Chapter 2
Variation

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Abstract Understanding the origins of biological diversity is one of the main challenge for biologists. But in evolutionary biology, variation is also a starting point: natural selection can generate evolution because populations are made of non-identical individuals, transmitting different genetic combinations to offsprings. The sources of these heritable variations are to be found in the structure of DNA, the molecule of heredity, which combines feature of stability with a potential for mutability at different scales. In addition, epigenetic mechanisms can provide another source of heritable variations and evolvability.

Variation lies at the core of Darwinian thought and the concept of natural selection.1 The rehabilitation of variation as a biological parameter is one of the major reason why Charles Darwin’s ideas remain so modern.2 For the English naturalist, though this modernity does not consist of having postulated the evolution of species. Others preceded Darwin, most notably Jean-Baptiste Lamarck, who formulated this hypothesis in 1809 (laying the foundations for it in 1802); Lamarck also suggested a largely discredited mechanism for evolution, the effect of use and non-use associated with the heredity of acquired traits. Far from being a stubborn idée reçue, this mechanism was of interest to Darwin—it is even the subject of one of his main works: The Variation of Animals and Plants under Domestication 1868 —, but he had also proposed another major mechanism, natural selection (simultaneously with Wallace) that he considered as complementary, and which proved to have the most powerful impact on the explanation of evolution, all the more than heredity of acquired characters would in the same time be largely disproved. Within the hereditary mechanism of acquired traits3, the appearance of a variation is the product

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1 See Huneman, Chap. 4, this volume.
2 On this crucial question, see Charbonnat.
3 See Heams, “Heredity”, Chap. 3, this volume.

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of a force: the giraffe stretches its neck in order to be able to reach the highest leaves, and, always according to this mechanism, this variation—provided that it was carried by both parents and under certain age conditions—can be transmitted to offspring. In this sense, Lamarckism, although it is a type of evolutionism, remains limited to a universe whose basic principle is stability. There must be a force that creates variety. Without one, there is no urgency and no evolution. In Darwin’s proposed mechanism of natural selection, nature carries out a selection from the variations that appear spontaneously. This distinction appears to be nuance at first glance, yet it is in fact a radical shift in perspective. The possibility for nature to always create variations yields a vision of a dynamic world in a permanent state of transformation, calling into question the notion of a fixed universe. The transformation of the world is intrinsically linked to the existence of variation rather than being the occasional consequence of favorable circumstances. One can keep a wondering why Darwin was the among the very first to suggest this new perspective. Global and the individual factors likely have produced this foundational moment in modern biology. For Darwin, the intersection of the Enlightenment’s far-reaching influence as a freedom from a previously fixed world, profound changes in Western social structures throughout the nineteenth century, and his random luck as an observer with unequaled curiosity led to his studies of animal husbandry in England as well as the finches in the Galapagos Islands. Even if it is clear that the idea had been ripened for the picking by earlier research, such as the works Alfred Russel Wallace was above to publish as soon as 1858.

1 Which Variations Can Be Transmitted via Evolutionary Pressures at Play?

Yet what, physically, are these inheritable variations Darwin referred to without having the experimental means to discover them? The issue is more complex than it first appears: in a population, in a living organism, in an organ, at all levels, everything varies all the time (Hallgrímsson and Hall 2005). This variability (the ability to vary) and this variety (this result of variability) are physiological and anatomical: there are around 250 types of cells in a mammal such as man. These cells are also temporal: despite the feeling our permanence, which founds our identity and our individuality, nearly every cell in our bodies is regenerated roughly every 15 years leaving our bodies are nearly wholly changed; our most essential cells are much younger than we are. If we move to the molecular or atomic scale, the exchanges are even more dynamic since even the perennial macroscopic structures like bones are periodically renewed in their totality. These constant exchanges between life’s entities, and which constitute metabolism, are the very subject of biological science in the broadest sense.

In the Darwinian paradigm that concerns us here, the goal is thus to reformulate the question “what varies?” into “what are the variations that can be transmitted by the evolutionary pressures at play?”. This is a drastic restriction of the first question,
but as we shall see, it still remains incredibly vast. Darwin and his contemporaries observed visual variations of traits. The mode by which these traits were transmitted remained a mystery, and when he attempted to define it, Darwin suggested hypotheses that ultimately were false. Far from diminishing the merits of natural selection formulated in *On the Origin of Species* using incomparably rich data, however, natural selection is all the more commendable for having been suggested when its physical evidence was inaccessible. Rapid development of genetics at the beginning of the twentieth century followed the rediscovery of Gregor Mendel’s work (already three decades old) on the transmission of material determinants, or genes, from generation to generation. “Material determinants” means that, on one hand, these are physical entities, and that, on the other, each one theoretically has a link with an elementary observable trait that it “determines”. Evolutionary biology in the twentieth century will use these two fields of research: finding modes of transmission and finding the link between these entities and the corresponding trait.

The historical periods of understanding transmission have been the following: over the course of the first half of the twentieth century, genes were progressively localized in the cell’s nucleus, then physically on the DNA molecule, present in each of our cells. When James Watson and Francis Crick uncovered in DNA’s structure in 1953, the completed the discovery by describing DNA as a linkage of small units of just four types (adenosine, guanosine, cytidine, thymidine) referred to by their first letter (A, G, C and T), in a long pearl-necklace pattern so that each chain comprises a sequence unique to each individual (Watson and Crick 1953, with Rosalind Franklin). Furthermore, this molecule has two strands: when a cell divides, in can thus transmit two identical batches of DNA to its daughter cells. This is as true of a bacterial division as it is of a liver cell. DNA led, therefore, to a broad understanding of how these determines are transmitted. In addition, many geneticists had not waited for this structural discovery to demonstrate that certain agents like chemicals or X-rays could cause changes in certain traits. Watson, Crick & Franklin's discovery finally allowed them to see concretely the mechanism by which what were then referred to as mutagenic agents could have an influence on genes: they did so by modifying the DNA sequence at certain crucial points at a certain point in time. Now called mutations, these are exactly the variations that can be affected by natural selection since they are both linked to a trait and transmissible. It is also to these broadly defined mutations that we will now turn in greater detail.

## 2 How Do Mutations Appear?

Nevertheless, if only X-rays or chemical products could cause mutations, then that would still not explain their occurrence in nature. Here, molecular biology provides the essential elements for understanding how these two things could themselves spontaneously appear. The main reason, the one that is universal in the living world, is that they are duplication errors. This is possible because the cell duplicates its
genome (all of its DNA) prior to cell division. This duplication occurs due to a battery of enzymes that will, base after base, synthesize the copy in question. In humans there are several billion base pairs to faithfully duplicate. It is reasonable to imagine that even one extremely reliable enzyme that will, through biological evolution, have progressively developed to photocopy will never be *totally* reliable. Every few thousand or even hundred thousand bases depending on the species, this enzyme will occasionally make errors, and thus create mutations. These mutations will, moreover, have another particularity that squares perfectly with what Darwin intuited and what was observed in the first experiments carried out in experimental genetics: their appearance, and thus their position on DNA, are random. The DNA copy will very closely resemble the matrix, but it will never be exactly the same. This is the key to genetic mutations, which we can look at with the same perspective as Darwin had on the organisms he observed: the finesse and sophistication of this copy’s molecular mechanisms begs the question of how variations do *not* appear more often rather than how they appear at all! The capacity for creating variation is intrinsic to the mechanisms at work and it is thus not necessary *a priori* to search for a specific mechanism that generates variation; it is even less necessary to seek a force that will have this effect.

At this point, this return to Darwin requires an explanation of the link between genes and traits. Molecular biology demonstrated it: each sequence of the gene codes for a specific protein according to a (quasi) universal correspondence called the genetic code. Modifying one sequence of DNA can thus lead to a modification of the corresponding protein sequence and then of the trait in question. The classic example is the following: a simple -well known- mutation of the genetic sequence of hemoglobin can cause a single amino acid to change, which is enough to modify the hemoglobin’s folds and affect its ability to carry oxygen. Individuals who carry this mutation, especially if they inherit it from both parents (not just one) can present a major respiratory pathology. The link is thus established between the variations Darwin observed and those that geneticists observe in DNA. Natural selection will act upon traits, also called phenotypes, and favor the corresponding genotypes (groups of genes) to the detriment of others. It has been clear for a long time, however, that the “one gene/one protein” relationship is much more complex than the one I have summarized here. One sequence may be read more or less partially, giving rise to different proteins, and thus to a supplementary variability. A gene can also act upon several traits, a phenomenon called “pleiotropy”. When mutations intervene in coding sequences and are not counter-selected, they create different copies of the gene involved. These copies can coexist in a population and may potentially have different corresponding proteins. These copies are called *alleles*. A given gene will be a homozygote if the paternal allele is identical to the maternal allele; it will be a heterozygote if they differ. Population genetics is the discipline that studies populations from the angle of allelic frequencies of certain genes under the effect of evolutionary pressures: mutation, selection, migrations or genetic drift (random variation of an allelic frequency best seen in small populations).4

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4See Huneman, Chap. 4, this volume.
What, more precisely, are these inheritable variations? If we return to the DNA sequence, these mutations are, globally, any change that can arise in this sequence. There are localized “errors” such as a nucleotide (or base) deletion, substitution for another (a T replaces a G for example), or the addition of a base. These modifications, which seem trivial given the billions of base pairs that make up a genome, can have, as we have seen, important consequences. These mutations will generally degrade the trait, since the corresponding gene is the product of an evolutionary history that has given it a certain adaptation: the disturbance caused by a mutation is frequently harmful. More rarely, it will reinforce the trait to make it more adapted to circumstances, and in this case contribute to an increase in the carrier organism’s selective value, therefore favoring its survival relative to its peers. This is the core mechanism of natural selection.

In eukaryotes, however, a large part of DNA is non-coding; more than 90% of the sequence does not code for genes. Since mutations are random, they will survive more often in the majority of the genome. These mutations will not then have any functional effect and are neutral. Nevertheless, such mutations are of interest to researchers as well, but for another reason: they create variability that is transmitted to offspring, since it is not counter-selected, which in turn allows for the measurement of relationships between organisms of the same species, or of proximities between species. This is the study of polymorphism ("many forms"), the modern name for “descent with modification” that was so important to Darwin and which forms the basis for genetic analysis. The principle of using this polymorphism is as follows: these localized mutations transmitted this way will remain in the DNA from generation to generation at positions that will logically take the name SNP (single nucleotide polymorphism); in effect, several possible bases—the “initial” base and the mutation-caused base (or absence of base) will be found there (from one individual to another, from one chromosome of a pair to another). Combination (and there are hundreds of millions in the human genome, for example) of these SNP positions is like a genetic identity map unique to each individual. Knowing how to routinely detect them has a clear use as a scientific policy, for instance. They are also useful for genomic selection in livestock. Today it is possible to associate certain SNP combinations with complex traits, such as the quantity of milk produced by bovines, even though the complexity of the molecular mechanisms involved in production remains relatively unclear. How is this possible? Among the SNP, a fraction will be situated near certain genes involved in this trait. These genes (possibly unidentified) will have several alleles, contributing more or less efficiently to the trait in question, explaining in part why some cows are better producers than others (only in part, because the environment plays a role as well). Rather than undertake a lengthy characterization of each of these genes, it is simpler to determine the positions of nearby SNP whose variations reflect those of an observable trait. Once the

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5 See Grandcolas, Chap. 5, this volume.
relevant SNP’s “play”, also called an “instructive” is determined, the combinations of these SNP in any given cow can be routinely obtained with a blood sample in order to product the value of the complex trait in question. This is of great interest for livestock breeders who can use the technique to make better crossbreeding decisions by carrying out genetic tests from birth on their animals before even seeing their specific role. Such practices carry more than a little irony, since it was precisely through a familiarity with efficiency of artificial selection that Darwin elaborated his own theory, appropriating the term “selection” that came from breeders’ practices.

Do these random variations in DNA have a homogeneous pattern in the way they appear? It is important to recall that the variations we see are those that have been selected, or at least those that have not been counter-selected. Considering the mechanisms discussed earlier, nothing really suggests that the appearance of these random mutations occurs in different patterns in a given genome (bacteria that challenge this pseudo-evidence will be discussed later on). Yet the mutations that we actually see are not homogenously distributed. Their frequency varies from one region to another (typically between coding and non-coding portions) on the genome, as well as between species (the mouse genome, for example, appears to be more variable than the human genome). Differential variability is useful in phylogenesis to establish a molecular clock connecting a group of mutations to a period of divergence between studied groups (Kumar 2005). This differential speed of variability of certain genes versus others provides a useful tool based on the specific time period in question. For instance, certain genes which intervene in the ribosome, the machine that “translates” RNA into proteins, are universal and only vary very little in the living world: the rareness of their variations allows for the study of divergences between large groups over a long period of time. Other genes whose variation frequency is more rapid are more useful for making comparisons between groups in the shorter term. Biologists have thus learned to turn these natural and multiform variations into instruments of research. From criminology to animal husbandry, biologists no longer sit back and observe; they also know how to create identity cards, performance predictors, or even the history of life from variation frequency, among other tools, even if they do not always have a direct functional impact.

Localized mutations, even if they are the easiest to conceptualize, are far from the only ones that exist in DNA. There are also repetitions, in various numbers, of tiny patterns on non-coding DNA portions: for example, the sequence “AT” repeated 20 times on one chromosome and 22 times at the same position on the other chromosome in the pair (one from the father, one from the mother). There are micro-or mini-satellites as a function of the base pattern’s length. Here again, DNA copying errors in one of the ancestors explain the appearance of these variations, and since these mutations have no functional effect, they are transmitted from generation to generation. They are the source of a polymorphism, which in this case is the number of the pattern’s repetitions, and, following the very same principle described above, they can be useful for laying out an individual’s genetic identity
card, for predicting a complex trait, or for finding phylogenic connections without actually sequencing the entire genome.

The progress of sequencing techniques, which have contributed greatly to the detection of many SNP, has also led to the discovery of large-scale variations. We have known for years that genes could be found in many copies on the same genome, in tandem (some behind others) or sometimes in distant positions. This leads to a range of situations: all copies could be truly identical, coding for the same protein, as long as it is produced in a comfortable quantity. In other cases, some copies can be degraded to the point of no longer functioning; these are “pseudogenes”, which are the trace of an old duplication whose durability was not or is no longer evolutionarily useful. There are also genes that code for slightly different proteins, for example, ones that are adapted to different stages of the organism’s life cycle. The copy with modification to an existing gene is in this case an effective evolutionary solution for creating a close variant. Certain genetic sequences are mobile elements or “transposons”. Their structures may resemble the genome of certain viruses and can thus transfer by duplicating themselves in the genome. The scale of these movements is rather large, since one estimates *grosso modo* that these more or less degraded mobile elements cover half the genome. It is probably useful to have so much “non-coding” DNA, since it lowers the probability that these elements will insert themselves into coding regions! The more recently observed scale of these variations, including those among individuals of the same species, challenges the previously held notion that a species’ genomic structure was much more stable. We now refer to “the copy number variation” (CNV) to describe a complex reality: from one individual to another entire portions of the genome (arbitrarily defined as more than 1,000 bases) may or may not be duplicated, causing important quantitative differences in length. These cumulative CNV may cover regions totaling several hundred megabases, including those that code, which is up to 10 % of the total length of the genome in the case of man! (Iafrate et al. 2004; Sebat et al. 2004). The CNV opens to the door to a redefinition of the concept of species\(^6\) from a genomic point of view, or at the very least to a more continuous perspective on the passage of one to another.

### 3 Variation, Ploidy and Sexuality

The genome is where variations on all scales takes place, from the simple base to portions with tens of thousands of bases that can differ from one individual to another. Sometimes, these are even entire “extra” chromosomes that are transmitted, with a functional consequence in some cases (cilia) or pathological one in others (Trisomy 21 in humans, caused by the transmission of an “extra” copy of

\(^6\)See Samadi and Barberousse, Chap. 8, this volume.
chromosome 21). We can imagine, especially in single-celled organisms, that these random variations can sometimes be the source of genetic innovations that are potentially retained by natural selection. Ploidy variations—the number of chromosome copies—raises the corresponding issue of the extent to which sexuality is a supplementary and fundamental source of variation. For example, the human species is diploid (or 2N). This means that each individual possesses in each somatic cell chromosomes that are active “by pair”. In humans, only reproductive cells are haploid (or N), since they have a half-set that will fuse with a set coming from a gamete of the opposite sex. In each generation, the meeting of these chromosome’s haploid sets results in a diploid embryo, creating a vast combination lottery. Each pair’s chromosomes will randomly re-divide in a given gamete over the course of meiosis (cell division that creates gametes). For humans, who have 22 “autosomes”, or pairs of chromosomes, in contrast with the sex chromosome pair (the famous X and Y), that means there are already \(2^{22}\), or millions of possible combinations. In addition, the portions that correspond to homologous chromosomes (those from the same pair), are exchanged during meiosis with know way of predicting the precise limits of these portions that change randomly from one gamete to another: this is recombination. The effect of these unpredictable exchanges is that the chromosomes an individual transmits to offspring are a patchwork of maternal or paternal portions, but which maintain their overall organization and thus their functional integrity. These chromosomes will meet up with those of the corresponding gamete having undergone a recombination according to the same principle. The resulting combinatorial analysis is truly staggering… Ploidy variations over the course of a life cycle are well documented. Other variations on a much larger scale are even more so (Parfrey et al. 2008). It is possible, for instance, to establish that in certain single-celled eukaryote species, these variations in ploidies can appear between individuals (from 4 to 40N chez in certain intestinal parasites) as well as on a spectacular scale during a cycle (from N to 1,000N – ! – in certain radiolaria). This means that some organisms can have up to 250,000 chromosomes! We also know that many plants are polyploids (though on a smaller scale), as are some animals that are phylogenetically close to humans: some rodents are tetraploids (Gallardo et al. 1999). Here again, nothing rules out ploidy variations as a source of genetic, and thus evolutionary, innovation. If sexuality is defined from a genetic perspective as the exchange of genetic material between individuals that leads to a new descendent, it is also worth mentioning that the mechanisms this definition implies also exist in bacteria. In effect, exchanges of genome portions between bacteria that “conjugate” are referred to as horizontal (or lateral) gene transfer (Gogarten and Townsend 2005). These mechanisms, which certainly played a predominant role as genetic mixing when life appeared, are still a major mode of adaptation in bacteria populations today.

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7 See Gouyon and Giraud, Chap. 23, this volume.
4 Action of Variations, Evolvability, Epigenetics

When we look at the nature of variations, why do some appear continuous and others discontinuous? Variations of traits (phenotypes) may fall into two broad orders. Some are discontinuous, such as being albino or not. Others are continuous, such as an individual’s size. Do these variations indicate that there are different mechanisms at work? Do Mendel’s peas, whose variations are continuous (“wrinkled” or “smooth”), only describe one aspect of variation? This is not an innocent question, since this still-nascent “discontinuist” notion at one time seemed to oppose the gradualist view of Darwin, who envisioned an accumulation of small variations transmitted to offspring through a game of chance and selection.\(^8\) This conflict is, however, simply an opposition of two facades. Continuous traits, which are also called quantitative, are actually often complex traits that result from the interaction of many genes, each with a limited contribution to the final phenotype. If there is “one gene” whose mutation leads to albinism, there is also not “one gene” that determines an individual’s size. Many genes are involved, which is easy to understand: those that act upon the skeleton, muscular development, dietary efficiency, etc. Furthermore, these complex traits are never entirely dependent on one gene combination, no matter how large it may be. An environmental component to variation also enters into play. The study of interactions between environmental factors and genetics on the individual variation of traits, or complex phenotypes, is the basis for “quantitative genetics”. This discipline has a very strong mathematical component and has proven very powerful in the context of genetic improvement of livestock even when the genes involve in a trait are totally unknown. The precise study of the performance of individuals and their relatives (ancestors, offspring, and collaterals) eventually allows for the separation of a trait’s environmental components from its genetic ones. Although this approach had obviously not been formalized at the time, it is nevertheless clear that its empirical premises used by breeders influenced Darwin’s observations as a proponent of gradualism. We know now how to explain these continuous variations by the sum of small cumulative effects of a large number of genes whose transmission remains, individually, classically Mendelian.

Do mutations act uniformly, independently of the position upon which they act? We noted earlier on the general framework: eventual impact on the coded protein’s sequence, modification of the protein’s effect, negative consequences (often) or positive ones (rarely) on the selective value of the organism carrying the mutation, selection in the second case, and evolution of the line. Recent \textit{in vitro} work on evolution with bacteria shows, however, that certain mutations can have a potentializing effect (Taddei et al. 1997). This is the case when mutations arise on genes involved in DNA repair and duplication management, genes whose role is, precisely, to control and limit the impact of mutations. There can also be a variation in mutability when these controlling genes are affected. Their general property of control will be modified, and the bacteria lines that carry these modified genes will

\(^8\)See Heams, “Heredity”, Chap. 3, this volume.
in turn become “mutators: that is, they will have a tendency to retain more mutations than others and thus to explore more possible avenues of evolution. The study of these lines, which involves the observation of competition between mutator lines or between mutator lines and non-mutator lines is of great interest. Such lines are potentially both very adaptable (exploring new genetic solutions) and very fragile (accumulating often harmful mutations). Their ability to be cultured in fixed or changing environments and to rapidly generate offspring make these bacteria a boon to *in vivo* modeling of evolutionary dynamics. They are choice material when it comes to laying the groundwork of what is called *evolvability*, or the ability of organisms to evolve via a balance between genome stability (and thus maintenance and transmission of evolutionary solutions) and exploratory capacities. A bacterium’s evolvability cannot, of course, directly inform our understanding of a sexually reproducing multi-celled organism; however, these bacteria still constitute a very useful source for productive observations.

When initially introduced, these “mutator” bacteria lines were provocatively presented as having a Lamarckian behavior because the environment could cause their mutability. In light of the mechanisms described, however, they function according to molecular mechanisms that broadly indicate a Darwinian paradigm: these bacteria begin with mutation that randomly appears. Yet this example illustrates the fact that the issue of neo-Lamarckism is often a sensitive one when it comes to tackling “new” transmissible modes of variation. Beyond the semantic debate, it demonstrates the stunningly vast scope of variations in the living world. What is at stake in this debate is chronologically and causally situating the order between the environmental variation and the associated genetic mutation. Beyond the confines of “Lamarckian” models (environmental variation causes mutation) and the “Darwinian” model (mutation already exist and environmental variation selects for it among others), are several other models, like that of James Mark Baldwin or Igor Ivanovich Schmalhausen, who sought a middle ground that recent authors Marc Kirschner and John Gerhart, have studied and updated as “facilitated variation” (Kirschner and Gerhart 2005). They start from the principle that all of an organism’s processes are not subject to the same constraints. Certain universal processes that occur in small numbers are essential and arise from the classical mechanism of natural selection. This is the case, according to the authors, of “large” processes like DNA replication, protein translation, and cell membrane functioning; they are all constrained. Many others involve regulations that can be much less constrained. These regulations act upon the combinations of essential processes’ effects and allow the exploration of new paths. In this model, since each organism has a broad exploratory behavior, one environmental variation could cause it to take on a range of given functions within its explorable range: this is facilitated variation. An important point is that mutations could only intervene in a second instance in order to stabilize and reinforce certain attained states. The authors, like their predecessors cited here, are straddling narrow territory between the two paradigms (the mutation

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*On evolvability, and among many other references: Griswold (2006), Hendrikse et al. (2007), Pigliucci (2008), Wagner (2005).*
comes chronologically after the environmental variation, but is not caused by it, and selection remains) that they that they reinforce with many arguments that are also quite convincing. Methods for determining the level of effective generalization of this proposition, which is doubtlessly a major contribution to the current debate on evolvability, still remain to be found.

Are genetic variations in the classical sense of the term the only transmissible variations? Nothing is less certain. The field of research that generically referred to as epigenetics, and which is has undergone a revival in recent years, is used to demonstrate that other variations may eventually be transmissible as well. Some modifications of gene methylation—chemical modifications that do not affect the DNA sequence itself but which can have a functional impact—may be, in certain circumstances, transferred to offspring. Similarly, the position of chromosomes inside the nucleus is partly heritable from mother to daughter cell and we know that this position can also affect expression of the genes in question. There are also sources there of possible heritable variations whose scope has yet to be measured. Epigenetic variations are also sometimes qualified as Lamarckian and loaded with the same polemic potential as that mentioned above.

5 Conclusion

This chapter has only touched the surface of variation. By now, though, at least the broad outlines of the connections between variations and Darwinian dynamics should be clear. In organisms there are at least three areas where the variation/selection pairing drives a process. The immune system relies on the possibility for an organism to synthesize countless combinations of antibodies, some of which will recognize an antigen, triggering a large-scale preferential sequence of copies. Some Darwinian dynamics can address such variability followed by a form of selection of certain variants. In the same way, the selective stabilization of neurons that originates with the development of the nervous system relies on these neurons’ exploratory behavior, followed by a reinforcement of a certain number of connections that are initially established randomly. This is another special form of variation/selection. Finally, the inherently random dimension of gene expression followed by the stabilization of certain combinations of these genes could be a major mechanism of cellular differentiation. At minimum, this randomness of expression is manifest in the generation of necessary and sufficient diversity for the functioning of certain organs.

If Darwinism’s applications cast a long shadow, as this book certainly shows, it is often because its adopters make the connection between the existence of a variation from initial states and a selection process of these states. In addition to the fields addressed in this work, there are many other theoretical proposals on very different scales, ranging from “quantum Darwinism” in particle physics (Zurek 2009), to “cosmological natural selection” in astrophysics (Smolin 1992, 2008), and

10 See Heams, “Heredity”, Sect. 5, this volume.
“mineral evolution” in geology (Hazen et al. 2008). Authors who use, more or less metaphorically, part or all of Darwinian dynamics, do so notably by assuming the existence of varied states on the scale considered and the finitude of “resources” that can cause a selection among some of these states. Without evaluating the pertinence of such exports, they certainly demonstrate the vitality of variation. As Friedrich Nietzsche stated upon his enthusiastic exploration of biology, notably the functioning of the human body, which he called the wonder of wonders, “uniformity is pure madness” (cited in Müller-Lauter 1998). It is perhaps the most beautifully pithy definition of life and its capacity to produce, by the play of natural selection, this wonder and so many others.11

References


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