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CETERIS PARIBUS – AN INADEQUATE REPRESENTATION FOR BIOLOGICAL CONTINGENCY

ABSTRACT. It has been claimed that *ceteris paribus* laws, rather than strict laws are the proper aim of the special sciences. This is so because the causal regularities found in these domains are exception-ridden, being contingent on the presence of the appropriate conditions and the absence of interfering factors. I argue that the *ceteris paribus* strategy obscures rather than illuminates the important similarities and differences between representations of causal regularities in the exact and inexact sciences. In particular, a detailed account of the types and degrees of contingency found in the domain of biology permits a more adequate understanding of the relations among the sciences.

1. *CETERIS PARIBUS* LAWS AND BIOLOGICAL CONTINGENCY

Biological systems are evolved, multi-component, multi-level complex systems. Their features are, in large part, historically contingent. Their behavior is the result of the interaction of many component parts that populate various levels of organization from gene to cell to organ to organism to social group. It is my view that the complexity of the systems studied by biology and other sciences has implications for the pursuit and representation of scientific knowledge about such systems. I will argue that a proper understanding of the regularities in biological systems should influence our philosophical views on the nature of causal laws and, in particular, the role of *ceteris paribus* qualifications.

A well-known problem for the special sciences, and biology in particular, is the failure of generalizations about evolved, complex systems to meet what have been identified as the defining characteristics of scientific laws. This is alleged to be a serious problem because of the special role that laws play in science. They are what science supposedly seeks to discover. They are supposed to be the codifications of knowledge about the world that enable us to explain why what happens, happens, to predict what will happen in the future or in other circumstances and provide us the tools to intervene in the world in order to reach our pragmatic goals. As such, they have been taken to be the gold standard of modern scientific practice. Philosophers have analyzed and re-analyzed the concept of a scientific

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law or a law of nature in the hopes of specifying a set of necessary and sufficient conditions that postulations of laws have to meet in order to be the "real thing" and hence be able to perform the functions of explanation, prediction, and intervention. The "received view" of what conditions are required of a law include:

1. logical contingency (having empirical content)
2. universality (covering all space and time)
3. truth (being exceptionless)
4. natural necessity (not being accidental)

Some hold that laws are not just records of what happens in the universe but are stronger claims about what must happen, albeit not logically, but physically in our world and hence have the power to dictate what will happen or what would have happened in circumstances which we have not in fact encountered. Thus laws are said to support counterfactuals. It is not clear that anything that has been discovered in science meets the strictest requirements for being a law. However, if true, presumably Newton's Laws of Motion, or The Laws of Thermodynamics, or the Law of the Conservation of Mass/Energy, would count. The closest candidates for being a law and test cases for a philosophical account of scientific law live most commonly and comfortably in the realm of physics. Many philosophers have pointed out the fact that few regularities in biology seem to meet the criteria for lawfulness enjoyed by the laws of physics.

How are we to think about the knowledge we have of biological systems that fail to be characterized in terms of universal, exceptionless, necessary truths? Their inferior status is sometimes blamed on the contingency of biological causal structures. The ways in which biological systems are organized has changed over time, they have evolved. Their causal structures thus not only could have been different but in fact were different in diverse periods in the evolution of life on the planet and in distinct regions of the earth and most likely will be different in the future. Thus exceptionless universality seems to be unattainable. The traditional account of scientific laws is out of reach for biology. Should we conclude that biology is lawless?

If so, how can we make sense of the fact that the patterns of behavior we see in a social insect colony or the patterns of genetic frequencies we see over time in a population subject to selection are caused, are predictable, are explainable, and can be used to reliably manipulate biological systems? The short answer is that biology has causal knowledge that performs the same epistemological and pragmatic tasks as strict laws without being universal, exceptionless truths, even though biological knowledge consists of contingent, domain restricted truths. This alone raises the question of

whether laws in the traditional sense should be taken as the gold standard against which to assess the success or failure of our attainment of scientific knowledge.

But perhaps we should not be too quick to abandon the standard. There is, after all, a well-worn strategy for converting domain restricted, exception ridden claims into universal truths and that is by means of the addition of a *ceteris paribus* clause. Take the causal dependency described by Mendel's law of segregation. That law says; in all sexually reproducing organisms, during gamete formation each member of an allelic pair separates from the other member to form the genetic constitution of an individual gamete. So, there is a 50:50 ratio of alleles in the mass of the gametes. In fact, Mendel's law does not hold universally. We know two unruly facts about this causal structure. First, this rule applied only after the evolution of sexually reproducing organisms, an evolutionary event that, in some sense, need not have occurred. Second, some sexually reproducing organisms don't follow the rule because they experience meiotic drive, whereby gamete production is skewed to generate more of one allele of the pair during meiotic division. Does this mean than Mendel's law of segregation is not a "law"? We can say that, *ceteris paribus*, Mendel's law holds. We can begin to spell out the *ceteris paribus* clause: provided that a system of sexual reproduction obtains, and meiotic drive does not occur, and other factors don't disrupt the mechanisms whereby gametes are produced, then gamete production will be fifty-fifty. Finer specifications about possible interference, *especially when they are not yet* identified, get lumped into a single phrase – "*ceteris paribus*" – when all else is equal, or provided nothing interferes. This logical maneuver can transform the strictly false universal claim of Mendel's law into a universally true, *ceteris paribus* law. With the *ceteris paribus* clause tacked on, even biological generalizations have the logical appearance of laws.

But, the cost of the *ceteris paribus* clause is high. First, although making a generalization universally true in this way can always be done, it is at the risk of vacuity. Woodward (this volume) makes this argument clearly and rejects *ceteris paribus* laws entirely, advocating instead a revision of our account of explanation that does not require universality. Others, like Pietroski and Rey (1995) have suggested that there are ways to fill out the *ceteris paribus* clause to make it contentful. However, the ability to fully fill in the conditions that could possibly interfere may well be an impossible task. Indeed, in evolutionary systems new structures accompanied by new rules may appear in the future, and hence we could never fully specify the content of potential interfering factors. Still others, Lange (2000, this volume) have argued that vagueness is not equivalent to vacuity.

He argues that scientists in their practice tacitly know what is meant by a *ceteris paribus* law. They know some cases of interfering factors and can extrapolate the nature of other factors by means of their family resemblance to the known ones. Earman, Roberts, and Smith (this volume) maintain that there are strict laws to be found, at least for fundamental physics, so there is no need for *ceteris paribus* laws there. Furthermore, they argue, that although the special sciences cannot discover strict laws, there are no such things as *ceteris paribus* laws. Their challenge leaves us with the problem of how to account for the explanatory and predictive power of biological generalizations if, as their account would entail, there are no laws in these domains. I will argue that it is only by providing a detailed account of biological knowledge claims that we can hope to address the problem that Earman, Roberts and Smith have posed.

Critics of the *ceteris paribus* clause correctly identify the fact that the clause violates the logical spirit of the concept of 'law'. I will argue that, more importantly, it violates a pragmatic aspect of 'laws' in that it collapses together interacting conditions of very different kinds. The *logical* cloak of *ceteris paribus* hides important differences in the *ontology* of different systems and the subsequent differences in epistemological practices. Where as *ceteris paribus* is a component of the statement of a causal regularity, what it is intended to mark in the world is the *contingency* of the causal regularity on the presence and/or absence of features upon which the operation of the regularity depends. Those contingencies are as important to good science as are the regularities that can be abstracted from distributions of their contextualized applications.

Indeed, a familiar way to mark the difference between exact and special sciences is by pointing to the contingency of the products of biological evolution, and the contingency of the causal dependencies to which they are subject. This is what Beatty dubs 'the evolutionary contingency thesis' (Beatty 1997). It is meant to capture the meaning of Steven J. Gould's metaphoric appeal to Frank Capra's "It's a Wonderful Life" (Gould 1990). That is, if we rewound the history of life and 'played the tape' again, the species, body plans and phenotypes that would evolve could be entirely different. The intuition is that small changes in initial 'chance' conditions can have dramatic consequences downstream. Sexual reproduction itself is thought to be a historically contingent development and hence the causal rules that govern gamete formation, for example, are themselves dependent on the contingent fact that the structures that obey those rules evolved in the first place. Biological contingency denotes the historical chanciness of evolved systems, the 'frozen accidents' that populate our planet, the lack of necessity about it all.

There have been different responses to the mismatch between the strict, ideal version of scientific law and the products of the special sciences.

- Biology has NO LAWS on the standard account of laws (Beatty, 1995, 1997, Brandon)
  - This may not be so bad, if as Cartwright argues science doesn't strict need laws or as Woodward argues we can have explanatory generalizations without universality.
- Biology HAS LAWS on the standard account of laws (Sober, Waters)
  - There are not many and these are *ceteris paribus* laws or are very abstract, perhaps mathematical truths or laws of physics and chemistry.
- Biology HAS LAWS on a revised account
  - We need to reject the standard account of laws and replace it with a better account (Mitchell)

Some have opted to accept the lesser status of biological generalizations and preserve the language of law for those venerable truths that people like Steven Weinberg dream will be few and so powerful as to make all the other knowledge claims we currently depend on part of their deductive closure. If laws are understood in the strict sense, biology doesn't have any, and the picture is even worse for the social sciences (Beatty 1995, 1997; Brandon 1997). Others while accepting this conclusion have gone on to suggest that laws aren't all they are cracked up to be, anyway, and so it is not so bad that the special sciences fail to have them – we don't need them (Cartwright 1994, 1999). Still others scramble to construe the most abstract of relationships within the special sciences or some physical or chemical regularity internal to biological systems as laws (Sober 1997; Waters 1998), thus there are some, though not many and not clearly identified as distinctively biological (Brown and West 2000). There are some, nevertheless, and hence the legitimacy of the special sciences can be restored according to this line of argumentation.

I find these responses less than satisfying. We need to rethink the idea of a scientific law pragmatically or functionally, that is, in terms of what scientific laws let us do rather than in terms of some ideal of a law by which to judge the inadequacies of the more common (and very useful) truths. Woodward (2002, this volume) also adopts a strategy to reconsider the nature of laws in the special sciences, rather than forcing those claims uncomfortably into the standard view, wedged in with the help of *ceteris paribus* clauses. He had developed an account of explanation that requires

generalizations less than universal in scope, but which can, nevertheless, support counterfactuals.

My strategy is somewhat different: I recommend that we look more closely at the character of the contingencies of the causal dependencies in biological systems that are often lumped into a single abstract concept of 'contingency' and singled out as *the* culprit preventing biological science to be lawful (Beatty 1997). I have argued elsewhere (1997, 2000) that general truths we discover about the world vary with respect to their degree of contingency on the conditions upon which the relationships described depend. Indeed, it is true that most of the laws of fundamental physics are more generally applicable, i.e., are more stable over changes in context, in space and time, than are the causal relations we discover hold in the biological world. They are closer to the ideal causal connections that we chose to call 'laws'. Yet, few of even these can escape the need for the *ceterus paribus* clause to render them logically true. Indeed, Cartwright has argued that we can't find a single instantiation of these laws and Earman et al. in this volume admit that we have yet to identify (or at least have evidence that we have identified) a law of physics. What's going on here?

The difference between fundamental physics and the special sciences is *not* between a domain of laws and a domain of no laws. Yet, I would agree, there are differences and those differences can inform our understanding of not only the special sciences but of the very notion of a 'law' and its function. By broadening the conceptual space in which we can locate the truths discovered in various scientific pursuits we can better represent the nature of the actual differences. The interesting issue for biological knowledge is not so much whether it is or isn't just like knowledge of fundamental physics, but how to characterize the types of contingent, complex causal dependencies found in that domain.

Recent developments in understanding scientific causal claims take as their starting point less than ideal knowledge. They explore how to draw causal inferences from statistical data, as well as how to determine the extent of our knowledge and range of application from the practices of experimentation. (Spirtes et al., 1993; Glymour 2001; Woodward 2000, 2001, 2002). When we see that some relationship holds here and now for this population in this environment, what can we say about the next population in the next environment? These new approaches recognize the less-than-universal character of many causal structures. If explanatory generalizations were universal, then when we detect a system in which A is correlated with B, and we determine that this is a causal relationship, we could infer that A would cause B in every system. But we often cannot. The difficulty goes beyond the correlation-causation relationship. Even when

we gave good evidence that *A* causes *B* in a system, say through controlled intervention, we still can't say that *A* would cause *B* in every system. When we look at the tidy behavior of Mendel's pea plants, where internal genetic factors assort independently and segregate fairly, we might wish to infer that would always happen, in any sexually reproducing population. But it doesn't. And since it doesn't, we need to understand more about the system of Mendel's peas and their relationship to other systems to know what about the original test case is exportable to the new domains. If we were so lucky as to have detected a universal exceptionless relationship, constitutive of the strict interpretation of law, we would know it would automatically apply to all times and all places. But that is not the world of the special sciences.

In systems that depend on specific configurations of events and properties which may not obtain elsewhere, and which include the interaction of multiple, weak causes rather than the domination of a single, determining force, what laws we can garner will have to have accompanying them much more information if we are to use that knowledge in new contexts. These are precisely the domains that the special sciences take as their objects of study. Thus the central problem of laws in the special sciences, and perhaps for all sciences, is shifted from what is a strict law, *ceteris paribus*, or no law at all, to how do we detect and describe the causal structure of complex, highly contingent, interactive systems and how do we export that knowledge to other similar systems (Mitchell 2002).

Let me then consider the parts of this situation in turn. First, what kinds of complexity are present in biological systems? Second, what is that nature of the contingency of causal structures in biology? Third, what are the implications of complexity and contingency for scientific investigation?

2. COMPLEXITY

While the term 'complexity' is widely invoked, what is meant by it varies enormously. Often linked with chaos and emergence, current definitions of complexity numbered somewhere between 30 and 45 in 1996 (at least according to Horgan's report of Yorke's list. See Horgan (1997, fn. 11, p. 197)). I believe the multiplicity of definitions of 'complexity' in biology reflects the fact that biological systems are complex in a variety of ways.

- They display complexity of ***structure***, the whole being formed of numerous parts in non-random organization (Wimsatt 1986; Simon 1981).

- They are complex in the *processes* by which they develop from single celled origins to multicellular adults (Raff 1996; Goodwin 1994; Goodwin and Saunders 1992) and by which they evolve from single celled ancestors to multicellular descendants (Buss 1987; Bonner 1988; Salthe 1993).
- The *domain* of alternative evolutionary solutions to adaptive problems defines a third form of complexity. This consists in the wide diversity of forms of life that have evolved despite facing similar adaptive challenges. (Beatty 1995; Mitchell 1997, 2002).

2.1. *Compositional Complexity*

Minimally, complex systems can be identified in contrast to simple objects by the feature of having parts. Simon defined a complex system as "made up of a large number of parts that interact in a nonsimple way . . . the whole is more that the sum of the parts" (1981 p. 86). Complex systems are also characterized by the ways in which the parts are arranged, i.e., the relations in which the components stand or their structure. The cells constituting a multicellular organism differentiate into cell types and growth fields (Raff 1996). A honeybee colony has a queen, and workers specialized into nursing, food storage, guarding or foraging tasks (Winston 1987; Wilson 1971). Such systems are bounded, have parts, and those parts differentially interact. In hierarchical organization the processes occurring at a specific level – for example the level of an individual worker in a honey bee colony – may be constrained both from below by means of its genetic make up or hormonal state and from above by means of the demographic features of the colony. In Simon's terms, the system is partially decomposable. Interactions occur in modules and do not spread across all parts constituting the system. For example, in a honey bee colony if a need develops for more pollen, the workers whose task it is to forage for pollen will increase their activity, but changes need not occur in all other task groups (Page and Mitchell 1991).

Different types of organizational structures will mediate the causal relations within a level and between levels. Some modular structures shield the internal operations of a system from external influences, or at least from some set of them. Other features may structure the way in which external information is transmitted through the module.

2.2. *Complex Dynamics*

The non-linearity of mathematical models which represent temporal and spatial processes has become, for some, the exclusive definition of com-

plexity. While this aspect of complexity is widespread it still captures only one aspect of process complexity. Process complexity is linked with a number of dynamical properties including extreme sensitivity to initial conditions (the butterfly effect) and self-organizing and recursive patterning (thermal convection patterns) (Nicolis and Prigogine 1989). Self-organization refers to processes by which global order emerges from the simple interactions of component parts in the absence of a pre-programmed blueprint.

The sensitivity of complex behaviors is further complicated by the fact that developing and evolving organisms and social groups are subject to the operation of multiple, weak, nonadditive forces. For example, in an evolving population natural selection may be operating simultaneously at different levels of organization – on gametes, on individuals with variant fitness relative to their shared environment, on kin groups of different degrees of relatedness, etc. (Brandon 1982; Sober 1984; Hull 1989). In addition to multiple levels of selection, genetic drift may influence the patterns of change in the frequencies of genes through time. Phylogenetic constraints which limit the options for adaptive response, as well as physical constraints, such as the thermal regulation differentials for land dwelling or sea dwelling endotherms, may also stamp their character on the types of change available in an evolving population. One, some, or all of these different forces may operate in varying combinations.

2.3. *Evolved Diversity of Populations*

The third sense of complexity found in biology is exhibited by the diversity of organisms resulting from historical contingencies. Given the irreversible nature of the processes of evolution, the randomness with which mutations arise relative to those processes, and the modularity by which complex organisms are built from simpler ones, there exists in nature a multitude of ways to 'solve' the problems of survival and reproduction. For example, a social insect colony adjusts the proportions of workers active in particular tasks in correspondence to both internal and external factors. If there is a destruction of foragers, the younger individuals may leave their nursing or food storing tasks to fill the vacant jobs. This homeostatic response is accomplished in different ways by different species. Honeybee colonies harbor sufficient genetic variability among the workers to generate variant responses to stimuli (Page and Metcalf 1982; Calderone and Page 1992). Ant colonies, on the other hand, may accomplish the same sort of response flexibility by means not of genetic variability by the variations in nest architecture (Tofts and Franks 1992). The ways in which information gets modulated through these systems depends on a host of properties. But what

is important to notice here is that the manner by which one social insect colony solves a problem may well be different from solutions found by similar organisms.

Historical contingencies contribute to the particular ways in which organisms develop and evolve. History fashions what mutational raw materials are available for selection to act upon; what resources already present can be co-opted for new functions; and what structures constrain evolutionary developments.

Complexity carries with it challenges to scientific investigation of causal dependence and the discovery of explanatory laws. For example, redundancy of systems to generate a functional state will make experimentation in the standard sense less definitive. What could we learn from a controlled experiment? Imagine we are trying to determine the consequences of a particular component, say of visual information on the direction of flight of honeybee foragers. We create two study populations that are identical for genetics, age, environment, food source, etc. We block the visual uptake of the individuals in the first population while leaving the system operative in the second population. Then we look to see what differences there are in the foraging behavior, ready to attribute differences to the role of visual information. It could well be that the foraging behavior in the two populations is identical. Does that tell us that visual information plays no causal role in foraging decisions? No. It is plausible to postulate that when visual information is not available, an olfactory system takes over and chemical cues in that mechanism generate the same behavioral responses – i.e., ones that are adaptive or optimal responses to foraging problems.

Redundancy of mechanism makes controlled experimental approaches problematic. This is not to say that each modality or mechanism cannot be studied to determine the contribution of each in the absence of the activation of the others. However, redundancy can take a number of different forms that make the ways in which the mechanisms mutually contribute to an overall outcome vary. For example as described in the hypothetical case above, there may be serial and independent redundant 'back-up' systems. There may be mutually enhancing or amplifying systems or, conversely, mutually dampening systems. Knowing the causal laws that govern each process in isolation by experimentation will not automatically yield sufficient information for drawing an inference to their integrated contribution in situ. As I will discuss below, this type of complexity introduces a special set of contingencies in which to consider the operation of any partially isolatable mechanism.

Another feature of complexity also affects epistemological practices. Consider situations of what we might call phase changes or nonlinear processes whereby the causal relationship governing the behavior of two variables changes completely at certain values of one of the variables. Consider the slime mold, dichtystelium, which lives much of its life as a collection of individual single cells moving through space in search of food. The movements of the cells are driven by the detection of food. However, should a group of cells find itself in a situation in which the value of the food variable is below a threshold, i.e., what would be close to starvation rations, then an entirely new set of causal dependencies kick in. Now instead of each cell moving toward food in a predictable way, the individual cells are drawn to each other, amass together and form a new organism, a multi-cellular slime mold made up of stalk and fruiting body. The latter emits spores to search for more nutrient friendly hunting grounds. The rules governing cellular behavior have changed.

Which rules apply, and when the rules apply is contingent. Complexity affords different kinds of contingency that must be understood to accommodate both the knowledge we have of complex systems and the practices scientists engage in to acquire this knowledge.

3. CONTINGENCY

It is not particularly useful to say *that* laws are contingent or that they can be re-written as *ceteris paribus* generalizations, without detailing *what* kinds of conditions they depend upon and *how* that dependency works. Only by further articulating the differences rather than covering them over with a phrase denoting the existence of restrictions, can the nature of complex systems be taken seriously. The problem of laws in the special sciences is not just a feature of our epistemological failings; it is a function of the nature of complexity displayed by the objects studied by the special sciences. Providing a more adequate understanding of laws in the special sciences requires a better taxonomy of contingency so that we can articulate the several ways in which laws are *not* 'universal and exceptionless'. In what follows I will detail a taxonomy of different sorts of contingencies that can play a role in the operation of a causal mechanism. Knowing when and how causal processes depend on features of what we relegate to the context of their operation is central to using our understanding of casual dependence to explain, predict, and intervene. I have grouped the types of contingency by consideration of logic, spatio/temporal range, evolution and complexity.

3.1. *Logical Contingency*

I have pointed out elsewhere (Mitchell 2000) the obvious point that there is a clear sense in which all truths about our world that are not logical or mathematical truths are contingent on the way our universe arose and evolved. Thus there is one type of contingency, i.e., logical contingency that applies to all scientific claims. The causal structure of our world is not logically necessary. This is as true for physical structures that might have been fixed in the first three minutes after the big bang, atomic structures that appeared as the elements were created in the evolution of stars, and the dependencies found in complex, evolved biological structures whose rules changed from self-replicating molecules with the subsequent evolution of single celled existence, multi-cellularity, sexual reproduction and social groups. All causal dependencies are contingent on some set of conditions that occur in this world, not in all possible worlds.

3.2. *Space/Time Regional Contingency (No Sexually Reproducing Organisms, No Mendelian Law)*

There is another sense of contingency that is attributed to biological laws to distinguish them from the laws of physics which refers to restrictions in spatial and temporal distribution (see also Waters 1998). Mendel's law of segregation of gametes, for example, did not apply until the evolution of sexually reproducing organisms some 2.5 to 3.5 billion years ago (Bernstein et al. 1981). The earth is 1 or 2 billion years older, the universe itself 10 billion years older. The conditions upon which causal structures depend are not equally well distributed in space and time. Biological causal structures are certainly more recent than some physical structures, and may be more ephemeral.

3.3. *Evolutionary Contingencies: Weak Contingency*

Even after the initial evolution of a structure and the associated causal dependencies that govern it, there may be changes in future environmental conditions that will break down those structures. With their demise the causal dependencies describing them will no longer apply. This is what Beatty dubs 'weak evolutionary contingency', i.e., that the conditions upon which the causal dependencies describing biological systems may change over time. They come and go. Thus there are types of historical contingency, or restrictions on the domain of applicability that attaches more often to biological regularities because life arose much later than other material forms, and may not hold on to its causal structures for as long. But universality never meant that the causal structure described in a law would

occur at every point in space/time, rather that whenever and wherever the conditions for a relationship between the properties did occur, it would occur according to the relation described by the law.

3.4. *Strong Contingency (Multiple Outcomes Contingency)*

Beatty identifies a stronger sense of evolutionary contingency with "the fact that evolution can lead to different outcomes from the same starting point, even when the same selection pressures are operating" (Beatty 1995, p. 57). Here the focus is shifted from how likely or widespread are the conditions under which a causal relation will be operant to the uniqueness of the causal relationship or structure being evoked by those very conditions.

There are two ways in which strong contingency could occur. The first is when systems are indeterministic. If quantum processes have effects on macroscopic phenomena, then there can be cases where all the initial conditions are the same and the outcomes are nevertheless variant. Given mutations may be generated by radiation effects, this could well be symptomatic of biological systems. The second is in deterministic systems that are chaotic. Thus there may be the 'same' initial conditions, that is the same in so far as we can determine them to be the same, that nevertheless give rise to widely divergent outcomes. Complex biological systems are paradigm cases of chaos.

For deterministic cases, the *ceteris paribus* strategy for forcing this type of irregularity into the form of a law has been invoked (see Sober 1997). The reasoning is that if systems are deterministic there *must* be specific conditions, even if we can never know what they are or determine if they have occurred, that will distinguish the causal structures of the divergent outcomes. Sober argued that those conditions that caused the selection of a given structure and its accompanying regularities, e.g., the variations and fitness differences for the evolution of sexual reproduction and gametic segregation, could then form the antecedent of a strict law.

Beatty's own response to this type of maneuver is to suggest that there is no way to enumerate the conditions in the *ceteris paribus* antecedent clause to transform the biological generalizations that describe the causal structures of evolved biological systems. Earman, Roberts and Smith (this volume) remind us that this is only an epistemological worry. It may be that we will be able to articulate the conditions that completely specify the numerous conditions upon which some evolved structure depended and depends and hence be able to articulate the strict law governing that domain. If it is just a matter of knowing or not knowing all the conditions, then the very existence of *ceteris paribus* laws that cover evolutionary contingency is not called into question.

While it is clear how this strategy would work for weak contingency, how would it apply to strongly evolutionarily contingent regularities? Here, even when the causal conditions are operant, different, functionally equivalent outcomes are generated. If the relationship between the antecedent conditions and the evolved structure is probabilistic, then one could invoke the *ceteris paribus* strategy and allow for strict probabilistic laws. If the multiple outcomes are the result of a deterministic, but chaotic dynamics, then one could still claim that although we cannot discover the strict deterministic laws that direct the system into its separate outcome states, they nevertheless exist. Once again evolutionary contingent claims can be embedded via the specification of the contingent antecedent conditions in a strict law.

The problem I see with invoking the *ceteris paribus* response to Beatty's evolutionary contingency thesis is that it collapses the different ways in which a causal regularity can fail to be strict. By so doing it obscures, rather than illuminates the nature of biological knowledge.

4. COMPLEXITY AND CONTINGENCY

So far I have discussed the historical dimensions of chance and change characteristic of the evolved complexity of the domain of biological objects that make lawful behavior of the universal and exceptionless variety hard to come by. In addition there are contingencies that confound the strict lawfulness of currently existing complex biological systems. These are the result of the multi-level compositional structure of complex systems and the multi-component interactions at each level of those systems – complexity of both composition and of process.

4.1. *Multi-Level Interactions*

The operation of some causal mechanisms is contingent on the constraints imposed by their location within a multi-level system. For example, one could detail the causal relations describing the ovarian development in female bees, something that may well be a conserved trait from solitary ancestors to social descendents. The developmental laws that describe this process depend upon both the appropriate genes and internal (to the individual bee) conditions for the triggering of the expression of those genes at certain stages in the process of cellular specialization. However, when individuals come to live in social groups, the context in which these internal processes have to operate may change. When a female honeybee develops in a colony in which there is a queen, then the worker's ovarian develop-

ment is suppressed by means of pheromonal control from the queen. If the queen should be killed, and the colony left without a queen, then the workers immediately begin to develop ovaries and produce haploid eggs. Thus the conditions upon which a causal mechanism operates depends upon the organizational structure in which it is embedded. This is not an incident peculiar to social groups.

The story of ovarian suppression in social insects is similar to the change of rules that occurs when the single celled stage of the slime mold ends with the aggregation to form the multi-cellular flowering stage I discussed above. Indeed, Buss (1987) has argued that the very origin of multi-cellular individuals from single-celled ancestors is one that involves the suppression of competition among the components (cells, or worker bees) of the new individual (multi-cellular individual, or colony) which permits the new individual to become the stable locus of evolutionary change. Thus the composition rules that characterize the various ways in which complex biological objects are formed will also affect the nature of the contingency in which the component mechanisms will operate.

4.2. *Multi-Component Causal Structures within a Level*

In addition to the type of contingencies arising with compositional hierarchies, there are also contingencies that affect specific causal mechanisms occurring within a level of organization. These are the result of the fact that most behaviors of complex biological systems are the result of the interaction of multiple mechanisms or causal factors. That there is more than one force acting to produce an outcome does not, by itself, threaten the existence, empirical accessibility, or usefulness of strict laws describing the individual components. Rather the problem arises with the nature of the interaction of these components. Positive and negative feedback loops, amplifying and damping interactions of a non-linear type are characteristic of complex biological systems.

For example, current research indicates that the behavior of individual foragers in a honeybee colony vary with genetic differences. This has been described in terms of the threshold levels for the stimulus required to initiate foraging behavior of individual bees. Experiments have supported the view that genetic variation accounts for behavioral variation via the genetic components determining individual threshold levels. However, it is now known that learning from the environment can also affect the behavior of foragers by moderating their threshold level. Indeed, the contribution of each of these very different mechanisms can amplify the expected probability of foraging behavior. Thus there may not be a 'regular' manner in which the contributions of different components generate a

resultant outcome. Under some values of the components, it may be that the contribution of genetic variation is much stronger than variant learning experiences and hence completely determines the pattern of foragers in a colony or between colonies. Other times, the reverse might be true, and still other times, the interaction of the two components is operative in generating the pattern. Interactions may take different forms, including additive, swamping, damping and amplifying. Thus the operation of a single causal component, its contribution to the resultant effect, can be contingent not just on background standing conditions, but also on the other causal mechanisms operating at the same time. The nature of the contingency may vary with the different values that the variables in the component mechanisms take.

Redundancy and phase change phenomena can also be present in complex biological systems (see discussion above). These offer two more types of complex contingencies that have import for understanding the range of contexts in which the regular behavior of a set of variables may be disrupted. In systems with redundant processes, the contribution of any one may be elicited or moderated by the operation of another. For phase change, like non-linear dynamical processes, the nature of the function describing the behavior of variables may itself change under certain values of the variables or changes in external conditions.

5. THE PHILOSOPHICAL CONSEQUENCES

I have presented a variety of different ways in which causal dependencies in biology are contingent. This is damning of biology only if one retains the strict notion that laws must be universal and exceptionless. Instead, we can turn the question around and ask not whether biological claims can be transformed into strict laws, but rather when and how do biological claims perform the functions that laws are thought to serve? That is, how can less than strict laws explain, predict and assist in intervention? Recent work on causal dependence has done much to develop answers.

In his 2001 and his paper in this issue, Woodward applies his notion of explanation being grounded not by universal, exceptionless laws, but by generalizations that are invariant under intervention to cases in biology. He argues that his notion of invariance is both distinct from my idea of stability of conditions upon which relations hold and that invariance is what is needed for explanation, not stability. I want to explore Woodward's argument, to see where and why the disagreement occurs. First of all, there are many similarities in Woodward's and my account of laws. We both reject universality and exceptionlessness as necessary for knowledge claims to be

deemed laws. We both want laws to have properties that come in degrees, rather than dichotomous values. What Woodward identifies as the relevant continuum is that of invariance (2000, p. 199) "... unlike lawfulness, invariance comes in gradations or degrees". For him, generalizations come with different degrees of invariance. It seems a bit odd to say invariance comes in degrees – since it seems to be the case that either the relationship between two variables is invariant or not, and how much it varies may be tracked, how much it fails to vary doesn't seem to make linguistic sense. However, what Woodward means does: he means that the relation between two variables is domain insensitive, such that if you change the value of the independent variable X, the dependent variable, Y, will stand in the same functional relationship say $Y = -kX + a$ for a range of changing values of X. There are however some values that X can take for which the function no longer is true. So the function is invariant under some but not all changes in the value of X. How many changes, how large the domain of invariance, of different functions will differ – and this is where I believe the degrees come in. Some functional relationships hold universally, some hold nearly universally – say except near a black hole – some hold for the majority of the time, and some hold some of the time. The value of X thus explains the value of Y by means of the functional relationship that describes the causal dependence of Y on X, in just those regions of the domain where the relationship is invariant. And for Woodward, having some domain of invariance is sufficient for explanation, even if it isn't universal, since some counterfactual situations – namely those changes in X where the function remains invariant – are supported.

I also attempt to provide a means of describing varying domains of applicability of different scientific laws (Mitchell 2000). To this end, I have identified a number of different continua that generalizations can be located within – in particular ontological ones of stability and of strength and representational ones of abstraction and cognitive accessibility. Ontological differences obtain between Fourier's law of thermal expansion, for example, and Mendel's law of segregation but it is not that one functions as a law and the other does not, or that one is necessary and the other is contingent. Rather one difference is in the stability of the conditions upon which the relations are contingent. Once the distribution of matter in the primordial atom was fixed, presumably shortly after the big bang, the function described by Fourier's law would hold. It would not have applied, if that distribution had been different, and indeed, will not apply should the universe enter a state of heat death. The conditions that both gave rise to the evolved structure of sexual reproduction and meiotic process of gamete

production are less stable. The strength of deterministic, a probabilistic and a chaotic causal relations also vary.

There are methodological consequences to these variations in stability and strength. There is a difference in the kind of information required in order to *use* the different claims. It would be great if we could always detach the relation discovered from its evidential context, and be assured it will apply to all regions of space/time and in all contexts. But we cannot. Causal structures are contingent and, as I have argued above, they are contingent in a number of different ways. In order to apply less than ideally universal laws, one must carry the evidence from the discovery and confirmation contexts along to the new situations. As the conditions required become less stable, more information is required for application. Thus the difference between the laws of physics, the laws of biology, and the so-called accidental generalizations is better rendered as degrees of stability of conditions upon which the relations described depend, and the practical upshot is a corresponding difference in the way in which evidence for their acceptance must be treated in their further application.

Woodward compares his idea of invariance with my idea of stability and finds mine wanting for the job of explanation. Stability for me is a measure of the range of conditions that are required for the relationship described by the law to hold, which I take to include the domain of Woodward's invariance. However, stability can be a feature of relationships that are not invariant under ideal intervention. "Mere stability under some or even many changes is not sufficient for explanatoriness" (Woodward 2001). His counterexample is the case of common cause. According to Woodward, while the relationship between the two effects of a common cause is stable in any situation in which the common cause is operative, the one effect does not explain the other effect. Woodward's notion of invariance is supposed to capture this distinction, since some ideal interventions on the common cause system will show that a change in the value of the first effect will not be correlated with a change in the value of the second effect. The relationship breaks down, and so is not explanatory. If the world were such that those types of interventions never occurred naturally nor could be produced experimentally, on my view stability would be maintained, but invariance would still be transgressed, since there could be ideal situations in which it broke down. So, on his view, we would not have a 'law', on mine, we would.

The empiricist in me finds it difficult to detect the cash value of the difference Woodward is drawing between invariance and stability. If we could produce or witness the breaking of the relationship between the effects of a common cause, then we would find that the law describing the

relationship between cause and either effect to be more useful than a law describing the relationship between effect and effect. Namely, the former would work in cases where the latter did not. But if there never were such cases to be found, then wouldn't they work equally well for prediction and intervention? If, on the other hand one requires a more substantial metaphysical warrant for explanation, as I believe Woodward does, then this constant conjunction or stable correlation would fail to explain why what happens, happens. The trouble is, I do not see how in practice you can distinguish the positions. If you have evidence for a common causal structure then on both accounts, the cause is a better predictor, and better at explaining the effects than either effect is of the other. If you have no evidence for a common causal structure, then the correlation, with the right sort of supporting evidence (like temporal order) would be taken to be explanatory.

I think the disagreement lies in the functions of laws on which we individually focus. Woodward admittedly attempts to account for only the explanatory function of laws. To perform this function, a generalization must report a counterfactual dependence. It has to describe a causal relationship that will remain true under certain episodes of "other things being different". It need not be true under *all* such episodes, i.e., exceptionless and universal, but to explain a particular occurrence, i.e., to say why a variable Y has the value it has by appeal to the value of a variable X, it must be the case that one could track x, y pairs in other circumstances, in particular, in interventions, *in the domain where the law was invariant*, and find the same relationship one finds in the explanandum situation. This is for Woodward what it means to say that Y is causally dependent on X and hence an occurrence or value of it is explained by appeal to X by means of the invariant generalization connecting X and Y. That is, Woodward lets domain restricted generalizations count as explanatory in just those domains where the relationship described in the generalization holds. Stability does just the same work, however it is weaker and includes what might turn out to be correlations due to a non-direct causal relationship. But for there to be a distinction between stability and invariance, then we would have to already know the causal structure producing the correlation.

Because the sciences I worry about embrace complexity, my goal has been to see how complexity affects the way we do science. Now if the world were hopelessly complex, or the dynamical evolution so rapid that we found ourselves in a Heraclitean Universe, we wouldn't be able to capture any knowledge that could be used downstream. But the history of science doesn't make this a plausible view – we can't be that deluded for that long that we actually can manipulate and predict events in the

world. But it is equally misguided to take as an assumption that the world is simple, and expect to find that simplicity at every turn, and blame the investigator or impugn the science when simple laws are not to be found. We can learn about the features of a complex world, it is just not easy, and no single algorithm is likely to work in all the contexts in which complexity is found.

So where does that leave us with respect to the implications of complexity and contingency for our epistemological practices? First of all, descriptions of causal dependencies to be useful for prediction, explanation and intervention need not be universally true and exceptionless. As long as we can detail the domains in which the dependency is stable or invariant, then we can explain why what happens in that domain happens, and what will happen when changes in the magnitude of the causal parameters changes. However, there are different ways in which domains are restricted, or universality is lost including temporal and spatial restrictions that are the result of the evolutionary process; contextual restrictions in which certain parameter values or background conditions change the functions that describe the causal dependency; and contextual restrictions in which the operation of other causal mechanisms can interact in ways in which the effects of a cause are amplified, damped, made redundant or evoked. Representing all that variety of contingency by means of an unspecified *ceteris paribus* clause will mask the different strategies required to elicit information about complex contingencies in nature. In short, the context sensitivity of complex dynamical systems, like those studied by biology, entails a shift in our expectations. We should not be looking for single, simple causes. We should not be looking exclusively for universal causal relationships. And we must record and use not only the casual dependencies detected in a particular system or population to understand other systems and populations, but also the features that define the contexts present in the system under study. Without that information, exporting domain specific, exception ridden general truths cannot be done.

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