the study of the phenotypic variation obtained under developmental perturbations. Particularly, they studied phenotypic variants that resulted from the same perturbation in the development of a homologous trait—digit number—in different amphibian species in order to compare the induced variation with extant phenotypic diversity among the two clades of limbed amphibians (Alberch & Gale 1985). Their goal was to experimentally explore the existence of "congruent patterns between phylogenetic diversity and the 'potentiality' of a given developmental system" in the formation of amphibian digits (Alberch & Gale 1985, p. 8). In other words, they studied the similarities between inter-specific variation and the potential of the developmental system for intraspecific variation. For doing this, the authors compared variation in the number of digits of several species of frogs and salamanders, the two orders of amphibians with limbs.[43] Most frog species—*Anura* order—have five digits in their hind limb, except for two species that have lost the first digit and thus have only four—numbers 2, 3, 4 and 5. Importantly, these species are not monophyletic, meaning that the digit loss has occurred independently in the evolution of both species, that is, the same change has occurred twice. On the other hand, most salamander species—*Urodela* order—also have five digits in their hind limb, but species with only four digits are not uncommon in this order, where digit loss has evolved independently several times. In those salamander species with four digits, either digit 4 or 5 has been lost, thus the conserved ones being numbers 1, 2, 3 and "4-5". The authors then compared the ontogenetic process of digit formation in the two taxa. They observed that frogs differentiate the central digits first (3 and 4), followed by the differentiation of digits 2 and 5, and finally—in those species with

[43] Their comparisons also included patterns in the number of phalanges in each digit, their level of cartilaginousity and the loss of skeletal elements, but I omit these factors for the sake of simplicity.

five digits—with the differentiation of digit 1. By contrast, salamanders develop digit 1 first, and then differentiate the subsequent digits one after the other.

In the experimental phase, Alberch and Gale perturbed the development of the limb bud—the structure in early development of tetrapods that precedes limb formation—in several species of five-digit frogs and salamanders through the injection of the mitotic inhibitor colchicine. This caused a temporal lack of proliferation of the cells in the limb bud, which resulted in the limb developing with fewer cells than usual. The results showed not only smaller limbs but also specific patterns of phalange and digit absence that differed between the two orders but were similar among the species belonging to each order. Specifically, they found that phalange reductions—and sometimes complete digit loss—in frogs primarily took place in digit 1, whereas they took place in digits 4 and 5 in salamanders. In combination, their results showed that *the same developmental perturbation*, namely reduction in limb bud cell number, had different effects in accordance with the developmental pattern of the phenotype, i.e. reductions in digit 1 for frogs and in digits 4-5 for salamanders. Moreover, they suggested an evolutionary significance of such developmental bias, insofar as the perturbed limbs resembled the phenotypes of species with digit loss within their order (Alberch & Gale 1985). In other words, in their experiments, a perturbation in ontogeny of the limb bud primarily affected the formation of the last digit that differentiates in development. Since frogs and salamanders have different patterns of digit differentiation, perturbations will typically affect digit 1 in frogs and digit 4-5 in salamanders. These are precisely the digits that are absent in those wild frog and salamander species with only four digits, suggesting that the genetic change that originated such evolutionary transitions has been facilitated by the developmental pattern. That is to say, the congruence between experimentally induced variation and phylogenetic patterns indicates that extant variation in digit number among the species of the same taxa may be the result of genetic changes affecting the same developmental pathways. The authors reason that a variety of different developmental parameters, such as migratory rates or cell proliferation, can reduce the number of cells in the limb bud and thus produce a reduction of it in development. Since many different mutations can bring about

changes in those parameters, the probability that a genotypic change in the development of the limb specifically reduces digit 1 in frogs and digit 4-5 in salamanders is high. This probability is relatively independent of the probabilities of different mutations arising, since it is not the nature of the mutation itself that matters. On the contrary, the rules governing the development of digits make it likely that *any* mutation affecting these rules will result in the same phenotypic change.

In his introduction of the G-P map, Alberch (1991) proposed that developmental processes such as this can be seen as pattern-generating systems with an associated parameter space. The systems have specific "form-generating potentialities" and robustness (p. 10), and thus will map genetic variation into phenotypic variation in specific ways. Therefore the developmental rules of digit differentiation in amphibians can establish a parameter space with different possible digit numbers as phenotypes, five digits being the most frequent phenotype. In his characterization, the parameter space includes neutral areas where every genotypic combination will map into the same phenotype. For example, if a population is in the neutral area that maps to 'five digits', small genetic variations in it can leave the number of digits unchanged. In addition, the parameter space will include so-called "transformational boundaries" (p. 8) demarcating those genotypic surfaces where small differences in genotype result in different phenotypes. In Figure 3.2a, areas *A* to *F* are neutral spaces each corresponding to a particular phenotype, while the lines demarcating them are transformational boundaries among them. Now, if *D* represents the pentadactyl phenotype, most species of frogs and salamanders will occupy a certain area inside *D* in the parameter space. However, frogs and salamanders will be nearby different transformational boundaries, insofar as they are more likely to encounter different phenotypes under perturbation—digits 2, 3, 4 and 5 in frogs; digits 1, 2, 3, 4-5 in salamanders. Thus, depending on the position in the parameter space—that is, depending on the pattern-generation process—, a species will have different potentialities for change. Therefore, they will map genotypic to phenotypic variation differently (Alberch 1991).

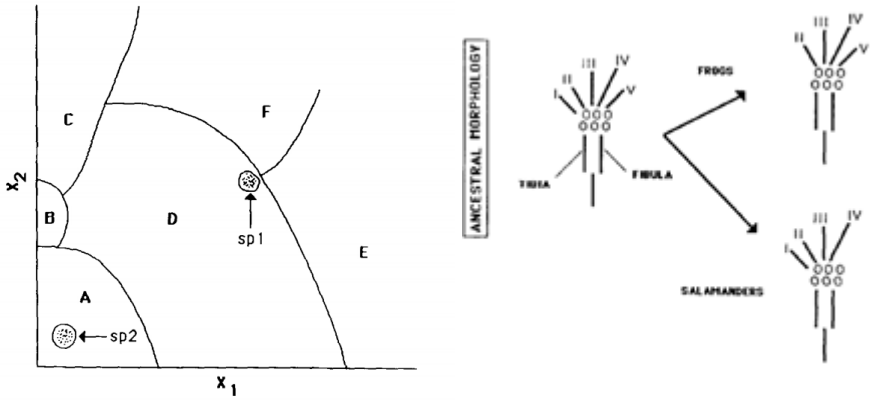

Figure 3.2. Left: Representation of two species in the genotypic space. Species 1 (sp 1) is located in phenotype D, very close to transformational boundaries leading to phenotypes F and E, respectively. Species 2 (sp 2) is located in phenotype A and relatively far from any other phenotype. **Right:** Digit loss experimental transformations of frogs and salamanders. Reproduced with permission from Alberch (1991).

The G-P map of the pentadactyl character or type—the five digits in tetrapod limbs—has been a very prolific area of study in evo-devo. Since tetrapods have typically and at most five digits in their limbs, an interesting research inquiry in developmental studies of evolution has been the digit identity across species and taxa of tetrapods, that is, the study of homologies within the type, and the developmental constraints that might be associated to it. A phenomenon under study is that even if most tetrapod limbs develop five digits, the way they map genotypic to phenotypic variation may differ by virtue of the available developmental pathways. A prominent model of the developmental evolution of tetrapod digits has been developed by Günter Wagner and his collaborators (Wagner & Gauthier 1999, Wagner 2005, Kohlsdorf & Wagner 2006, Wagner 2014, Stewart et al. 2019). Some of the questions they have addressed are how likely it is to induce digit loss as well as the the re-evolution of a lost digit in different amphibian species (Kohlsdorf & Wagner 2006, Stopper & Wagner 2007, Stopper et al. 2016, Wagner et al. 2018), and what are the homologous identities of the three digits present in the avian wing (Wagner & Gauthier 1999, Wagner 2005, Young et al. 2011, Stewart et al. 2019). The general approach is to study both how genetic variation maps into phenotypic variation in specific cases and how developmental perturbations affect the generation of variants,

and to evaluate the evolutionary significance of the developmental biases found. That is, they combine statistical methods with interventionism (cf. Woodward 2003) and theoretical significance.

Another salient example of the developmental approach to phenotypic variation can be found in the work of Isaac Salazar-Ciudad and collaborators (Salazar-Ciudad & Jernvall 2002, 2010; Salazar-Ciudad 2012; Marin-Riera et al. 2018), who have produced a model of developmental evolution for teeth in mammals. Dental shape—specifically the size, morphology and position of teeth cusps—is very variable among mammals and is taxa-specific (Salazar-Ciudad & Jernvall 2010). Salazar-Ciudad and colleagues have developed a computational model of developmental variation for dental shape and, complementarily, they have experimented with *in vitro* dental tissue growth. In their computational model, they have implemented "parameters of genetic and cellular interactions" that produce three dimensional teeth from a simple precursor (Salazar-Ciudad & Jernvall 2010, p. 1). In such model, they carried out perturbations as inputs and derived different morphologies as outputs, thus generating a mapping relationship between variation in initial conditions and phenotypic variation. Experimentally, they have produced different tooth morphologies *in vitro*, producing similar patterns of variation than the ones found in nature and in their simulations (Salazar-Ciudad 2012). Both in their computational model and experiments, the effect of mutations highly depends on which developmental parameters are affected by them. For instance, in their models, different perturbations of the same given parameter lead to similar tooth morphologies, while similar perturbations derive in very different tooth morphologies depending on the developmental parameter they alter (Salazar-Ciudad & Jernvall 2002).

Interestingly, the G-P maps of some complex traits that have traditionally been the focus of attention in quantitative genetics are increasingly being approached as well through a developmental perspective. For instance, in order to gain an understanding of evolutionary patterns of variation in the wing of the model organism the fruit fly *Drosophila*, the study of the phenotypic effects of developmental perturbations has recently been incorporated, in particular with

regards to the mechanisms responsible for wing shape and vein formation (Ray et al. 2015; Matamoro-Vidal et al. 2015). Patterns of wing pigmentation in butterflies are also being explored in this way (Nijhout 2001, 2017), as well as highly complex traits such as cranial integration (Martínez-Abadías et al. 2012, Attanasio et al. 2013) and facial shape (Porto et al. 2013) in mammals.

However, as mentioned above, the developmental view of the G-P map is not constrained to adult phenotypic traits. A growing research agenda within evo-devo is interested in phenotypes at different levels of organization and developmental stages. For example, some evo-devo models of G-P relationships study the variational tendencies of different cell types in multicellular organisms (Arendt et al. 2016). Moreover, the study of gene regulatory networks is a very influential and prolific area of evo-devo (Fischer & Smith 2012). In these models, genetic variation is mapped into different levels of expression and regulation in the molecular interactions that bring about phenotypes in development. Thus a mapping relationship can be built between genetic or (intra-organismic) environmental changes and changes in gene regulatory networks. These networks are the result of the interactions among a set of genes that code for transcription factors and the genes those factors regulate. An interesting feature of these G-P maps is that they set the target phenotype at the level of non-protein coding genes, thus distancing themselves from the view that only coding regions of the genotype are important for explaining phenotypic differences (DiFrisco & Jaeger 2019). In studying how genotypic variation maps into variation in the regulation of certain transcription factors, these evo-devo models take into account the variational potentiality of specific developmental pathways, the previous stage to morphological and phenotypic variation.

One such model is the G-P map of the so-called gap gene system (Jaeger 2011; 2018). Gap genes are involved in the embryonic segmentation of some arthropods, such as flies. These genes are expressed when they interact with maternal morphogenetic gradients,[44] and determine the pattern of segmentation in the body of the fly through complex regulatory interactions. A G-P model of the gap gene system

[44] A morphogenetic gradient is the spatial distribution of a substance (the morphogen) in the embryo that determines the formation of a morphological pattern.

thus consists of a "dynamical network model" that represents regulatory interactions among its components and describes "change in gap gene product concentration" resulting from variation in the inputs of the model—activation or repression of regulatory parameters (Jaeger 2018, pp. 67-8). Variation in some of the genetic components of the network thus maps into variation in the segmentation pattern of the embryo, which will ultimately map into variation in the resultant morphologies.

All these G-P maps can help in building up local-specific developmental morphospaces (cf. Eble 2003), establishing a space of possible morphologies to explore by the developmental system as based on its dynamical properties. Nonetheless, a further question concerns whether or not there are general tendencies of G-P mapping relations that may serve for talking about the general probabilities of variation. In other words: can we identify in evo-devo G-P maps those properties that are responsible for variational probabilities in the sense of the previous chapter?

1.2. Developmental encoding and the G-P map

We have seen how G-P map models instantiate a particular way of approaching the space of evolutionary possibilities that is developmentally grounded. Instead of navigating through an infinite space of possible genotypes or phenotypes, developmental systems navigate through a constrained space of possible developmental changes. Let us remind that this is the key conceptual contribution of a developmental morphospace as exemplified by Raup's cube of shell morphologies (1966): phenotypic possibilities are constituted by virtue of the developmental pathways available. Indeed, the idea underlying Alberch's introduction of the G-P map is that the space of possible phenotypes "is a property of the internal structure of the developmental system." (Alberch 1991, p. 7). In this regard, the phenotypic area explorable in a particular morphospace corresponds to the end state of a particular G-P map, that is, to the phenotypes in its parameter space, as defined by the dynamical properties of a developmental system. In turn, it is the very properties of this dynamical system—the properties of development—that determine what is the space of phenotypic possibilities.

In this context, we may vindicate with Pigliucci "the end of the blueprint metaphor" of genes (2010a), namely the end of the informational genetic paradigm governing classical evolutionary genetics. According to Pigliucci, the idea of a genetic encoding of organismal information is advocated to failure, and should be replaced by "developmental encoding" (p. 557) instead. In the previous chapter (section 4), we saw that genes are a key causal component of developmental processes, but that the properties of development are not reducible to the properties of genes. Indeed, we saw that, unlike the abstract statistical entities of population genetics, developmental genes form intricate networks that are better conceived as *pathways* (Gilbert 2000). Thus in modeling a G-P map, we take those developmental properties that remain stable and specify morphogenetic pathways, despite their partial dependence on gene expression, and abstract away the generating rules (Alberch 1985b) that they "code for" (i.e. developmental encoding). Consequently, in a G-P map we may distinguish between the genotypic variation under analysis on the one hand (i.e. the genotypic variants as initial states of the mapping relation), and those genotypic properties involved in the particular developmental encoding under interest. Developmental encoding thus does not demarcate a neat distinction between genes and developmental properties in any metaphysical sense, for genes are clearly involved in developmental processes. Nevertheless, it serves as an abstraction of those relatively stable properties that cause specific patterns of phenotypic variation.

Developmental encoding can be thus seen as "a small number of 'instructions'" (Roggen et al. 2007) that maps the genotype to the phenotype through a generative process. The origin of this approach can be traced back to Turing's diffusion model of morphogenesis (1952), arguably the first mathematical model of ontogenetic pattern formation. This model presents a simple inhibitor-activator model that regulates the diffusion of a chemical reaction. By changing the values of inhibition-activation reactions, the diffusion pattern will change in nonlinear ways: the same system can generate a homogeneous pattern, a 'dots' one and a 'striped' one depending on the values of inhibitors and activators. The inhibition and activation *instructions* are developmentally encoded, that is, they are codified by the developmental properties of the system. The values that these instructions will take

nevertheless depend on mutational inputs. A wide array of morphological patterns can arise from generative rules of this type determined by mechanical and chemical properties of the cells and tissues under development (see e.g. Forgacs & Newman 2005). In the line of Turing's model, computational approaches to evolution have incorporated the G-P map for developing so-called evolutionary algorithms, which simulate an evolutionary process where genetic inputs generate phenotypic variants upon which selection can be applied. Generally speaking, in evolutionary algorithms phenotypic variants depend on the organizational rules that relate the genetic (input) to a particular phenotypic solution or "offspring" (output) (Wagner & Altenberg 1996). Unlike those genetic algorithms mentioned in the previous chapter where selection searches fitness values in a space of possible genotypes (e.g. Mitchell et al. 1996), evolutionary algorithms understood in the present sense consider a space of possible phenotypes that a developmental system can explore given certain organizational rules.

Following the urn analogy as modified in Section 4 of the previous chapter, we may consider that the building properties of new balls—either assembly followed by painting or vice versa—are developmentally, rather than genetically, encoded. Of course, these properties depend on genetic properties prone to changes through mutations. However, we can distinguish regular mutations, such as changes in color, from a change in the way the balls are built, i.e. a change in the developmental encoding—for instance, a transition from assembly followed by painting to painting followed by assembly. In turn, genotypic changes—i.e. mutations—can be regarded as providing variation of very different types, depending on how they affect developmental properties. Thus G-P maps consider the genotypic space of certain *kinds* of mutations, defining a parameter space of their possible effects when every other genotypic, developmental and environmental aspect interacting with or affecting the expression of those effects remains constant. Let us illustrate this with Richard Dawkins' *Blind Watchmaker* program (2003/1988), where he implemented a simple evolutionary algorithm for drawing phenotypes. Dawkins noticed that each model in his program needed to include a set of growing rules in order for random mutations to specify biologically meaningful phenotypes. As he mentions,

> Genes do not control small fragments of the body, the equivalent of pixels. Genes control growing-rules, developmental processes, embryological algorithms (Dawkins 2003/1988, p. 243).

Thus in order to add "general biological principles" of growth (p. 245) such as symmetry, segmentation, or size gradients, he regarded useful to distinguish between "ordinary mutations" in his program and mutations entailing "changes in the genetic system" (2003/1988, p. 252). While the former could fluctuate randomly, the latter implied a re-coding in the program. We can consider each 'genetic system' in Dawkins' sense, namely each parametrization of the program, as a different model of a G-P map. In this sense, the coding needed for establishing the genetic system is the building of the parameter space of the map. Accordingly, every time a change in the code is introduced—such as the introduction of a 'mirror' gene type producing a symmetry pattern—a new G-P map is generated with a distinct developmental encoding assigning a specific function in the *growing process* (i.e. the drawing of phenotypes) to a possible mutation. In this case, the new G-P map will generate phenotypes with a new symmetric property. Once the code is set and the program is running, there are different genetic combinations that can obtain through random mutations, such as larger or smaller branches following the same symmetry pattern. These are the 'ordinary mutations' representing different points in the space of possibilities generated by the parametrization. In turn, 'ordinary mutations' would correspond to changes in the variables of the mapping function, while a change in the genetic system—therefore a re-codification in the program setup—would imply a change in the parameter space defining the function itself. In this case, the distinction between these two types of mutations corresponds to their different roles in the model: changes in the genetic system produce different growing processes, while ordinary mutations change the particular instantiation of such processes. The former affect the developmental encoding of the model, while the latter influence a particular implementation of it.

The use of computational metaphors for development is not accidental. As pointed out in the previous chapter, the theoretical programme of evo-devo is widely

influenced by the computational approach to evolution (Müller 2007). Indeed, an important component of the developmental view of evolution has been the implementation of models that simulate embryonic rules of growth. Although the significance of simulations has at times been put into question (see Humphreys 2009), the capability of these models of generating patterns that resemble the patterns of phenotypic evolution suggests that they can be analogous to the organismal structures that brought them about. As the systems biologist Hiroaki Kitano puts it,

> The scientific goal of systems biology is not merely to create precision models of cells and organs, but also to discover fundamental and structural principles behind biological systems that define the possible design space of life (Kitano 2007, p. 1)

The assumption underlying these computational models is that finding the coding rules needed for simulating the evolution of phenotypes can provide an understanding of the actual causal processes responsible for it. The general approach is "reverse-engineering", namely the inference from the properties of a target object to the process that generated it. With this approach, it is possible to derive a hypothetical mechanistic explanation that specifies "the dynamic causal chain of events" that leads to the target output "from its initial state" (Jaeger 2018, p. 66). These causal hypotheses are later indirectly testable through a comparison with empirical interventions. This is the idea behind, for example, the computational models of the evolution of mammalian teeth mentioned above (Salazar-Ciudad & Jernvall 2010, Marin-Riera et al. 2018). In order to generate patterns of variation similar to the ones found in nature, a particular computational model will implement specific developmental rules of teeth formation, such as determinate rules for "differential tissue growth and differential cell adhesion" (Marin-Riera et al. 2018).

Besides the potential for suggesting actual evolutionary and developmental pathways that may have taken place, the computational approach allows for exploring general properties of evolving developmental systems. An interesting feature of developmental encoding is that it represents an abstraction of those internal properties of the developmental system that transform certain inputs—be

them random or not—into nonrandom outputs. At this point, we are in the position of approaching evo-devo G-P maps as abstractions of the chance setups that determine the phenotypic sample space of evolution in the sense articulated in the previous chapter. Thus developmental encoding can be seen as the implementation of a certain kind of developmental chance setup. In the remainder of this section, I present in more detail a very simple model of a G-P map that will serve for illustrating how this causal, developmental understanding of the genotype-phenotype relation can be fruitful in the representation of variational probabilities.

1.3. Molecular evolutionary models as models for evo-devo

The field of molecular evolution has undergone a boost in the last decades. Since the mid 20th century, the evolution of the molecular components of organisms has both provided important experimental results and theoretical insights to evolutionary biology. Moreover, as mentioned in the previous chapter (sec. 4.2), molecular studies constitute a substantial part of the evo-devo research agenda, the comparative developmental genetics approach being one of the most prolific areas in the field. Although some of the most important components of the evo-devo research lack an analog at the molecular level—such as the importance of physical properties of cellular organization—even molecular models need to introduce a distinction between genotypic or mutational inputs and the "developmental encoding" of a system.

At the molecular level, evolutionary algorithms have the advantage of specifying the entire genome sequence that maps into a specific output. Thus the models of molecular evolution serve as minimal models for illustrating the main conceptual components of evo-devo variational tendencies (Fontana 2002, Nuño de la Rosa & Villegas 2019). They do so while they instantiate the idea that these tendencies can be responsible for precise probabilistic measures of variation: in specifying how each genome sequence maps into a particular phenotype, molecular

G-P maps can be used to derive variational probabilities in the sense depicted in the previous chapter with mathematical precision.

Here I present the most basic model of a G-P map in the field of molecular evolution: the RNA model (section a). As an extension to a previous account (Nuño de la Rosa & Villegas 2019), here I deepen into the properties of this model that are relevant for evo-devo probabilities. This model will serve in the remainder of the chapter as an illustration of the probabilistic potential of G-P map properties at other levels of organization, such as those mentioned previously in this chapter. After introducing the model in some detail, I will point at some of its limitations and I will sketch a slightly more complex model that introduces spatiotemporal developmental rules, in order to exemplify how the analogy between molecular and evo-devo models can be drawn: the minimal cell model (section b).

a) The RNA model

The RNA model (Fontana & Schuster 1998, Ancel & Fontana 2000, Schuster 2001) is a random graph relating the nucleotide sequences of RNA molecules (e.g. AGA GCG CCG) to their two-dimensional shape or secondary structure (see Figure 3.3). The model takes nucleotide sequences as the "genotype" of the molecules, and their secondary structure as their "phenotype". The secondary structure of a molecule refers to the shape in which it folds, that is, to how the nucleotides are organized with respect to each other in a hypothetical two-dimensional space.[45] The folding relating sequences to shape represents the general base pairing rules, namely the combinations A-U, G-C and G-U. Although these rules typically allow for more than one secondary structure given a particular sequence, only the most energetically stable among the possible ones, as estimated by its free energy, is usually considered. Importantly, the secondary structure does not include three-dimensional properties of RNA. Such properties, considered as their 'tertiary structure', would multiply the level of structural complexity of the molecules, and their representation and

[45] More precisely, a secondary structure is a graph of contacts between nucleotides that provides information on their spatial arrangement and relative distances in the folded molecule, particularly on the way the different bases pair. Thus a secondary structure graph consists of a number of stacks, or alignments of pairs, and loops, or groups of unpaired bases.

computation is by far too complex to be integrated in simple genotype-phenotype relations, for they do not present the level of tractability and empirical accessibility as the base pairing structure (Fontana 2002).[46] Nonetheless, in considering the sequence-to-shape (i.e. secondary structure) relation as one of a genotype-phenotype, it is possible to draw an analogy between a hypothetical folding process and the process of development in multicellular organisms. In this case, the algorithmic rules coding for the folding process of molecules correspond to the developmental encoding of the RNA model.

The RNA model captures a series of properties of G-P maps and their potential evolutionary consequences that are representative of the variational properties of interest in the field of evo-devo. Considering that the developmental properties of complex multicellular organisms are not exhaustively tractable at the molecular level, having a simple model relating RNA sequences to shapes helps in visualising the evolutionary importance of these properties at a certain level of abstraction. As Fontana (2002) claims,

> the RNA model is an abstract analogue of development that grounds a discussion of these [evo-devo properties] within a simple biophysical and formal framework (p. 1164).

This model is particularly useful for illustrating how variational probabilities are conceptualized in evo-devo (Nuño de la Rosa & Villegas 2019, p. 12). Firstly, the model is logically independent of and prior to natural selection. This enables to represent the probabilities of generation separately from the probabilities of being sampled. Despite the fact that these are not always empirically discernible in natural populations, separating them is conceptually indispensable in order to analyse the role of variation in evolution, as argued in Chapter 2. Secondly, this model allows defining these variational probabilities independently of mutational probabilities as well. In relating nucleotide sequences with their correspondent phenotype, this model can establish the probability of phenotypic variation even under the

[46] An additional level of complexity is added when considering the so-called quaternary structure of polynucleotides, which refers to their relation with other molecules in its environment and thus to a higher level of organization. An example of such a structure is the chromatin form of DNA molecules in the cell nucleus and their consequent interaction with histones.

assumption of mutational randomness (in the sense of lack of pattern given a null model, see Chapter 1) and without any assumptions on mutational rates. Finally, in well-defining both the genotypic and the phenotypic spaces, the RNA model allows for a precise quantification of variational probabilities. Most genotype-phenotype maps in evo-devo have a qualitative character with regards to probability (that is, they do not specify numerical probabilities), or they derive quantitative estimations of probabilities only epistemically. The specification of numerical ontological probabilities is possible through the RNA model, where complex developmental interactions are abstracted away but still represented. Given these considerations, in the RNA model we can quantify with precision the *variability* of a molecule type in terms of how likely it is that a nucleotide change produces a change in secondary structure.

A typical way to illustrate the RNA sequence-to-shape or G-P map is through a topological space of a neutral network (see Figure 3.3). Neutral networks are common in the study and modeling of molecular evolution; for example, they have been used for modeling the G-P maps of proteins and gene networks (Bastolla et al. 2003, Ciliberti et al. 2007). These representations consist of a genotypic space formed by a network of genotypes connected by virtue of their mutational closeness. Each node in the network has a corresponding phenotype represented by a different color, thus the location of phenotypes in the genotypic space is easily visualized. When genotypes of the same phenotype are mutationally connected, they form a neutral network, namely a region in the genotypic space where mutations are neutral with respect to the phenotype—thus an area homogeneously colored. In the RNA model, the genotypic space, namely the space of all possible sequences considered, is a network of RNA sequences arranged in virtue of their sequential similarity. Each node in the network corresponds to an RNA sequence, and edges connect the sequences that differ only in one nucleotide. Neighbor sequences are in turn separated by a single point mutation, meaning that a nucleotide change in replication can lead from one point to the ones in its immediate neighboring area. Due to the assumption of genome length conservation, point mutations and combinations of them, that is, replacements of specific nucleotides in a sequence by a

different one, are the only genetic changes considered in these representations, where genome duplications, insertions and deletions are neglected. A key aspect of neutral networks representations is that they allow us to visually represent those areas of the genotypic space leading to a given phenotype, in turn illustrating the lack of a 1-to-1 correlation between genotypes and phenotypes. A neutral network in this model is a network of mutationally connected sequences that map into the same RNA secondary shape. In other words, it is an area of the network where all sequences lead to the same point of the phenotypic space, and consequently where some mutational changes will be neutral with respect to the phenotype. Notice that this neutrality is prior to and logically independent of the neutrality considered in genetic drift models, for it does not refer to fitness values but to phenotypic changes (see chapter 2, section 2).

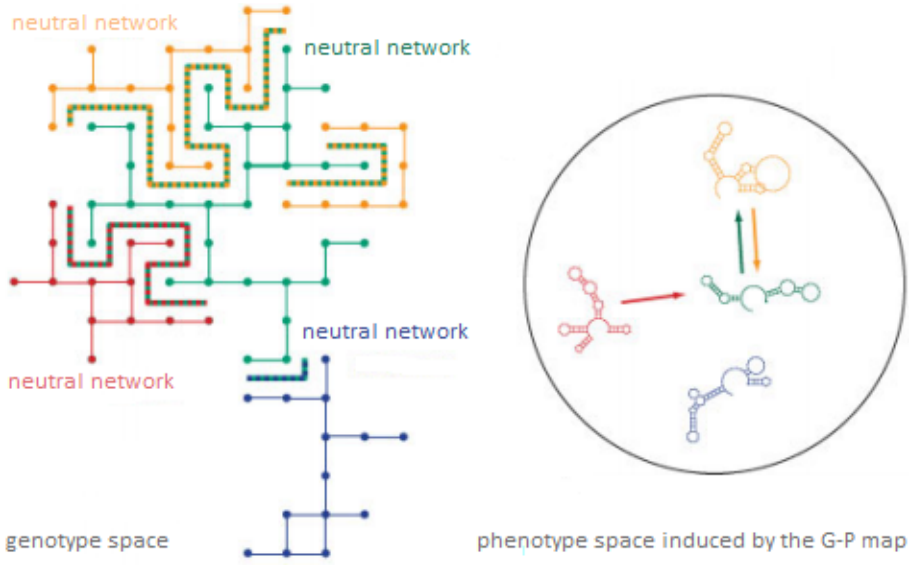

Figure 3.3. A neutral network representation of the RNA Genotype-Phenotype map. The left image shows a genotypic space, where each point corresponds to an RNA sequence differing from their neighbors by a single point mutation. The space has four different neutral networks, clustering those sets of sequences that fold into the same phenotype or RNA shape, each of which is represented by a different color. Four lines of phenotypic change are also illustrated. The right image corresponds to the phenotypic space, where the four different phenotypes are represented. Reproduced with permission from (Fontana 2002).

Moreover, through the model, it is possible to visualize the context-dependency of genes, thus representing epistatic effects of mutations. An RNA model can illustrate how the effects of a change in sequence are dependent

upon the rest of the sequence. For instance, any specific point mutation will lead to a change in phenotype or to a phenotypically neutral result depending on where the entire sequence is situated in the neutral network. More interestingly, a neutral network representation can help represent variational probabilities, insofar as it includes phenotypic possibilities and their accessibility through mutations in quantifiable terms. That is, in this model, it is possible to measure variational probabilities through an estimation of the accessibility of different neutral networks upon mutation. In the sections that follow, I will deepen into this feature of the RNA model in order to explore the philosophical consequences of having an evo-devo model of variation where variational probabilities can be easily exemplified.

b) The minimal cell model

The RNA model presents certain limitations when it comes to representing variational tendencies of development in evolution. As Fontana (2002) claims, "after all, an RNA sequence does not code for the base pairing rules" (p. 1166), that is, folding rules are based on chemical principles that are independent of the particularities of each genome. A key limitation is the lack of analogue for any aspect of organization in development. The absence of any feature with regards to control and regulation of the developmental process in this model has indeed lead to some researchers to go a step further and develop a mapping encrypting a dynamical system that "itself is a minimal version of a gene regulatory and metabolic network" (Flamm et al. 2007, p. 1832). Christoph Flamm and coworkers built a cell model that incorporates the insights from the RNA model but attempts to overcome its limitations in terms of dynamical control of metabolism and environmental interactions by genes. The cell model is inspired by Stuart Kauffman's use of boolean networks for modeling gene regulatory networks (1993). In a nutshell, gene regulatory networks are conceived of a system of dynamical gene interactions where different components can be switched on and off. The authors modeled the phenotype as a minimal cell whose components interactions are described as regulatory networks of this type.

In this model, the genotype is an RNA string, whereas the phenotype is a regulatory and metabolic network where all molecular types are RNA molecules. Each possible genotype has the same sequence pattern, where two regulatory sites are followed by a coding region of a fixed length. When they are expressed, these genotypes produce either structural RNA composing a regulatory network, or transcription factors accomplishing metabolic functions in it. Just like in the RNA model, the minimum free energy target shape of an RNA sequence is taken as its phenotypic structure, which will determine its metabolic function. On the other hand, the transcription factors are responsible for regulating the activity of the genes. Finally, metabolism is modeled as RNA polymers with an auto-catalytic cycle. The metabolic functions enable, on the one hand, the reactivation of RNA building blocks, which are present in a fixed amount. On the other hand, they fulfill the construction of a cell membrane. Consequently, each phenotype—each regulatory and metabolic network—builds up a self-contained cell.

The G-P map relationship underlying this complexity is the assignment of a cell metabolism (phenotype) to each RNA strand (genotype). This model, in representing metabolic functions and cell membrane growth, incorporates key aspects of development that are not present in the simple RNA model. Instead of general nucleotide folding rules, the developmental encoding embedded in this model considers building rules that are spatiotemporally sparced: the construction of a cell through transcription and metabolic interactions. Analogously to the generative rules of amphibian digit pattern formation in Alberch's G-P maps (1991), the rules for cell growth are codified in the minimal cell model. The insight gained from this model is that these rules can be coded at the molecular level, providing for a space of phenotypic possibilities almost as mathematically tractable as the space generated in the RNA model. Therefore probabilistic patterns of changes in metabolism (phenotype) under changes in the RNA string (genotype) can be empirically and computationally studied. Moreover, in addition to the establishment of a phenotypic space, this model can undergo evolution in its simulations. Unlike the RNA model where the space of possible changes is constrained by the kinetic rules of base pairing, the minimal cell model can bring about an enormous amount

of variation. The genotype and the phenotype in this model—namely the RNA string and the metabolic system—are distinct objects, there being a complex causal chain between the two allowing for a vast array of possible combinations. The result is a system where relatively complex evolutionary dynamics can be traced while all the relevant causal components of the G-P map are known, and thus where the role of internal factors in nonrandom patterns can be studied:

> In contrast to prior exclusively RNA-based autocatalytic systems, the genotype and the phenotype in the presented model constitute separate objects. This allows an unhindered evolvability of the minimal cell on the way from a random dynamical network to an adapted functional system. (Flamm et al. 2007, p. 1838)

In addition, unlike the models where artificial selection is required each generation, the cell model is self-contained: no additional external inputs are needed for its evolution. The system can therefore increase its complexity without external limitations and "freely explore the genotypic space" throughout simulations (Flamm et al. 2007, p. 1832). In their simulations, the authors verified that random mutations, in the sense of lacking any order of pattern (see section 2 in Chapter 1), can give rise to viable regulatory networks. In other words, given a set of developmental rules for cell building, the changes in the inputs of the model can be random and still derive in non random phenotypic results.

2. Variational dispositions of G-P maps

We have seen that G-P maps abstract away the causal properties of development that are relevant for probabilistic patterns of variation, and that some simple models of them allow for probability measures of variational tendencies. In order to characterize the propensities responsible for the sample space of evolutionary variants in these variational tendencies, let us now see which are the general properties of G-P maps accounting for patterns of variation.

The variational properties of G-P maps are an increasing focus of attention among evolutionary biologists. On the one hand, since studying how the pathways

linking genotypic changes to phenotypic variants influence patterns of variation is one of the research goals of evo-devo, the identification of general variational capacities accounting for those patterns is arguably as central as depicting specific evo-devo mechanisms. On the other hand, the evolutionary origin and significance of genotype-phenotype general relations is a key aspect for the study of the evolution of complex systems.

Variational G-P map properties have been accounted for from a dispositional point of view in the recent philosophical literature (Austin 2017; Austin & Nuño de la Rosa 2018; Brigandt 2007; 2015b). Philosophers of evo-devo tend to consider the variability, modularity, robustness and plasticity of developmental systems as dispositions to bring about certain types of variation. In this sense, evo-devo can be regarded as "a science of dispositions", where the evolutionary potential of developmental systems is captured by their dispositions to undergo changes under different types of perturbations (Austin 2017). Particularly, evo-devo mechanisms show different dispositions to react to modifications, namely differences in robustness, modularity and plasticity, in turn playing an explanatory role that cannot be accounted for from the classical mechanistic point of view (Brigandt 2015b). In other words, the dispositionality embedded in evo-devo variational properties seems to be central to the explanatory agenda of evo-devo (Austin & Nuño de la Rosa 2018). In turn, recent philosophical reflections agree on the promising nature of understanding these properties as dispositions.

In this section, and following a recently published account (Nuño de la Rosa & Villegas 2019), I introduce these properties as propensities of developmental systems. Moreover, as an expansion to this account, in this section I consider the particularities of phenotypic variability and further dispositional properties such as robustness, modularity and plasticity, dealing as well with the connections between these properties (section 2.1). This will serve me as the first step for providing a causal propensity perspective of variational probabilities that will follow in the next sections of the present chapter. In addition to this, I consider the evolutionary origin of these properties in order to explore their nature and relationship with other products of the evolutionary process (section 2.2).

2.1. The variety of variational dispositions

Let us now turn to the general dispositions that developmental systems show, as represented in G-P map models, that are relevant for evolutionary patterns of variation: their variability, robustness, modularity and plasticity.

a) Variability

We shall start from the general capacity to generate variation. As we saw in the previous chapter (section 4), extant variation is only a fraction of the possible variants a developmental system can generate, namely its variability. For instance, we have seen in Alberch's model of amphibian digits how some phenotypic transformations are more likely than others as a result of developmental tendencies. Consequently, some possibilities will be realized with more frequency than others, while others may not even be realized at all even if they constitute possible transformations of the developmental system. This is because developmental systems determine their own phenotypic space of possibilities, which include specific phenotypic biases. Moreover, not only biases are present with regards to which phenotypes are accessible, but also with regards to *how accessible* they are, which leads evo-devo biologists to make probabilistic considerations about the potential of developmental systems to generate specific types of variation in evolution. Phenotypic variability thus refers to the general disposition of living systems to undergo a phenotypic change, and in turn can be defined, as Lauren Ancel and Walter Fontana (2000) do, as the capacity to produce a different phenotype upon mutation:

> Phenotypic variability describes the extent of phenotypic variation accessible to a genotype through mutation. (Ancel & Fontana 2000, p. 243)

In this regard, phenotypic variability is typically referred to in dispositional terms. For instance, it has been defined as "the potential or the propensity to vary" (Wagner and Altenberg 1996), or, more specifically, as "the *tendency* of developmental systems to change, amplify, or reduce the expression of genetic

variation at the phenotypic level" (Hallgrímsson et al. 2005, p. 526). We can thus say that variability refers to the disposition of the system to change under mutation, regardless the type of phenotypic exploration involved, as well as to *what* particular phenotypes can be achieved through mutation and *how diverse* they are (Salazar-Ciudad 2007). Accordingly, given a G-P map, phenotypic variability is the tendency of the represented developmental system to generate different phenotypes upon mutation. Phenotypic variability therefore concerns what phenotypes can be generated, how strong is the disposition to generate them and how diverse the phenotypes are from one another. The biases in the distribution of phenotypes in the evolutionary space of possibilities can result in differences in propensities such as how gradual the variation a developmental system can generate is, or how prone to changes it is upon mutation.

Despite its meaning being intuitive (mutations bring about phenotypic changes), this property is very variable across different traits. Let us recall that, while some traits show high variability—such as feather color in birds—, other traits remain highly stable across species—such as feathers themselves—or higher taxa—e.g. the dorsal nerve cord in the phylum *Chordata*. As we saw in the previous chapter (section 4), the evo-devo research agenda concerns inter-specific patterns of unity and diversity, thus recognizing homologous characters and their variable specifications across species, and in turn considering how traits manifest their distinct variabilities differently. These differences in the variability of certain systems will be instantiated in differences in their G-P maps. In this sense, a variable G-P map is one where possible genotypes map to a variety of diverse phenotypes. In other words, a G-P map shows variability if genotypic changes tend to lead to phenotypic changes (see Figure 3.4).

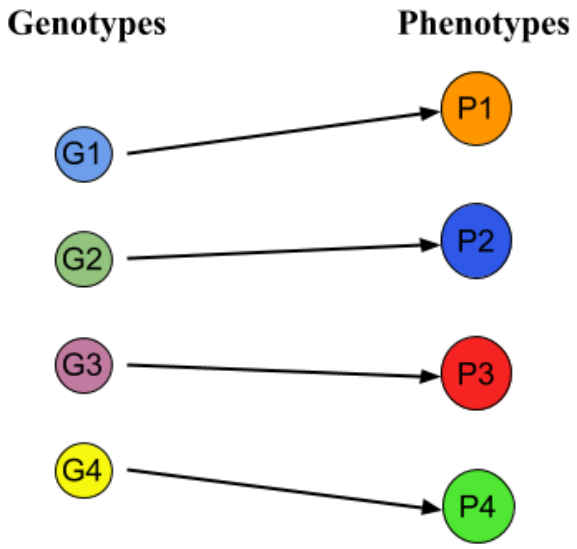

Figure 3.4. A simple variable G-P map, where every genotypic variant leads to a different phenotype.

The irregularities in the explorability of the morphospace have been conceptualised in a variety of ways: phenotype distribution bias (Borenstein & Krakauer 2008), facilitated variation (Gerhart & Kirschner 2007), developmental constraint (Alberch 1985a, Amundson 1994), or simply variability (Salazar-Ciudad 2007). Although these notions may seem to refer to different phenomena, it is believed that the same processes bringing about variability are responsible for its limitation (Willmore et al. 2007). In this regard, and as mentioned in the previous chapter, variability and developmental constraints can be seen as the two sides of the same coin: by limiting possible phenotypic transformations, the properties of development favor others (Brigandt 2015a). This is also the idea underlying Dawkins' introduction of a developmental encoding in computational evolution and the notion of evolvability. In limiting the ways in which phenotypes can be constructed, building rules also make particular phenotypes largely more accessible than others (Dawkins 2003/1988).

In turn, the idea behind variability is that the same properties that channel phenotypic transformation are responsible for a reduction in the relative amount of genetic changes needed for acquiring a novel phenotype. For example, regulatory changes in development have constrained effects, even if a small number of them

may suffice to "redeploy core processes" (Gerhart & Kirschner 2007, p. 8588). That is, even when variation is limited to changes in regulatory sites, the number of possible combinations of regulations is very large and their phenotypic effects may be extremely multifaceted. In this regard, developmental repatterning refers to the evolutionary change in the pattern of development due to mutations affecting regulatory sites (Arthur 2011). The potential for these changes is restricted in kind though highly versatile: changes in the timing—heterochrony—, the spatial arrangement—heterotopy—, the magnitude—heterometry—, or the type—heterotypy—of regulatory patterns are constrained in kind but their potential phenotypic results are manifold. These developmental mechanisms enhance the exploration of the morphospace: possible phenotypes will be more or less accessible depending on the developmental properties linking genotypes to phenotypes.

The neutral network representation of the RNA model allows for establishing phenotypic change probability measures under the consideration of genotypic closeness (Fontana & Schuster 1998). Through this model, a number of variational properties can not only be defined with precision, but also computationally explored. For example, the variability of a phenotype *j* towards another phenotype *k* can be defined as the probability that any point mutation from any sequence in its neutral network (S_j) results in a specific change in the phenotype, i.e. in the RNA secondary structure (S_k). The following equation describes this probability (Schuster 2001):

$$\rho\,(S_k\,;\,S_j) = \gamma_{kj} \,/\, |\,B_j\,|$$

In this equation, γ_{kj} corresponds to the number of point mutations that lead from phenotype *j* to a different phenotype *k*, and B_j corresponds to the set of all possible genotypes forming phenotype j.[47] This equation can be applied to the RNA model in Figure 3.3 as follows. Let us suppose that we want to calculate the variability of the red structure to the green one. In order to do so, we consider the green neutral network S_g and the red one S_r, and apply the formula to it such that ρ

[47] $|B_j|$ is proportional to the number of all possible genotypic combinations forming phenotype *j*, i.e. proportional to (G_j), and the number of pair bases (κ) of the sequence: $|\,B_j\,| = [\,1\,(\kappa\text{-}1)\,|\,G_j\,|\,]$ (Schuster 2001).

$(S_g ; S_r) = \gamma_{gr} / | B_r |$. That is, the variability of red to green is equal to the number of point mutations that lead from red to green (γ_{gr}) divided by the number of sequences folding into the red secondary structure. Taking this measure as reference, a generalization of it can be made so that the variability of a given phenotype to any other phenotype in its neighbouring area is well defined:

$$\rho (S_k ; S_{\neg k}) = \gamma_{k\neg k} / | B_{\neg k} |$$

Here $\neg k$ corresponds to the set of all neighbouring phenotypes of *k*. This equation quantifies the variability of a specific phenotype as a probability function over the phenotypic transformations it can undergo upon (single-point) mutation. In the model of Figure 3.3, it could be thus possible to define the probability of the green phenotype giving rise to any other phenotype (blue, yellow or red) upon mutation. This illustrates that, when G-P maps are well defined, as it is the case in the RNA model, a probability measure can be established over phenotypic changes in virtue of the variational properties of the mapping (i.e. developmental) relation between genotypes and phenotypes. Thus, in the RNA model, the representation of sequence-to-shapes relations allows to define the variability of a specific phenotype, but also of a trait with different possible phenotypes. For instance, an RNA G-P map can represent the variability of several mutationally related secondary structures. That is, not only the variability of a single neutral network can be defined: the variability of an entire G-P map could be established by considering how neutral networks are distributed in it. Such variability can be measured as the probability that any mutation in the entire genotypic space will result in a phenotypic change. Taking the previous variability function as a reference, we can establish the variability of a simple G-P map *m* consisting of two neutral networks *k* and *j* through the following equation:

$$\rho [(S_k ; S_j) \vee (S_j ; S_k)] = (\gamma_{jk} + \gamma_{kj}) / | B_m |$$

That is, the probability of a change from phenotype *j* to *k* ($S_k ; S_j$) or a change from phenotype *k* to *j* ($S_j ; S_k$) is equal to the sum of point mutations leading from *j* to *k* (γ_{jk}) and leading from *k* to *j* (γ_{kj}) divided by the number of point mutations

inside m (| B_m |)—where B_m equals $B_j + B_k$. This equation measures the proportion of point mutations in m that would result in a change in the phenotype—either from k to j or from j to k. In the model of Figure 3.3, a similar equation would give the proportion of point mutations leading from either green, red, blue and yellow phenotype to a different neutral network.

Another interesting possibility of probability measures in G-P maps is the definition of probability distributions over different possible phenotypic results. Departing from a given phenotype, a probability distribution of phenotypic results upon mutation can be assigned. Considering again the model in Figure 3.3, it should be possible to derive the probability distribution of phenotypic changes upon mutations departing from the green phenotype, combining the different probabilities of phenotypic change (i.e. the probability of a change towards red, towards blue and towards yellow). Such distribution would include a proportion of phenotypically neutral mutations inside the green network, a proportion of changes to the yellow phenotype (change to yellow network), a proportion of changes to the red phenotype (change to red network) and a proportion of changes to the blue phenotype (change to blue network).

The ways in which a G-P map can show variability are very diverse, other general variational tendencies such as robustness, modularity and plasticity contributing to the overall variability of a given system (Hallgrímsson et al. 2002, Willmore et al. 2007). From a philosophical perspective, this variational potential of developmental systems has recently been accounted for in terms of dispositions, precisely for constituting "intrinsic generative capacities" that cause phenotypic novelty (Austin 2017, p. 5). As a matter of fact, biologists seem to make use of dispositional terms in order to refer to the potential of developmental systems to provide variation. In this regard, developmental units of variation can be seen as dispositions insofar as they are defined by their generative potential in the construction of the morphospace. However, phenotypic variability as a general disposition of G-P maps has been less examined. In particular, the consequences of considering this disposition from the point of view of variational probabilities and chance have only been tentatively explored very recently (Nuño de la Rosa & Villegas

2019). In the remainder of this section, we shall see how robustness, modularity and plasticity, as general dispositions of G-P maps, provide developmental systems with different kinds of variability that allow for distinct ways of generating the developmental morphospace.

b) Robustness

A central dispositional property of biological systems that seems to be in opposition to variability is their robustness. In broad terms, a system is robust if it remains unchanged under a range of circumstances. Of course, this property is a matter of degree and will highly depend on what it is considered to be a wide enough such range. At first sight, this property may seem the opposite of any kind of disposition: given certain stimulus conditions, no manifestation is met at all. However, recall the contrastive nature of the causal and explanatory role of dispositions discussed in section 3 of Chapter 1: a disposition explains why something is the case instead of not being the case. Therefore something is robust when it shows persistence when non-persistence is expected, that is, when it manifests a behaviour—i.e. persistence—that other, non-robust systems would not manifest under the same triggering conditions—i.e. disturbances. The dispositional nature of this property is now clear-cut: a system is robust if it *manifests* resistance to change given certain *stimulus* conditions. Robustness can therefore be thought of as a tendency to remain constant in a certain aspect when other aspects are changed. For example, the chimney is a robust part of a building, for it tends to remain stable when hit, as compared to the fragile window (cf. Chapter 1).

When it comes to biological systems, homeostasis, the general capacity of organisms to maintain their condition through the compensation of changes, is a disposition of this kind. However, individual dispositions of organisms are not responsible for patterns of variation. From the variational point of view, the important feature is the robustness of *potential* phenotypes under different genetic compositions and environmental contexts in development. This type of robustness is different from homeostatic properties, for it refers to the invariance with respect to the potential result of the developmental process. In other words, the system is

robust insofar as it will bring about the same phenotypic result under many different circumstances. Following Christian Klingenberg's recent review of this phenomenon (Klingenberg 2019), I will lean on the notion of 'target phenotype' (Nijhout & Davidowitz 2003) to summarize the main insights on the topic in contemporary biological literature. A target phenotype is a theoretical construct that refers to the—hypothetical—specific phenotype that is expected to develop given a particular genotype and an environmental arena in the absence of stochastic perturbations. With this in mind, robustness can be seen either as the multiple realizability of a target phenotype, or as the tendency of actual phenotypes to remain close to the target.

Thus a phenotype is robust if it persists in the same way under genetic or environmental perturbations. Although the degree of robustness may vary vastly for different phenotypes, this property seems to be ubiquitous in living organisms: phenotypic variation is less abundant than variation in genotypes and environmental conditions in virtually every case. This observation dates back at least to Conrad H. Waddington's notion of canalization (Waddington 1942), and is currently widely accepted and experimentally supported in a far-reaching array of species and levels of biological organization, such as molecular secondary structure, gene regulatory and metabolic networks (de Visser et al. 2003, Kitano 2004, Felix & Wagner 2008). Canalization was first introduced as the evolved ability of development in a population to "bring about one definite end-result regardless of minor variations" (Waddington 1942, p. 563). The notion of phenotypic robustness is an extension of this concept. As defined in an influential perspective article on the topic, phenotypic robustness is "the reduced sensitivity of a phenotype (...) with respect to perturbations in the parameters (genetic and environmental) that affect its expression" (de Visser et al 2003, p. 1960). That is, since a phenotype is the result of genetic and environmental interactions, robust phenotypes are those that result from many different such interactions, or, in other words, they are those developmental outputs that remain relatively invariant under a number of different inputs (Masel & Siegal 2009). For example, the body axis determination of flies mentioned in the previous chapter (section 4.2) and the segmentation it allows for are genetically

robust traits in the fly species *Drosophila melanogaster*, which develops a stable segment pattern under different initial molecular values (von Dassow et al. 2000). Robustness is the result of a variety of so-called "buffering mechanisms", only a few of which are well understood, acting at different stages of the developmental process. A buffering mechanism is whatever mechanism—such as gene redundancy—that interacts with a genetic or environmental perturbation and either avoids its interference with the rest of the developing process or compensates for its effect.

Genetic and environmental robustness are often seen as two faces of the same coin. However, their role is arguably different, as involved by their different conceptualization and use in the scientific literature. Environmental robustness is this stability with respect to diverse environmental conditions. These conditions are all non-heritable factors that influence the development of the phenotype, be them the external environment or those internal aspects that are not part of the heritable material. To this regard, developmental systems show "developmental stability", namely the capacity to produce the target phenotype under stochastic perturbations constituting the so-called "developmental noise". Developmental noise is a chancy factor that is explanatorily relevant for the final phenotypic result in particular ontogenies (Merlin 2015). However, large deviations from the target phenotype are oftentimes buffered against, most traits showing some degree of developmental stability and thus of phenotypic robustness.[48] For example, de Visser and coauthors point at "developmental noise caused by fluctuation in the concentration of relevant gene products" as an internal environmental perturbation of the developing system that can be overcome by environmental robustness mechanisms (de Visser et al. 2003, p. 1960). On the other hand, the possible target phenotypes of a particular genotype will vary with environmental conditions following specific "reaction norms". Even if developmental systems are plastic—i.e. they show variation under different environmental conditions, see epigraph *d* below—this plasticity is canalized and will typically bring about a limited array of phenotypic outputs. The homeostatic behavior of gene regulatory networks and the 'kinetic proofreading' of

[48] By contrast, the degree of deviation of a phenotype from the target due to developmental noise is called developmental instability. A typical way to quantify these properties for morphological traits is by measuring their fluctuating asymmetry, i.e. their deviation from a symmetric form.

transcriptional mechanisms are some of the basic characters that provide organisms with environmental robustness (Masel & Siegal 2009).

In opposition to environmental robustness, genetic or *mutational robustness* is the stability of a target phenotype with respect to diversity in genotypes. This property is at the core of the lack of 1-1 correlation between genotypes and phenotypes, because it entails that many different genotypic combinations map into the same phenotypic result, that is, it entails the multiple *genotypic* realizability of a given target phenotype (Figure 3.5). Genotypic robustness is tantamount to the phenotypic neutrality of some mutations as mentioned in the previous chapter (section 2). However—and importantly—, this neutrality does not merely refer to a lack of effect on fitness, but to the more fundamental lack of effect in the resulting phenotype. A phenotype can change while preserving its fitness value, and thus many neutral mutations with respect to fitness may not be neutral in the relevant sense for phenotypic robustness. For example, the genetically grounded variation in color and shape in the shells of grove snails is completely random with respect to their probability of being stepped on by larger animals (Futuyma & Kirkpatrick 2018). A phenotypically neutral mutation, by contrast, is a genotypic change that is either not expressed or buffered against through development, regardless its potential effect on fitness if expressed.

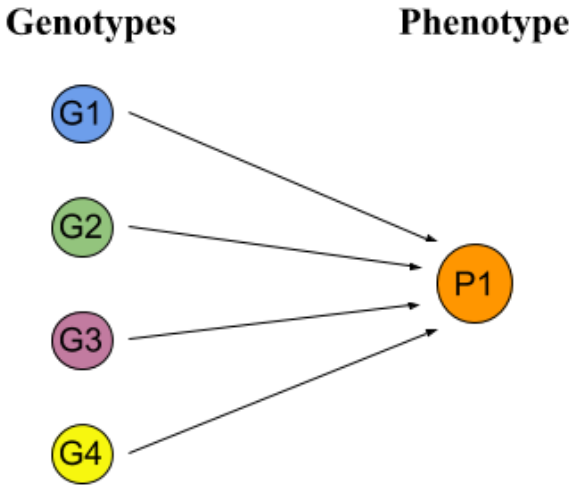

Figure 3.5. A mutationally robust G-P map where many genotypes map into the same phenotype.

A buffering mechanism against mutational perturbations is epistatic by definition, that is, its nature is interactive with the expression of the mutation. The most basic form of mutational robustness is dominance, where the dominant phenotype is more robust to mutations than the recessive one. Other forms of robustness include the redundancy of parts on the one hand and the complex interactions of a given system on the other (de Visser et al. 2003) (see Figure 3.6). For instance, redundant genes and proteins evolved by gene duplications enhance robustness by reducing the impact of those mutations affecting them. Since the same genotypic expression can be carried out by different copies of the same genetic material, mutations that may modify such expression will have a lower effect on the phenotype (A. Wagner 1994; 1996b). By contrast, non-redundant buffering mechanisms are responsible for "distributed robustness" (Felix & Wagner 2008), namely the phenomenon that many components of a system contribute to a particular effect without none of them being redundant. For example, the suppression of individual reactions in metabolic networks may not have effects on the metabolic overall function even when individual enzymes catalyze different chemical reactions.

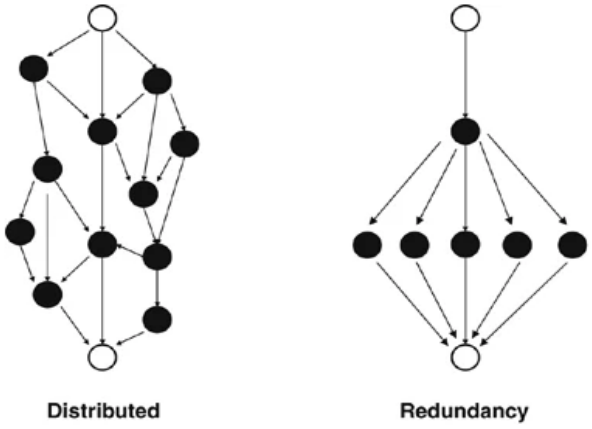

Figure 3.6. Representations of distributed and redundant robustness. "If a pathway like this shows distributed robustness (left), it is robust because the flow of information is distributed among several alternative paths, with no two parts performing the same function. In contrast, if robustness is achieved through redundancy (right), several components perform the same function." Reproduced with permission from (Felix & Wagner 2008).

This variety of mechanisms highlights the non-reducible nature of robustness as a general tendency or disposition of both individuals and types of developmental systems. Particular mechanisms may realize robustness in one or other situation. However, robustness is a disposition to manifest a target phenotype under different stimulus and generating conditions, and thus cannot be reduced to any particular mechanism. In this regard, this property is conceptualized as a disposition to recompose the developmental system under perturbations in turn conserving its integrity and showing persistence, regardless of the particular mechanisms instantiating it (Austin 2017). Moreover, genetic robustness is not a property of particular individuals—contrarily to homeostasis and environmental robustness—but a property of a G-P map, that is, a property of the developmental pathways mapping genotypic to phenotypic variation. In this regard, robustness is a property of the genotype-phenotype relation, insofar as it refers to a many-to-few mapping function. That is, the robustness of phenotypes is manifested in the perseverance of the target phenotype in many different genotypic contexts, and thus cannot be seen as a property of one single organism.

In the neutral network approach to evolution introduced with the RNA model, robustness—also referred to as *neutrality*—is represented by the degree of connectivity of the genotypic space (see Figure 3.3). A high connectivity in a G-P map accounts for the robustness of the phenotypes represented in it. So the larger the range of genotypic space a neutral network occupies, the most robust to perturbations its target phenotype is. A node inside a neutral network is thus typically robust to mutational changes, unless it lays very close to a neighbouring area. This is easily perceived in the RNA model, where large areas of the genotypic space of nucleotide sequences map into the same secondary structure. Through this characterization, it is easy to see how relevant it is to distinguish genotypic from phenotypic robustness. While genotypic robustness is "the fraction of neighbors with the same native phenotype as a given (...) genotype *G*," phenotypic robustness is "calculated by averaging the genotypic robustness of all [the] genotypes" composing a specific phenotype (Wagner 2014 p. 961). If we focus on a particular point of the genotypic space—thus in an individual genotype—robustness clearly imposes a

limitation to variability. A genotype with a very robust phenotype will probably produce offspring with the same phenotype, since it is surrounded by other members of the same neutral network. Thus the probability that a point mutation for a given sequence results in a phenotypic change can be low. However, if we focus on the entire phenotype, the limitation does not hold. On the contrary, since it will occupy a wider area in the genotypic space, a robust phenotype is more likely to have a higher number of neighbors than a non-robust one. Thus the individual disposition to produce offspring with the same, canalized phenotype contrasts with the G-P map disposition to bring about variation. This observation has been highlighted by the evolutionary and systems biologist Andreas Wagner, one of the most relevant authors arguing for the importance of phenotypic robustness in bringing about novel phenotypes and enabling adaptive change (A. Wagner 2008a, 2008b, 2012b). The evolutionary significance of this property lies in its being predicated of the developmental pathways linking genotypic to phenotypic variation. In turn, whether or not particular individuals—or populations—are prone to generate offspring with phenotypic change is not as explanatorily relevant in the long term as the fact that some G-P relationships are more robust or variable than others. This is because an individual or a population may be confined in a remote neutral area of the genotypic space and thus have a very high local robustness. However, in the long term the population may drift to other areas of the neutral area closer to neighbor phenotypes, therefore showing a higher degree of variability. In turn, robust phenotypes are more likely to have higher values of variability in the sense that a higher number of neighbors will surround the neutral network.

The main idea behind this connection between robustness and evolutionary potential is that populations can explore larger regions of the genotypic space while conserving their phenotype, which in turn increases their probability of encountering novel, selectable phenotypes. Recall that a population can move inside a neutral network without changing its phenotype. However, since different nodes inside the network represent distinct genotypes, the result is that populations can accumulate a variety of genetic backgrounds that do not affect the original phenotype but may become important for the expression of a different phenotype if it reaches a

neighboring area. In population genetics, this is usually labeled as "cryptic variation", namely genetic variation that is not expressed under the environment it evolved—thus evolved neutrally or as a side-effect—but may express itself under specific environmental or genetic (epistatic) changes (Payne & Wagner 2014, Zheng et al. 2019). In sum, the robustness of phenotypes, even if it represents a limitation of phenotypic variation by definition, plays a central role enabling the viability as well as the variability of complex biological systems in a broader sense.

c) Modularity

An important capacity from an evolutionary point of view that developmental systems show is their modularity. Organisms are modular, that is, they are composed of "quasi-independent" units that function with certain independence of each other but are "tightly integrated within themselves" (Schlosser & Wagner 2004, p. 1). This property refers to the relative autonomy of different aspects of a system from one another, and to the relative internal integration of each of these aspects. In turn, modularity refers to the extent of decomposability of the system: a system is modular if it is possible to decompose it into different parts with a certain grade of autonomy. As in the case with most propensities, modularity is not only a dispositional property—the disposition to be separated into parts—but also a matter of degree: some systems are more modular than others. Modularity is a key concept in evo-devo, as shows the recent literature, including two highly influential edited volumes (Schlosser & Wagner 2004, Callebaut and Rasskin-Gutman 2005). In addition, it has gained attention among philosophers of biology (Brigandt 2007, Austin 2017). The reason is that modularity enables us to reflect on the units of development and evolution beyond the gene and population levels.

It is easy to find modularity in designed objects. An extreme example is construction block toys, which enable the assembly of modules in order to build different structures. Every structure can be separable into its building modules without any loss of the integrity of each of them—unlike the integrity of the whole structure they are part of. The blocks can also be functional units on their own. As an example, if you build a castle with these blocks that has a big tower in it, it is possible

to separate the tower from the rest of the castle without damaging its "functionality" as a tower. This is not the case with other modular structures. For example, a watch can be separated into its different components, but most of them will not be functional in the absence of the rest. A watch piece such as a minute hand does not perform any function independently of other parts composing the watch engine (Leibniz 1978/1695, see Wolfe 2010).

Not surprisingly, organisms are modular in a much more complex way. Both the integration and the autonomy of organisms and their parts show a high degree of complexity, a variety of factors being relevant for the individuation of biological modules. To an extent, biological modules can be individuated by their insensitivity to certain changes in their context. Interestingly, this context-insensitivity of modules will depend on the frequency of certain types of changes taking place in the particular environment, rendering modularity a relative concept (Schlosser & Wagner 2004). Moreover, modules may be embedded in higher-order modules in a hierarchical fashion, there being many levels for their individuation: gene regulatory networks, cell types, organs or body parts (e.g. limbs) are examples of modules at different levels of organization. Therefore the same organismal component such as a specific tissue can be a relatively integrated part of a particular organ, and also be part of an autonomous module at the organismal level. Similarly, a gene regulatory network can constitute an independent module in the development of a specific part, but it might be still considered an integrated component of this part with respect to a different level of organization, such as the whole organism. Moreover, the ways in which organismal modules are connected may be manifold:

> The connections [between modules] can be physical, for instance, protein–protein interactions or amino-acid contacts within a protein, or they can be dynamical, as in the case of gene regulatory networks, or statistical, such as the pleiotropic effects of genes causing correlations among phenotypic traits ... Modularity thus refers to very different kinds of connections and elements; however, it can still be considered a uniting principle. (Wagner et al. 2007, p. 921)

Therefore modularity is a property that abstracts away from particular mechanisms and their complexity, and intends to capture a basic rationale of

biological organization: organisms are composed of differentiable parts. Far from a triviality, this idea is central for the individuation of biological traits. On the one hand, since different organisms can be composed of the same parts, understanding modularity is crucial for the recognition of body plans and homologous characters (Wagner 2014). The same module can be found under varied forms in different species or even higher taxa, the concept of modularity thus providing a basis for the study of homology. As we saw in the previous chapter (section 4), studying homologous traits is a key aspect of the evo-devo research agenda, as exemplified in the paradigmatic example of the many forms in which the vertebrate limb type is instantiated across different taxa: the same module of organization is present in avian, reptile and mammal species. Why has the same part been preserved throughout evolutionary time in such different groups? Has it evolved in a specific way? As we shall see, the evolutionary importance of modularity resides in that modules articulate not only the internal organization of living beings (organismal modularity), but also the ways in which organisms can evolve (variational modularity). On the other hand, it is not uncommon to find several instances of the same module in an organism. As also mentioned in the previous chapter, serial homology refers to the phenomenon where the same modules are repeated in different locations and at times as part of different functions in the same organism. The segmentation of arthropods and vertebrates is paradigmatic: in their bodies, segments are repeated several times across the anterior-posterior axis, such as our own vertebras arranged across the column. Leafs in plants and feathers in birds are also examples of this repetition of modules. In this context of versatility of organism components, which is an increasing focus of attention among evolutionary biologists, Gerhard Schlosser and Günter Wagner reflect:

> The recent excitement about modularity stems largely from accumulating evidence that some of the modular units of development were highly preserved but promiscuously recombined during evolution (Schlosser & Wagner 2004 p. 6)

Thus the decomposability into modules of living beings has an important evolutionary significance: modules can be units of evolutionary variation and

recombination, not only in the sense of being variably present throughout species, but also because modules can be repeatedly present in the same organism. Thus, there are several senses in which a living being can be considered decomposable, and it is common thus to distinguish different ways in which organisms can be modular (Wagner et al. 2007). On the one hand, organisms may show functional modularity, meaning that the functions of their parts can be relatively abstracted away from one another. This implies that the performance of some particular feature can be relatively autonomous from other functions. It is an important notion from the point of view of evolution, since it allows for the relative autonomy in the *selection*—broadly considered—of traits. Environmental changes, be them internal or external, may select for the adjustment of a certain organismal function without needing for changes in any other functional aspect. For example, the environment of a species can change so that a change in its diet is beneficial. Selection would thus enhance changes in the nutritional function, without necessarily favoring any other type of functional change. The characterization of this type of modularity is typically dynamical: certain processes—regardless the mechanisms underlying them—can be individuated with respect to others as having a separable, relatively context-independent function (Wagner & Mezey 2004). Moreover, functional individuation of modules can also take place at the level of molecular processes. In this regard, the field of comparative developmental genetics is full of examples of homologous molecular processes or modules that retain the same function in different organisms. An example is similar genes performing the same function in different species, such as Hox genes in the formation of the anterior-posterior axis of animals, as we saw in the previous chapter (section 4).

On the other hand, organisms can show developmental modularity, or dissociability (Gould 1977). This type of modularity refers to the relative autonomy of body parts when undergoing their generation independently from other ones. It refers to embryonic parts "that can develop all or most of its structure outside its normal [developmental] context" (Wagner & Mezey 2004, p. 339). Developmental modules are related to the historical notion of morphogenetic field, referring to the spatially differentiated, ontogenetic precursor of a particular morphological structure

of the organism. Morphogenetic fields correspond to differentiable collections of cells in the embryo that are "isolatable, transplantable, and well-characterized landmarks" (Gilbert et al. 1996, p. 365). For example, the morphogenetic field of the limb bud is a collection of mesoderm cells formed in early development. Once this structure is present, limb formation constitutes a relatively independent developmental module insofar as the formation process takes place with a high level of invariance with respect to the organismal environment, as empirical research shows (Young et al. 2010).

Finally, and in connection with the two preceding modes of modularity, organisms show *variational* modularity, or the capacity to vary in modular ways. This sense of modularity is the most important one from the evolutionary point of view (Wagner & Altenberg 1996). Unlike functional and developmental modules, variational modules are components of a G-P map: it is the disposition to translate genotypic variation into phenotypic variation in a modular way (Figure 3.7). Modularity refers in this context to the fact that the variation affecting a particular module will be decoupled from the variation affecting a different module, while variation affecting parts of the same module will be integrated. Variational modularity can be understood as the propensity of the G-P relation to generate a "coherent and autonomous response to heritable variations" in the different modules that compone it (Schlosser & Wagner 2004, p. 7). The integration of variational modules is achieved through the pattern of pleiotropic effects characterizing the G-P relationship. Pleiotropy refers to the phenomenon where the same gene or genome fraction affects different traits of the organism. Thus the more pleiotropic relationships between organismal elements—that is, the more they are affected by the same mutations—the higher their integration into a variational module. This property allows for the evolutionary individuation of modules or characters, namely their definition as units of evolutionary significance. For example, limbs constitute a variational module in most tetrapod species. Variation in limbs is typically highly integrated—it will affect the four limbs in a coordinated fashion—as well as relatively independent from variation in other body parts. However, in some tetrapod species, hindlimbs and forelimbs act as two distinct variational modules because of the lower

level of pleiotropic connections between the two. In humans, for instance, variation affects arms and legs distinctively (Young et al. 2010, Hallgrímsson et al. 2002). In contrast, the high pleiotropy between left and right arms makes very likely that variation is coordinated in the two. The same goes for left and right legs.

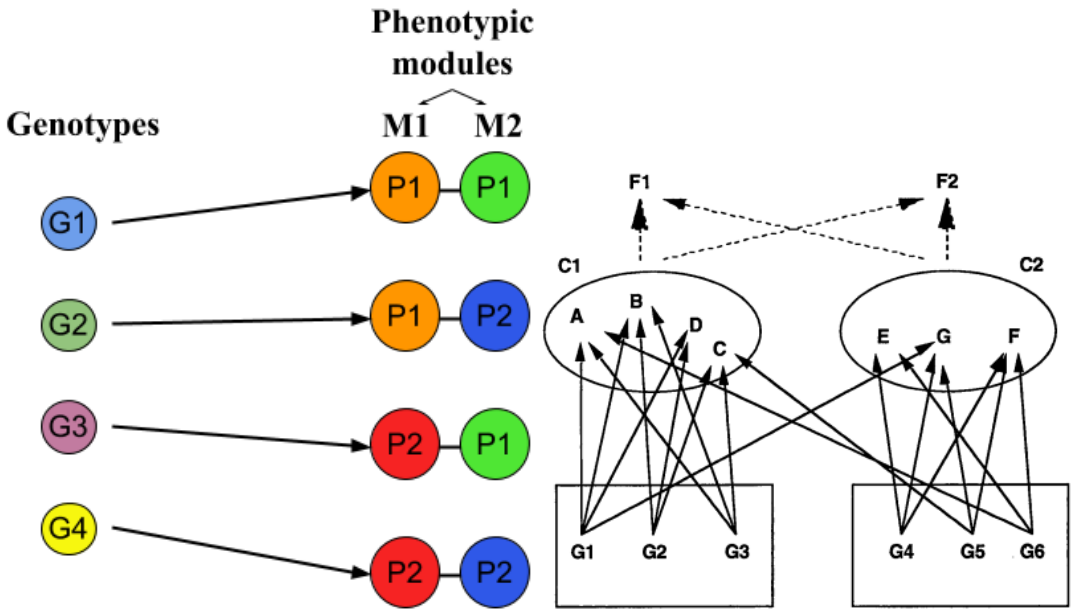

Figure 3.7. Left: a very simple modular G-P map, where different variants of modules 1 and 2 are not correlated. **Right**: modular G-P variation, where each genotypic variant has pleiotropic effects that are (mostly) confined to a particular module (Reproduced with permission from Wagner & Altenberg 1996).

The capacity to vary in a modular way is in practice connected to both functional and developmental modularity, although there is no complete overlap between the different types of modularity. On the one hand, developmental modularity allows for evolutionary changes such as heterochrony and heterotopy, namely changes in the timing and espacial location, respectively, of the expression of developmental modules (Schlosser & Wagner 2004). Moreover, a module that is selected independently is more likely to vary independently and vice versa. On the other hand, variational independence is not ensured by neither developmental nor functional modularity. The fitness effect of a variational module need not be separable from other units of variation, while a developmental module is not always a unit of variation either. In the human limbs example above, left and right arms constitute different developmental modules despite comprising only one variational

module. Even if mutations tend to affect both limbs together, their ontogenetic formation occurs with high independence from each other. Therefore, it need not be the case that everything that constitutes a unit of variation—a separable unit that varies independently of others—behaves as a unit at the developmental level.

Nevertheless, and despite this lack of complete overlap, the distinct degrees of physical connection in development influence variational linkage among phenotypic traits (Wagner et al. 2007). In order to get variational modularity, the developmental pathway relating genetic to phenotypic variants has to be autonomous from other types of variation. Thus variational modularity—and not only developmental modularity—is a property of developmental systems: developmental pathways allow for the modular variation of body parts and organismal elements. The difference resides in that variational modularity is a property of how *variation* at the genotypic level transforms into *variation* at the phenotypic level—i.e. it is a property of the G-P map. In this regard, a module can be seen as "a unit of heritable phenotypic variability" (Brigandt 2015a, p. 712), thus showing the capacity to vary its phenotype under genetic variation in a relatively autonomous fashion. In turn, variational modularity provides means for the individuation of characters as units of morphological evolution. Indeed, developmental modules have been characterized as the "ontological elements" of evo-devo (Austin 2017, p. 377), inasmuch as they constitute discrete units of evolutionary and developmental transformation. In the study of homology, Günter Wagner (2014) differentiates between character identity (a module) and character state (different instantiations of the same module). Following this terminology, a variational module is a character with the capacity to instantiate itself in a variety of character states independently from other modules. Thus a G-P map showing variational modularity has the disposition to vary the different modules that compose it (Austin & Nuño de la Rosa 2018), that is, to explore the different areas of the morphospace separately. As a consequence, the dimensionality of the explorable morphospace for a given G-P map will be proportional to the number of variational modules composing it (Brigandt 2015a).

Modularity has been studied in the RNA model. Some RNA molecules have a modular secondary structure where two or more substructures can be differentiable.

In their study of RNA variational properties, Lauren Ancel and Walter Fontana (2000) studied the reaction of secondary structures to strong temperature changes, reporting RNA sequences with "a highly localized melting behavior" (p. 276), thus showing a dispositional property analogous to developmental modularity. The secondary structures of these sequences were composed of differentiable structural components with specific response to temperature change. They found that some parts of the RNA molecule structure can undergo variation without compromising the general integrity of the molecule. In this study, they define the modularity of an RNA molecule as "the thermophysical independence of a structural trait from other traits over a wide temperature range" (Ancel & Fontana 2000, p. 277). Moreover, they studied the behavior of these molecules under mutations, and found that their structural components also showed variational modularity. In their review of modularity in RNA, Ancel and Fontana (2005) define RNA modularity in the following terms:

> the partitioning of molecules into subunits that are simultaneously independent with respect to their thermodynamic environment, genetic context, and folding kinetics (Ancel & Fontana 2005, p. 129)

For the present purposes, the independence of these units is particularly interesting with respect to the genetic context, insofar as it points at the capacity of RNA to target the effects of genetic changes to particular structural subunits. Thus in the RNA model, variational modularity corresponds to alterations of nucleotide sequence that affect only a particular subunit of the secondary structure of the molecule, leaving the rest of the shape unchanged. Therefore the partitioning of RNA molecules into independent subunits reflects their variational modularity as a disposition to vary molecular secondary structure in a modular way, that is, to produce structural variation in the subunits composing them independently. In this regard, given a specific RNA G-P map, it is possible to derive a probability measure of modular change under mutation or change in the thermodynamic environment. Perhaps more interestingly, it is possible to regard modularity as the relation of independence between the probability of a change in one structural module and the

probability of change in another module. For instance, in a further study Ancel and Fontana stress that variational modules in RNA "maintain their original shape with a much higher likelihood than the fragments of random sequences with the same shape" (Ancel & Fontana 2005, p. 136). In particular, the same RNA secondary structures presented 0.83 and 0.94 probabilities of remaining stable upon mutations when they had evolved modularity, contrasting with the 0.017 and 0.015 probabilities respectively when they were instantiated by random, not modular sequences.

Variational modularity is a disposition of developmental systems represented in G-P relationships that cannot be reducible to specific mechanisms. In particular, the mechanisms underlying pleiotropic patterns in species are extremely diverse. Instead, variational modularity makes reference to the capacity of a developmental system to generate variation in the different modules that compose it independently, without need of specification of the mechanisms implementing it. A modular G-P map enables the exploration of different regions of the phenotypic space in combination: exploring the region of a specific trait need not affect the exploration of any other region. In sum, the modularity of a G-P map is a central dispositional property that enables the independent evolution and recombination of traits.

d) Plasticity

A different dispositional property of developmental systems of interest may sound in conflict with what has just been said above at first sight: phenotypic plasticity. Phenotypic plasticity has a straightforward relevance for variability insofar as it refers to the ability to reach different phenotypes by the same genotype, therefore referring to the capacity of an individual system to generate variation. Plasticity then manifests itself in an array of possible states the same developing system can achieve under different triggering—environmental—conditions.

In a general, non-biological sense, plasticity is the disposition to undergo changes—typically in shape—in response to different stimuli. Plastic materials are contrasted to elastic ones—although almost every material shows a degree of both—in the sense that they are irreversibly modifiable whereas elastic materials recover their original form once they have been perturbed. For example, moised clay

responds to the application of forces by changing its shape (plasticity), while a rubber band is only temporarily deformed under the application of forces (elasticity). Similarly, unlike the general homeostatic capacity of organisms, phenotypic plasticity typically implies the permanence of the plastic response: rather than showing resilience, the organism will manifest a particular phenotypic variation that may remain even in the lack of the stimulus that generated it in the first place. Accordingly, phenotypic plasticity can be seen as an "environmentally sensitive production" of phenotypic alternatives (DeWitt & Scheiner 2004, p. 2) (Figure 3.8).

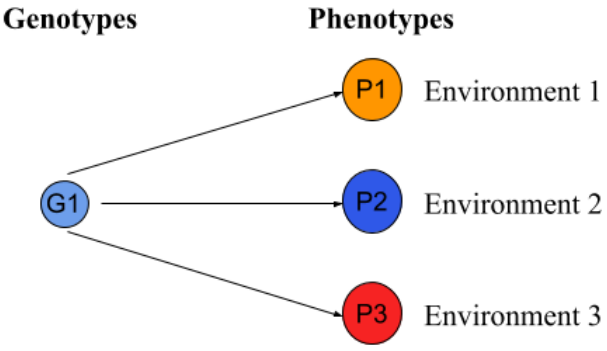

Figure 3.8. A simple plastic G-P map, where the same genotype maps into three different possible phenotypes, depending on the environment where it develops.

Since every biological phenomenon is to some extent influenced by the environment, it is important to distinguish kinds of plasticity from the mere absence of robustness. While genotypic robustness refers to the invariance of a target phenotype for different genotypes under the same environment broadly considered, phenotypic plasticity refers to the variance of target phenotypes for the same genotype under different environments. As in the case of robustness, therefore, defining the plasticity of a particular genotype will highly depend on how the environment is considered: how plastic a developmental system is depends on what is perceived as a sufficiently different environment. For example, judging a phenotype as non-plastic demands that a wide enough range of alleged different environments leave the phenotype unchanged. In this regard, plasticity can be contrasted to *environmental* robustness, although, as we shall see, the contrast does not imply that both concepts are the exact opposite. Very simple cases of phenotypic plasticity include linear functions of the environment, such as plant height within a

range developing under different levels of nutrient concentration in soil: the higher the concentration of nutrients, the higher the plant will grow—within a certain range. Extreme cases, known as "polyphenisms", can be found, for instance, in some beetle species in which males develop horns depending on the diet they follow as larvas (Moczek 2006), or the distinct types of leaves that some semi-aquatic plants develop under and above water (Wells & Pigliucci 2000). The existence of different degrees of plasticity and the fact that some plastic responses are adaptive suggests that, at least in some cases, this property has evolved (Pigliucci 2010b).

Plasticity can take many different forms. Some plastic responses are considered "active" while others are "passive", the distinction being in the anticipatory nature of the response. For instance, exploratory behavior in plants, where they seek for light in order to develop in the most convenient direction, is a case of active plasticity (West-Eberhard 2003). This leads to the question of how to distinguish plasticity both from behavior and physiological reaction. Although there may be some overlap among the three phenomena, the basic idea is that phenotypic plasticity involves development (Pigliucci 2001). In fact, Mary Jane West-Eberhard, one of the most influential authors on this topic, calls it *developmental* plasticity. Thus phenotypic plasticity involves some sort of morphological change that is not only mediated through hormonal and transcriptional reactions, but that "may require a complex cascade of genetic switches and epigenetic effects" in development (Pigliucci 2001, pp. 42-3). Another important distinction is that between continuous and discontinuous plasticity (West-Eberhard 2003). On the one hand, continuous plasticity or phenotypic modulation is a change in the degree of expression of some developmental parameter. Thus the phenotypic change can be seen as a linear function of an environmental variable. On the other hand, discontinuous plasticity, or developmental conversion, is the existence of discrete states of a phenotype. It involves the existence of a threshold in the value of the environmental variable from which a particular phenotype is expressed. Furthermore, plastic responses can be adaptive, if they confer some advantage to the organism under the environmental trigger; or nonadaptive, if they are random in this respect.

Phenotypic plasticity is often represented through a so-called "norm of reaction". A norm of reaction is a function that maps environmental conditions in ontogeny to the range of phenotypes that a particular genotype gives rise to under those conditions. In other words, given a genotype, a function can be drawn between the possible environments it can develop in and the phenotype that will be generated in each of them (Pigliucci 2010b). Note that the mere existence of such a function does not imply plasticity. A reaction norm can be a function mapping from every environment to the same phenotype, and thus show no plasticity at all. However, it is not unusual that a reaction norm will show a certain "slope" in its linear representation, meaning that some characterization of the phenotype is environmentally-dependent.

As stated above, the existence of more or less systematic reaction norms implies that organisms typically show environmental robustness. Thus rather than a mere absence of environmental robustness, phenotypic plasticity refers to the environmental aspect of canalization (A. Wagner 1996a). In other words, reaction norms are canalized and typically confine the phenotypic possibilities to a restricted area of the morphospace. In this sense, plasticity and developmental stability (a key component of environmental robustness) can be seen as different aspects of the same phenomenon, namely the existence of specific target phenotypes for every genotype-environment pair. Moreover, developmental systems show different degrees of plasticity, namely different probabilities of developing an environment-specific phenotype in a given range of environmental conditions. A developmental system is more plastic than other if, under the same range of environmental conditions, it shows a higher number of environment-specific phenotypes than the other, or a broader range of change in a quantitative scale. A measure of plasticity will have to take into consideration aspects such as the relevance of the environment considered or the degree of similarity among the different target phenotypes. In any case, measures of this property will be a probability distribution of target phenotypes, given a specific genotype and a range of environments.

As it is the case with other G-P map dispositions, phenotypic plasticity can be implemented through a variety of mechanisms, and it is thus not reducible to any of

its particular mechanistic instantiations. A significant proportion of them are known as hypervariability mechanisms, typically followed by so-called developmental—also 'somatic' or 'epigenetic'—selection (West-Eberhard 2003). These mechanisms induce random variants in development that are subject to selective preservation, namely differential growth of tissues. As a result, a functional pattern emerges without need of central control. These "exploratory systems" (Kirschner & Gerhart 1998) produce many small, undirected perturbations, only a subset of which will be retained by virtue of its functionality under the environmental condition that triggered the mechanisms in the first place. Examples of exploratory mechanisms are the aforementioned exploratory growth of plants, which grow their parts on a random basis until a particular stimulus channels growth direction; and the formation of the immune system (West-Eberhard 2003). The immune system is formed through the overproduction of random types of antibodies in certain precursor cells, and the subsequent selection of particular antibody-producer cells under the presence of a foreign protein. Another type of mechanism for plasticity are threshold responses in hormone systems, that is, the existence of hormone switches that are activated only under certain environmental conditions. Lastly, there is an increasing body of work that studies the molecular mechanisms responsible for developmental plasticity. An example is the so-called alternative splicing, namely the sensitivity of mRNA translation to conditions of the cell environment (Brett et al. 2002). Other conditions-sensitive molecular mechanisms include transposable elements (Pedersen & Zisoulis 2016) and the folding-mediator chaperone molecules (Sangster & Queitsch 2005).

Plasticity has been studied in the RNA model as well (Fontana 2002). The repertoire of possible phenotypes that a genome can develop can be considered analogous to the energy landscape of an RNA sequence. Although random graphs usually map sequences to its minimum free energy state, a given sequence has a range of possible secondary shapes that move away from thermodynamic equilibrium in different degrees. There exists then a configuration space of these possible shapes a sequence can adopt, determined by the dynamics of the folding process. In this sense, two sequences folding into the same secondary structure in

equilibrium can differ in their energy landscape—thus in their reaction norm. A given sequence can realize a range of alternative structures, navigating its energy landscape, therefore manifesting phenotypic plasticity (Fontana 2002). These changes are a consequence of energy fluctuation, the stochasticity of molecular processes paralleling developmental noise (Ancel & Fontana 2000). A 'plastic map' can therefore be elaborated, where each sequence maps to a well-defined set of secondary structures, and a probability is assigned to every possible shape in virtue of its free energy. Consequently, plasticity can be regarded as a G-P map propensity, namely the propensity of a G-P map to generate variation that is environment-dependent.

As we have seen, phenotypic plasticity is typically defined as a property of individuals. It is individual genotypes that have the capacity to develop particular phenotypes under distinct environmental conditions. However, this capacity is based on the one-to-many G-P relationship this individual potentially has. In other words, plasticity is *trivially* a property of a G-P map insofar as, by definition, only G-P relationships can be phenotypically plastic. That is, phenotypic plasticity is precisely a specific way to map genotypes to phenotypes, namely on a one-to-many basis. In turn, phenotypic plasticity will only manifest itself in a plurality of phenotypic responses to different environmental conditions. However, phenotypic plasticity can be conceptualized at the level of variational modules. That is, units of variation can be units of plasticity as well, a single module being conceived as responsible for producing its own environmentally-based reaction norm (Austin 2017). Now, what about G-P mapping functions with a wider domain? Can they exhibit plasticity that is non-reducible to the plasticity of individual genotypes? For instance, is the plasticity of an RNA model a property of the entire G-P map or a mere aggregate of individual plasticities? Or, is the plasticity of *Arabidopsis* a G-P map property or a sum of the plasticity of each *Arabidopsis* specimen? Importantly, the plasticity of a G-P map can only be considered an abstraction of a cluster of propensities rather than an abstraction of extant properties. In other words, plastic G-P maps are those that assign different phenotypes to genotypes in an array of potential environments. In this sense, plasticity cannot be manifested in particular individuals but in similar individuals under different environments. This observation serves for emphasizing

the fact that determining the bearer of dispositions is an important philosophical task in their characterization as properties. In the next section, we shall deepen further into the bearers of variational dispositions.

Plasticity allows for a more exhaustive exploration of the phenotypic space. If each genotype has an array of possible phenotypes, a broader area of the morphospace is always reachable than in the absence of plasticity. In this regard, plasticity can be seen as the environmental counterpart of variability. That is, while variability regards the reaching of distinct phenotypes under mutational changes, plasticity regards the reaching of distinct phenotypes under environmental perturbations. In addition, the evolutionary relevance of plasticity has been stressed on many occasions, especially with regards to the phenomenon of "plasticity first" evolution (West-Eberhard 2003), namely when a phenotype gets genetically canalized in a population only after it has been first reached through environmental induction. As we have just seen, plasticity is predicated as a disposition of the entire G-P relationship. That is, G-P maps have the capacity to explore the morphospace in a plastic way, an explorative capacity that cannot be reduced to the many mechanisms realizing it in different biological contexts.

2.2 The evolution of G-P map dispositions

In the previous section, we have dealt with the main dispositions of G-P maps that are relevant for the production of variation: modularity, variability, robustness and plasticity. Now, are these properties inherent to all evolving systems or are they evolved properties themselves? Recall that they represent distinct variational tendencies of developmental systems as dependent on the structure that relates their genotype to their phenotype. As we have seen, different systems show different degrees and types of robustness, plasticity, modularity and phenotypic bias, which suggests that these properties are the product of distinct evolutionary processes. But how do these dispositional properties of G-P maps evolve? A major question in the evo-devo literature is the evolution of the developmental properties affecting evolution themselves. This question lies at the crossroads between the two central

research questions in the field: how does development evolve and how does it affect evolution. Interestingly, this question—although mentioned (e.g. Nuño de la Rosa & Villegas 2019)—has been much less explored philosophically, perhaps due to the general lack of consensus in the scientific literature about what is the evolutionary origin of these properties. Saying that G-P map dispositions are the product of evolution is not tantamount to saying that they have evolved by natural selection—that is, that there has been selection *of* them—, and surely not to saying that they have been the direct target of selection—or that there has been selection *for* them (Sober 1984). There is, as a matter of fact, a number of ways in which G-P map dispositions may have evolved, and the scientific literature has been much concerned about finding plausible scenarios for their evolution. In this section I summarise the main alternatives evolutionary biologists consider when they theorize about the origin of these properties, with the intention of exploring the philosophical consequences of them being both a product and a determinant of evolution.

The first scenario to explore is the direct selection for variational properties. This adaptive possibility can be seen as entailing a higher level of selection than the individual, where the tendency to vary in certain ways provides a selective advantage to the group. Directly selecting for variational properties means that these properties have a direct impact on fitness. For example, phenotypic robustness can enhance the fitness of a species. Although group selection is highly controversial (see Okasha 2006), it could be argued that variational properties may increase the relative fitness of certain groups. Interestingly, this scenario was postulated by Dawkins (2003/1988) when he coined the notion of evolvability. According to Dawkins, selection of types of embryologies—he later refers to them as *lineages* (Dawkins 1996)—may explain the evolution of evolvability:

> Perhaps there is a sense in which natural selection favors, not just adaptively successful phenotypes, but a tendency to evolve ... It now seems to me that an embryology that is pregnant with evolutionary potential is a good candidate for a higher-level property of just the kind that we must have before we allow ourselves to speak of species or higher-level selection. (Dawkins 2003/1988, pp. 253-254).

For sure, variational properties have an impact on the evolutionary potential of populations and higher level groupings of organisms. However, this does not entail that they affect fitness at the group level. As discussed in the previous chapter, fitness refers to the capacity for survival and reproduction of individuals, populations and traits, and not to other aspects that are important for evolution, such as the divergence or integrity of parts. Whether variational propensities affect only the latter or group fitness as well is not entirely clear. In this regard, the idea that variational properties are the direct target of group selection is very contested, and most authors agree that other means are more plausible. Nonetheless, variational properties can enhance long-term fitness, in the sense of benefiting offspring in the longer term, for instance by affecting the rate of mean fitness increase in a population (Wagner & Draghi 2010). This way, variational properties can be selected at the individual, rather than the group level.

A second possible scenario for the evolution of variational propensities is their indirect selection, or their evolution as side-effects of adaptations (Hansen 2011). Indirect selection includes a wide array of possible origins for each mechanism instantiating a particular variational propensity. In this scenario, the particular mechanisms are selected at the individual level, in the sense that there is a selective advantage for those individuals bearing the particular mechanisms responsible for variational dispositions. In other words, individuals carrying a specific mechanism for robustness, plasticity, variability or modularity are selected because of the individual ontogenetic effects of the mechanism, entailing the selection of a certain genotype-phenotype relationship at a higher level. The idea behind this is that there is a certain correspondence between environmental and genetic variation, that is, between how environmental and genetic changes affect the development of a trait. For example, the so-called congruence hypothesis states that selection for environmental robustness leads to genetic robustness. An instance is selection on developmental stability, which can result in higher mutational robustness (Siegal & Bergman 2002). For example, individuals with buffering genes for a particular phenotype may be selected when the phenotype they preserve is under stabilizing selection, thus moving the population to a more robust area of the morphospace and

providing the G-P map with genetic robustness (Hermisson & Wagner 2004). Plasticity, on the other hand, can evolve under environmental fluctuations that give an advantage to hypermutable individuals (Pigliucci 2001), while variational modularity can evolve under the selection of functional and developmental modularity (Wagner & Altenberg 1996).

Finally, it is possible that these properties are intrinsic to genotype-phenotype relationships, and that their evolution is a side-effect of the broader phenomenon of the evolution of complexity (Hansen 2006). For instance, robustness can evolve as an intrinsic feature of complex organisms that undergo stabilizing selection (A. Wagner 1996a). That is, biological systems can evolve insensitivity to mutations as a result of their developmental complexity. In this sense, the nonlinearity of developmental interactions has been claimed to be responsible for the emergence of robustness at different levels of organization (e.g. Green et al. 2017). The modularity of traits may be an intrinsic property of G-P maps as well, as illustrates the modular structure of simple molecules such as RNA. As for plasticity, although it is likely that the environmental dependence of traits be an intrinsic property of genotype-phenotype relationships, canalized norms of reaction are harder to fit into this picture, and they are typically considered as evolved under—either direct or indirect—selection (Pigliucci 2005).

The intrinsicness of phenotypic variability is somewhat harder to approach. Is phenotypic variability the result of adaptive evolution or is it intrinsic to some systems? Notably, some important properties that enable variability are intrinsic to the cell and tissue level of organization (Forgacs & Newman 2005). For instance, the physical properties leading tissue formation and growth in the development of organs both limit the possible morphologies the organ can acquire through mutation and enable a specific set of possible morphologies that can be reached. In this regard, both constraints and variability may be a necessary property of multicellular organisms. However, whether or not phenotypic variability evolves as an adaptation remains unclear, for some phenotypic biases are the result of the evolution of other variational properties such as robustness or modularity. For example, the extension

of a neutral network in the genotypic space through robustness can lead a drifting population to approach different areas, making different phenotypes more accessible.

As variational properties are instantiated in a variety of mechanisms, a plausible scenario is that the distinct mechanisms have evolved in different ways irrespectively of which of the variational capacities they instantiate. In any case, these properties are the result of the complex genetic architecture of organisms. In turn,

> Because genetic architecture is a function of general organismal development and structure, it can be affected by basically any evolutionary change in the organism. (Hansen 2006, p. 136)

Thus the study of the evolution of G-P map properties will very likely bring about a very heterogeneous landscape of different, historically-dependent origins for variational propensities. For example, some specific plastic responses may have evolved through selection, while hypervariability mechanisms may be general intrinsic properties of G-P maps. This is not surprising considering the diversity of mechanisms that can be responsible for this property, and the same rationale goes for each of the discussed variational dispositions. Despite this plurality, it is interesting to note that these capacities are ubiquitous in the living world, and that their evolutionary significance, as we shall see, is comparable across very diverse species and taxa.

Another subject to note is that the evolution of these properties may entail intricate connections among them, such as selection for one resulting in indirect selection for the other. For instance, the connections between the evolution of robustness and variability (A. Wagner 2005), as well as between modularity and variability (Wagner & Altenberg 1996) have been considered in depth in the literature. Moreover, robustness and phenotypic plasticity have been argued to be "two cases of the same phenomena", namely the "evolution of the dependency of phenotype on some environmental factor" (de Visser et al. 2003, p. 1961), where a trait can be considered plastic or robust depending on the level of organization where the focus is made. Other forms of connections have been defended. For example,

West-Eberhard (2003) notably argued that developmental modularity follows from plasticity:

> As differentiation evolves to produce specialized parts and an internal division of labor, internal heterogeneity gives rise to conditional switches between developmental pathways. The result is a structure characterized by somewhat discrete parts—modularity (West-Eberhard 2003, p. 34).

The evolution of G-P map dispositions has been explored through the RNA model. Computer simulations in this model, as well as experimental settings, have proven fruitful in the exploration of how variational properties can originate in evolution. An important property of this model is plastogenetic congruence, namely the fact "that plasticity and variability mirror each other" (Ancel & Fontana 2000, p. 243). Thus in a neutral network representation of the model, such as the one introduced in section 1.3a above, the shapes in the plastic repertoire of a particular sequence coincide with the target shapes of their mutational neighbors. That is, there is a correlation between the target phenotypes of environmental and genotypic neighbors. As a consequence, when phenotypes are canalized, a correspondence between genetic and environmental robustness will obtain in this model, as well as a negative correlation of each type of robustness with phenotypic variability and plasticity, respectively. Although there are severe limitations for generalizing this property due to the complexity of other G-P maps, the results are in line with theoretical approximations to the evolution of the G-P map (e.g. Wagner et al. 1997, de Visser et al. 2003) and have empirical support (e.g. Gibson & Hogness 1996). Another interesting result of these studies is that RNA molecules evolve modularity under environmental canalization (Ancel & Fontana 2000, Fontana 2002). Moreover, they tend to increase their robustness under stabilizing selection, creating big areas of neutrality (A. Wagner 1996b). Importantly, these evolved properties increase the variability and possibly the evolvability of the system. For example, selection on plasticity "indirectly curtails phenotypic novelty accessible by mutation" in the genotypic space (Ancel & Fontana 2000, p. 280). More generally, as mentioned above, the evolution of robustness may enhance variability by enabling a wider—and "safer"—exploration of the phenotypic space (A. Wagner 2005, 2008b).

Although directly extrapolating from the RNA model to all evo-devo G-P maps can be misleading, the fact that a simple model such as this evolves variational tendencies that are analogous to the properties of complex G-P maps throws some light into the mere possibility of their evolution. As Pigliucci (2010a) puts it,

> RNA folding... may yield important clues to how historically mapping functions [between genotypes and phenotypes] got started and became more complex and indirect. (Pigliucci 2010a, p. 558)

In other words, studying the evolutionary behavior of a simple G-P map whose properties can be depicted is a first step towards understanding how more complex G-P maps are built up in evolution. To sum up this section, although the precise evolutionary origin of variational tendencies is for the most part unknown, several possibilities are contemplated in the literature, manifesting that their evolution is plausible in many possible scenarios and pointing at the rather surprising fact that they are present in very distant biological systems under diverse mechanisms.

The presentation of G-P map variational dispositions and their evolution in this section shall serve for considering what are the aspects of variational tendencies studied in evo-devo models. Throughout the section, we have seen that these properties characterize the evolutionary and developmental behavior of all living systems to a larger or lesser degree, and that the main approximations to them in the evo-devo literature are made in dispositional terms. In order to articulate the evolutionary significance of these variational dispositions, in the next section I approach them from the causal propensity framework developed in Chapter 1.

3. Causal propensities of developmental types

So far, in this chapter I have dealt with the representation of developmental variation in evo-devo models (section 1) and with the kinds of variational dispositions of developmental systems these models represent (section 2). In this third section, I explicitly apply the framework of causal propensities to these properties. This exercise is an implementation of the ideas presented in Chapter 1 about chance and causal explanation to the problem depicted in the second chapter, the so-called

problem of variation in evolution. My will is to illustrate how a causal understanding of developmental tendencies, as responsible for the patterns of variation encountered in evolution, can make sense of the nonrandom generation of the "sample space of variation", from which only a small subset represents extant variation on which selection and drift act upon. The existence of different probabilities to vary that depend on the mapping relationship between genes and phenotypes suggests that, as it is the claim of evo-devo advocates, development indeed plays an irreducible role in evolutionary explanations and, consequently, in the generation of this sample space.

As it has been patent in the previous section, variational tendencies have been interpreted in the philosophical literature as dispositional properties (e.g. Austin & Nuño de la Rosa 2018). Moreover, a relationship between variability and causal probability and propensities has recently been vindicated (Nuño de la Rosa & Villegas 2019). In this section, I consider these ideas in more depth, expanding them to the dispositions reviewed in the previous section, and fit them into the causal propensity view developed in the first chapter. Moreover, I also provide a first step towards the inclusion of the philosophical treatment of population dynamics probabilities—such as the causalists-statisticalists debate reviewed in the second section of Chapter 2—to the discussions on how evolutionary variation is generated. This endeavour will lead me to revise the typological nature of the evo-devo approach in some detail, and its consequences for a notion of chance in variation. In other words, in introducing a causal propensity view of the so-called problem of variation, I expand the range of application of some philosophy of probability and chance conceptual tools to the other main component of the evolutionary process, namely the generation of variation. In order to do so, I first explore the dispositional nature of these properties in terms of their manifestations, their triggering conditions and their effects, as well as the causal modeling leading to their postulation (section 3.1). Then, I review how these properties enable a causal understanding of the generation of variation in similar terms to those developed in the previous chapter for ecological causes (section 3.2). Finally, I introduce developmental types as the bearers of these probabilistic dispositions (section 3.3).

3.1. Characterizing variational propensities

In contrast to the general statistical approach of evolutionary genetics, evo-devo has been generally understood as a mechanistic science (Müller 2007, Wagner et al. 2000, Baedke 2020), where developmental mechanisms explain phenotypic transformations. However, we have seen that, considered as capacities to generate variation in specific ways, evo-devo variational tendencies are typically referred to in dispositional terms in the scientific literature. Indeed, the classical mechanistic approach (Craver 2006) seems to fail short in accounting for the variational potential of developmental properties (Brigandt 2015a). In this situation, and as mentioned above, Christopher Austin argues that evo-devo is "a science of dispositions" (Austin 2017). He identifies developmental modules, defined as the regulatory networks giving rise to specific morphological units, such as the eyes or wings, as the basic "ontological elements" in evo-devo. Austin argues that the motifs of an ontology of dispositions capture the basic features of these modules, understood as units of development and evolutionary variation. In particular, the functional individualization, multiple realizability and modal nature—in Austin's terms, goal directedness—(see section 3 of Chapter 1) of developmental modules are their key characterization. Developmental modules or dispositions *à la* Austin are individualized by the manifestation "of a definite range of variations" on a specific morphological structure, in turn "specifying a demarcated morphospace" (Austin 2017, p. 379). As dispositions, developmental modules have the capacity to generate phenotypic stability and diversity by virtue of their causal structure, which is dynamically oriented to the consequence of a phenotypic outcome. Their significance is both developmental and evolutionary, insofar as they are conceived as both units of ontogenetic variation and stability and units of evolutionary diversification and unity.

However, let us recall that units of evolutionary variation need not be identified with units of developmental variation. As reviewed in the previous section, the variational dispositions of G-P maps are not *developmental modules* in the sense

of units of development. Rather, they are abstract properties of developmental systems as determined by their possible *ways to vary upon reproductions*. Moreover, these dispositions reflect properties of development, but they don't imply that developmental systems *are* dispositions *per se*. Even if it may be argued, with Austin, that developmental units of transformation are dispositions, my claim is about the nature of certain developmental inferred properties, and does not regard the ontological status of developmental processes or systems. In turn, although Austin's approach is a decisive starting point for thinking about evo-devo dispositions, I believe it is important to separate G-P map dispositions from developmental modules. General variational tendencies of G-P maps such as variability, modularity, robustness and plasticity have specifically been analysed as dispositions only very recently (Austin & Nuño de la Rosa 2018). According to this view, the dispositional language ingrained in evo-devo studies of variation manifests its interest for the possible in evolution, a reflection of how their models tend to "appeal to the efficacy of the intrinsic, dynamic capacities of developmental systems to shape the course of evolution." I take this recent perspective as my starting position, from where I further want to argue that these dispositions are causal propensities in the sense depicted in Chapter 1, in turn explaining the probabilistic potential of evo-devo models of variation.

a) Variational dispositions are causal propensities

It should be clear by now that G-P map variational properties are functionally individualized by their manifestation, and that they are multiply realizable by an undefined number of possible developmental mechanisms. On one hand, variability, robustness, modularity and plasticity are identified as those properties responsible for variational, robust, modular and plastic behavior in evolutionary change, respectively. In other words, what individualizes and defines these properties is their function in providing specific types of variation. On the other, developmental mechanisms realizing these properties are very diverse, different mechanisms providing the same disposition to change in evolution in different contexts. For example, we saw that allelic dominance and redundancy in regulatory networks are

two different mechanisms that confer robustness to a developmental system. These mechanisms act as the causal bases of G-P map dispositions, in turn being causally efficacious every time they are triggered. They are responsible for particular effects of them but, importantly, these mechanisms are not identifiable with variational dispositions. Besides being functionally individualized and multiply realizable, we saw in the third section of Chapter 1 that the characterization of dispositions includes their modal nature and the distinctions between manifestation and effect, between intrinsicness and extrinsicness, and between surefire and probabilistic dispositions. Let us see this now in some detail.

The dispositional language of variational properties is a manifestation of evo-devo's modal epistemic agenda, that is, of its interest in the possible in evolution as contrasted with the actual (Austin & Nuño de la Rosa 2018, Nuño de la Rosa 2014, Eble 2003). Evo-devo study of variational tendencies is concerned with the exploratory nature of developmental systems with respect to the morphospace, and, in this regard, variational dispositions are an instance of evo-devo's interest in possible evolutionary trajectories as determined by developmental systems.[49] Thus the properties under interest for the study of developmental tendencies are not reducible to categorical properties from which the possible may supervene. As dispositions, G-P map variational properties play a particular theoretical role (see Choi and Fara 2018 for the theoretical role of dispositions) that comprises the functional and dynamical behavior of complex biological systems, without demanding any kind of essentialist definition that could reduce them to categorical properties. In this sense, and in the line of many other dispositions in biology (see Hüttemann & Kaiser 2019), variability, modularity, robustness and phenotypic plasticity are not only functionally *individualized* but also, and importantly, functional in nature. What identifies G-P map variational properties is their causal role in dynamical processes—e.g. bringing about different types of phenotypic variation—rather than what they are essentially. For instance, the causal nature of

[49] To be sure, comparative evo-devo does care about extant differences among developmental systems. It is the developmental evolution research agenda that primarily focuses about the possible: which possible evolutionary transformations can developmental systems bring about (see section 4.2 in Chapter 2 for this distinction).

variational modularity, namely enabling modular variation, is an epistemic target of evo-devo on its own, playing an irreducible explanatory role and at the same time being an independent *explanandum*. The causal activity *per se* is an essential component of the explanatory agenda of evo-devo, independently of the additional interest that specific causal bases—perhaps categorical in nature—and effects of this causal activity have in other branches of evo-devo.

The causal role of these properties is, of course, relative to a given context. We have seen throughout the previous section that G-P map variational dispositions are context-dependent and gradable, in turn manifesting with different strengths and always relative to a state of affairs. Thus while these dispositions are intrinsic to a developmental system as represented in a G-P map—that is, their causal bases are intrinsic to the developmental system—, they are necessarily relative to a given context. Recall the contrastive nature of explanations (Lipton 1990) remarked in Chapter one (section 3), according to which we typically explain why something is the case *rather than not being the case*. That is, explanations tend to point to salient causes of those phenomena that seem unlikely under default background conditions. In this sense, variational dispositions are explanatory insofar as they explain a kind of behavior that is unexpected. For instance, we have seen that mutational robustness is identified whenever a phenotype manifests invariance when variation is expected, e.g. when it is affected by mutations. This means that the system is robust to mutations by contrast to a default expectation of phenotypes being phenotypically affected by them. In turn, while the causal bases of G-P map dispositions are typically intrinsic, their manifestation will depend on background conditions, some of which may be extrinsic to them. For example, we may expect more plasticity in the semi-aquatic plant *Arabidopsis* (Wells & Pigliucci 2000) if the presence of water fluctuates in the environment—insofar as it develops different types of leaves under and above water—than if what fluctuates is the amount of sunlight hours.

Not only the characterization of these dispositions is relative, but also their magnitude. Just like some objects are more fragile than others, some developmental systems are more variable, robust, modular or plastic than others. That is to say, some systems tend to show variational properties *more often* or with a *stronger tendency*

than others. This observation situates G-P map dispositions in the scope of propensities, insofar as they do not have just one specified outcome when triggered. Changes in reproductive conditions, as triggers of these variational dispositions, bring about a range of possible different results. In the first chapter we saw that this is what distinguishes propensities from surefire dispositions (sections 1 and 3). Propensities manifest probabilistically, meaning that they can be understood as tending towards their manifestation with a certain probability, as manifesting in a probability distribution, or as a disposition to manifest a pattern in the long run. I believe that variational dispositions are propensities insofar as they not only tend to provide variation of a certain kind, but they do so with different associated strengths, in turn disposing towards an array of possible results whose degree of likelihood can be weighted in principle. Thus the possible for G-P map dispositions is not merely a given region of the morphospace, it is a range of possibilities that can be considered as a sample space with definible probability measures under different contexts. For instance, variability can manifest itself in a probability distribution of possible results upon reproductions, that is, in the probability of obtaining different types of phenotypic changes throughout generations. Indeed, as we saw with the RNA model (Fontana 2002), this is what the characterization of variational properties in G-P maps enables us to do in principle.

Characterizing variational dispositions as propensities facilitates the recognition of their manifestation and their effect as distinct things (Molnar 2003, Mumford 2009). Let us recall that the manifestation of a disposition is the function that defines it, such as breaking being the manifestation of fragility. In the case of propensities, a manifestation is a weighted set of possibilities or a probabilistic behavior. For instance, the propensity of a coin to land heads is manifested in the equipossibility pattern of heads and tails. These manifestations are abstract behaviors, and they contrast with the particular effects of dispositions and propensities when they are actually triggered. Effects of fragility may be particular ways to break when hit with different emphases and types of rocks. Effects of the propensity to land heads are particular, actual instances of my coin landing heads when it is tossed. Similarly, in the case of variational propensities we may define the

manifestation of variability as probabilistic patterns of possible variations, but only extant variation will constitute its effect. To be sure, a number of other factors may influence particular outcomes, such as different propensities acting together in the same trial. For example, variational modularity and developmental robustness with respect to the same set of traits can be present in the same reproductive trial, in turn both influencing the particular phenotypic outcome of that trial.

It is now easier to see that not every developmental disposition will qualify as a variational propensity, for there are other developmental dispositions that can manifest in a variety of ways. For example, the disposition of an individual to develop in a certain way is not a variational propensity. It is not so because in order for developmental properties to qualify as variational propensities, they must be functionally defined through the right pair of triggering and manifestation conditions, which differs between variational propensities and developmental dispositions more generally. The manifestation of a variational propensity is a probabilistic pattern of changes upon changes in reproduction, that is, given the triggering conditions of changes in the reproductive process (see Nuño de la Rosa & Villegas 2019). Under this view, mutational robustness manifests in the probability of reproducing the same phenotype; variability manifests in the probability of reproducing a distinct phenotype; plasticity manifests in the probability of exploring different areas of the morphospace under different environments; and variational modularity manifests in the probability of reproducing a phenotype with a modular change. The individuation of a particular variational propensity thus is not a particular phenotype, nor a probability distribution of possible phenotypes. It is, on the contrary, the probability distribution of *ways to generate* variation in reproduction.

As stated above, in this view, extant variation refers to the particular events to which variational propensities contribute. Extant variation in a given trait can be studied at many different levels: the population, the species, the clade, etc. This variation represents the *frequency of outcomes* of variational propensities. For example, the phenotypic variance of a trait in a population can be seen as the (partial) effect of the variability of its G-P map. In turn, the properties of extant

variation can be seen as the particular effects that variational propensities contribute to when they are triggered. As we saw in Chapter 1, frequencies may not match the probabilistic expectations derived from propensities, especially when trials are limited or when other factors are involved. Thus, just like in the case of genetic drift reviewed in the previous chapter (section 2), the actual frequencies of variants may not be representative of the possibilities ingrained in variational propensities. Moreover, while propensities explain general chancy behavior, they do not explain particular outcomes or series of them. Tendencies are used for explaining general patterns, but they are futile for explaining particular causal chains (Cartwright 1994/1989, see section 3 in Chapter 1). In this regard, variational propensities explain the variational tendencies of developmental systems, but they cannot be used to explain the appearance of specific variants. For example, the modularity of hindlimbs and forelimbs in some vertebrate groups such as humans or bats (Hallgrímsson et al. 2002) explains the higher probability of having a modular change in the former without affecting the latter. However, this is not tantamount to saying that modularity is the difference-maker explanas of particular modular changes such as the emergence of bat wings independently of bat hindlimbs. Modularity indeed enabled such an independence in the transformation, that is, the fact that bat forelimbs could change independently of bat hindlimbs. However, there were particular genetic changes and selective pressures implied that made the difference for the appearance of *precisely* wings in bats. Modularity could have enabled a different modular change in bats forelimbs, for example independent changes in length, if different mutations or environmental conditions had been met. In turn, just like we saw in the general case of dispositions (Chapter 1) and in the particular case of fitness and drift (Chapter 2), variational propensities explain regularities rather than particularities. For particular outcomes to be explained, further particular factors must be introduced.

As in the case with other propensities, particular outcomes must be explained alluding to triggering conditions in addition to the propensity. Let us see this through the fair coin example. My coin has a propensity to land heads/tails as based on its structural properties, namely the causal bases of this propensity, such as its

symmetry. This propensity explains a space of possibilities where heads and tails are equally possible. If I toss my coin, I run an entire chance setup consisting of a fair coin, a tossing hand and a set of many other contextual factors. The trial will specify the particular triggering conditions of the propensity, namely the initial velocity and position of the toss. The specific result of this trial—e.g. tails—is causally explained by the entire setup and the particular initial conditions leading to the result. In other words, explaining why the coin lands head in a particular coin toss demands specifying, besides the properties of the coin, the initial velocity and position of the tossing hand. In turn, explaining particular outcomes is beyond the scope of general properties and, specifically, of propensities. Similarly, variational modularity can explain the space of modular possibilities for phenotypic changes, but not specific modular changes, or at least not entirely. In the case of modularity, the specific reproductive conditions, and notably the mutations involved, are a necessary component of the causal story of particular modular changes. In the next epigraf, we shall see how each of the variational propensities here reviewed demand specific characterizations in terms of the chance setups and the triggering conditions involved in their manifestations.

The explanatory role of variational propensities is independent of any analysis of probability ascriptions. Recall that we saw in Chapter 1 that identifying propensities with probabilities entailed difficulties associated with the calculus of reverse conditional probabilities (section 2). Perhaps more importantly, we arrived at the conclusion that propensities were better understood as explanatory causes of probabilistic behavior. Thus deriving specific probabilistic measures is not necessary for propensities to be explanatory. This is particularly important for variational propensities, since, as we have seen, precise probability statements are typically far from reach. Although the probability of specific kinds of phenotypic variations can be in principle specified through evo-devo G-P models of variation—as it is indeed the case in the RNA model—, complex G-P maps tend to specify qualitative rather than quantitative probabilities. Variational propensities explain probabilistic behavior that is not necessarily modeled mathematically with precision. What they explain is the existence of variational possibilities beyond the range of extant variants and as

specified by developmental causes. In turn, these propensities play the 'chance role' in the sense of establishing the possible as previous to and more fundamental than the actual in evolution (see Chapter 1).

Interestingly, this chance role in variation is similar to the role of chance as sampling. As it was the case with the probabilities of evolutionary genetics models, the probabilities of evo-devo models of variation are not associated with any fundamental indeterminism. On the contrary, they refer to higher-level properties that are independent from the underlying ontology. To be sure, the role of stochasticity in development is not minor. As we have seen, some triggering aspects of developmental dispositions can be considered stochastic components. Mutations are one such source, where chemical and physical factors might underdetermine their very nature (Millstein 2011, Brandon & Carson 1996). While mutational mechanisms are well studied (Chen et al. 2012), the connections between the causes and the nature of mutations is largely unknown, there being room for fundamental indeterminism in their emergence. Developmental noise (Willmore & Hallgrímsson 2005) is another important source of stochasticity in direct connection with developmental propensities, for it refers to random fluctuations of development caused by normal environmental conditions. Central as it is, this stochasticity is not essential to the nature of variational probabilities. Indeed, the indeterminism potentially underlying these developmental components does not affect variational propensities. On the contrary, these (possibly) indeterministic factors act as triggers of particular trials of the developmental chance setup. However, were these factors completely deterministic, the probabilistic character of the chance setup would still hold. This is because the probabilistic component lies in the mapping between initial conditions and end-states. In other words, the probabilistic nature of variational properties is based on the causal, higher-level structure of genotype-phenotype relationships (Nuño de la Rosa & Villegas 2019). The higher level here refers to the level of organization of developmental pathways (Gilbert 2000), whose probabilistic properties, in the sense articulated in this chapter and the final section of the previous one, are independent of any stochasticity in the lower level. Consequently, developmental systems show different probabilities of producing variation by virtue

of how they connect lower-level inputs—be them stochastic or not—to higher-level, phenotypic possibilities. Thus the probabilities embedded in the generation of variation are higher-level dynamical patterns that do not entail any assumption on indeterminism. In the coin example, there being a $\frac{1}{2}$ probability of landing heads has nothing to do with ontological indeterminism in initial positions and velocities. Rather, it has to do with the structure connecting possible initial states with possible termination conditions. Similarly to the coin, and to the probabilities of population dynamics, variational probabilities are causal in nature inasmuch as the causal structure of a chance setup determines an output probability space. Since the causes responsible for variational probabilities are stable throughout generations, they are so throughout the iteration of a variational chance setup (Hacking 1965, Abrams 2015). It is this chance setup that bears a particular variational propensity by virtue of its dynamical structure.

A final consideration to make about variational propensities is how they are inferred. Since variational tendencies do not refer merely to extant variation within populations, variational propensities are not extrapolated from frequencies and other statistical properties of intra-species variation. Rather, they are inferred as the best explanation (cf. Lipton 1991, see Chapter 1) for morphological patterns of unity and diversity—thus inter-species variation—on the basis of the empirical research of evo-devo. In the first two sections of this chapter we have reviewed evo-devo models of variation and its methodological approach. In broad terms, we have seen so far that evo-devo studies rely, besides micro and macroevolutionary statistical data, on comparative methods, experimental approaches and computational modelling (Müller 2007). The combination of these methods is what enables the inference of variational propensities. Statistical data serves as a preliminary approximation to the capabilities of developmental systems, insofar as extant variation is necessarily a fraction of what is developmentally possible. Particularly, since the variation under the prism of evo-devo is typically inter-specific, phylogenetic statistical knowledge is essential for establishing specific research goals in the study of variation. The comparative scrutiny of different developmental systems—as contrasted with statistical and phylogenetic knowledge—is the first step towards associating

conserved developmental pathways with their variational potential, as well as for identifying those pathways that provide versatility to the developmental process. Thus it is in the comparative analysis that similarities and differences among developmental systems are primarily inferred, providing the grounds for implementing the experimental and the computational approaches. Interventions, either experimented, simulated and conceived (cf. Woodward 2003, see Chapter 1, section 3.1), play a key role in the subsequent phase of identifying causal bases for variational propensities in evo-devo. For instance, the experiments driven by Alberch and Gale in amphibians (1985, see section 1.1 above), where they manipulated a developmental precursor in order to study patterns of variation, demonstrate the importance of constructing and testing causal hypotheses in evo-devo studies of variation. In this regard, and as it was reviewed in the first section of this chapter, evo-devo G-P maps are usually constructed on the basis of experimental data (e.g. Alberch 1991). Finally, the computational approach of evo-devo is an important step towards the generalization of developmental rules, abstracting away from particular developmental mechanisms. For example, the computational model of developmental evolution for teeth in mammals (e.g. Salazar-Ciudad & Jernvall 2010, see section 1.1 above) sets a series of minimal developmental rules capable of constructing different teeth morphologies, abstracting away from the particular developmental mechanisms giving rise to them in living systems. In this regard, developmental encoding acts as an abstraction of the growing rules responsible for patterns of variation, serving as means for recognizing the abstract properties of G-P maps and their causal potential for generating variation. The combination of these methodologies exemplifies how evo-devo studies of variational tendencies are concerned with the possible as determined by developmental *causes*. In turn, developmental propensities such as variability, modularity, robustness and plasticity are inferred as abstract causes of variational possibilities. Whereas they enable the construction of probabilistic generalizations about the variational potential of developmental systems, these propensities do not exhaust the study of specific causes of variation. On the contrary, they enable the scrutiny of the specific mechanisms realizing these propensities in different systems, while they point at the structural

features shared by all these mechanisms: their tendency towards specific kinds of manifestations.

b) The plurality of variational propensities

Although the above characterization intends to apply to all variational propensities, their differences certainly stand in the way of having a strictly unified account of them. The notions of robustness, modularity, plasticity and phenotypic variability capture particular aspects of *how* the morphospace can be explored: through finding new phenotypes under mutation, through navigating neutral phenotypic spaces, through acquiring different environment-specific phenotypes, or through changing modular components of the phenotype independently. These means of exploration have particularities with regards not only to manifestation conditions, but also to triggering causes and, more generally, to the trials involved. Here I want to briefly articulate the differences between these properties that affect their conception as causal propensities.

Phenotypic variability is the general capacity to generate a different phenotype upon reproduction and, as such, it ecompasses different kinds of variational capacities. Developmental systems show variability when the same morphological unit—be it a character or an entire body plan—is prone to generate phenotypic variation on itself when reproduced. Notice that this propensity is not only estimated in short-term evolution. That is, variability does not only concern the variability a given trait can produce in a particular population. On the contrary, it primarily concerns the inter-species variability of the trait, defined through a range of possible manifestations in different developmental contexts, or, in other words, in different types of organisms. Thus reproductive trials need to be iterated in the long evolutionary run in order to manifest a significant portion of the variability of developmental systems. This may seem like a limitation for the experimental study of variability. However, the long evolutionary record of every living system can be regarded in fact as an experimentation of reproductive trials, and we may study variability over the inquiry about phylogenetic relations and through macroevolutionary comparisons. The triggering conditions for the propensity to vary

to be manifested are for the most part genetic mutations. Changes in the genetic material activate the capacity of the system to either recreate the same phenotype—i.e. robustness—or to manifest a particular modification of it—i.e. variability. Therefore reproductions in the long run manifest variability when mutations tend to generate phenotypic changes. In this regard, we may talk about the variability of the dorsal nerve cord in the genus *Chordata*, or the variability of digits in tetrapods. Through phylogenetic, comparative, experimental and computational approaches we may estimate whether these systems have been more or less prone to phenotypic changes under mutational and environmental changes, and whether their variability is stable across the different taxa sharing these homologous traits. With regards to the former, its variability has changed throughout evolution, showing several evolved modalities, such as the dorsal hollow nerve cord in cephalochordates and the spinal cord in vertebrates. Once evolved, the spinal cord has remained highly robust across different vertebrate species, especially with regards to function and location, and less so when it comes to length. The latter, on the other hand, is relatively robust with respect to digit numbers (typically five), but is highly variable in function. Moreover, some modalities show modularity across digits (Wagner 2014).

Mutational robustness involves the same trials and triggering conditions than variability, but, in contrast to it, it manifests itself in the perseverance of the phenotypic target throughout reproductive events. Thus mutations in reproduction are the triggers of mutational robustness, activating the capacity of the developmental system to generate a particular instantiation of the phenotype under different initial conditions. However, and as mentioned above, robustness is not necessarily the opposite to variability. Indeed, phenotypes tend to be robust in the short term, which may enable variability in the long term by virtue of the exploration of a wider area of the genotypic space (A. Wagner 2005, 2008b). In other words, the mutations triggering robustness may accumulate without phenotypic changes, providing different genetic backgrounds that may facilitate the manifestation of variability in subsequent trials. In general terms the same (abstract) system can provide robust variations of the same phenotype under different genetic

backgrounds, thus showing an evolutionary history of variability while being robust in the short term.

The propensity to vary modularly is, by contrast to variability and robustness above, predicated of a single trait only if it is with reference to another one—perhaps to every other trait in the organism. Typically, the chance setup realizing modularity will consist of several phenotypic traits reproducing together, and the triggering conditions will be mutations affecting only a subset of those phenotypes. For example, a system composed by two different traits, such as a forelimb consisting of three different regions—stylopod, zeugopod, and autopod regions—, will manifest modularity in reproduction if the mutational inputs affecting its development tend to cause phenotypic changes in only one of those regions.

The above variational propensities are all triggered by mutational inputs. The case of phenotypic plasticity differs from the preceding ones in a very significant way. Recall that phenotypic plasticity refers to the capacity to produce different environmentally induced phenotypes from the same genotypic input. Consequently, mutations need not be involved in the manifestation of plasticity. In order for plasticity to be triggered as a variational propensity—that is, as a property of a phenotype rather than an individual—what is needed is a reproductive trial where environmental conditions during development are different but genotypic inputs remain stable. Thus plasticity is the propensity to generate a different phenotype in reproduction *upon environmental differences during development*. In other words, a phenotype is plastic if it is prone to undergo modifications when developed under different environments.[50] Phenotypic plasticity enables a navigation through the morphospace that does not necessarily demand mutational inputs in order to be manifested. However, it does demand reproductive trails just like the other

[50] Notice that the same could be argued about environmental robustness and developmental modularity—as opposed to mutational robustness and variational modularity, respectively—, namely that they could be characterized as variational propensities if we considered different environmental conditions in development as triggering conditions. I omitted this possibility for the sake of simplicity, as based on the relative importance of the propensities presented here—i.e. mutational robustness, variational modularity and phenotypic plasticity—in evo-devo studies of variational properties.

variational propensities, for it refers to a potential of the developmental system for generating a different phenotypic outcome.

3.2. Variational chance: expected, realized and vernacular

Let us briefly turn back to how variation is considered in the classical framework of evolutionary genetics in order to grasp the significance of evo-devo variational propensities for the notion of chance in evolutionary variation. As we saw in the previous chapter (section 3), the evolutionary genetics picture endorses a statistical treatment of variation, where the received notions of chance refer to the randomness of mutations with respect both to phenotypes and to the directionality of evolution. However, we have seen that once variation is defined at the phenotypic level, its randomness is challenged from a morphological point of view: the exploration of the morphospace is not random but highly structured by the properties of development, regardless the nature of mutations. The overall situation is that phenotypic patterns of variation are assessed in different ways in the research agendas of evo-devo and evolutionary genetics. In addition, the language found in the literature on variational properties is ambiguous about their characterization, sometimes referring to tendencies, others to probabilities, and yet others to statistical summaries or correlation frequencies. As mentioned in section 1 of this chapter, some G-P maps can be considered as statistical tools that indeed refer to the correlation patterns of specific events (Sendhoff et al. 1997, Hansen 2008). Thus certain usages of variational properties actually refer to the frequencies of specific results, in the sense of the relative frequency of an outcome in a specific reference class.

I argued in the preceding chapter (section 3) that this statistical treatment, while instrumental for the purposes of evolutionary genetics, is not explanatory of evolutionary variation. Let us see this through the statistical characterization of variational properties. When it comes to represent and measure phenotypic variation, evolutionary genetics is mostly concerned with so-called quantitative traits, namely those whose values that can be mapped into a metrical scale for comparative purposes. Quantitative geneticists identify a trait in a given population, and from

measures of extant variants they define its mean quantitative value and variance. The phenotypic variance (V_P) is a statistical measure of how much deviation from the mean value a certain trait shows in the population. For instance, two different populations of wheat can have an average height of 1 meter. However, individuals of population *A* may be very diverse among one another, there being many individuals measuring 60 centimeters or 1,30 meters, while individuals in population *B* may be all very close to being 1 meter tall. In this case, the phenotypic variance of *A* would be greater than that of *B*, even if their average phenotypic value coincides. The phenotypic variance is typically divided into the genetic variance (V_G) and the environmental variance (V_E), which represent how much of this diversity is attributed to genetic effects and to the environment, respectively. Thus,

$$V_P = V_G + V_E$$

The ratio of genotypic variance over phenotypic variance of a trait is called its heritability, which is typically used to estimate the capacity of the trait to respond to selection, that is, to change its mean value in the direction of selection.[51] To this standard characterization, we may include a stochastic factor in the variance due to developmental noise (V_{error}), and a gene-environment interaction component ($V_{G\times E}$). The variance due to gene-environment interactions ($V_{G\times E}$) is that fraction of variation that depends on specific environment-dependent individual developmental properties, namely due to differences in the norms of reaction of individuals. A more complete characterization of the phenotypic variance of a trait is thus (DeWitt & Scheiner 2004):

$$V_P = V_G + V_E + V_{G\times E} + V_{error}$$

Following this statistical approximation to phenotypic variation, let us see how the properties reviewed in the previous section can be understood statistically. Environmental robustness can be seen as the weak correlation between the environmental variance (V_E) of a phenotype and its phenotypic variance (V_P). That is, it is the low impact of variation in the environment in phenotypic variation.

[51] This measure cannot be used to compare responses to selection across different traits and species unless it is scaled to the mean (Houle 1992, Hansen & Houle 2008).

Similarly, mutational robustness is the weak correlation between the genetic variance (V_G) of a phenotype and its phenotypic variance (V_P), namely the low impact of genotypic variation in phenotypic variation. On the other hand, phenotypic plasticity can be understood as the gene-environment interaction component ($V_{G\times E}$) combined with the environmental component (V_E) of the phenotypic variance (DeWitt & Scheiner 2004). In this sense, it is different from developmental noise (V_{error}), which refers to random deviation of extant phenotypes from target phenotypes. The distinction between V_E and $V_{G\times E}$ reflects the difference between those systematic effects of environmental variance in a population (V_E) and the effect of specific genotype-environment interaction in different individuals, thus of differences in plasticity ($V_{G\times E}$). Variational modularity, by contrast, cannot be associated with a component of the variance of a trait. Rather, it is related to patterns of covariation among different traits. In the statistical approach to variation, it is the covariance that serves as a measure of the manifestation of modularity: the more independent the variance of a trait is with respect to variation in other traits, the higher modularity it shows with respect to it. For considering relations among traits in their response to selection, quantitative geneticists make use of the genetic variance-covariance matrix, the G-matrix, which is an estimate of the genetic correlations among the traits.

The above characterization estimates variational properties from extant variation within a given population. Estimations of this type are successfully used for short-term microevolutionary predictions. This is because the patterns of additive genetic variances and covariances can be very stable locally (Lande 1979; see Hansen 2008). Recall that genes in evolutionary genetics are abstractions based on the average deviation from the mean phenotypic value of organisms carrying them, and that they are assumed to combine additively, namely to add their quantitative effects to the effects of other genes without restriction. When statistical approximations of this type are used in microevolutionary studies, the developmental interactions among genes are completely abstracted away and neglected, assuming that the extant pattern of additivity and correlation provides enough information about future patterns. However, developmental nonlinearities can produce changes in the statistical properties of variation even in short scales. In this regard, genetic

correlations are a good first approximation to the local G-P map, but they lack any long-term projectability (Hansen 2008, Müller 2007) and, moreover, they are inapplicable to groupings at higher taxonomic levels than the species.[52]

These considerations bring the statistical approach to variation of evolutionary genetics close to a frequentist view of probability, where the probabilistic modelling is exclusively based on the frequency in which events take place (Reichenbach 1938). As a consequence, the problems associated with frequentism about probabilities affect evolutionary genetics models directly. For example, these models face the reference class problem insofar as conceiving the probability of a change in the mean of a trait in a population is determined by the statistical trend the population already shows. Recall that frequentists assume that probabilities are assigned through the establishment of a reference class of trials with a defined frequency of outcome events (see Chapter 1, first section). Thus a problem arises with regards to the establishment of the right class of trials to consider for defining probabilities. Similarly to the statistical view of fitness, which fails to assign a fitness value when the number of trials is small, a statistical view of variation fails to provide the probabilities of generating variation when few trials are considered. For instance, how many trials—i.e. reproductions—are needed in order to estimate if traits are really varying in a coordinated fashion or they are otherwise changing together by chance? In other words, how do we estimate the right reference class for the variational events under study without knowing their generating conditions? In this regard, I believe that recent criticism to the statistical nature of evolutionary genetics models (Pigliucci & Kaplan 2006, Pigliucci 2010a) can be better understood as pointing at the explanatory pitfalls of endorsing a frequentist view of probability.

As we mentioned in the previous chapter (section 4), evolutionary genetics models do not rely on any causal knowledge about the role of genes in the building

[52] In this regard, we can consider, instead of microevolutionary statistical measures of variation, the macroevolutionary patterns of diversity among species. However, notice that the frequency of variational changes in long evolutionary scales cannot serve for stipulating variational probabilities. This is because reproductive trials are necessarily imbricated with ecological trials, namely with the sampling due to natural selection and drift. Extant macroevolutionary patterns, therefore, need not be representative of variational potentialities. I will get back to this crucial point in the next section.

up of phenotypes (Pigliucci 2010a). However, as it has been argued throughout the present chapter for variational tendencies, and in Chapter 1 for probabilities more generally, in order to capture the generative processes leading to probabilistic behavior, it is necessary to consider their causal structure. Statistical approaches to the G-P map, insofar as they only concern extant patterns of variation—or particular, context-dependent effects of variational tendencies—can only be regarded as a first approximation to the probabilistic modeling of variation. In this regard, there is a general agreement that, in order for the G-P map to account for long-term variational patterns, the causal structure of development linking genotypes to phenotypes must be introduced (e.g. Hansen 2008, Pigliucci 2010a).

In direct connection to this, and interestingly from the point of view of this thesis, in this statistical approach, variational properties are unable to explain extant variation: they are identified with one another. Just like in the case of a statistical understanding of population genetics, extrapolating patterns of variation from extant correlations lacks explanatory power. This is not a problem *per se* for quantitative and population genetics: they don't intend to explain variation but short-term ecological trends as based on the properties of extant variation. The problem resides, nonetheless, in extrapolating from this fact to the explanatory structure of evolutionary biology more generally. That is, this statistical situation of variation fails to adequately exemplify the role that chance plays in evolutionary variation in a general sense. Perhaps more importantly, it fails in providing an articulation of the causal impact that the very production of variation has in the course of evolution. The microevolutionary success of evolutionary genetics models was sometimes mistaken for a trustworthy representation of how evolution works at all scales. The idealizations ingrained in the classical case of Fisher (1930) are paradigmatic, such as the idea that developmental nonlinearities tend to average over in large enough populations and thus can be ignored with no harm (Hansen 2006).[53]

What I want to argue now is that a separation between vernacular, expected and realized variational tendencies, analogous to the distinctions already present in

[53] In order to deal with persistent nonlinearities, in any case, the statistical treatment of variation needs to incorporate "ad hoc properties, such as pleiotropy, penetrance, covariance, etc." (Alberch 1991).

the philosophy about fitness (see section 2 of the previous chapter), can help in introducing a causal notion of chance into the probabilistic view of evolutionary variation. This separation coincides with the distinction between developmental propensities (section 3.1 above), variational probabilities (section 4.3 of previous chapter) and extant patterns and frequencies of variation, both within populations and in phylogenetic relations. The patterns of variation as presented in this section, in turn, do not represent variational probabilities, let alone propensities. On the contrary, they represent the first, frequency-like approximation to them. Let us recall that observed frequencies can provide "the only evidence for propensity statements" when there is no "well-developed theoretical background" (Giere 1973, p. 504). But as I argued in section 4 of the previous chapter and throughout the present one, evo-devo provides a solid, empirically-based theoretical background for talking about variational propensities and probabilities.

Mathematical models of variational probabilities are limited. We have seen that few simple G-P maps allow for specific probability measures, such as the RNA and the minimal cell models (see section 1.3 above). However, one of the goals of evo-devo models of variation is deriving probability measures of phenotypic transformation in the relatively near future (Jaeger et al. 2015). In any case, it is clear now that both statistical knowledge *and* propensity statements must be involved in order to derive meaningful probabilistic predictions of phenotypic transformation beyond the short, microevolutionary term. In this regard, the 'predictive' or 'expected' version of variational tendencies—the probabilities of varying in certain ways—can only be different from 'realized' variation once there is causal modeling involved, that is, once there is a 'vernacular' sense of variational tendencies being considered. The variational propensities of G-P maps are explanatory of developmentally possible evolutionary scenarios from which specific probability measures can be derived in principle. Realized variation, in contrast, refers to the particular effects of variational propensities in different contexts, similarly to the realized fitness of organisms.

What we can see now more clearly is that the 'source laws' of variation are not only found in genetics, as mentioned by Sober in his classical depiction of evolutionary biology (1984). On the contrary, they can be mostly found in the

evo-devo study of developmental tendencies, insofar as it provides the theoretical framework from which phenotypic possibilities can be derived on causal grounds. Thus in order to construct a causal view of chance at the level of how variation is generated, I believe that it will be fruitful to incorporate evo-devo variational propensities as developed here. These variational propensities can work as 'source laws' for probabilistic patterns of variation susceptible of being mathematically measured under different contexts. This is because these propensities determine a sample space of possible variants by virtue of the causal structure they represent.

Let us finally return to our urn example developed in the previous chapter to illustrate the distinction between vernacular, expected and realized variational tendencies introduced here. We had established that chance in the generation of variation refers to how sampled balls are reinserted into the urn—or into a new one—and the way they are reproduced. Recall that, as a matter of fact, the variants sampled by natural selection and drift do not reproduce exact copies of themselves. Rather, their offspring typically shows phenotypic deviations from them. The generation of the sample space of evolution refers to this fact, that is, to the construction of phenotypic variants in reproduction. Thus acknowledging variational propensities in this analogy means to consider the potential transformations the balls may suffer as based on the way they are constructed. Let us recall that we exemplified this through the idea of a building hand for the reintroduced balls, where some balls may be colored before they are assembled—perhaps they consist of two different pieces—while others are assembled before they are colored (section 4.3 of previous chapter). Variational propensities, in the vernacular sense, represent the properties of these constructing hands. They are hands in charge of the construction of each ball—i.e. of individual developmental processes—, but they are also hands whose *building rules* determine a space of possible ways to vary, such as those hands constructing potentially multicolor balls. Only the actual frequencies of changes in the coloration of balls resulting from these building processes will constitute the realized variational tendencies of balls. The expected sense of these tendencies, on the other hand, corresponds to the mathematical model predicting the probability of

a change in ball colors, as based on our knowledge of both extant variation and the properties of building hands.

However, as it was also introduced in the previous chapter, evo-devo is not specially focused on the generation of variation in microevolutionary processes. In other words, it is not typically concerned with how balls are reinserted into particular urns. Rather, it is interested in the variational tendencies of *types*. That is, evo-devo's variational probabilities regard how distinct developmental types *tend to reinsert* their balls in their respective urns, and what are the evolutionary consequences of these different general tendencies. Consequently, an important epistemic goal in evo-devo will be more accurately conceived as a *type of hand* rather than a particular one.

3.3. Developmental types and chance setups

I have talked thus far about developmental systems as bearers of these variational propensities, and stressed that these systems cannot be identified with individual organisms, inasmuch as individuals do not vary upon reproductions—rather, they reproduce other organisms. In this sense, I have mentioned that the developmental *chance setup* for these tendencies has to be characterized by means of the right pair of manifestation and triggering conditions, namely by ways to vary phenotypically and changes upon reproductive conditions, respectively. Thus the 'vernacular' sense of the variational propensities here analysed is a causal property of a system that persists temporally while reproducing under different conditions. Since these properties are represented in G-P maps, we may wonder what is the bearer that G-P maps refer to in a way or another. I now want to make the case for developmental types, in the sense introduced in the previous chapter (section 4.1), being the bearers of variational propensities.

Let us recall what it means for evo-devo to inherit the typological perspective of the morphological tradition (section 4.1 of previous chapter). It entails that the patterns of unity and diversity under evo-devo's purview are not reducible to the intra-specific differences of individuals—and thus to a population kind of thinking

(cf. Mayr 1959). Rather, they refer to the possible variations within a *type*, that is, within a specific developmental logic underlying the production of a particular character or phenotypic trait. In this regard, a G-P map can be seen as representing the variational possibilities of a particular developmental type. Structural aspects shared by different organisms and species, such a specific body plan or a morphological character are what is represented in evo-devo models of variation. For example, we saw in section 1.1 above that the G-P maps of the pentadactyl plan or type are very influential in the evo-devo literature. Alberch introduced the G-P map (1991) after studying variational patterns of digit loss in amphibians with Gale (Alberch & Gale 1985) and, after that, tetrapod digits have been abundantly studied from an evo-devo perspective (e.g. Wagner & Gauthier 1999, Stewart et al. 2019, Galis et al. 2001). In these models, G-P maps abstract away from specific developmental mechanisms in order to associate genotypic possibilities with phenotypic ones, typically encompassing a variety of species showing the same morphological unit, i.e. the tetrapod digits. In turn, it is these units represented in G-P maps that bear the variational propensities here described: they show variability, robustness, modularity and plasticity throughout reproductions of their structures.

The ontological status of these types has been explored by philosophers and theoreticians of biology in the last few decades. In this sense, approaching the traditional views of comparative morphologists from a contemporary perspective, compatible with current standards about causation, has sometimes been received with reluctancy (Amundson 1998, Lewens 2009b). However, most current discussions agree that understanding developmental types ontologically need not be problematic (Shubin & Alberch 1986, Amundson 2005, Rieppel 2005, Wagner 2014). As Shubin and Alberch (1986) put it, there is a need to "exchange the metaphysical concept of the Bauplan" for a mechanistically grounded one (p. 377, cited in Etxeberria & Nuño de la Rosa 2020). Similarly, Brian K. Hall claims that

> The need is not to regard the *Bauplan* [or the type] as the idealized, unchangeable abstraction of Geoffroy, but to treat it as a fundamental, structural, phylogenetic organization that is constantly being maintained and preserved because of how ontogeny is structured (Hall 1999, pp. 98-99)

In this regard, it is worth pointing at the recent advances on the mechanistic bases that may underlie the existence of shared types across species (e.g. Davidson & Erwin 2006, Graf & Enver 2009). A salient example is Günter Wagner's proposal that character identities or types can be associated with gene regulatory networks inhibiting the expression of alternative characters and controlling the gene expression of the cells forming the character (Wagner 2007b, 2014). Importantly, "[t]he information for character identity is *within* the cells that react to the signal and not in the inductive signal itself" (2014, p. 93, stress added). For example, the highly conserved "segment polarity network" across insects plays a key role in segment development, despite the variety of particular signals activating such process in different insect species. In particular, there is considerable variation in the developmental stages preceding the activation of this network across species, as well as in the genes actually carrying out segmentation in later stages, after the network is activated. Nonetheless, the basic features of this network, namely how it reacts to signals for building morphological features, are conserved, thus pointing at its relevance in the insect segment type or character identity (Wagner 2014, p. 98-).

An important intent to conceive these developmental types—or homologous characters—in a metaphysical sense is their understanding as natural kinds, particularly as "homeostatic property clusters" (Boyd 1991). Olivier Rieppel (2005) has argued that the similarity in developmental pathways and the patterns of gene expression among different species sharing the same character can be seen as a cluster of homeostatic properties (see also Wagner 2001). What this means is that instantiations of these kinds need not share essential properties. Rather, they share homeostatic mechanisms responsible for a certain cluster of properties, none of which is necessary for instantiating the type (Boyd 1991, Rieppel 2005). In other words, the developmental mechanisms instantiating a particular type need not share any essential component, but tend to show instead some of the properties—but not necessarily all—of the cluster that characterizes the developmental type. Interestingly, some authors have pointed out that this view enables the understanding of kinds as individuals (Wilson et al. 2007, Asiss & Brigandt 2009). In this regard, natural kinds such as homologous structures or developmental types can

be seen as individuals in the sense of units of evolutionary change forming materially continuous lineages of living beings (Wagner 2014).

Regardless of the precise characterization of these kinds, the evo-devo conceptual framework allows for a distinction between a type and specific realizations of it, what Wagner calls character identity and state, respectively (Wagner 2014, see section 2.1c above). For example, human arms are an instantiation of the forelimb type. In addition, and since identities may overlap, human arms are also instantiations of the broader type the tetrapod limb. This leads to the idea that there may be different "variational modalities" of a specific type, namely "sets of character states of the same character" that share similar developmental mechanisms, and where transitions among states are more likely than transitions from a set to another (Wagner 2014, p. 63). For example, the avian wing may be a variational modality of the tetrapod limb. But within the avian wing there is still a wide range of different character states instantiating limbs, such as the wings of ostriches and robins. Types like this understood are considered as relatively independent units of evolution, themselves being subject to evolutionary change and showing different modalities (Brigandt 2007, Amundson 2005).

But types need not be characterized as ontological units in order to play an explanatory role in evolution. In contrast to the above characterization, some authors have pointed out that the nature of developmental types may be merely nominal (e.g. Lewens 2009b). Perhaps less radically, philosopher Alan Love has argued that a "reconfiguration" of the philosophical approach to types, from the metaphysical to the epistemological, can be fruitful in broadening our views about the roles that typological concepts play in biology (Love 2009). According to Love, typology is a kind of representational reasoning that is relevant for explanatory purposes. In representing biological phenomena, it is useful to idealize conditions and develop approximations in the pursuit of explaining. Thus a type is an idealized structure that captures abstract, shared features, and is constructed from knowledge of a wide range of its instantiations. In this regard, the specific goals of scientists when deriving and using a typological explanation must be considered. For example, developmental biologists picture development as consisting of differentiable normal stages that are

commonly held across species (Love 2009). This conception of a continuous ontogenetic process as partitioned into different periods is only achieved through assumptions and idealizations, but it is useful in representing real commonalities under scientific scrutiny. Similarly, in the particular case of developmental types, Amundson argues that their nature "is inferred", that is, it is primarily the result of an epistemological, inferential process derived in the very scientific practice "from comparative morphology and experimental embryology" (Amundson 2005, p. 232).

Let us see now how these types are identifiable with the chance setups responsible for variational propensities. In Chapter 1 we saw that we typically assign propensities to chance setups rather than to particular trials. Thus, for example, we attribute the propensity of landing heads to coins, an entire setup, instead than to any actual toss of it. This might be a convention (Mellor 1971), but it is representative of how propensities are usually conceptualized. Similarly, we may consider that it is only tokens or particular reproductive trials—although not individual *organisms*—that bear variational propensities. However, the general types represented in G-P maps—even if their nature may be inferential—are better understood as the bearers of variational propensities (Nuño de la Rosa & Villegas 2019). This is because when explaining probabilistic behavior and modeling variational probabilities we are not concerned with the possible outcomes of specific reproductive trials. Instead, we are concerned with the potential results of the long-run iteration of these trials in different contexts. In this regard, every reproductive-developmental event, that is, every entire cycle of reproduction, can be regarded as a particular trial of the typological chance setup. When organisms reproduce, their generative properties are instantiated back with small variations introduced—such as mutations—and under different environmental circumstances. It is these properties that constitute the chance setup that is relevant for variational propensities.

The small variations in reproductive conditions are represented by the input space of these chance setups. The input space is a genotypic space that includes all possible genotypic combinations that can affect the same developmental structure. In the case of phenotypic plasticity, moreover, the input space includes different

environmental inputs triggering and affecting the reproductive trial. Thus every time a reproductive event takes place, a trial with a different input state is instantiated. Lewens (2009b) nicely illustrates the variational properties of developmental types making reference to the properties of dice rolling. As we saw in Chapter 1, when rolling a die, certain structural properties, such as its mass distribution, play a fundamental role in establishing the probability distribution of different results. Lewens states that some developmental properties play the same role when considering the probability distribution of different phenotypes: the structure of developmental processes will make some biological forms more or less likely to arise in a population, independently of the mutational and environmental inputs of each reproductive event. Following this analogy, each reproductive trial followed by a developmental process is analogous to one roll of the die, while the different genotypic and environmental combinations that constitute the input space correspond to the possible initial velocities and positions of the roll. This analogy illustrates how development channels the way different possible mutations relate to phenotypic effects. When rolling a die, a probability distribution of possible results can be objectively established independently of the specific initial conditions of the roll. In particular, the velocity and position of the die in each roll is deliberately neglected for resulting explanatorily unhelpful. Given that such initial conditions satisfy certain general requirements (see Chapter 1), it is the dynamical properties of the process that generate the probability distribution of results. Similarly, in evo-devo explanations, the occurrence of particular initial developmental conditions (i.e. mutations and environmental perturbations) are not considered in detail for establishing the probability distributions of phenotypic results, as long as these initial conditions satisfy certain general requirements (Lewens 2009a, Nuño de la Rosa & Villegas 2019).

Now we can see how initial conditions are not particularly explanatory for the purposes of modeling developmental types. Let us recall that mutations are not random, in the sense that there are biases in their production (Merlin 2016, see section 3.1 of previous chapter). However, these biases are not necessarily considered in the variational probabilities of developmental types. Let us notice that evo-devo

can model mutational changes as random in an equiprobability sense, and still will derive non-equiprobable distributions of phenotypes in virtue of the general developmental paths available from these conditions to phenotypic outputs. In other words, even if every possible mutation was equally likely, the structure of the G-P map would render phenotypic results non-random. We have seen this in the RNA model (section 1.3 above), where point mutations from one point in the genotypic space to a neighboring area could be modeled as equiprobable without implying that the phenotypic results obtaining from them are equiprobable as well (A. Wagner 2012a). In this regard, talking about the variational tendencies of development is radically distinct from talking about mutational biases—which arguably could play an important role in evolution as well, an issue that is not explored in this thesis. It is clear now that saying that developmental propensities affect the direction of variation is not "just another way to say that mutations are not occurring in other possible directions", as some authors have suggested (Lenormand et al. 2016, p. 198). Just like in the case of a loaded die, the low frequency of some results is not a matter of low frequencies in specific initial conditions. Rather, it is a matter of the low presence of structural pathways leading from *any* initial condition to these particular results.

Nonetheless, let us recall that initial or triggering conditions, while they may be unexplanatory of general patterns, are part of the causal story of specific events (Cartwright 1994/1989, see section 3 of Chapter 1). In the case of variational propensities, specific mutations and environmental conditions do explain, together with the properties of types, the particular variants actually arising in specific reproductive trials. In this regard, the history-dependence of developmental chance setups is of great importance. Their trials are connected insofar as, of all possible initial inputs of the system, those that are closer to the initial conditions of the previous trial will be more likely. When reproduction takes place, the inputs for development are the result of a deviation from parent to offspring, therefore radically different inputs being extremely rare. If we picture this through the loaded die analogy, we may have to assume that, when a trial takes place, the initial velocities and positions of the die can only change *extremely little* with respect to the velocity

and position of the previous trial. That is, even if the type allows for a wide space of morphological possibilities, each reproductive trial, inasmuch as it departs from the *re-production* of the previous one, will deviate from it only slightly. Perhaps, this may entail that only those faces in the die surrounding the one that resulted in the previous roll are actually available, while the face in the opposite side is only reachable for other series of trials—such as the reproductive trials of other species belonging to the same type.

If the initial conditions of actual trials of variational chance setups are history-dependent, one can imagine an ideal trial of the complete G-P map that sets the initial conditions *at random*. I believe that this erasing of initial conditions, similar to the "shaking" of a dice, where initial conditions are completely randomized (Strevens 2013), represents the developmental type. In other words, when evo-devo models of variation conceive a type, they are abstracting away not only from particular mechanisms instantiating developmental pathways, but also, and importantly, from the distinct initial conditions triggering the type in different history-dependent instantiations of it. Thus even if variational events are necessarily history-dependent, the developmental types explaining them are considered without the different specifications triggering particular results. This is particularly important at very high levels of abstraction, for example when dealing with types such as the vertebrate limb: its variational propensities are considered abstracting the differences in genetic background of vertebrate species. If we are modeling a particular limb such as bat wings, the level of abstraction from possible initial conditions will be lower. As a consequence of this process of abstraction, the developmental type can serve for conceiving unrealized phenotypic changes, inasmuch as the initial conditions potentially triggering them have only contingently not been the case.

Finally, as we have seen in section 2.2 above, variational propensities themselves evolve. The stability of the developmental chance setup accounting for these properties is relative and limited in time, and thus a change in its structural properties is definitely possible—although certainly way less frequent than a change in its instantiations. For example, the tetrapod digits are an evolved type whose variational tendencies have changed in evolutionary history. While the first tetrapod

species had six to eight digits, early on tetrapods started to show only up to five digits, a tendency that has remained stable thereafter (Wagner 2014). Thus tetrapod digits have changed in robustness and variability. Interestingly, there exists a second-order evolutionary dynamics that determines the evolution of developmental types. The emergence of new variational modalities or even new character identities in the evolutionary history is totally compatible with there being relatively stable types that explain variational possibilities (Lewens 2009b, Wagner 2014). In this regard, it is worth recalling, with Popper, that propensities are not only properties of a particular physical situation, but also "sometimes even of the particular way in which a situation changes" (1990, p. 17, see section 1.1 of Chapter 1). In other words, the variational propensities of developmental types structure the possible ways in which the type can be instantiated, but also, and importantly, they structure the possible ways in which the type itself can give way to a new one. Types do not evolve at random, but accordingly to what is more developmentally possible and ecologically viable. Thus the properties of types influence the ways it can evolve and even transform into a new type as much as they influence its different instantiations across species. In the next and final section, I will present some of the consequences that follow from this understanding of developmental types as evolvable chance setups for the very explanatory structure of evolutionary biology.

4. Variational propensities in evolutionary perspective

In the preceding sections I have argued that the causal structure of development is responsible for variational probabilities, determining thus the sample space of variation. Is this sample space of variation the sample space of population dynamics? In other words, is there a *unique* sample space of evolution? We have seen so far that extant variation, the sample space of population dynamics—the balls inside the urn—is only a restricted area of the sample space of developmental chance setups. Quite trivially, selection cannot act on unrealized morphologies, so what is not in the space of extant variation—what is not in the sample space of population dynamics—is not part of what can be fixated. However, the strong dependency of

extant variation on the existence of a developmental sample space of the possible has interesting consequences for the very explanatory structure of evolutionary biology.

In this last section, I present what I consider some interesting connections between developmental type propensities and the explanatory structure of evolutionary biology, specially regarding the dynamics of populations as depicted in Chapter 2. In so doing, my goal is to make sense of some of the present discussions on the explanatory and causal structure of evolutionary theory from a causal propensity point of view. If in Chapter 2 I adopted the simplifying idea that evolution was a "two steps process" for the sake of argument, in this section I shall abandon it and explore some consequences of the entanglement of the production and the fixation of variation. In order to do this, I first relate the preceding ideas on developmental types to arguably one of the most important notions now widely present in the evolutionary biology literature: evolvability (section 4.1). This will be an attempt to articulate what is the evolutionary significance of developmental propensities beyond the establishment of a space of possibilities, additionally considering their potential to affect adaptation itself. Then, I explore some consequences that follow for the explanatory structure of evolutionary biology and the role that chance plays in it (section 4.2). In particular, I argue that variational propensities are a way to consider developmental properties as ultimate, rather than proximate, causes of evolution.

4.1. Evolvability: the evolutionary potential of developmental types

For evolution to be possible at all, biological systems need to fulfil certain requirements. Let us recall that the minimal conditions for evolution to take place are generally considered to be heritable variation and differential reproduction (Lewontin 1970). Thus biological systems must be able to generate heritable variants that can be subject to ecological sampling—be it by natural selection or drift—in order to undergo evolution. In other words, they must be able to generate the sample space of evolution, in the sense articulated in this thesis. While so far I have focused on how developmental properties affect the distinct ways in which this sample space

can be produced, the fact that it can be generated at all in successful ways has remained unproblematized. Evolvability is gaining attention among evolutionary biologists and philosophers as a notion that generally captures this idea of a capacity to generate heritable and potentially successful variation. Early usages of evolvability in the context of evolutionary biology, starting from the 1930s, refer to a general, fundamental capacity of living beings to evolve (Crother & Murray 2019). However, references before the late 1980s are scarce, and it was only in the 1990s that evolvability was constituted as a research agenda on its own (Nuño de la Rosa 2017). This is no puzzle considering the assumption of 'chance variation', supposedly abundantly and unbiasedly present at all times, reigning the conceptual framework of the M.S. (see previous chapter, sections 1 and 3). Thus, in the classical picture of evolution, evolvability is "an assumption rather than the subject of study" (Wagner & Draghi 2010, p. 395), in the sense that variation acts as a precondition for explaining evolutionary change instead of as a phenomenon demanding an evolutionary explanation on its own grounds. In this panorama, the emergence of evolvability as a subject of study, not only theoretically but empirically and computationally as well (Payne & Wagner 2019), has implied an increasing focus on the nature and composition of evolutionary variation. Consequently, evolvability is nowadays regarded as a central component of the ongoing debate over the explanatory status of M.S. and its potential extension (Pigliucci & Müller 2010), insofar as it refers to a phenomenon that acted as background condition in the M.S. explanations, but seems to act both as a central *explanans* and an *explanandum* in contemporary research, especially in the field of evo-devo.

Evolvability first appeared in the modern sense of the word in a proceedings article by Dawkins (2003/1988), who associated it with the "evolutionary potential" of some types of embryologies. According to Dawkins, some embryologies—exemplified in the "genetic systems" parameterizing his computational simulations (see section 1.2 above)—are capable of producing very diverse and successful variation relatively easily because of the way they construct phenotypes. The potential of embriologies to provide variation is a traditional vindication of evolutionary morphologists and embryologists, as we saw in the

previous chapter (section 4.1). However, the coining of the term 'evolvability' to refer to this fact proved successful, the decade of 1990s being very prolific in the emergence of some key works that inaugurated the research agenda of evolvability (e.g. Alberch 1991, Houle 1992, Wagner & Altenberg 1996, Kirschner & Gerhart 1998; see Nuño de la Rosa 2017 for a careful study about the literature on evolvability). In their now classical piece, Wagner and Altenberg (1996) define evolvability as "the ability of random variations to sometimes produce improvement" (p. 967). Importantly, this influential definition refers to the production of improved or *better* phenotypes, which seems to be a key component of evolvability in most approaches. Similarly, Kirschner and Gerhart (1998) refer to "the capacity to generate heritable, selectable phenotypic variation" (p. 8420), thus they explicitly relate this capacity to the potential ecological success of variants.

Evolvability is considered a key concept in evo-devo, to the point of constituting its "proper focus" (Hendrikse et al. 2007) or "central target" (Minelli 2010). The centrality of evolvability in evo-devo research resides in its encompassing nature. If the developmental evolution branch of evo-devo (cf. Wagner 2000)—as contrasted to the comparative developmental biology branch (see section 4.1 of previous chapter)—is concerned with the variational tendencies of developmental systems, evolvability can be regarded as their most central variational tendency. Indeed, in the context of evo-devo, evolvability is associated to the G-P map and its variational properties (Wagner & Altenberg 1996, Pigliucci 2008). Thus evolvability refers to the distinct biases in the production and modulation of variation that enable different ways to evolve (Hendrikse et al. 2007), in turn containing variability, modularity, robustness and plasticity as reviewed in this chapter. In this regard, I believe that what talking about evolvability adds to the capacities to generate the sample space of evolution in distinct ways—i.e. to the variability, modularity, robustness and plasticity of developmental types—is that this sample space can be generated in a way that facilitates evolutionary change in iterations of reproductive trials. In other words, evolvability refers to the capacity to generate variation *such that evolutionary changes are likely*.

By no means is evolvability exclusive to evo-devo. Rather, it can be seen as a notion comprising "a family of related concepts" (Pigliucci 2008, p. 75) used in different branches of evolutionary biology. From the point of view of quantitative genetics, evolvability has been associated with heritability and G-Matrix studies (Pigliucci 2008, Nuño de la Rosa 2017), which, as we saw in section 3.2 above, are statistical measures of the genetic variation responsible for phenotypic change. David Houle (1992) refers to evolvability as "the ability of a population to respond to natural or artificial selection" (p. 195), directly relating it with the genetic variance of traits. However, the use of genetic coefficients rather than heritabilities to estimate selection response enables comparisons among different traits and species, which seems to be a key aspect of evolvability in quantitative genetics (see also Hansen et al. 2011). In addition, an interesting area within the quantitative approach to evolvability is the study of "conditional evolvability" (Hansen 2003), namely the ability of a trait to respond to directional selection conditioned upon other traits being stabilized. Classical population genetics, on the other hand, studies evolvability from the point of view of mutational robustness and cryptic variation (Nuño de la Rosa 2017). Cryptic variation refers to the potential, not expressed variation of a certain grouping of organisms, typically populations. Thus it refers to the unexpressed genotypic variation of robust phenotypes, which can be realized by many different genotypes with distinct cryptic variants. In this regard, "different populations stochastically accumulate different cryptic variants" (Zheng et al 2019, p. 352), namely mutations that are neutral in the environment where they emerge and get fixated by drift, but whose phenotypic potential in other possible environments is unknown. The particularity of evolvability in these evolutionary genetics approaches is that it points to the evolutionary significance of extant variation beyond the microevolutionary interest. That is, it points to its significance beyond being a mere precondition for selection to act upon in a given population. In considering non-encountered environments, comparisons among non-inbreeding populations and statistical properties of the G-P map, evolvability studies reflect a shift in the importance that variation has in evolutionary genetics.

Finally, in the field of macroevolution, evolvability has been associated with the ability to generate major innovations and phenotypic novelties (Maynard Smith & Szathmáry 1995). In this regard, evolvability has been defined as "the proportion of radically different designs created by mutation that are viable and fertile" (Brookfield 2001). This definition remarks the phenotypic versatility of biological systems and, importantly, their potential ecological success. Thus the origin of great evolutionary innovations, known as 'major transitions' (cf. Maynard Smith & Szathmáry 1995), such as the origin of multicellularity, and of major body plans are considered the result of a high evolvability of certain biological systems (Pigliucci 2008). In turn, those characters that have shown prolific throughout evolutionary history are considered to be particularly evolvable in this sense. For instance, tetrapod limbs are highly evolvable insofar as they have diversified in a variety of successful and adaptive ways in different tetrapod species and under distinct ecological circumstances. In this context, it is interesting to note that the structure of the G-P map has been vindicated as a way to link microevolutionary events to macroevolutionary patterns of change (Polly 2008, Hansen 2008). That is, studying how development transforms genotypic into phenotypic variation is considered key to linking evolutionary potential in the short term and the long-term patterns of macroevolution.

What all these ideas seem to embed is that the nonrandom dynamics represented in variational tendencies can condition the ecological dynamics of populations in a significant way. In other words, the variational properties of G-P maps can affect population dynamics—i.e. selection and drift—in a non-random fashion. For example, Mihaela Pavlicev and Günter Wagner regard evolvability as "the probability that random mutation will improve the phenotype" (Pavlicev & Wagner 2012, p. 232). This is similar to our conception of variability above, but it includes the condition that the new phenotype is, in a broad sense, fitter than the previous one. This probability is different in distinct systems and, importantly, it is affected by the variational propensities of the G-P map. Variability, modularity, robustness and plasticity are typically considered properties that enhance evolvability. In this regard, evolvability is the study of how the complex machinery

constituting living beings is able to transform seemingly random inputs—i.e. mutations—into viable, potentially successful phenotypic variation. Wagner and Draghi put it this way:

> The most fundamental reason why research into evolvability is important for evolutionary biology is that it addresses one of the most basic assumptions of contemporary evolutionary theory: that complex organisms can arise from selection on random genetic variation. (Wagner & Draghi 2010, p. 380)

Thus the core interest of evolvability is that some general trends of how development generates variation facilitate adaptation occurring *more than it might be expected by chance*. Let us recall that, in discussing the adaptive potential of mutator mechanisms (section 3.1 of previous chapter), I have already problematized the idea of a default fitness expectation for induced mutations, from which a judgement of their randomness with respect to fitness to the local environment can be drawn. However, one has at least to admit that developmental systems are generally able to maintain highly complex systems under random genetic and environmental changes. Fisher (1930) already noticed that mutations should be less likely to be advantageous in complex enough systems, but that still these systems tend to preserve their functionality. This problem is known as the "cost of complexity" (Orr 2000),[54] namely the risk of maintaining complexity against random changes. An intuitive default expectation is that highly complex systems will be very likely to be altered by deleterious mutations, simply because there are more ways to degenerate a complex functional system than to improve it (A. Wagner 2012a). For example, there are arguably infinite many more ways to break a watch than to make it work more precisely. However, highly complex multicellular organisms are not constantly being affected by devastating deleterious mutations. Rather, they seem to be less affected by deleterious mutations than proportionally expected, which constitutes a puzzle for evolutionary biologists (e.g. Wagner & Altenberg 1996, Wagner et al. 2008). In this regard, the variational propensities of developmental

[54] Complexity can be understood in terms of the diversity in the parts of a whole (McShea & Brandon 2010). For example, Bonner (2011) associates organismal complexity in multicellulars with number of type cells. Interestingly, he argues that the more complex a biological system is, the less random are the possible changes it can undergo.

systems seem to enhance, or at least to enable in a very significant way, the emergence of viable, adapted complex organisms. Thus evolvability points to the organizational principles of development that are able to transform random inputs into phenotypic novelties and versatile traits at a sufficient rate, in turn capable of undergoing evolution by natural selection.

Philosophers of biology have recently started to pay attention to this notion (Love 2003, Sterenly 2007, Brown 2014, Brigandt 2015b, Nuño de la Rosa 2017). In the philosophical literature, evolvability has been claimed to be the evolutionary potential of specific lineages, as based on their developmental properties (Sterelny 2007); or the propensity to evolve of populations (Brown 2014). These views reflect the variety of definitions and bearers of evolvability in the scientific community, ranging from genetic systems (Hansen 2006, Salazar-Ciudad 2007) to organisms (Kirschner & Gerhart 1998, Yang 2001) to populations (Maynard Smith & Szathmáry 1995, Flatt 2005). However, from the point of view of G-P map variational propensities, evolvability is better predicated of developmental types (Brigandt 2015b, Nuño de la Rosa & Villegas 2019). From an evo-devo perspective, evolvability can be seen as a central G-P map variational property, thus predicated of the type represented by the G-P map. Under this view, different developmental types are more or less evolvable—i.e. they are more or less likely to generate fit variation—by virtue of their variational propensities. In this regard, the variability of a developmental type highly affects its evolvability, insofar as only variable developmental systems can provide fit variation in the first place. On the other hand, the robustness of a developmental type enables the safe exploration of the genotypic space of different populations, in turn facilitating the encountering of a fitter phenotype without risking the preceding one (e.g. A. Wagner 2008b). In addition, modular G-P maps are fitter when there is modular selection, thus endowing trait-by trait selection while preserving the overall integration of the organism (e.g. Pavlicev & Hansen 2011). Finally, plastic types can navigate a broader range of the phenotypic space under different environments, facilitating so-called "plasticity first" evolutionary changes (Levin & Pfennig 2016, West-Eberhard 2003), where a plastic phenotype becomes genetically assimilated in a population when environmental conditions enable it.

Philosopher Alan Love (2003) has argued that extrinsic components can also be in the causal basis of evolvability. For example, mutation rates, which are extrinsic relative to G-P maps, seem to be an important component of evolvability in some contexts (Bedau & Packard 2003). Nonetheless, they can be regarded as an intrinsic factor if evolvability is predicated of a particular population rather than of a developmental type. The most important external contributor to the propensity to evolve is the referent environment. Having more phenotypes accessible says nothing about whether or not these phenotypes will be viable, far less about whether or not they will be fitter. Thus in order to explore how variational tendencies affect adaptation, any conception of evolvability must have a potential environment considered. While it is true that evolvability does not typically refer to any particular environment, it surely cannot be an entirely intrinsic property "in the sense of environmentally invariant" (Love 2003, p. 1022). In this regard, it is interesting to note that evolvability has been associated with providing "non-lethal" variation, namely variation that doesn't necessarily destroy the functionality of the organism, in turn potentially fit to a possible environment but not restricted in any other sense (Kirschner 2013). In any case, the capacity of variational properties to affect evolutionary fate is usually regarded in abstract terms that do not specify particular environments nor populational properties. In this regard, the attribution of evolvability entails similar idealizations of the environment than the attribution of fitness and drift values (Abrams 2009), as we saw in the previous chapter (section 2). The work on the properties of Simpson's adaptive landscape (see section 4.3 of previous chapter), and of the so-called phenotype-fitness map (Salazar-Ciudad & Marín-Riera 2013) are instrumental in the generalization of relevant environmental properties for evolvability.

Much has been said in the literature about the evolution of evolvability. Indeed, the very origin of the term was introduced in the context of explaining its evolution. When Dawkins' used the notion to refer to evolutionary potential, he was explicitly concerned about how such a potential can evolve by natural selection (Dawkins 2003/1988). The main controversy behind the selection for evolvability is that it may seem to entail a teleological view of evolution. For, how can natural

selection benefit traits based on future, still-not-encountered environments? A solution to this conceptual puzzle seems to have arrived from the field of computational learning algorithms. Computational simulations have cast some light into this by showing how some general, structural features of the selective environment can be internalized by evolving systems, who can extrapolate ways of generating more successful phenotypes on average throughout the iteration of a selective process (Parter et al. 2008, Watson et al. 2016). The key to this is that those general properties of the environment that are important for understanding evolvability, are also important for understanding its evolution. This is because, while some aspects of environmental changes are certainly unpredictable, others don't vary at random. Rather, environments tend to fluctuate—for example in temperature or in number of predators—within given ranges, which enables the evolution of versatile systems that are better prepared for generating the right kind of adaptive variation under new ecological pressures. In turn, the fact that some developmental systems are better at generating new adaptive variants needs not entail any teleological puzzle, inasmuch as they have evolved in the iteration of recurrent environmental changes (Watson et al. 2014).

Consequently, similarly to the variety of scenarios that are considered for the evolution of other G-P map propensities, evolvability can be argued to be the result of many different evolutionary processes, without necessarily entailing any controversial, teleological claim. The propensity to evolve is a natural result of evolution, in a combination of developmental tendencies to produce variation and the long history of natural selection on developmental systems. Evolved propensities explain, in turn, the existence of distinct tendencies to evolve without any need of teleology or backwards means of causation. Evolvability can be thus the result of direct selection, either at the group level or at the level of individuals, who can be fitter inasmuch as their long-term offspring is fitter (Wagner & Draghi 2010). Moreover, it can be the result of indirect selection, due to the ontogenetic effects that any of the mechanisms underlying it may have (Earl & Deem 2004). Finally, evolvability can evolve as an intrinsic capacity of complex G-P interactions (Patridge & Barton 2000). In all of these possibilities, the developmental system can

internalize—be it by natural selection or not—ways to generate viable responses to the general properties of changing environments, in turn affecting how successful the sample space they provide may be in ecological terms. In the next and final subsection, I rough out some philosophical consequences of this entanglement of developmental and ecological propensities.

4.2. Variational chance and evolutionary explanations

The above discussion on evolvability serves for introducing the complex and imbricated relation that variational propensities hold with population dynamics. They not only serve as the causal grounds for chance in the sample space of evolution in each single generation. In addition, they can affect chance in evolution in a broader sense. My position here is that the variational propensities of developmental types, including their evolvability, enable a reconsideration of our ways to understand chance in evolutionary explanations.

First of all, variational propensities serve as a bridge between typological and population thinking, challenging the explanatory scope of the 'chance variation' view. Although we have seen that developmental types ought to be considered the bearers of these propensities, we have also considered how these types are epistemically motivated as based on the conserved nature of some structural developmental properties in evolution. Typology like this understood has nothing to do with there being essential causes for the type and contingent causes for its modifications—as we saw it has been sometimes represented (see introduction to the previous chapter). Rather, it has to do with some causes being conserved *and structuring an array of phenotypic possibilities* that determine the non-random character of variation. Despite typological thinking being traditionally regarded "as the very antithesis of scientific evolutionary thought" (Amundson 1998, p. 174), evo-devo's typology is instead a vindication of commonalities in structure across the living world as a deep biological reality that transcends adaptation to the local environment and thus population thinking. In this regard, the variational propensities here presented blurry the frontiers between the adaptationist, population-based view of evolution and the

typological, potentiality-based one. This is because the types bearing these propensities are partially the result of population processes as well as they influence population probabilistic dynamics. Indeed, the existence of types and their phenotypic versatility is the result of an evolutionary process where populational factors such as natural selection play an important role. Moreover, and perhaps more crucially, the variational propensities of types can influence ecological propensities—i.e. chance as sampling—in a non-trivial sense, as we have just seen is the case with evolvability and its components. The result is a non-random exploration of the morphospace that completely opposes the production of variation as a separate step "where chance reigns supreme" (Mayr 1963, see section 1 of previous chapter).

The key contribution of evo-devo studies of variational tendencies to general evolutionary biology is the consideration of the evolutionary origin, stability, versatility and ecological success of developmental types. I believe that understanding their variational tendencies in terms of causal propensities can help in integrating this central contribution of evo-devo into the causal and probabilistic understanding of classical evolutionary thought. Rather than a process where chance brings new variants and selection and drift decide on their fate, evolution can in turn be seen as a process where development creates variation in a probabilistic fashion, and selection and drift accumulate types of developmental processes. As Lewens (2009a) has argued, "the typologist claims that relatively few basic organic configurations are stable" (p. 790), which is—as we have seen—a fundamental aspect of the explanation of phenotypic evolution and the role that chance plays in it, and not merely a contingent result of descent with modification. Not only this phenomenon has to be incorporated into probabilistic explanations of short-term evolution, in the sense of accounting for the production of a sample space of evolution. In addition, it has to be regarded as the very product of an evolutionary process where developmental construction and ecological success together constitute phenotypic possibilities.

We have seen in the previous section that types like this understood can be seen as analogous to the structural properties of a die when rolled. Some authors have pointed out, in addition, that they remind of the position of Francis Galton,

ironically one of the most influential neo-Darwinians that preceded the times of the M.S. (Lewens 2009b, Gould 1993). Galton (1869) envisioned a polyhedron with limited 'positions of stable equilibrium', corresponding to the faces where it can rest upon, representing the discrete phenotypic possibilities of biological systems. The evo-devo perspective is that these structural possibilities are "active participants in the pathways of evolutionary change" (Gould 1994, p. 385). But let us see now how these structural properties, in addition to being creative of probabilistic evolutionary pathways, are the result of an evolutionary process where ecological propensities have a necessary—though definitely not exclusive—role. For doing this, we can briefly imagine that the shape of the die that is thrown has been sculptured in iterations of rolls and 'selections' on it. Let us suppose that we start with round spheres that represent developmental systems, which will go through a developmental process each time they are rolled on a board. The point at which a sphere stops represents the resulting phenotype it generates. As compared to Galton's polyhedron, which represented the interaction of selection with phenotypic results, this analogy incorporates development: the phenotypic result is the point on which the system rests after being rolled. Let us additionally suppose that, in each roll, a small number of tiny bumps and flattened surfaces can appear on the sphere. As a consequence, some points on the surface will become more stable than others, increasing the probability of the ball resting upon them at the end of the roll. The result will be that, once they are rolled, those spheres sampled for 'reproducing' new balls to roll—i.e. those systems with reproductive success—will reproduce their flattened surfaces, thus determining the very shape of the next sphere to be rolled. But importantly, in the iteration of this process, selection not only reproduces the flattened surface—i.e. the phenotype—nor the initial conditions of the roll—i.e. the genotypic inputs. Additionally, it reproduces the entire sphere, with its flattened surface and its internal structure, letting it turn into an irregular die or polyhedron in the long run, with its own probabilities of surface varying. We can see through this analogy how the evolution of a developmental type, or a polyhedron with positions of stability, constitutes a case of reciprocal causation (Laland et al. 2011), inasmuch as both internal and external factors explain its emergence. In turn, the production of a

sample space of phenotypic possibilities is the evolutionary result of a reciprocal process where conserved types and ecological versatility play equally central roles. In addition, this analogy enables us to see how propensities also structure the possible ways in which the chance setup itself can change. For example, the possible changes in the sphere will highly depend on the shape it has.

This leads to another interesting consequence of variational propensities for evolutionary explanations. We have seen that evo-devo entails an introduction of the proximate causes of development into ultime, evolutionary explanations. This can be viewed as reciprocal causation between developmental mechanisms and ultimate causes as just mentioned, or as introducing new types of questions such as those demanding lineage explanations in Calcott's sense (2009), e.g. those questions referring to the evolutionary transformations of particular developmental mechanisms (see section 4.3 of previous chapter). Indeed, one of the promises of incorporating developmental mechanisms into the explanatory structure of evolution is the possibility of raising *how questions*, and in turn proximate biological causes, into the scope of evolutionary biology (Pigliucci & Müller 2010). However, from the point of view of this dissertation, the imbrication of proximate with ultimate causes can also be seen as a broadening of the range of ultimate evolutionary questions. If ultimate questions refer to the evolutionary forces of biological phenomena rather than to their proximate causes, we can consider variational propensities of developmental types as evolutionary forces in the broad sense of being ultimate causes. Let us recall once again that evolutionary explanations are those that show how an evolutionary product may have plausibly resulted given certain causal hypotheses (Sober 1984). This can certainly be achieved by postulating specific developmental mechanisms responsible for particular transitions, thus introducing developmental proximate causes. But a probabilistic explanation involving chance instead demands postulating the causal propensities responsible for a suitable space of the possible, as we saw in sections 2 and 3 of Chapter 1. While fitness has been traditionally considered in this way, my position here is that variational propensities can be regarded with the same perspective.

This is in line with current views about evolutionary explanations as abstracting away those aspects of the complete evolutionary causal process—i.e. development *and* ecological processes—carrying with more explanatory salience for the particular type of phenomenon to be explained (Amundson 1989, Wagner 2000, Scholl & Pigliucci 2014). Interestingly, this is precisely what contemporary ideas about explanation in general hold: explanations have a contrastive character, in the sense that they explain by contrast to a background expectation (Lipton 1990), see section 3 of Chapter 1). In this regard, evolutionary processes would be explained by a plurality of causes, each of which shows a distinct degree of salience depending on the phenomenon to be explained, a view that is typically endorsed in discussions over the extension of the synthesis of evolution (Pigliucci 2007, 2009, Pigliucci & Müller 2010, Laland et al. 2011). In a pluralistic view of evolutionary explanations, we must consider for each evolutionary phenomenon the causes that make a difference for explaining it, without prioritizing the ontological commitments of the neo Darwinian understanding of evolution (Wagner 2000). For example, in the case of the vertebrate limb, its morphological stability across species has to be explained in terms of the developmental mechanisms responsible for the osteological pattern that characterizes it. Its versatility, on the other hand, will demand an explanation in terms of both the variational and ecological possibilities of limb development, as based on how variable, modular, robust and plastic it is in its different modalities, and how evolvable it is in different environments. Thus, a plurality of evolutionary causes, including both ecological and variational propensities, are necessary for having an encompassing understanding of chance and probabilistic behavior in evolution. In our case, abstracting away the variational propensities of developmental types is of explanatory relevance for explaining phenotypic evolution beyond the short-term and populational senses and, importantly, beyond the mechanistic approach to particular evolutionary transitions that represents other aspects of evo-devo and the extension of the evolutionary synthesis (e.g. Pigliucci & Müller 2010). Let us recall that, while developmental types certainly refer to developmental properties, they do so without referring to specific proximate causes of individual ontogenetic processes. As Amundson puts it:

> Even though the ontogeny of each individual salamander involves proximate processes, the urodele [i.e. salamander] limb is an abstract theoretical entity that is embedded in a theory that links evolution to ontogeny (Amundson 2005, p. 232).

In other words, considering developmental *types*—instead of mechanisms—in evolutionary explanations is not a way to include proximate causes in them but, rather, to embrace the general evolutionary forces acting beyond populations of extant variants, namely *developmental type causes*. Thus introducing variational propensities into evolutionary explanations means abstracting away from particular proximate causes in order to conceive their evolutionary potential. While they rest upon the properties of developmental systems, variability, modularity, robustness, plasticity and evolvability are general propensities instantiated in one way or another in every developmental system. In this regard, they are as general as fitness and drift: all living systems have a capacity to produce variation just like they have a capacity to survive and reproduce. Fitness values can be stronger or lighter, and certainly grounded in very different physiological and ecological mechanisms—compare the proximate causes grounding the fitness of a wheat plant and that of a jellyfish. Similarly, all developmental systems show some degree of these variational propensities, which can in turn be regarded as ultimate causes of evolution regardless of particular instantiations. In turn, while the variational propensities of types are the result of a process of reciprocal causation between developmental and ecological mechanisms, their explanatory power lies in their abstraction as general ultimate causes of phenotypic change.

From this pluralistic approach to evolutionary propensities, it is clear now that chance in evolutionary variation cannot be considered neither as opposed to natural selection nor as merely the 'raw material' of evolution (cf. section 3 of previous chapter). Rather, it has to be considered as the causally grounded phenotypic potential of reproductive and developmental systems. Consequently, variation refers to the realized effects of a developmental potential of evolutionary transformation, but it is not 'chancy' in the senses implied by the received view of evolution. In this regard, even if contingency certainly plays a role in evolution

(Beatty 1984, Gould 1989), it does not exhaust the non-selective ultimate causes of biological unity and diversity. While some features of development may be considered contingent, some of its structural properties, as we have seen, definitely are not. In addition, their evolutionary emergence may be contingent in a historical sense, but they can become highly conserved once they have emerged, to the point of turning necessary relative to a specific clade.[55] In turn, the sense of chance reflected in variational propensities is also different from chance as contingency, for it refers to the open possibilities of developmental systems rather than to their historical origin.

The potentiality of development, in its opposition to the classical externalist picture of evolution, could be seen by some as suspect of a teleological picture of evolution. The framework presented here would in turn be a naturalization of this alleged teleology and its explanatory potential, but ought not to be considered as a vindication on any goal-directedness in evolution. Instead, I believe that this framework is a step towards considering *variable inheritance*, or reproduction—in a very broad sense—, as opposed to *heritable variation*, as a fundamental component of the evolutionary process. Thinking of selection and drift as preservers of reproductive and developmental systems with the capacity to vary in certain ways, rather than as preservers of variants that can be inherited, widens the scope of the ultimate causes of evolution in a way that encompases internal tendencies to change in a demystified way.

5. Concluding remarks

In this chapter, I have argued for a causal propensity view of developmental types, stressing that the general variational properties of development, as depicted in evo-devo models of genotype-phenotype maps, allow for a causally grounded conception of chance in the evolutionary generation of variation. In the first section, I have presented the genotype-phenotype (G-P) map as the core tool in evo-devo for

[55] This idea is behind, for example, the "generative entrenchment" of some early stages of development, according to which the downstream cascade events that would follow the modification of these stages would generally render the developing organism non-viable (Wimsatt 1986).

the study of variational tendencies, where a mapping relation between genotypic inputs and phenotypic outputs is modeled. I have shown the versatility of G-P maps in representing the variational tendencies of several types of biological systems, ranging from general body plans to species-specific patterns of gene expression. I have then argued that these maps are not merely statistical, but they serve as abstractions of the type of causal processes entailing what I have called "developmental encoding", namely the set of morphogenetic rules determining a particular type of phenotypic construction. G-P maps, in translating genotypic variation into phenotypic variation, abstract away the properties of development through developmental encoding. Finally, I have presented two models of molecular evolution that serve as toy models for evo-devo variational probabilities: the highly influential RNA model and the more dynamically complex minimal cell model. These have served for showing how relatively simple developmental rules can ground in principle a mathematically consistent probability space of possible phenotypic changes.

In the second section, I have reviewed the main general variational tendencies studied in evo-devo models of G-P maps: their variability, their robustness, their modularity and their plasticity. I have presented them as general dispositions to bring about kinds of variation, regardless of the particular mechanisms instantiating them in specific developmental systems. Variability refers to the general capacity of developmental systems to generate phenotypic variation upon mutations, being typically associated with the existence of biases in the direction, magnitude and quality of potential phenotypic changes and their accessibility through mutations. Robustness is the capacity of a developmental system to generate a target phenotype under different genotypic combinations. Robust G-P maps thus attribute the same phenotype to many genotypes. Variational modularity is the disposition to generate variation in specific parts or modules composing a developmental system in an autonomous way. In this regard, it is the capacity to translate genotypic variation into phenotypic variation in a modular way. Plasticity is the ability of developmental systems to produce different environmentally specific phenotypes, thus referring to the disposition to develop different phenotypes under distinct environmental

conditions. Finally, I have presented an overview of the main hypotheses about their evolutionary origin, showing that variational dispositions may have been the target of selection, they may have evolved through indirect selection, or they may be intrinsic to genotype-phenotype relations. Since there is no scientific consensus on their evolutionary origin, and given the variety of mechanisms realizing them, a plausible scenario is some combination of these three distinct postulated mechanisms for the origin of variational dispositions.

In the third section, I have presented what I consider to be the central contribution of this dissertation, namely a developmentally grounded conception of chance in the generation of evolutionary variation. For doing this, I have stressed that variational G-P map dispositions are individualized by probabilistic patterns of ways to vary in reproduction, presenting different degrees, thus being better characterized as probabilistic dispositions or propensities. While the probabilistic behavior of developmental systems is the manifestation of these propensities, their particular effects are specific frequencies of phenotypic variants. These propensities are the result of a process of causal inference where several evo-devo methods—comparative, experimental, computational—are involved, and they refer to higher-level properties that have no direct connection with any indeterministic ontology. From this position, I have then introduced the distinction between the vernacular sense of variational chance, identifiable with these propensities; the expected sense, corresponding to specific models of variational probabilities; and the realized sense, which refers to extant frequencies of phenotypic variants. This enables to stress the distinctive role of evo-devo explanations as contrasted with statistical views of evolutionary variation. Finally, I have presented developmental types as the bearers of these variational propensities. Developmental types are idealized abstractions of the developmental pathways shared by a manifold of distinct species bearing a particular character. These types, I have argued, are the chance setups responsible for developmental trials, where variational propensities are in turn manifested.

In the fourth and final section, I have briefly explored the significance of variational propensities and developmental chance for the general explanatory structure of evolutionary biology. I have first introduced their relation with one of

the most central dispositions now encountered in evolutionary biology, namely evolvability. Evolvability is the capacity to generate variation that is potentially successful in an ecological sense. Thus evolvability is a variational as well as an ecological propensity, insofar as it is a tendency towards the production of fit variants. In this regard, developmental types bearing evolvability are the result of an evolutionary process where the chance setup for variation is able to generate a sample space where evolution by natural selection is likely. This view is a bridge between population and typological thinking, inasmuch as both conserved types and natural selection are responsible for the production of phenotypic variants. Additionally, it serves for considering variational propensities as ultimate, rather than proximate, causes of evolution. In conclusion, these remarks manifest the capacity of variational propensities to change our view of chance in evolutionary variation, from random generation to the phenotypic potential of development.

Conclusions

Throughout this dissertation, I have discussed the role of chance in the models and explanations of distinct fields in evolutionary biology, where probabilities are either informally or explicitly invoked, with a special focus on the generation of variation. I have done so in order to cast some light into the discrepancy between the classical evolutionary genetics approach and evo-devo claims about the chancy character of variation and its role in guiding the course of evolution. My main argument has two parts. First, the classical evolutionary genetics picture of evolution enables the recognition of probabilistic causes at the level of the ecological interactions of living organisms, as based on its celebrated population thinking, namely the belief that extant differences among individuals in a population ground evolutionary change. Following Millstein (2011), I have labelled this 'chance as sampling', in the sense of representing the probabilities of being sampled for generating offspring. In addition, this classical picture forbids any recognition of probabilistic causes at the level of the generation of evolutionary variants, precisely because of the population thinking embedded in its explanations. Second, some evo-devo models of phenotypic variation, by contrast, identify the developmental probabilistic causes of variation responsible for a space of possible phenotypic variants, in turn enabling the study of chance in the generation of variation. The development of this argument has departed from the construction of a suitable conceptual framework about the representative role of probabilities in the life sciences and the type of explanatory causes that can be responsible for them, namely that of causal propensities. The principal result of this dissertation has been the construction of a causally grounded notion of chance based on the different propensities to vary of what I have called, following Amundson (2005) and others (e.g. Wagner 2014, Lewens 2009), developmental types. These propensities, notably variability, robustness, modularity and plasticity, structure a space of possible ways to generate variation, in turn providing developmental systems with distinct capacities to evolve, or evolvabilities,

under different environments. This conception of chance in the generation of evolutionary variation is the result of incorporating several conceptual tools from the philosophy of probability and chance into current discussions on the nature of evolutionary variation and the ongoing debate over the explanatory structure of evolutionary biology.

In the first chapter, I have presented the ideas about probability, chance, dispositions and explanation that constitute what I consider a consistent philosophical framework from which to assess explanatory notions of chance in evolutionary biology. I started by discussing the main received traditions concerned with objective probability, namely the frequency and the propensity interpretations, inasmuch as they are representative of the two opposing views about the relation that probability holds with empirical research and scientific explanation typically found in the philosophy of biology literature. The frequency interpretation identifies probabilities with the relative frequency of an event type in a series of events, be them real or hypothetical. The propensity interpretation, by contrast, identifies probabilities with the generating conditions of frequency events. In making use of dispositions—widely used in biology—, the propensity view seems to be particularly helpful for biological explanations. Both interpretations, however, derive in a series of complications that, in my view, prevent them from being the right paradigm for discussing chance in evolution. In turn, I have considered that, while frequentism and (classical) propensionism are representative of the main positions about probability in the philosophy of evolution, addressing the significance of probabilities in evolution indeed demands a broader notion of chance than these received views on objective probability.

Since contemporary debates about evolutionary probabilities mostly concern the causal structure underlying evolutionary explanations and models, my view has been that a characterization of chance in evolution demands depicting how it relates to causation. This observation has led me to the notion of causal probability, which encopasses the philosophical literature on probabilities derived from Poincaré's Method of Arbitrary Functions, and more generally from the idea that deterministic systems can determine a probability space by virtue of their causal structure. As we

have seen, this conception enables to consider how complex dynamical systems can show relatively simple probabilistic patterns because of the way they causally relate initial to termination conditions. This broader notion of probability is a good ally for chance in evolution, inasmuch as it can make probabilistic sense of the classical Laplacian tradition of the philosophy of chance, which regards chance as measures of the possible. In particular, I have considered how it is useful for regarding what Millstein (2011) calls a unified chance concept in evolution, according to which different future states are possible given a causal arrangement. The connection of chance like this understood and causal probability is that possible future states are typically modelled through the tools of probability calculus.

Although a view such as this can certainly make sense of probabilistic models, it does not assess the seemingly explanatory role that dispositions play in biology. This has led me to develop the causal propensity framework, a proposal that combines the idea of generating conditions of probability associated to propensities with the possibilist considerations about how chance relates to causation. From this position, I have defended that dispositions, and specifically propensities, can play an important explanatory role in the establishment of spaces of possibilities. As we have seen, the characterization of dispositions, such as their functional and dynamical individuation, the distinction between their manifestation and effect that they allow for, as well as their multiple realizability, confere propensities the ability to explain probabilistic phenomena. Thus propensities can take part in regularity explanations, which differ from causal-mechanical explanations, in turn explaining probabilistic behavior. Under this view, a causal propensity is *a probabilistic disposition that structures a space of possibilities in such a way that it is possible—in principle—to derive a probability measure from it in different contexts*. In other words, a causal propensity is the capacity of a chance setup to manifest a probabilistic behavior, susceptible of being represented by probabilistic models. Importantly, the probabilistic representation is *in principle* possible but not necessary, in the sense that it need not be formalized.

The causal propensity framework developed in the first chapter has enabled me to philosophically approach some central ideas about chance in variation and the

probabilistic nature of evolution in the second chapter of this thesis. For doing this, I first reviewed the central Darwinian notion of 'chance variation', according to which variation is always abundant and unbiased, constituting the 'raw material' of evolution and waiting for its ecological opportunity to spread by means of natural selection. This conception led, almost a century later, to the Modern Synthesis understanding of variation as random copying errors of the genetic material, with gradual phenotypic effects, and which accumulated in reproduction. As we have seen, the process of evolution was established at this time as consisting of two distinct steps, namely the generation of variation, where "chance reigns supreme"; and the spread of variation, which is responsible for any directionality. Such a view constituted the celebrated 'population thinking' of evolutionary biology, where evolutionary changes are grounded on extant genetic differences among individuals within a population, as based on their fitness values. In turn, this classical perspective enabled the externalist and adaptationist picture of evolution that inseminated the models of evolutionary genetics and the philosophical concerns about them.

This revision has enabled me to apply the causal propensity view to the 'chance as sampling' ingrained in the models of evolutionary genetics, which include the probabilistic processes of natural selection and genetic drift. The main philosophical theme in the contemporary literature about population dynamics is whether the probabilistic models of evolutionary genetics refer to the causes of evolution at this level (causalist position) or they refer to statistical aggregates (statistical position). Following the general framework developed in this thesis, I have aligned with causalist advocates, arguing against the statisticalist position on the basis of their endorsement of a frequency view of probability and a non-causal understanding of explanation. I have defended that the models of population dynamics are explanatory by virtue of referring to ecological factors through pragmatic, theoretical and experimental considerations. While they generally do so through a propensity understanding of the fitness of individuals, my view is that causal propensities can also be applied to higher-level properties in evolution, such as properties of populations or types. This enables a causal understanding of the notion of chance as sampling, in the sense that ecological causal propensities structure a

space of possible populational changes that are responsible for the construction of evolutionary probabilistic models.

However, the same 'chance variation' view ingrained in the classical models of evolutionary genetics prevents any interpretation of the "first step" of evolution, namely the generation of variation, in terms of causal propensities. In evolutionary genetics models, the treatment of variation is merely statistical, as implied by the main notions of chance typically applied to its generation, and there is no recognition of the causes of variation. My position has been that there is a lack of connection between the origin and nature of variation and their impact on evolution in these models. As I have argued, this situation leads to characterize chance in variation in the same terms than chance at the ecological level, namely as opposed to the directionality of natural selection. On the one hand, evolutionary chance mutation refers to the lack of causal connection between the environmental induction of mutations and their impact on fitness. On the other, the idea that mutations bring about completely random phenotypic changes refers to the lack of causal impact of the nature of mutations in their phenotypic effects. These ideas, together with the adaptationist inclination of the classical picture of evolution, culminated in the perception of selection as a creative force, responsible not only for preserving fitter variants but crucially for generating new possible variants. However, I have defended that none of these notions refer to the causes of variation at the phenotypic level, but merely reflect a general philosophical trend that stood in the way of having a more pluralistic and empirically-based view of variation in evolution.

By contrast, I have defended that evo-devo models and explanations of phenotypic evolution do enable a causal understanding of the production of variation in evolution. Evo-devo concerns the developmental generation of phenotypic variation, in turn considering the causal structure responsible for extant variants. As we have seen, the historical background of this evo-devo agenda is situated in the tradition of morphologists, who envisioned the unity and diversity of forms as explained by typological units instantiated in different organisms in a variety of ways. In this regard, it is located in the antipodes of Darwinian population thinking and chance variation. This radically different approach has led me to

consider the causal bases of variational tendencies, namely tendencies to produce types of phenotypic variation. However, just like a mechanistic understanding of ecological interactions may serve the purposes of ecology but not of general tendencies in the models of population dynamics, a mechanistic understanding of development serves the purposes of comparative evo-devo but not of the general variational tendencies of developmental evolution, or devo-evo. For overcoming this difficulty, I have advocated for the inclusion of developmental causes into the notion of chance at the level of variation, emphasizing the role that development plays in articulating possible phenotypic changes. In order to make sense of this position from the point of view of the philosophy of probability and chance, I have introduced the idea of variational probabilities as the formal counterpart of variational tendencies. Variational probabilities are *the probabilities of generating distinct types of variation from a generation to another in the process of reproduction.* These probabilities, I have defended, determine a space of possible variants that may arise in reproduction, and only those actually arising will have a probability of being sampled by selection or drift in the "second step" in the evolutionary process.

The third and final chapter of this thesis has been devoted to the articulation of a causal propensity view of these evo-devo variational probabilities. I have first presented the main tool in evo-devo models of variation, namely the genotype-phenotype (G-P) map. G-P maps define a mapping function from specific genotypic variants to specific phenotypes, in turn capturing general properties of how genetic changes can bring about phenotypic transformations. I have argued that, in evo-devo, these maps are not merely statistical, but they are abstract representations of morphogenetic rules determining a particular type of phenotypic construction, thus entailing what I have called "developmental encoding". In order to illustrate how G-P maps can represent evo-devo variational probabilities I have presented two minimal models of a G-P map coming from molecular evolution: the RNA and the minimal cell models. In these models, how each genome sequence maps into a particular phenotype is specified with precision, therefore enabling accurate probability measures of possible phenotypic changes.

The main variational dispositions of G-P maps are variability, robustness, modularity and plasticity, whose nature and evolutionary origin are increasing focuses of attention among evolutionary biologists and philosophers of biology. On the one hand, identifying general variational capacities influencing patterns of variation is as central an evo-devo task as depicting specific developmental mechanisms. On the other hand, the evolutionary origin and significance of genotype-phenotype general relations is a key aspect for the study of the evolution of complex systems. Variability is the general capacity of developmental systems to generate phenotypic variation upon mutations, and it is typically associated with the biases present in the direction, magnitude, quality and accessibility of potential phenotypic changes. Robustness is a developmental system's disposition to produce the same phenotype under different genotypic combinations, thus showing persistence despite mutational changes. Variational modularity is the capacity to translate genotypic variation into phenotypic variation in a way that affects specific parts or modules of the developmental system with relative independence from other modules. Plasticity refers to the ability of developmental systems to develop different phenotypes under distinct environmental conditions. The evolutionary origin of G-P map dispositions seems to depend on a combination of their evolution by natural selection, their emergence through indirect selection, and their intrinsicality to genotype-phenotype relations.

After introducing these dispositions, I have applied the framework of causal propensities to them, what I consider to be the central contribution of this dissertation. In doing this, I have argued that G-P map variational dispositions are better understood as propensities, insofar as they are individualized by a probabilistic behaviour, namely different ways of producing variation with distinct degrees under reproduction. The individuation of a particular variational propensity thus is not a particular phenotype, nor a probability distribution of possible phenotypes. Instead, it is the probability distribution of *ways to generate* variation in reproduction. This probabilistic behavior is their manifestation, which differs from the particular effects they may produce in different contexts, i.e. extant variation. As we have seen, the causal modeling leading to the postulation of these properties involves several

evo-devo methods, such as the comparative, experimental and computational. In this regard, my position has been that variational propensities refer to a vernacular, thus causal, sense of variational chance. The bearers of these variational propensities, I have argued, are developmental types, as idealized abstractions of the developmental pathways common to a variety of species. By contrast, specific models of variational probabilities refer to the expected sense of variational chance, while extant frequencies of phenotypic variants refer to its realized sense. This conception is, I believe, the first step for expanding the range of application of some philosophy of probability and chance conceptual tools to the problem of the generation of variation.

I have concluded the last chapter by exploring some interesting consequences for the very explanatory structure of evolutionary biology that follow from this approach. In particular, I have considered how there is a strong dependency between extant variation acting as the sample space of evolution and the developmental sample space of possible variants, variational chance in turn affecting our views on evolution more generally. To this regard, we have seen that evolvability refers to the capacity of developmental types to generate variation that is potentially successful in an ecological sense. This renders evolvability both a variational and an ecological propensity, in the sense that developmental systems can tend to vary in fitter ways. Interestingly, developmental types, as the chance setup for variation, seem to have evolved in a way that they generate a sample space where evolution by natural selection is likely. As a consequence, the alleged dichotomy between typological and population thinking gets blurred, insofar as both conserved types and natural selection are responsible for the production of phenotypic variants. Moreover, I have argued that developmental causes like this understood act as *ultimate* causes in a similar way that natural selection does. In sum, variational propensities can change our views on chance in evolutionary variation, from random, unbiased generation to the phenotypic potential of development.

The argument developed in this thesis concerns the explanatory aspects of evolutionary biology from a causalist side, that is, as based on the belief that it is causes that we use to explain evolutionary change. To this respect, let us recall the

two fundamental explanatory claims of evo-devo with regards to evolutionary variation. On the one hand, when it comes to explain the *origin* of phenotypic variation—thus of one of the fundamental components of evolutionary change (cf. Lewontin 1970)—, the models of evo-devo overcome some of the strong limitations of the models of evolutionary genetics. In particular, while the evolutionary genetics statistical treatment of variation is very productive in the prediction of short-term evolutionary trends, they do not concern the causes of variation, which renders their long-term applicability fairly limited. Evo-devo models, on the contrary, allude to the developmental causes responsible for tendencies in phenotypic change. On the other hand, the directionality of evolutionary change is not fully considered under the adaptationist program, which includes movements along (and against) the direction of natural selection, but leaves aside questions regarding changes in the morphological dimensions of phenotypes. In this regard, evo-devo models of phenotypic variation refer to the tendencies in the exploration of the morphospace, which can entail as many dimensions as attributed to phenotypes, regardless of their fitness values.

From the perspective developed in this thesis, there are important consequences that follow for these two key aspects of variation, namely the causes of its origin and its driving role in evolution. Regarding the origin of variation, the randomness of mutations, as the ultimate source of variation, has to be carefully considered before we jump into relating it with the role of chance in evolution. The origin and nature of mutations must be, as it certainly is, examined by mutational studies. Their phenotypic effects, nonetheless, are a developmental concern. While the relation between origin and effect of mutations can be generally 'random' in the local adaptive sense, nothing precludes us from saying that the effects of mutations are extremely—and necessarily—biased by what developmental pathways they affect. The variational propensities of developmental systems, moreover, significantly affect the fitness values of the phenotypic effect of mutations. Let us recall that mutations are not only the ultimate source of variation, but the inputs of the developmental chance setups that actually generate it. If we regard mutations in this way, the fact that they may be 'random' in the first place will say nothing about the randomness of

phenotypic variation. However we end up relating source laws of variation with their consequence laws—that is, the mutational origins of phenotypic variation with its evolutionary effects—developmental causes will have to be considered as grounding evolutionary probabilistic trends. This takes us back to the notion of randomness as lack of pattern with respect to a null statistical model (Eagle 2014). Only when our evo-devo models of phenotypic evolution predict the probabilities of producing distinct phenotypic variants, we will be in the position to call variation random with respect to these models (see A. Wagner 2012a). In sum, there is still much to be done before we can safely say that variation is random, in the sense that our models predict all the patterns present in their generation. And even when—or *if*—we achieve this, phenotypic variation will only be as random as the results of rolling a fairly well-known but extremely loaded die.

Regarding the driving role of variation in evolution, the non-random occupation and exploration of the morphospace by developmental systems affect the probabilities of phenotypic change in a way that drives the evolutionary process. Importantly, this is not a claim about what selection can explore given that it is contingently positioned in a given historical pathway of the morphospace. It is, on the contrary, a claim about what things are likely to be produced so that selection—or drift—can act upon them. Historical contingency has been vindicated as an important agent of evolutionary change by critics of the MS traditional view (Gould 1989). The introduction of variational propensities is a way out, however, from the apparent dichotomy between necessity and contingency. In a very trivial sense, the survival of the fittest is a lawful fact, while every other aspect of evolution is contingent, in the sense that it is a historical product that wasn't a necessary result of evolution. In this regard, we may consider the agents of biological unity such as genes and major body plans as "frozen accidents", contingently biasing the possibilities of the only necessary true cause in evolution, namely natural selection. However, we have seen that the variational aspects of developmental types are general properties shared by developmental systems of different taxa, grounded in the physical properties of development. I believe that the position developed here is a way to deepen into the generalities of contingency (Beatty 1995, 2010), and, perhaps

more importantly, to regard the constructive role of development in evolution as a component of our probabilistic view about it. The fact that developmental types might have evolved differently than how they did, and that they are subject to evolutionary change, does not exclude that there are explanatory generalities about them that can ground our general—in the sense of not exclusive of a particular system—probabilistic models of evolution. Nonetheless, the consideration of the contingency thesis under the view of variational propensities remains underdeveloped, and shall be considered as an interesting pathway to explore in future research.

The variational propensities of developmental types are evolutionary causes that interact with the propensities of populations. The traditional philosophical interest in populations and their properties, and their relation with individuals as basis of evolutionary potential is justified by the prevalence of population thinking in classical evolutionary thought. However, we have seen that typological propensities play a fundamental explanatory role for those evolutionary phenomena that transcend the population level, such as the existence of body plans and homologues, and crucially for the very nature of variation in populations. Rather than being an alternative explanatory approach to the same evolutionary phenomenon, the typological view presented here complements the traditional populational view of evolution. It does so overcoming the limitations that a view focused on individual differences and population properties may have with respect to the nature of variation. But importantly, while the propensities of developmental types explain possible evolutionary pathways, they need to be combined with the properties of populations in order to predict particular evolutionary outcomes. Neglecting the crucial, irreducible role of natural selection and drift in evolution is as objectionable as neglecting the role of developmental properties.

From the approach developed here, natural selection, drift and variational propensities are all probabilistic ultimate causes of evolution. They have an explanatory role for the probabilistic evolutionary behavior of living systems, and must be separated from—but also related to—the mathematical, predictive models of evolution that make use of probabilities, as well as from the actual results of

evolution. Explaining evolution through ultimate causes is, as we have seen throughout this thesis, a complex process of causal inference where statistical tools are important but certainly not sufficient. Incorporating developmental dispositions, and not only ecological ones such as fitness, is an endeavor that I believe will improve our understanding of the complexity of causes determining the evolution of living systems. Let us recall that the received historiographical reconstruction of the development of biological thought envisages Darwinian theory of evolution by natural selection as a turning point in how the stunning diversity of living beings is conceptualized. However, we have seen that many of the components that are able to explain the role of variation in evolution actually precede the Darwinian tradition and have been developed relatively independently until quite recently. The indisputable historical importance of Darwin's 'chance variation' view and his principle of natural selection ought not to obscure the explanatory significance of other evolutionary components. In particular, the fact that variation is heritable may not be particularly explanatory of certain evolutionary phenomena, such as the existence of homologues. By contrast, it could be more explanatory to say that reproductive systems are variable in certain ways. In addition, if rather than envisioning heritable variation as a brute fact and a necessary condition for evolution—as the Darwinian tradition seems to imply—, we regard it as an evolutionary product susceptible to being explained, we may demand different explanatory tools than classically assumed. Importantly, the evolutionary explanation of variation, and perhaps of developmental types and their variational propensities, increases the scope of evolutionary biology without lessening their own explanatory salience in evolution (Okasha 2018).

As advanced in the introduction, the argument presented here is the result of introducing some conceptual tools of the philosophy of probability and chance into the discrepancy about the chanciness of variation between the classical and the evo-devo pictures of evolution. The ongoing discussion about the extension of the MS is indeed a very controversial one among biologists and philosophers. The position defended here certainly aligns with the Evolutionary Extended Synthesis (EES) view in important aspects, a view that is sometimes accused of not being fair

with the scope of evolutionary genetics models (e.g. Wray et al. 2014). However, this thesis does not concern the empirical applicability nor the predictive capacity of evolutionary genetics versus evo-devo. As a matter of fact, evolutionary genetics models can be, and have been, relatively well adapted to the complexity of developmental effects in phenotypic evolution (Huneman 2017). What this thesis concerns, however, is the explanatory purposes, strategies, capacities and tools of each domain. In this regard, I consider that evo-devo has many important claims about the evolutionary salience of phenotypic variation that are much better explained and included under its own modeling and theoretical tools. Exploring this problem through notions such as causal probability or explanatory propensities has enabled me to separate this explanatory concern from the broader discussion over how exactly to extend the domain of evolutionary biology. In a sense, I believe that the arguments developed here did not regard which idealizations or explanations are better in an abstract way. On the contrary, they regard what we consider to be more explanatory of specific evolutionary phenomena and, crucially, what we consider to be worth of an evolutionary explanation. This is an aspect that necessarily has to be part of the EES discussion, and indeed is recently being considered (Baedke et al. 2020). I hope that the ideas of this dissertation can be minimally helpful with regards to this.

In sum, I consider this thesis to be a well grounded starting position for future research in the philosophy of evolution. But in addition to the explorable aspects of evolutionary biology mentioned above, I believe there is also a potential in the study of propensities in biology more generally. While the explanatory role of dispositions is a hot topic in general metaphysics and epistemology, the explanatory potential of propensities in particular domains is typically less explored. But there is certain agreement about dispositions being an important epistemological tool in the life sciences, and I believe that deepening into how dispositional biological terms relate to probabilistic models of biology can be a prolific area of research. Interestingly, the philosophy of chance and probability is typically discussed by alluding to physical systems and examples from gambling games, which raises a difficulty when it comes to applying its conceptual tools to biological systems. We

have seen throughout this thesis that, while the biological realm certainly complicates these analogies, it is philosophically stimulating to build bridges between the two domains. Considerations about whether some propensity views are more convenient than others for biological purposes, or whether the complexity of living systems demands a rethink of how we understand propensities can be an interesting source of philosophical study. Given the tentative nature of the causal propensity view developed here for evolutionary purposes, there is much work to be done in this respect.

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