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Causal Regularities in the Biological World of Contingent Distributions*

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Abstract. Former discussions of biological generalizations have focused on the question of whether there are "universal laws" of biology. These discussions typically analyzed generalizations out of their investigative and explanatory contexts and concluded that whatever biological generalizations are, they are not universal laws. The aim of this paper is to explain what biological generalizations are by shifting attention towards the contexts in which they are drawn. I argue that within the context of any particular biological explanation or investigation, biologists employ two types of generations. One type identifies causal regularities exhibited by particular kinds of biological entities. The other type identifies how these entities are distributed in the biological world.

Key words: causal, contingent, distribution, essentialism, explanation, generalization, kind, law, regularity, ultimate and proximate explanation, universal

Every biological generalization seems to admit exceptions. Apparently, even Mendel's *Law* of Segregation is not universal; some sexually reproducing organisms are not disposed to segregate all their genes in Mendelian ratios. Most philosophers of science have therefore settled for an understanding of biology as a piecemeal application of abstract models, rather than a systematic application of universal laws (e.g. Beatty 1981; Brandon 1990; Kitcher 1984a; Lloyd 1988; Rosenberg 1985; Thompson 1988).[1] This view is at odds with traditional philosophies of science which held that universal laws play

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a central role in scientific knowledge (e.g. Nagel 1961; Hempel 1965, 1966). While numerous aspects of the traditional philosophies have been rightly rejected, many philosophers and biologists would agree that scientific understanding requires general statements of an empirical nature regardless of whether these statements fit all the criteria traditionally attributed to "laws". Yet, once philosophers decided that biology lacked genuine laws, they seem to have lost interest in analyzing the empirical generalizations of the science.[2]

Meanwhile, biologists continue to generalize. Their textbooks celebrate discoveries of important generalities such as Harvey's discovery that hearts pump blood through closed circulatory systems (Wilson 1972: p. 6), Darwin's observation that taxa on remote islands (e.g. finches on the Galapagos Islands) take on a greater variety of forms than corresponding taxa on the continents (Dobzhansky et al. 1977: p. 186), and Chargaff's finding that the ratio of purines to pyrimidines in DNA is one-to-one (Watson et al. 1987: chapters 3 and 9).[3] Discussion of such generalizations is not limited to textbook pedagogy; research journals throughout the biological sciences are filled with announcements about generalizations and discussions of their potential implications. In the field of genetics, for example, investigators are actively searching for and finding consensus sequences in genomes from different taxa. Surely, empirical generalizations, even if not formulated as true universal statements, play important roles in the scientific investigation and understanding of the biological world. The aim of this paper is to analyze the nature and role of these generalizations.

My account will distinguish between two types of empirical generalizations. I will argue that both types play important roles throughout the biological sciences and that much confusion has arisen from not distinguishing between them. Generalizations of the first type are historically-based contingencies which represent current or former distributions of biological entities of various kinds. I call these generalizations *distributions*. The second type of generalization presupposes the existence of causal regularities. Although biologists never *fully* articulate statements about these regularities, their explanatory and investigative practices identify them. Generalizations of the second type exhibit many of the features traditionally attributed to scientific laws, but I will resist the temptation to use this loaded term, and will call them, instead, *causal regularities.*[4]

The distinction between distributions and causal regularities can be illustrated by examining textbook accounts of biological reasoning as well as the research literature. Take, for instance, J. A. Wilson's textbook discussion of animal circulatory systems (1972). His account is filled with generalizations about distributions of biological entities of various kinds. It includes generalizations about the prevalence of particular kinds of circulatory systems

across taxa. For example, he writes: “A closed circulatory system – one that is formed of a continuous circuit of blood vessels – is found in vertebrates, some annelids, cephalopod molluscs, some echinoderms, nemerteans, and trematodes.” (p. 482). Wilson’s account also includes generalizations about the prevalence of certain kinds of entities within individual organisms of a taxon. For instance, he generalizes about the distribution of tissue types in different kinds of blood vessels within individual organisms. Wilson reports that blood vessels contain four kinds of tissues. He provides a figure detailing the distribution of tissue types in eight different kinds of blood vessels (Figure 12-11, p. 493). Among other trends, his figure shows that elastin is distributed in far greater amounts in the aorta than in other vessels.

Not all of Wilson’s empirical generalizations are about *distributions* of entities belonging to various biological kinds. Some of his generalizations concern *causal regularities.* For example, he explains that tissues with a high content of elastin expand and contract when subjected to increases and decreases in internal fluid pressure. This regularity, which is exhibited by the major arteries because of their high elastin content, smooths out differences in fluid pressures and blood flow in downstream vessels. Wilson’s explanation of the uniform circulation of fluids includes distributions about various kinds of things (e.g. tissue types) and causal regularities exhibited by them (e.g. expansion and contraction).

The distinction between distributions and causal regularities is easily overlooked because the two kinds of generalizations are typically presented in a seamless manner. In fact, a single sentence is often used to express both kinds of generalizations at once. Consider the following sentence from Wilson’s account:

> The major arteries possess a thick layer of elastic tissue that allows them to expand when blood is injected into them. (p. 494)

This sentence implies two different generalizations. One is a *distribution* about the prevalence of a thick elastic layer (i.e. a layer with a high proportion of elastin) in the major arteries. The other generalization concerns the disposition to expand when filled with a fluid, a *causal regularity* of vessels containing a high content of elastin.

Wilson’s overall explanation of the workings of the vertebrate circulatory system depends on generalizing about both distributions and regularities. He presents generalizations about the distribution of entities of various kinds within the elements of vertebrate circulatory systems (e.g. the distribution of blood vessels with a high content of elastin tissue in the aorta); he describes causal regularities applying to the various kinds (e.g. blood vessels with a high content of elastin tissue regularly expand and contract as internal fluid pressure increases and decreases); and he explains how these causal regular-

ities mesh to produce the regularities exhibited by the circulatory system as a whole (e.g. how regularities of heart pumping and arterial expansion and contraction bring about uniform blood flow in the peripheral tissues). Drawing the distinction between distributions and regularities clarifies the overall structure of biological explanation. Biological explanations invoke empirical generalizations that refer to causal regularities exhibited by various kinds of biological entities. The application of these generalizations is systematized, not by their apparent universal form, but by the establishment of empirical generalizations that describe the distribution of the relevant biological entities.

The distinction between distributions and causal regularities should be drawn between generalizations as they are employed within particular explanatory or investigative contexts. Consider, for instance, generalizations about tissue types in mammalian blood vessels. One might ask, do they involve simple distributions about the prevalence of various tissue types? Or, deep down, do they *really* concern causal regularities about the propensity of organisms to develop vessels with certain tissue types? Such questions cannot be answered except with respect to particular contexts. Within the context of Wilson's account of circulation, generalizations about the prevalence of tissue types do not concern causal regularities of tissue development. Wilson cites actual distributions of tissue types simply as a basis for explaining the workings of the circulatory systems in which they are distributed. Within other explanatory contexts, however, distributions that Wilson takes as the bases for explaining circulation, might be treated as things to be explained. For example, the prevalence of elastin tissue in major arterial vessels might be treated as a phenomenon to be explained in the context of developmental biology. The developmental biologist might cite a causal regularity to the effect that mammalian embryos have a propensity to develop major arterial vessels with a high content of elastin. Hence, in order to understand whether a sentence designates a distribution or a regularity (or both), we need to examine the context in which the generalization is employed. Any attempt to analyze biological generalizations out of their particular explanatory or investigative contexts will result in confusion.

The distinction between distributions and causal regularities probably applies to physics as well as to biology.[5] Although I am unaware of any philosophical discussions about distributions in physics, Nancy Cartwright's (1983) account of phenomenological laws seems to correspond to my conception of causal regularities. Cartwright distinguishes phenomenological laws from the theoretical laws commonly attributed to physics. She argues that the truth lies in the narrower phenomenological laws, which model the causal regularities observed in actual experimental systems. If I am correct, the iden-

tification of such regularities is just one of the two modes of generalizing in biology. I suspect that the other mode of generalizing, the identification of distributions, is as central to the physical sciences as it is to the biological ones, but I will leave the pursuit of such thoughts to others. I intend to focus my attention on biology.

My account begins with a brief metaphysical discussion, which sets the stage by clearly distinguishing my metaphysical assumptions from those of naive essentialism. This is important because discussions often draw a link, which I intend to sever, between the idea that there are modal regularities in biology and the naive metaphysics of an extreme essentialism. Section 2 covers the first kind of generalization, generalizations about distributions of biological entities of various kinds. I show that this kind of generalization, which has usually been overlooked in the philosophical literature, is central to biological thought.[6] Section 3 discusses the other kind of biological generalization, causal regularities. I argue that these generalizations, unlike the distributions discussed in section 2, exhibit many of the features traditionally attributed to scientific laws. Section 4 responds to the philosophical literature on laws, or rather the literature on the alleged lack of laws, in biology.

1. A non-essentialist metaphysics of biological kinds

Perhaps the easiest way to introduce my metaphysics is to contrast it to the naive metaphysics of extreme essentialism. According to the naive metaphysics, the world of possible things is divided into neatly delineated natural kinds. Suppose the kinds in such a world were represented in a multi-dimensional state-space with each dimension representing a set of alternative states or properties. Each point in the state-space represents a particular combination of properties. Only a subset of these points represents physically possible combinations of properties. Of course, there are combinations of properties that are physically possible, but have not been exhibited together in any real entity. That is, not all possible combinations of properties have been *realized*. Hence, an even smaller subset of points designates combinations of properties that have been exhibited by real things. One can visualize a state-space representation as consisting of shaded regions designating sets of points each of which represents a physically possible combination of properties. These regions contain subregions designating sets of points each of which represents a combination of properties that have actually been realized (exhibited together in a real entity). The regions of physically possible state-space are surrounded by "empty space" that represents sets of points, each of which designates a physically impossible combination of properties.

An extreme essentialist would insist that the right state-space representation of the physical world would "carve nature at its joints" by revealing well-separated, compact regions of physically possible combinations of properties. That is, extreme essentialism implies that if the right variables for a domain were included, the state-space representation would reveal well-defined dots of physical possibility. Actual entities would *necessarily* fall into one or another of these "natural kinds" and follow whatever physical laws apply to the particular kind. Such a state-space representation, according to essentialism, would be yielded only if all essential variables for a domain were included. If essential variables were not represented by a dimension in state-space, entities falling in the same dot might exhibit different properties and follow different physical laws. If non-essential variables were included, the regions representing possible combinations of properties might appear as broad regions rather than compact dots.

When applied to physical chemistry, extreme essentialism implies that the chemical elements represent discrete combinations of physical properties. The elements are clearly distinct from one another, according to this view, because it is impossible for atoms to exhibit the various combinations of properties represented by the empty regions of state-space surrounding the compact dots designating elements. The points that would form continuous transitions between elements in state-space presumably represent physically impossible, or at least highly unstable, combinations of properties.

According to extreme essentialism, science should be a tidy affair. Natural kinds are presumed to be so well-defined that members of any particular kind can be identified by different subsets of that kind's characteristic properties. For example, a token might be identified as copper by its microscopic make-up, or by its lawlike behavior, or by its exhibition of telltale macroscopic properties. This would mean that scientists could begin partitioning a domain into natural kinds before identifying the truly essential variables. Hence, one could discover the natural kind called copper and laws governing its behavior before identifying the essential properties that presumably separate this kind from others in state-space. After determining the essential properties, one could explain why entities with those properties are subject to the relevant laws and why they tend to exhibit the telltale markers. In fact, the essentialist might argue, the copper kind was specified well in advance of theories about atomic structure. And afterwards, according to the essentialist story, physicists observed that copper conducts electricity, discovered the atomic structure of copper, and constructed an explanation of why entities with the internal structure of copper conduct electricity.

The copper kind is not as well-defined as extreme essentialism would suppose. Extremely hot tokens of copper do not exhibit all the usual properties.

For example, they do not conduct electricity. One metaphysical interpretation of such exceptional behavior is that there is no causal regularity about copper conducting electricity. An alternative interpretation, and the kind to be advanced in this paper, is that the conduction behavior does involve causal regularity, but that the causal regularity is exhibited by a poorly delineated kind under poorly delineated conditions. The copper kind that conducts electricity is not simply copper. Extremely hot tokens of copper do not conduct electricity because they have a different internal structure or state than cooler tokens of copper. Hence, with respect to electrical conduction, cool tokens of copper and extremely hot tokens of copper belong to different kinds. The copper kind that conducts electricity is copper in some ill-defined, perhaps even disjunctive state. I call such ill-defined kinds *theoretical kinds* to contrast them with the natural kinds posited by essentialism. I propose we reject the metaphysics of extreme essentialism, but retain the idea that the physical and biological realms exhibit causal regularities. Causal regularities are difficult to express fully because the theoretical kinds exhibiting them do not form neatly delineated natural kinds (and as others have emphasized, the conditions under which they exhibit the regular behavior are ill-defined). Instead of corresponding to dots in state-space that are cleanly separated by regions of unrealizable state-space, *theoretical kinds* correspond to ill-defined subregions within blotches of physically realizable regions of state-space. What unites the entities falling within such an ill-defined subregion is their disposition to exhibit a causal regularity because of their similar internal structure or make-up. I favor this metaphysics because it seems to offer the most realistic perspective for making sense out of contemporary biology.[7]

Extreme essentialism provides a poor metaphysics for biology. According to this naive view, kinds in the biological world correspond to the regions in state-space designating physically possible *and biologically viable*[8] combinations of properties. An extreme essentialist would maintain that if the right dimensions were included, the resulting state-space representation would reveal well-separated, compact dots of biological viability, the natural kinds of the biological world. Cuvier advanced this kind of essentialism (with respect to species) when he argued against the possibility of evolution by claiming that organisms representing transitions between species would be biologically inviable.

Contemporary biology, however, has proven Cuvier's essentialism wrong. The regions of state-space representing biologically viable possibilities are not neatly delineated. In the state-space of the biological realm, there is an endless gradation of biologically viable possibilities; all kinds of species, organisms, organs, cells, organelles, and macromolecules might have been. The biologically viable regions of state-space would look more like smears

that blend into one another than the well-defined islands in the state-space posited by extreme essentialism. These are too poorly-delineated to represent kinds neatly individuated by discrete sets of dispositional properties and macroscopic characteristics. Furthermore, the portions of the smears that have been realized by actual entities are equally ill-defined regions of state-space that also blur into one another. The biological world is a messy place.[9]

Each theoretical kind, i.e. each kind exhibiting some causal regularity, corresponds to one or more smears of realized and non-realized state-space contained within larger smears of biological viability. Such sloppy smears of state-space do not generally represent kinds that can be precisely specified in terms of a telltale set of macroscopic properties. In addition, such kinds are not necessarily stable. An individual that exhibits a combination of properties falling within a smear representing one theoretical kind may change so that its new combination of properties falls outside the smear representing that theoretical kind. Nevertheless, these smears do represent kinds; tokens of these kinds have internal characteristics that dispose them to exhibit regular causal behavior. For example, the kind of inheritance system that segregates genes in Mendelian ratios cannot be easily identified (independently of segregation behavior) by a set of well-defined characteristics. The Mendelian segregation kind is an ill-defined smear in state-space. Nevertheless, tokens of this theoretical kind regularly segregate genes in Mendelian ratios because their similar internal make-ups cause them to segregate genes in these ratios.

The situation is even more complicated than I have suggested. The additional complication arises because a set of biological entities that form a theoretical kind with respect to one causal regularity will not form a unified theoretical kind with respect to some other causal regularities.[10] For example, the set of organisms that form the theoretical kind with respect to Mendelian segregation behavior do not form a theoretical kind with respect to crossing over. It is well known that crossing over occurs very frequently in female *Drosophila*, but not in male *Drosophila*. Hence, although most *Drosophila* belong to the Mendelian segregation kind, only about half belong to the theoretical kind that recombines genetic material according to the causal regularities represented by the genetic maps of classical genetics (the females). And presumably some females that follow the regularities of crossing over do not follow the regularity of Mendelian segregation. The smear representing the Mendelian segregation kind does not neatly coincide with the smear representing the crossing over kind. The two smears overlap. Some organisms fall under one, some under the other, and some under both.

The biological world is much sloppier than extreme essentialism implies and this has important ramifications for the practice of generalizing in biology. On the essentialist account, one could generalize about the world by identi-

fying a few central laws that apply universally within the same well-defined, natural kinds. Each natural kind and the laws governing its behavior would be important elements of knowledge because the kind would represent one of the limited number of discrete possibilities. Even if, by chance, the universe had never produced copper atoms, copper would (according to essentialism) be a natural kind arguably as important as any other for our overall understanding of physical chemistry. But in the sloppiness of the real world, not every physically or biologically viable kind is important for science. What counts as an important kind in biology depends not just on whether the corresponding combination of properties is biologically viable, but also on (1) whether evolution has produced items with that particular combination of properties and (2) whether this combination of properties results in distinctive causal behavior with explanatory significance or practical utility. One cannot generalize about such a domain by identifying a few central laws. One must generalize, as biologists do, by describing the distributions of real entities and specifying the causal regularities exhibited by important kinds.

2. Distributions

The biological literature is chocked full of generalizations about the distribution of biological entities. The four year cycle in the number of organisms in Canadian populations of small herbivores, the preponderance of arrowleaf plants with structurally rigid leaves (rather than flaccid leaves) on land and the converse of arrowleaf plants in water, the prevalence of organisms with Mendelian segregation systems among diploid taxa, and the abundance of introns in vertebrate genomes and their absence in genomes of prokaryotes are all important generalizations about prevailing distributions in the biological world. These generalizations say something about the distribution of actual tokens. It is important to distinguish them from generalizations about the causal behavior of kinds. So, for example, we need to distinguish generalizations about the distribution of arterial valves (their location within various circulatory systems) from generalizations about the causal regularities of such valves (their disposition to open and close depending on differences in fluid pressure). Analysis of the latter sort of generalization is postponed until section 3.

Generalizations about biological distributions are so prominent that one would have difficulty finding a research article, textbook chapter, or grant proposal in any of the biological sciences that does not make important use of them. Nevertheless, this type of empirical generalization has largely escaped the attention of philosophers, perhaps because we have been trained to think that the important generalizations of science must take the form of

lawlike statements. The aim of this section is to analyze these non-lawlike generalizations. Since they generalize about the prevalence of *kind-tokens* over *domains*, I begin by examining the kinds (or types) to which the tokens belong and the domains over which they are distributed. My analysis shows that biological distributions are accidental, rather than lawlike. I conclude my account of distributions by describing the roles they play in biological knowledge.

Distributions are generalizations about the prevalence of actual tokens. But tokens of what? Are they, for example, tokens of "natural kinds"? Biologists generalize about the distribution of tokens of a wide variety of types, though none of these types have the strong essentialist properties often associated with natural kinds. I use the term *type* in a very broad sense to designate the things of which entities might be tokens. I do not assume that the tokens of a particular type must share some internal make-up, structure, or set of outward characteristics or dispositional properties. For some types, tokens might be distinguished from non-tokens solely by historical relations. I limit my use of the term *kind* to cases where tokens share an internal structure or make-up, or have a common set of outward characteristics or dispositional properties that distinguish them from non-tokens. The term *theoretical kind* is restricted to cases where tokens exhibit a common causal regularity because they share a similar internal structure or make-up. Hence, as I use the terms, the category of theoretical kind is nested within kind, which is nested within type.

Distributions are generalizations about the prevalence of actual tokens of a wide variety of types and are not restricted to generalizations about tokens that share a uniform internal structure or make-up.[11] Nevertheless, many distributions do concern the prevalence of theoretical kind tokens (e.g. distributions about particular kinds of hemoglobin). Other distributions concern tokens of kinds that are determined by outward characteristics such as dispositional behavior (e.g. distributions about eusociality). And some distributions concern members of sets that are not determined by internal make-up or by outward characteristics, but by being part of a taxon (e.g. generalizations about the distribution of members of the sugar maple species). The latter kind of distribution deserves special scrutiny because of the obscure status of species and higher level taxa.

A number of biologists and philosophers, led by Michael Ghiselin (1974) and David Hull (1976), claim that members of a species are united by genealogical relations rather than by a set of common features. They argue that species should be thought of as individuals rather than kinds. (Dupré 1981, 1993; Kitcher 1984b among others have criticized the view; Sober 1984b and many others have defended it.) But even if Ghiselin and Hull are correct and species are individuals, the organisms making up such an

individual are still members of a set (the set consisting of the organisms that are constituent "parts" of the species). Hence, generalizations concerning the distribution of members of a species, under the Ghiselin-Hull view, turn out to be generalizations about the distribution of members of such sets. Calling such sets "types" and the members of such sets "tokens" may stretch the use of these terms, but it should not cause a problem provided we remain clear that generalizations about the distribution of taxa may concern the distribution of members of sets rather than tokens of genuine kinds. The use of the term token is perfectly fitting for the many cases that do not concern the distribution of taxa. These distributions concern kinds that are determined by uniform internal make-up and dispositional behavior (what I call *theoretical kinds*) or kinds that are determined simply by outward characteristics (so they would count as *kinds*, but not as *theoretical kinds*).

Another question concerning the nature of distributions pertains to the domain of generalization. Distributions generalize about the occurrence of actual tokens, but over what kind of domain do they generalize? As is so often the case in biology, the answer entails variety. Some distributions generalize over geographical regions (e.g. distributions about the pattern of marsupials over the continents) or habitats (e.g. distributions about the comparative prevalence of flaccid leaves among arrowleaf plants on land and in water), but many generalize over various taxa (e.g. distributions about the occurrence of compound eyes among vertebrates and invertebrates). Other important distributions generalize over cell lineages (e.g. those about the distribution of cytoplasmic factors in cell lineages of *C. elegans*) or over spatial regions within the individuals of a taxon (e.g. those concerning the distribution of homeotic transcripts within *Drosophila melanogaster* embryos). And a host of distributions generalize over periods of time (e.g. Williston's *Law* about the increasing prevalence of more specialized organisms). Biology provides a rich bank of generalizations about the distribution of tokens. These generalizations take on a wide variety of forms, including the spatial, ecological, taxonomic, organismic, and/or temporal distribution of various biological entities.

Distributions can be expressed in different ways. For example, the distribution that introns are prevalent in vertebrate genomes could also be expressed, perhaps more precisely, by saying that most vertebrates have multiple introns. And the distribution that nearly all *Drosophila* have Mendelian inheritance systems might also be expressed by the sentence "Mendelian inheritance systems are distributed very prevalently in the *Drosophila* taxon." These are simply different ways of expressing generalizations about the distribution of tokens in various domains.

Philosophical traditionalists might be tempted to dismiss distributions because, as my analysis clearly shows, they are not lawlike. Distributions

simply generalize about current evolutionary fashions. The process of evolution has changed the distribution of tokens in the past and undoubtedly will continue to do so. Although vertebrate genomes are filled with introns today, vertebrates of the distant future may lack introns. Hence, distributions are accidental generalizations that do not themselves represent any sort of timeless regularity, causal generality, or physical necessity. Like the generalization that actual tokens of pure gold do not weigh over 10,000 kilograms, the generalization that actual tokens of the intron type are common in vertebrates is, as far as biologists know, an accident or contingency of history. But unlike the generalization about gold, the generalization about the distribution of introns is scientifically significant. I conclude this section by describing some of the roles that distributions play in biology.

One role involves fruitfulness; the identification of distributions often leads to insights about structure, mechanism, or ecological relations. Chargaff's rule that the ratio of purines to pyrimidines in DNA is one-to-one led to the idea that DNA is structured in a way that pairs purines and pyrimidines. Another example illustrates how knowledge of distributions can lead to insights about mechanisms. Molecular biologists have recently discovered that the helicase motif (a conserved sequence found in proteins that unwind nucleic acid duplexes) is also contained in proteins coded by genes associated with DNA repair. This finding is providing hints about a mechanism that combines the functions of transcription and repair. (Buratowski 1993 discusses the relevance of the findings, which are reported by Selby and Sancar 1993; Schaeffer et al. 1993.) The insights provided by examining distributions are not limited to the molecular level. Population cycles of herbivores and carnivores, for example, reveal predator/prey relations. The temporal distribution of predators follows the temporal distribution of the prey on which they feed. Canadian predators feeding on herbivores with four year population cycles themselves have four year population cycles whereas Canadian predators feeding on herbivores with seven year population cycles have population cycles of seven years. Hence, understanding the distribution of various tokens, such as purines and pyrimidines in DNA, consensus sequences across genomes, or herbivores and carnivores over various periods of time, often lead to important advances in knowledge.

Generalizations about distributions play special roles within evolutionary biology. The distribution of species on oceanic islands differs in telling ways from the distribution of species on continents. The Galapagos Islands contain as many as fourteen species of finches ("Darwin's finches") which are much more diverse than genealogical groups on the continents. Why is there such a peculiar distribution of finches on this remote group of islands? The answer, according to evolutionists, is that ancestral finches arrived on the islands

before other birds and thus had the opportunity to adapt to many unoccupied ecological niches on the islands. The result was the extensive radiation marked by the morphological differentiation of beaks, which adapted to different ecological niches. The specification of distributions provides evolutionary biologists with fruitful information that can serve as a basis for inferences about the past course of evolution and as a store of things-to-be-explained by evolutionary theory.

Generalizations about prevailing distributions play another kind of role as well: they systematize our biological knowledge and characterize the scope of our theoretical understanding. The distribution that nearly all sexually reproducing diploid organisms have Mendelian segregation systems sets the general scope of Mendelian theory. Since the vast majority of models in population genetics are based upon the assumption that segregation is Mendelian, specifying the distribution of Mendelian segregation systems helps systematize the understanding provided by these population-level models. As becomes clear in the next section, this is crucial in biology because the scopes of biological regularities (and perhaps those of physics) are not antecedently determined. When the scope of a causal regularity is not universal over some well-delineated natural kind, determining the distribution of tokens to which the regularity applies is an important task of scientific inquiry.

These roles of generalizing about distributions are absolutely central to the development and articulation of biological knowledge. More could be said about the variety of distributions and the different roles they play in various contexts within biology. Nevertheless, the discussion here suffices to show that distributions are scientifically significant. It also partially explains what is distinctively biological about sciences like biochemistry. For like ecology, physiology, genetics, and evolutionary biology, biochemistry is linked to the world by the identification of biological distributions. Despite their centrality, generalizations about distributions have not received explicit attention in philosophical accounts of biological knowledge. This is unfortunate because any account of biology that fails to highlight the importance of generalizations about prevailing distributions is seriously incomplete.

3. Causal regularities

Generalizing about the distribution of actual tokens is central to biological thought, but explanation requires more. Citing the prevalence of helicase motifs, vessels with high elastin content, or Mendelian inheritance systems does not explain anything unless we can generalize about their behavior. Biologists do so by identifying causal regularities. For example, Wilson's account of vertebrate circulation, discussed in the opening section of this

paper, invokes a number of generalizations about causal processes including the pumping action of hearts, the expansion and contraction of arterial vessels, and the opening and closing of valves. His account is typical; explanations throughout the biological sciences appeal to generalizations about causal regularities. Ecologists cite causal regularities (e.g. tendency of broadleaf trees to grow canopies which cast shadows on rival saplings) to explain succession, evolutionists invoke causal regularities (e.g. the tendency of birds to prey on moths whose color differs from that of common resting spots) to explain the evolution of particular characteristics, classical geneticists appeal to causal regularities (e.g. regularities of chromosomal segregation) to explain the inheritance of genetic differences, and biochemists cite causal regularities (e.g. ATP's tendency to transfer chemical groups) to explain metabolism. *Nothing in biology makes sense without causal regularity.*

It is important to distinguish the regularities themselves from descriptions of the regularities. I will argue that the regularities exhibit many of the features traditionally attributed to scientific laws even though sentences describing those regularities do not exhibit the features attributed to law statements. Hence, in subsection 3.1, I focus attention on the nature of the regularities and show that they exhibit several features traditionally attributed to scientific laws. In 3.2 and 3.3, I take up some philosophical issues related to the description of these regularities. My account implies that many causal regularities are not scientifically important. I discuss the explanatory and practical features that distinguish the scientifically important regularities in 3.4.

3.1 Lawlike features of causal regularities

Generalizations such as those mentioned above represent regularities that exhibit several features traditionally attributed to scientific laws. First of all, the generalizations represent more than the actual behavior of particular entities. Each represents the potential behavior of a particular kind of entity, a potential that is determined by the internal make-up of tokens belonging to the kind. For example, the regularity involving the expansion of blood vessels with a high content of elastin tissue is lawlike in the sense that actual or potential entities belonging to the theoretical kind are causally disposed to behave in accordance with this regularity. Their common internal make-up causes them to expand and contract under the relevant conditions. Some readers might think there is something odd about attributing causal force to standing conditions, such as the condition of having a certain kind of internal make-up. But Elliott Sober (1984a) and Donald Davidson (1963) provide compelling arguments in favor of the idea that standing conditions can be causally efficacious.

Sometimes, biologists generalize about similar patterns of behavior that are exhibited by entities that do not share a common kind of internal make-up. Such generalizations, which I call *functional generalizations*, play an important role in biology. I discuss these generalizations in subsection 3.3. This subsection focuses on regularities of theoretical kinds whose tokens share a common internal make-up that causes them to behave in the specified way.

Biologists frequently discover a causal regularity before learning what internal make-up determines the relevant theoretical kind. For example, classical geneticists discovered regularities of gene expression before they had any idea what constituted a gene. They knew that *Drosophila* embryos homozygous for the mutant *w* allele develop into white-eyed adults. Morgan and his collaborators labeled the difference in make-up the "*w* allele" and could trace the difference's transmission and effects, but they did not know what constituted this difference. Nevertheless, the embryos (homozygous for the *w* allele) did indeed belong to a theoretical kind whose actual (and potential) tokens were causally disposed to develop in accordance with this regularity because of their shared internal make-up. This regularity is lawlike in the sense identified above; like the regularity of blood vessel expansion, this developmental regularity applies to actual and potential tokens of a theoretical kind (because having the formerly unknown internal make-up of the kind disposes tokens to behave in the specified ways under certain conditions).

Critics are quick to point out that sentences generalizing about biological regularities are false. Consider, for example, the following sentence:

> Blood vessels with a high content of elastin expand as internal fluid pressure increases and contract as the pressure decreases.

Strictly speaking, this statement is false; there is a possibility that because of genetic defect, aging, or injury, some vessels with a high content of elastin might nevertheless not be elastic. There is also the possibility that conditions external to vessels might prevent expansion or contraction. But these situations represent *counter-examples to the sentence, not exceptions to the intended causal regularity*. The underlying causal regularity admits no exceptions. This regularity applies to a messy kind, poorly delineated in nature and only partially specified by the phrase "blood vessels with a high content of elastin." Furthermore, it applies under a certain set of environmental conditions, which are not fully specified by the phrase "as internal fluid pressure increases and decreases." The latter point is emphasized in the philosophical literature on scientific laws, but the first point is more important in the case of biology.

I do not claim that sentences in the biological literature (or in this paper) fully describe causal regularities applying to neatly defined kinds under pre-

cisely defined conditions. What I claim is that explanations in the literature presuppose the existence of causal regularities applying to ill-defined theoretical kinds (under partially specified conditions). As I explain in the next subsection, the relevant kinds will rarely be linguistically specified in complete detail; biological kinds are too sloppy for that. But, biologists explain insofar as they succeed in targeting the sloppy kinds and identifying their causal regularities (how this is possible will be explained later in this paper). My major arterial blood vessels and yours, do indeed, share a common internal make-up of elastin tissue that causes them to expand and contract with lawlike regularity. And if our future descendants contain the same kind of circulatory system, their major arteries will also expand and contract with the same lawlike regularity. Of course these claims might be mistaken, but if so, physiologists have given us an incorrect explanation of how our circulatory systems work.[12]

The causal regularities exhibit another feature commonly attributed to laws; they support counterfactual conditionals. Consider the regularity of blood vessel expansion. Vertebrate veins contain little elastin tissue and presumably are relatively inelastic. But the regularity described by Wilson supports the counterfactual that if the veins did (contrary to fact) have a high content of elastin, then they would expand as internal fluid pressure increased. The regularity also supports counterfactual conditionals of a different sort. We can say of the major arteries of certain naturally aborted fetuses, which have a high content of elastin but have not been subjected to sudden increases in fluid pressure: if they had been subjected to sudden increases in pressure, they would have expanded. Perhaps this talk of supporting counterfactuals is just another way of saying that biological regularities apply to possible as well as actual tokens of the theoretical kind under possible as well as actual triggering conditions.

There is another sense in which the causal regularities identified by biologists are lawlike. They are neither temporally nor spatially bound. For example, blood vessels containing a high content of elastin tissue will expand and contract under the appropriate triggering conditions whenever and wherever they are so triggered. This is not an earthbound regularity, as was demonstrated by the Apollo astronauts, and we have no reason to think it will cease to be true in the future. Of course, evolution could bring an end to blood vessels as we know them, but it would still be true that if there were blood vessels with the relevant internal make-up, they would expand if subjected to an increase in internal fluid pressure (under appropriate conditions). It would be the *distribution* that such blood vessels are prevalent in higher organisms that would cease to be true.

Perhaps the most important sense in which the causal regularities are lawlike is that they have special explanatory relevance. Wilson's explanation of the circulatory system cannot stand on the simple statement that the major blood vessels have expanded when blood was pumped into them. It requires the idea that they have expanded and will continue to expand under such conditions because they have a kind of internal make-up causing them to behave in this way. Wilson doesn't provide a proximate explanation of the regularity itself, but the regularity is cited to deepen our understanding of the workings of the circulatory system of which the arteries are a part. I will have more to say about what makes causal regularities explanatorily significant in subsection 3.4. It suffices for present purposes to note that generalizations about causal regularities play an important explanatory role in biology.

I have argued that the causal regularities identified by biologists are lawlike in several important senses of the term. But I have neglected one of the most prominent features traditionally attributed to scientific laws: universality. Many philosophers seem to think that the issue of whether a generalization is truly lawlike comes down to the question of whether it is universal. I have postponed this question because the concept of universality is slippery in a world of sloppy distributions. To see why, let's consider an example. The law 'copper conducts electricity' is often said to be universal because it allegedly applies to all tokens of the copper kind, everywhere and always. The fact that the law does not apply to entities made of rubber is irrelevant. Universality does not require that regularities apply to everything, only that they apply to everything falling within particular kinds. But to what sort of kind must regularities apply in order to count as universal?

Presumably a universal regularity must apply to a kind that is well-delineated in nature. For naive essentialists, this is important because they believe that the world of possibilities consists of neatly delineated natural kinds. But if the world (or relevant domain) has no such kinds, the concept of universality does not add anything to our understanding of what it means for a regularity to be lawlike. In section 1, I claimed that the state-space of biological possibilities consists of poorly-delineated smears, rather than neatly-separated, compact dots. I suggested that the causal regularities apply to kinds that correspond to sloppy regions of realized space within the messy smears. It follows that universality is not the salient category that naive essentialism presumes. Calling a regularity universal in this context simply means that it applies to possible as well as actual tokens of some messy theoretical kind that is not well-delineated in nature or precisely defined by science. To require anything more of universality would be tantamount to invoking a metaphysics of extreme essentialism.

I conclude that causal regularities identified by biologists exhibit the most important features traditionally attributed to scientific laws. They apply to potential as well as actual tokens, they support counterfactual analysis, they are neither temporally nor spatially bound, and their identification plays a special role in biological explanations.

3.2 Generalizations describing causal regularities

I have already mentioned that sentences describing causal regularities are rarely, if ever, complete. They do not fully describe the kind whose behavior is to be explained or the conditions that trigger the specified behavior. Theoretical kinds are not well-delineated in nature and therefore are exceedingly difficult to specify in words. Philosophers often argue that the linguistic formulations used to designate alleged biological regularities are either false or "true by definition." Take the generalization about the causal behavior of blood vessels with a high content of elastin to expand and contract when subjected to increases and decreases in internal fluid pressure. As stated, this generalization is probably false because an individual organism could survive (in fact some probably have) with arteries containing defective elastin tissue that does not cause vessels to expand and contract in the specified way. One might try to rescue the generalization by revising the description of elastin to exclude defective forms. But philosophers have fertile imaginations and could easily conjure up all sorts of possibilities that would render the new formulation false (e.g. vessels made rigid by other defective layers, flexible vessels that can't expand or contract because of external coatings, vessels subjected to increases and decreases in external pressure that counterbalance changes in internal fluid pressure, etc.). Attempts to revise our description of the causal regularity from the barrage of real and imagined counter-examples would probably succeed only by producing a linguistic expression that was true by definition.

Two points should be made. First, the primary question is whether biologists have identified causal regularities that exhibit important features commonly attributed to scientific laws. Second, we can grant that linguistic formulations by themselves do not provide descriptions that fully specify such regularities and yet maintain the idea that biologists somehow manage to identify the regularities. Perhaps the contexts within which the formulations are expressed makes the specifications sufficiently complete, in a manner somewhat similar to the way contexts help fix the referents of indexical expressions. Stalnaker takes this tack to defend the idea that science supports counterfactual conditionals (1984: pp. 149–50). By appealing to a causal (or descriptive-causal) theory of reference, we can maintain that biologists refer to causal regularities that have lawlike features even if biologists never fully

describe them.[13] It is these partially described regularities that underwrite biological explanations.

We have good reason to believe that biologists have discovered causal regularities that exhibit lawlike features. For example, we have laboratory evidence that a high content of elastin causes blood vessels to expand and contract in response to changes in internal fluid pressures. Nevertheless, our knowledge is not sufficiently complete to formulate a full description of the relevant kind of make-up and triggering conditions. In a world like the one posited by extreme essentialism, it would be easy to specify fully the relevant kind and the inability to do so would be telling. But in the real world, theoretical kinds are not so well-delineated. The challenge for biologists is to identify the causal regularities of sloppy kinds. This challenge is frequently met in the absence of linguistic rigor. The philosophers' demand to express biological regularities in the form of statements that fully describe the theoretical kinds and triggering conditions simply confuses the issue of whether biologists have discovered causal regularities with lawlike features.

The lack of linguistic rigor raises questions of whether generalizations describing causal regularities are testable. Couldn't we protect our favorite such generalization from refutation simply by claiming that the counter instances are not tokens of the relevant theoretical kind? The answer is yes, but such a move carries the risk of marginalizing the disputed claim. Biologists do not explain simply by positing generalizations about causal regularities. They must also establish that these regularities are responsible for the relevant phenomena. Rescuing generalizations by claiming that the alleged exceptions are not of the posited kind is potentially self-defeating. Suppose, for example, that laboratories begin to discover that arteries with a high content of elastin taken from a variety of vertebrates do not expand upon an increase in internal fluid pressure. One could rescue the regularity about the expansion and contraction of tissues with a high content of elastin by saying that the exceptional samples do not belong to the posited theoretical kind. But such a response would immediately raise serious doubts about the explanatory relevance of the causal regularity.[14] The epistemic value of finding causal regularities with high explanatory relevance and the practical need to establish regularities that can be depended on in further research provide strong checks against the ad hoc rescue of generalizations from empirical refutation.

3.3 Generalizations describing functional regularities

My analysis assumes that causal regularities apply to tokens of a theoretical kind that share a common internal make-up. This ensures their empirical nature. But many biological generalizations describe patterns of similar

behavior exhibited by entities with quite different internal make-ups. I call these *functional generalizations* to distinguish them from individual causal regularities. The so-called *law of dominance* is a functional generalization. It can be expressed as follows: "whenever allele *a* is dominant to allele *b*, organisms with the *ab* genotype will exhibit the trait associated with the *a* allele." This generalization does not pick out a theoretical kind marked by a uniform internal make-up that causes its tokens to behave in uniform ways. The set of dominant alleles do not share any internal structure that the set of recessive alleles lack. Furthermore, there is no uniform relation between the internal structures of dominant/recessive allelic pairs. There are a variety of molecular mechanisms responsible for dominance (Hodge 1993). What the law of dominance represents is a common causal pattern resulting from several different kinds of causal interactions resulting in similar effects. That is, it picks out a common pattern of behavior resulting from several distinct causal regularities. Recognition of the dominance/recessive pattern has helped biologists find phenomena susceptible to genetic analysis. The pattern has evolutionary consequences and the generalization has therefore played an important role in the construction of evolutionary models. The appeal to functional generalizations is common in biology, especially in evolutionary biology, as Sober's (1984a) and Robert Brandon's (1990) accounts of evolutionary theory show.

Insofar as explanations appealing to functional generalizations are causal, they ultimately presuppose the existence of individual causal regularities. This follows from the idea that every causal assertion implies a potential regularity. If an entity is causally disposed to exhibit a pattern of behavior specified by a particular functional generalization, then it is disposed to do so because of one or another causal regularity.[15] Such individual regularities, combined in functional generalizations, are ultimately responsible for entities exhibiting the pattern of behavior identified by the higher level analysis. Often, as in the case of the law of dominance, biologists investigate the individual regularities. In many contexts, however, biologists are not interested in identifying the underlying causal regularities. In some situations, it is probably not in their immediate epistemic interest to try. But regardless of whether a causal explanation invokes functional generalizations or causal regularities, it ultimately rests on individual regularities each of which stems from the causal dispositions of entities sharing some kind of internal make-up.[16]

Functional generalizations exhibit at least two features associated with definitions. First, they seem to be true by definition. Second, they tend to define a pattern of behavior, rather than attribute the pattern to an independently specified kind of entity. The conceptualization of such patterns is an important part of biological theorizing and has dominated the attention of many philosophers. It has become popular to treat all propositions about

alleged regularities or laws as definitions rather than generalizations about causal regularities (for example, see Beatty 1981; Thompson 1988; Giere 1988). Marc Ereshefsky and I have argued (independently) that this view goes too far (Ereshefsky 1991; Waters 1989). While some biological generalizations about the behavior of tokens appear to be "true by definition" (i.e. appear to describe regularities exhibited by an abstract model), others, such as the one about the expansion behavior of blood vessels express something different. They posit the existence of a theoretical kind whose tokens share a common internal make-up that causes them to behave in a uniform way. The distinction between functional generalizations and individual causal regularities is not a sharp one because the difference between sharing a common internal make-up and exhibiting different ones is vague. But as we can say of the difference between rich and poor, the distinction is nevertheless a real one.

Whether a particular generalization is a functional generalization or a generalization about an individual causal regularity is an empirical question. Many geneticists originally believed that the law of dominance identified the causal consequence of having a uniform kind of genetic make-up. As Lindley Darden (1991) recounts, Bateson argued that dominant alleles involved the presence of something uniformly absent in organisms with the recessive trait. One of the pieces of evidence used against Bateson was the discovery of alleles that were dominant in relation to one alternative allele and recessive in relation to another. Linguistic appraisals will not suffice to settle the philosophical issue of whether biologists have identified causal regularities that are empirical in nature. A careful investigation of the empirical findings on a case by case basis is necessary. I believe that such investigation will reveal that some of the causal generalizations represent individual causal regularities and others serve as functional generalizations that represent common patterns of behavior resulting from several distinct causal regularities.[17]

3.4 The explanatory significance and investigative utility of causal regularities

Not all causal regularities are scientifically important. This follows from the fact that practically any true causal assertion is underwritten by a regularity to the effect that actual and possible tokens of some theoretical kind behave in the relevant manner out of causal necessity or propensity. The achievement of discovering a causal regularity, critics might complain, is trivial. "All one needs to do is make a simple observation and *voilà*, one has discovered a causal regularity. See that blade of grass with a single yellow dot? It must belong to some ill-defined theoretical kind and there must be a causal regularity to the effect that tokens of that kind grow a single yellow dot under appropriate

triggering conditions. Their internal make-up causes them to do so." The critic might also point out that the causal assertion need not even refer to actual things; my account implies that there exist causal regularities that apply to possible kinds that will never be realized in the real world. There is, admittedly, something odd about describing the potential behavior of a very rare or non-existent kind of thing a "regularity". On the other hand, there is nothing odd about calling the significant regularities discovered by biologists, such as the regularity of segregation in Mendelian inheritance systems and the expansion and contraction of arterial blood vessels, regularities. The lesson to draw is that it takes more than causality to make a regularity epistemically significant.

The scientific importance of a causal regularity is a function of both its explanatory significance and investigative utility. Three factors determine the explanatory significance of a causal regularity: explanatory scope, horizontal integration, and explanatory grounding. I will illustrate these factors by examining a regularity from classical genetics. *Drosophila* embryos containing two copies of the *w* allele and the normal genetic background characteristic of laboratory *Drosophila* develop into adults with white eyes, a regularity that I will dub the "*w regularity.*" *Explanatory scope* is the least important of the three factors, at least in the case of the *w regularity.* The scope of a regularity is related to the distribution of the relevant tokens and triggering conditions. As explained in section 2, part of the value of keeping track of distributions is that they say something about the explanatory significance of causal regularities and help systematize our application of the regularities. Causal regularities concerning kinds whose tokens (and triggering conditions) are prevalent have more potential explanatory significance, everything else equal, than regularities concerning kinds whose tokens are rare. In addition, the scope and potential significance of a causal regularity will be greater if its tokens are distributed in ways that make the regularity relevant to a wide variety of phenomena. The prevailing distribution of the *w* kind shows that its tokens are very rare and occur in a rather narrow variety of laboratory populations. The scope of the *w* regularity is not particularly broad or varied; hence, its explanatory import must lie elsewhere.

The second factor determining the explanatory significance of a causal regularity concerns its *horizontal integration* within an appropriate explanatory network. In the case of the *w regularity,* classical geneticists explained patterns of inheritance by constructing models that combined a number of regularities about the behavior of various kinds.[18] These models represented the transmission of particular gene differences from one generation to the next and the impact of these differences on development. The impact of particular gene differences was cashed out in terms of the *classical dogma* that

differences in genes cause differences in phenotypes within a uniform genetic and environmental context. Morgan and his contemporaries understood that the entire genome (as opposed to a gene) was responsible for the phenotype (within an environmental context), but they also realized that two genotypes differing with respect to only a single difference in one gene often exhibit alternative states of some phenotypic character. Their explanatory models traced the transmission of such differences from parents to offspring and related these differences to differences in phenotypic character. The explanatory basis of these models included generalizations of the form "organisms with two copies of allele *x* (with a particular difference in a particular gene) exhibit phenotype *X* and organisms with two copies of the wild-type allele (not containing the difference) exhibit the wild-type phenotype." According to my analysis, generalizations of classical genetics instantiating this form are partial descriptions of causal regularities. Organisms with allele *x* (and other unspecified internal features) form a theoretical kind whose internal make-up causes them to exhibit the distinctive phenotypic characteristic (under laboratory conditions). The *w regularity* is such a generalization and it fits neatly into the explanatory network of classical genetics.

When one examines the role of the *w regularity* within the context of classical genetics, the source of its explanatory significance becomes apparent. Its explanatory importance does not come from isolated explanations of white eye development in single organisms, but from its role in explanations of intricate patterns of inheritance. These explanations rest on a number of regularities about the transmission of genes and the development of phenotypes. The explanations, and the models on which they are based, themselves fit into the general explanatory and investigative framework of classical genetics, which adds additional significance to the individual causal regularities invoked by the various models.

The third factor determining the explanatory significance of a regularity concerns the extent of its *explanatory grounding*, that is, the extent to which scientists can explain the specified regularity in terms of the causal interaction of internal parts and external factors. This is not a necessary condition; causal generalizations may have explanatory significance even if they have not been grounded in a mechanistic explanation. The *w regularity,* for example, had explanatory significance even when geneticists knew very little about the internal make-up of the *w* kind. Morgan understood that its make-up consisted of genes on chromosomes and cytoplasmic factors. He did not, however, know what the genes themselves were made of or how they influenced development. Furthermore, he did not understand how the difference in the relevant gene affected eye color. He did not even know what the difference consisted of. The best he could do was determine the genetic location of the difference, label the

mutant version of the gene in which the difference occurred the "*w* allele", and base his explanations on the classical dogma that this difference somehow caused the difference in eye color within the genetic and environmental context of his laboratory population.

Today, we understand much more about the *w* mutation. Biologists have determined the molecular identity of the difference distinguishing flies of the *w* kind from wild-type flies. Furthermore, they have a general account of how such molecular differences cause differences in phenotypic characteristics such as eye color.[19] Many of the details of the biochemical reactions leading to the wild-type eye color in red-eyed flies (and their disruption in mutant flies) are being worked out. The law that *w* kind-tokens develop white eyes is being grounded. Insofar as geneticists have identified the underlying make-up of the *w* kind and are developing an account of why entities with that make-up have a propensity to develop white eyes, the explanatory significance of the *w regularity* is being further enhanced. In short, the understanding provided by the causal regularity is being deepened.

Regularities are often important, not just because of their explanatory significance, but also because knowledge of them has led to fruitful avenues of research.[20] In addition to their explanatory significance, regularities such as the *w regularity* had tremendous *investigative utility* for classical genetics. For one thing, they enabled geneticists to locate the relative genetic positions of gene differences. More importantly, they made it possible for geneticists to investigate chromosomal mechanics and a host of other biological processes. The *w regularity* is not scientifically important simply because it is causal, or because it applies to potential as well as actual tokens under potential as well as actual conditions, or because it is neither spatially nor temporally restricted. Its importance comes from its explanatory significance and investigative utility within the context of classical genetics.

Rich explanatory contexts and practical utility do not come cheaply even if partial descriptions of causal regularities do. The contrived generalization about the regularity of grass blades growing yellow dots is an insignificant regularity. The regularity involves a theoretical kind and triggering conditions that are rarely realized. It is not integrated within any explanatory network, and is not grounded. Furthermore, it has no investigative utility within a scientific practice. The causal generalization is rightly dismissed as trivial, *not* because it is based on a regularity that fails to have many of the important features attributed to scientific laws, but because it is based on a regularity that has absolutely no explanatory significance or investigative utility.

4. What's wrong with the arguments against laws

Several prominent philosophers of science have argued that there are no genuine laws of biology. While their conclusion may be taken as true (depending on how one chooses to interpret the term "genuine law"), their arguments go too far. Their general patterns of reasoning could also be used to argue against the existence of biological regularities with the features identified in section 3.1 (such as the feature of applying to potential as well as actual tokens). My aim in this section is to explain where the reasoning against laws tends to go wrong by critiquing three arguments that deny the existence of genuine biological laws. The first was given by J. J. C. Smart (1963) and centers on the claim that biological generalizations are not universal. The second is due to John Beatty (1981 also see 1995) and contends that biological generalizations represent historical contingencies rather than physical necessities. The third argument, which resembles Beatty's, has been advanced by Alexander Rosenberg (1985).

Smart's argument against the existence of biological laws is based on the assumption that genuine laws are universal in the sense that they apply everywhere in space and time and do not make essential reference to proper names. Smart claimed that biological generalizations are not laws because they are tacitly restricted to Earth. But, as I showed in section 3.1, there are a class of biological generalizations, those concerning causal regularities, that are not tacitly restricted to Earth. So where did Smart go wrong? By way of example, he argued that the generalization "all albinotic mice always breed true" cannot be a law because it makes tacit reference to the planet Earth by using the term "mice", which he claimed is defined by the animals' "place in the evolutionary tree." Smart admitted that it might be possible to redefine mice in a way that does not make tacit reference to Earth. This might be achieved by specifying a set of properties A_1, A_2, ... A_n possessed by all mice, and on this planet only by mice. But this would render the original generalization false, claimed Smart, because "on some planet belonging to a remote star there may well be a species of animals with the properties A_1, A_2, ... A_n and of being albinotic but *without* the property of breeding true." (Smart 1963: p. 54) So, he concluded, (1) biological generalizations are not universal and (2) if their scope is broadened in order to render them universal, the resulting generalizations are false. I criticize these claims in turn.

Smart's claim that biological generalizations are not universal rests on the mistaken assumption that the theoretical kinds to which the candidate generalizations apply preserve the divisions between genealogical lineages. In section 3.1, I argued that calling a causal regularity universal simply means that it applies to possible as well as actual tokens of some messy theoretical kind that is not well-delineated in nature. Causal regularities in

biology do not preserve the divisions between taxonomic groups. Although the *w regularity* might be treated as a regularity of the species *Drosophila melanogaster*, the regularity would probably apply to organisms that are not contained within the *Drosophila m.* lineage (recombinant DNA research has confirmed similar claims). Furthermore, not all members of the *Drosophila m.* lineage are of the *w* kind. In functional biology, there is nothing sacred about the divisions between genetic lineages drawn in conventional taxonomy. The relevant kinds are not species and phyla, but kinds of communities, organisms, cells, inheritance systems, biochemical systems and so on, each of which has tokens whose shared internal make-up causes them to behave in uniform ways. The tokens of these latter kinds are not distributed in a perfectly clear way among the taxonomic groups, which is part of the reason why investigating distributions is such an important and challenging part of biological investigation.

Smart's claim that universal biological generalizations would be false rested on the idea that there could be counter-examples on other planets. But if a particular causal regularity applies to Earth creatures, it does so because they have a uniform internal make-up that causes them to behave in the specified regular manner under appropriate triggering conditions. If the creatures on some distant planet have the same internal make-up, then that make-up would cause them to behave in the same regular manner under the same triggering conditions. If the creatures do not share that make-up, then they might not behave in accordance with the regularity, but since they did not share the relevant make-up, the misbehavior would not count against the generalization's universality.

Beatty's influential argument against the existence of biological laws is based on evolutionary considerations (1981, also see 1995). Although the argument misses the intended target, it contains an important insight about the historical contingency of biology. The argument begins with the premise that any genetically based trait of a species or collection of species is subject to mutational change. For example, meiotic drive shows that the regularity by which genes are segregated in one-to-one ratios is subject to genetic mutation. Just as other genetically based characteristics have evolved, so could the characteristics of gene transmission. Beatty explains, "Mendel's law may in fact be a good approximation of present inheritance patterns. But that principle is not the approximation of any physically necessary regularity. The evolution of non-Mendelian inheritance is theoretically possible." (Beatty 1981: p. 409) Hence, as Beatty correctly concludes, it is possible that Mendel's law will not be a good approximation of future inheritance patterns. But this does not imply that Mendel's law does not approximate any "physically necessary regularity." If what I argued in section 3.1 is correct, there is a class of bio-

logical generalizations that do approximate physically necessary regularities (or regularities maintained by weaker modalities such as propensities) and Mendel's law is such a regularity.

What the evolutionary argument shows is that the *distribution* that *nearly all sexually reproducing organisms have Mendelian inheritance systems* represents a historical contingency. It does not provide evidence against the idea that organisms found to segregate their genes in one-to-one ratios share some kind of internal make-up that causes them to regularly do so. The relevant generalization is not the one about the distribution of tokens with the particular kind of internal make-up; rather, it is the generalization that all tokens with the particular make-up have a propensity to segregate their genes in the specified way. The problem with the evolutionary argument is that it does not distinguish distributions from causal regularities and jumps to the conclusion that there are no modal regularities on the basis of the premise that prevailing distributions are historically-based contingencies (Waters 1989).

The evolutionary argument has been persuasive for a number of reasons; I will mention just two. First, the theoretical kinds in biology are obscured by the more salient categories of taxonomy. Hence, when one member of a species segregates genes in accordance with Mendel's law and another member of the same species does not, the non-conforming individual *seems* to provide a counter-example to the regularity, especially to those who harbor an allegiance to extreme essentialism. But, if the theoretical kinds to which causal regularities apply are not aligned with the division between species (as argued above), then there is no more reason to think that the non-Mendelian organism provides a counter-example to Mendel's law than there is to think that an eraser's failure to conduct electricity counts against the regularity that copper conducts electricity. In both cases, the causal regularity applies to a kind determined by a shared internal make-up that causes tokens to behave in the specified lawlike manner. Ereshefsky (1991, 1992) and Sober (1989) criticize the evolutionary argument against laws for this sort of reason.

Another reason why the evolutionary argument is so plausible is that a large part of biological theorizing involves uncovering, explaining, and determining the ramifications of non-lawlike distributions. The evolutionary argument's analysis of these generalizations is correct. It is possible that distributions of the future will differ from those of today. And this possibility has interesting ramifications. It means that causal regularities that are of little explanatory significance today, may be of great explanatory importance in the future.

The fallacy of the evolutionary argument against universal laws is to conclude that a generalization about the behavior of some theoretical kind does not identify a regularity based on modality because distributions about the

prevalence of its tokens are historically contingent. The third argument against biological laws commits the same fallacy. Rosenberg claims that Mendel's so-called laws are not truly laws, but rather "follow as empirical generalizations or particular facts about the character of terrestrial evolution, to be explained in and not assumed by evolutionary theory." (1985: p. 134) By way of justification, he invites readers to consider the two avenues for explaining why Mendel's law obtains. The first avenue involves a cytological and ultimately biochemical account of the underlying mechanism. This explanation, Rosenberg notes, "leaves unanswered the question of why normal meiosis predominates and nondisjunction [resulting in non-Mendelian segregation of genes] constitutes a rare exception." (p. 134) The answer to this question, he explains, is an evolutionary one. The second avenue of explanation answers the evolutionary question and might, he suggests, proceed on the view that normal meiosis is more adaptive and has been selected over widespread nondisjunction.

The two avenues of explanation cited by Rosenberg actually explain different generalizations. The first kind of explanation, the mechanistic one, explains the causal regularity that Mendelian inheritance systems segregate genes in one-to-one ratios. The causal disposition is determined by the internal make-up and external conditions; it is not determined by natural selection. Natural selection cannot establish the causal fact that an inheritance system with the internal make-up of the usual fruit fly has the causal disposition to segregate genes in one-to-one ratios. What natural selection can do is select for inheritance systems that have such a disposition. Hence, the second kind of explanation, the evolutionary one, explains the historically-based distribution that most inheritance systems in sexually reproducing organisms are Mendelian. It should be stressed that it is not the case that one and the same generalization is explained in two different ways. Rather, there are two different kinds of generalizations and the causal one is explained one way (mechanistically) and the contingent one is explained another way (historically). By conflating these two different generalizations, Rosenberg commits the fallacy of concluding that the first generalization does not identify a lawlike regularity because the second generalization is a contingent result of evolution.

The common view among philosophers that there are no biological laws needs to be modified as follows: there are no generalizations of the type one would expect in the naive essentialist's world. That is, there are no lawlike regularities applying to naturally delineated kinds. But the lack of such laws stems from the lack of well-delineated kinds, not from the lack of causal regularities exhibiting the most important features traditionally attributed to scientific laws. Research throughout the biological sciences has

revealed a vast number of such regularities. Some are admittedly trivial. But others are important because of their potential explanatory relevance and investigative utility. Were it not for contingencies of the history of science, these might be called the *laws of biology*. They are much narrower and more numerous than logical empiricists might have expected. And their application is systematized, not by their logical form, but by a second class of biological generalizations, generalizations concerning the distribution of tokens. But they do exhibit the most important features associated with scientific laws.

5. Conclusion

The failure to distinguish between two kinds of empirical generalizations in biology has systematically muddled the debate about biological laws. An adequate account of biological thought must distinguish the causal regularities of biology from its historically-based distributions. This difference has also been a stumbling block for those who have struggled with understanding how the reductive approach of molecular biology could possibly contribute so much to an admittedly historical discipline. Those concentrating on historical explanation, interpret biological generalizations as statements about distributions, which are properly explained in terms of evolutionary history, not in terms of the proximate causal interaction of internal components (see Mayr 1961 for a related discussion). Those focusing on the molecular advances interpret the same passages as statements about causal behavior, which are properly explained in mechanistic terms.

Part of the reason for the impasse between reductionists and their foes is that they read the generalizations of biology so differently. But neither reading has priority; both kinds of generalization are important throughout the biological sciences. What warrants emphasis is the difference between causal regularities and contingent distributions. Causal generalizations concern the behavior of theoretical kinds; distributions concern the prevalence of tokens. Causal generalizations are about the causal behavior of actual and possible tokens under actual and possible conditions; they support counterfactual conditionals, contribute explanatory force, and are themselves explained in terms of the causal interaction of components and external elements. Historically-based distributions are about the way actual tokens are, and have been, distributed in the world; they are contingent on the course of evolution, provide fruitful information that leads to important advances in biological knowledge, and are explained by historical/evolutionary considerations. Practicing biology involves identifying and explaining both kinds of generalizations.

Notes

[1] Some philosophers apply the label "law" to certain biological principles, but they have dropped the idea that laws are universal claims and have not analyzed in what sense, if any, the things they call laws are genuinely lawlike. Others use the term "law" in the traditional sense, but argue that biology lacks laws. A few have sought to retain the traditional idea of laws, but have had difficulty identifying compelling examples (Ruse 1973; Hull 1974; Rosenberg 1985). Ereshefsky (1991), Carrier (1995), and Schaffner (1995) offer the strongest case for the idea that there are genuine biological laws.

[2] Two important exceptions are John Beatty (1995) and Kenneth Schaffner (1993, 1995).

[3] I am using the term generalization liberally. I consider all of these statements to be generalizations in the sense that each of them contains a general conclusion about one or more sets (i.e. set of mammalian hearts, sets of birds, and sets of molecules).

[4] Some skeptics, such as van Fraassen 1989, seem to think they can rule out the existence of laws on what seem to be *a priori* grounds. This paper is not intended to address their concerns. I favor a more empirical approach to philosophy and a somewhat less empirical interpretation of science.

[5] I thank readers of earlier drafts, especially Erich Reck, David Hull, and Jeffry Ramsey, for convincing a stubborn author of this point.

[6] It is the kind (distributions), not tokens of the kind that have been overlooked. That is, philosophers have examined statements describing particular distributions, but they have not stepped back to consider the nature and role of this kind of statement in general. Often, their aim has been to show what these generalizations are not (genuine laws), rather than to determine what they are or what role they play in biological thought. David Hull's examination of generalizations about the distribution of forms across taxonomic groups goes further, but even Hull stops short of analyzing the general nature and role of distributional generalizations in biology. John Beatty (1995) and Kenneth Schaffner (1993, 1995) show the greatest concern for understanding the distributional generalizations of biology for what they are, rather than for what they are not.

[7] Many philosophers of science assume that matters of metaphysics ought to be decided on the basis of what makes sense of quantum mechanics, rather than what makes sense of biology. I believe they are mistaken. My confidence in our knowledge of evolution, physiology, and molecular biology is much stronger than my confidence in our understanding of quantum physics. I think it is risky to base our metaphysics, or our general views about scientific understanding, on the quirks of quantum physics.

[8] Biologically viable possibilities are physically possible combinations of properties that would be viable under the range of Earth habitats (or perhaps the wider range of Earth habitats that could result from different combinations of living forms).

[9] Dupré also criticizes a strong version of essentialism (1993). He elects to redefine "natural kind" so that it no longer carries the baggage of essentialism and can be applied to the myriad of kinds in biology. I have taken a different approach. Instead of redefining "natural kind", I introduce new terms including "theoretical kind" and (in the next section) draw distinctions between different kinds of kinds.

[10] Marcel Weber tells me that cross-classification of causal regularities also occurs in chemistry and physics. I believe that many of the claims I make about by biology are probably true more generally.

[11] David Hull has pointed out that distributions could be analyzed by applying distinctions that biologists have found helpful, rather than the philosophically motivated distinctions I draw (personal communication). For instance, one might distinguish distributions about the prevalence of entities representing homologies from distributions about the prevalence of entities representing homoplasies.

[12] Or one of us lacks either arterial vessels of the ill-defined kind or bodily conditions (external to the vessels) of the partially-specified kind.

[13] Causal theories of reference are not without their problems, but most of them arise in

the context of pure-causal theories. As Devitt and Sterelny (1987) explain, the causal fixing of a referent can be more easily accounted for on a causal-descriptive theory. Such a theory assumes that grounders associate terms with some descriptive element during the dubbing events (but not with necessary and sufficient conditions). In the case of theoretical kind terms, what I have called the partial description could play that role. Of course this simply pushes the "qua problem" onto the terms in the partial description, but our only concern here is with the question of whether there is a *special* problem of reference for theoretical kind terms.

[14] The significance of this kind of check is substantiated by John Beatty's observation that the major controversies in biology have often been waged over the "relative significance" of opposing explanations (Beatty 1995).

[15] Schaffner makes the same claim. His analysis of biological generalizations includes a category of universal claims which seems to correspond to my category of causal regularities. See Schaffner (1993, 1995).

[16] Neven Sesardic has pointed out that this account might be too narrow because it seems to imply that Newton's Laws are not causal regularities because the laws presumably apply to entities with an indefinite variety of internal structures and make-ups. But perhaps simply having a physical make-up is the relevant shared feature of entities to which Newton's Laws apply.

[17] Dupré (1993) takes a similar meta-position, but he concludes that the existence of uniform theoretical kinds is very rare, whereas I think they are common. I don't necessarily disagree with his account of the examples he discusses, but I believe his choice of examples is loaded.

[18] This discussion is based on an account of the explanatory patterns of classical genetics developed in section 2 of Waters (1994).

[19] I describe the surprisingly unified molecular-level explanation of the classical dogma that gene differences cause phenotypic differences in Waters (1994).

[20] I owe this point to Art Caplan.

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