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Introduction: Biological Constraints on Learning

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This introduction presents a special issue on biological constraints on learning. The special issue includes a number of papers about various conceptual, methodological, empirical, and theoretical aspects of this important topic. The papers are summarized here after a brief historical and conceptual outline. The introduction ends with a discussion of some conceptual aspects of the topic. This special issue stemmed from a symposium on biological constraints on learning that was organized for the XVII Biennial Meeting of the International Society for Comparative Psychology in Bogotá, Colombia, September 2014. The symposium sought to revisit the topic of biological constraints on learning. Such is the aim of this special issue as well, guided by the conviction that the topic demands further study. Some of the most important researchers in the area were invited to contribute. Their acceptance indicates the topic's enduring importance.

Brief Historical and Conceptual Outline

Historically, the notion of biological constraints on learning arose from Darwin's (1859) thoughts on the distinction between *instincts* (behaviors that required no experience) and *habits* (behaviors that required experience), in terms of his theory of evolution by natural selection. Several discussions ensued between biologists and psychologists about how instincts and habits related to one another. Peaking during the 1950s and 1960s, these discussions gave conceptual focus to much of the empirical work in both disciplines. On the side of psychology, such focus paved the way to the notion of biological constraints against a view that made it difficult to smoothly integrate learning with Darwin's theory.

Biological constraints were widely discussed during the 1970s in relation to experimental research on associative learning in Pavlovian and instrumental conditioning (e.g., see Bitterman, 1975; Hinde & Stevenson-Hinde, 1973; Rozin & Kalat, 1971; Seligman & Hager, 1972; Shettleworth, 1972)[1]. Not all these authors saw eye to eye on the topic, but they agreed on the need to reexamine a fundamental assumption of that research that was challenged by evidence.

[1]Some (e.g., Kamil & Yoerg, 1982; Timberlake, 1990) have questioned the expression *biological constraints* as narrow, equivocal, or non-explanatory. But we continue to use it here, as it has become standard in the literature and a better label remains elusive. It should be recognized that it embraces not only limitations, but also predispositions and exceptionalities.

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The assumption in question stated that learning followed laws (or theories, principles, processes, explanations, or mechanisms) that were general across all stimuli, responses, reinforcers, and species. That is to say, the laws of learning were assumed to be independent of specific stimuli, responses, and reinforcers. There also was this notion that species differences in learning were theoretically less relevant than the similarities. The assumption can thus be called the *generality assumption*. It was challenged by evidence of certain systematic learning differences across stimuli, responses, reinforcers, and species. The evidence included phenomena such as species-specific learning (e.g., misbehavior, innate defense reactions, autoshaped keypecking in pigeons), cue-to-consequence effects (more ambiguously, *selective associations*), and specialized learning (e.g., birdsong learning, imprinting).

These phenomena suggested that the generality assumption needed reassessment. The reassessment, it was proposed, should seek to explain learning more biologically. *Biological* here refers not only to present animals' anatomy and physiology (e.g., brain structure and functioning). Perhaps more importantly, *biological* also refers to the evolutionary history and adaptive value of learning (Domjan, 1983; Hollis, 1982). Thus, such phenomena were to be reinterpreted as reflecting evolutionary, adaptive specificities, rather than laws that cut across stimuli, responses, reinforcers, and species.

Learned behavior was thus viewed as a phenotypic trait that was adaptive (i.e., conferred current organisms a reproductive advantage) and/or an adaptation (i.e., present in current organisms as products of natural selection)[2]. The idea, then, was that evolutionary function influenced (by hindering or enhancing) the effectiveness of procedures that were used to study learning. This idea came to be known as *biological constraints on learning*. The idea can thus be viewed more precisely as an *adaptationist explanatory hypothesis*, more than a description of phenomena (cf. Timberlake, 1990, p. 52).

Precision aside, the idea brought about a strong evolutionary thinking into the psychology of learning, promoting a link to comparative psychology. A fertile crossover between the two fields thus ensued. To be sure, there were important antecedents in, for example, Baldwin (1896) and Morgan's (1896) speculations over the learning-evolution link, Lubbock's (1898) studies on way-finding learning in hymenoptera, and Thorndike's (1898) studies of *belongingness* in instrumental learning. However, the experimental research tradition on learning that prompted the discussion over biological constraints was still decades away. A tradition was well in place when Bitterman (1960) propounded the need for collaboration, but it was before biological constraints were a discussion topic (cf. Bitterman, 1975), although he anticipated its spirit.

[2]Logically, a trait can be both, adaptive and an adaptation. Thus, the expression, *adaptive adaptation*, is not redundant. However, a trait can be adaptive without being an adaptation and vice versa. The distinction can help make those phenomena less puzzling, by allowing them to be interpreted as adaptations that become non-adaptive, due to changes in the selective factors form those in force when learning naturally evolved.

These antecedents suggested the potential, perhaps even necessity, of collaboration between comparative and learning psychology. Still, a greater sense of urgency for collaboration arose when biological constraints became an official discussion topic in the psychology of learning. It was then when the two fields really hit it off, as it were, and their partnership remains strong today, according to a recent bibliometric study (Blaser & Bellizi, 2014).

This partnership, however, is not a union but an intersection of the two fields. Many learning psychologists do not study evolution and adaptive value as part of their everyday research. Nor do many comparative psychologists actively investigate learning. This situation is not necessarily bad if motivated by a division of labor and personal interest. After all, no one can do or like everything, and science is no exception to this. It thus is quite legitimate and common in science to focus on something and ignore everything else.

Actually, such a focus can be motivated by deeper, methodological reasons, above and beyond a division of labor or personal interest. Studying something *X* in isolation of something else *Y* often means counterfactually thinking how *X would be* or *have been if Y* did not exist (or exerted negligible influence). Such isolation can also seek a purer understanding of *X* by controlling or avoiding *Y* as an external, temporarily unwanted influence. In either form, this sort of counterfactual, what-if thinking is a very common way to scientifically understand why reality is the way it is.

As an example close to home, the Hardy-Weinberg law in population genetics describes expected gene frequencies (after an infinite number of generations) given initial allele frequencies, as if there were no other evolutionary influences like sexual reproduction, mutation, and even natural selection. In doing this, the population geneticist is not saying that those other influences are unimportant, let alone inexistent. Of course they exist and are important, but may (in fact must) ignored temporarily to assess their importance in the dependence of gene frequencies on allele frequencies, or seek a purer understanding of such dependence.

The same goes for learning and evolution. By studying how learning would be without evolution, we might better appreciate why evolution is so important in, or achieve a purer understanding of, learning. In doing this, no one is saying that evolution is unimportant, let alone inexistent. Rather, the goal is to understand its importance by excluding it, or seek a purer understanding of learning. Likewise, by studying how evolution would be without learning, we could perhaps better understand why learning is so important in evolution, or achieve a purer (maybe more phylogenetic and less ontogenetic) understanding of evolution.

Troubles begin when scientists take what they have chosen to study (be it for division of labor, personal interest, or what-if thinking) as being more real, valid, or *essential* (whatever that means) than what they have chosen to ignore. In particular, things go awry when adaptive value and evolutionary history are thought to be inessential to learning. Maybe this is the root of the generality assumption about learning: What began as a focus on studying learning sans biological factors was

elevated to the status of a law of nature. This move could only result in perplexity about phenomena where adaptive value and evolutionary history are important.

In any case, initial discussions about biological constraints on learning were only the beginning and far from reaching a proper resolution to the issues raised. The overarching issue of what biological constraints mean for the generality of a science of learning remains unresolved. Such resistance, we should think, is due to a multifaceted, intricate character that does not admit a simple, quick treatment. As is often the case in worthy issues, this character results from the intersection of various conceptual, empirical, methodological, and theoretical dimensions that are difficult to unravel. They are all intertwined, feeding onto one another in complex, hidden, unintended, often surprising ways. The papers in this special section of the journal vividly exemplify such intricacy.

The Papers

In the first paper, Michael Domjan (2015a) gives an illuminating historical and personal account on some behind-the-scenes intricacies of John Garcia's seminal research on selective associations and conditioned taste aversions, one of the phenomena that prompted the hypothesis of biological constraints on learning. Philosopher George Santayana has been attributed the oft-paraphrased saying "Those who cannot remember the past are condemned to repeat it." But—if the past was good, perhaps we are blessed to repeat it. Domjan tells a story that has much good deserving of repetition. Of course, he also tells the inevitable cautionary tale that is wise to remember, on the pain of repeating pain. Specifically, he points out methodological traps awaiting those who unselectively infer the associative character of selective associations. These problems imply that selective associations may well have a substantial non-associative aspect to them. He also recommends ways in which such problems can be addressed.

In the second paper, Karen Hollis and Lauren Guillette (2015) discuss two views of the conditions for the evolution of learning. On the received view, environmental predictability is the primary condition for learning to have evolved. The newer, alternative view that has emerged from the behavioral and neural science of insects is that the ability to learn is an emergent property of all nervous systems. On this view, nervous systems, especially their plasticity, endow animals with a learning potential that is fulfilled under the right environmental conditions. This potential means that models of the evolution of learning should perhaps use *not-learning*, rather than *learning*, as the assumed default. Thus, instead of viewing learning as *having been selected for*, learning could be viewed as *having not been selected against*. When learning is selected against, hard-wired responding evolves. Therefore, learning does not evolve. Rather, what evolves is hard-wired responding.

In the third paper, Mark Krause (2015) examines two seemingly conflicting views on the evolution of adaptive memory in humans. One comes from human evolutionary psychology and assumes that current human brains consist of specialized modules that were (and presumably still are) selected for. Such modules, the assumption continues,

underwrite domain- and species-specific behavioral and cognitive processes that are adaptive adaptations (see Footnote 2). An alternative view, coming from a comparative perspective, is that learning and memory processes show quantitative, rather than domain specific qualitative variation, among species. Using the putative domain specific phenomenon of adaptive episodic memory in humans, Krause offers the alternative view that memory for survival relevant, episodic information is phylogenetically widespread. Thus, claims for domain specific memory processes in humans (or any species) cannot be made in the absence of comparative viewpoints.

In the fourth paper, Chana Akins and Brian Cusato (2015) discuss an application of a behavior systems approach to sexual conditioning in the male domesticated Japanese quail. On this approach, conditioning--Pavlovian and instrumental--is multiply caused. That is, conditioning results from complex, functional, hierarchical systems of causal interactions among various types of environmental events and responses, rather than a single, one-directional causal relation (e.g., conditioning as a result of just some kind of contingency). Much in those systems evolved as basic innate survival adaptations. Associative learning, as experience-dependent modifications of such systems, evolved to allow them to be more finely tuned to the particular ecological demands of evolutionary niches. Learning thus became as species-specific as the basic survival systems it fine-tunes. Akins and Cusato show how these ideas can be profitably applied to the male sexual behavior of Japanese quail and draws out future implications.

In the fifth paper, Dorothy Kwok and Robert Boakes (2015) discuss the issue of how conditioned taste aversion is possible despite the atypically long delay between taste and illness. This is one of the most perplexing features of this phenomenon, vis-á-vis the assumption that temporal contiguity between conditioned and unconditioned stimuli is necessary for Pavlovian conditioning. Long-delay taste aversion learning is often viewed as a qualitatively different type of associative learning. The authors summarize evidence to support an explanation that makes such feature less perplexing. The working hypothesis is that long-delay conditioning in conditioned taste aversion is possible because of a minor influence of serial overshadowing, compared to other preparations. The authors thus make assumptions to modify learning-deficit models of Pavlovian conditioning so that they can account for conditioned taste aversion (e.g., sickness occurs in bouts that emulate the discrete presence of an unconditioned stimulus).

In the sixth paper, Stanley Weiss and Leigh Panlilio (2015) review research on selective associations, differential learning with different cue-reinforcer pairs. The confound is between the type of reinforcer (food vs. shock) and the relative hedonic state (positive vs. negative, respectively) that becomes conditioned to a tone-light (TL) compound in traditional selective associations studies. Consequently, these studies did not allow for an unequivocal explanation of the conditions for producing this phenomenon in traditional paradigms. The authors present impressive evidence that with the same shock-avoidance contingency operating on a TL compound, when its context made TL relatively positive, L control resulted. When its context made TL relatively negative, T control resulted. On this basis, the authors propose a comparator hedonic model of selective associations according to which animals assess the various

environmental situations in their *world*. This model also links selective associations to other phenomena such as choice behavior and behavioral contrast.

In the second-to-last paper, I, (Burgos, 2015) present an account of misbehavior with an existing neural network model. The model makes no distinction between Pavlovian and instrumental learning (synaptic-change) mechanisms, while it preserves the usual distinctions between types of responding (emitted vs. elicited) and contingency (response-dependent vs. response independent). The model also takes into account the modulatory roles that dopaminergic and hippocampal systems have been observed to play in synaptic changes in both types of conditioning. A neural network used for the simulation included an inhibitory connection from a Pavlovian to an instrumental output unit. This network simulated the sort of reinforcer loss observed in misbehavior as a result of interference of operant by Pavlovian conditioned responding. Another network had no such connection and, hence, showed no interference. The model also predicts that misbehavior depends on feeder training and context.

In the last paper, Bruce Overmier and Julia Meyers-Manor (2015) question the substantiveness of some claims of selective associations, pointing out the lack of appropriate experimental controls in the designs typically used to study this phenomenon. The authors raise a number of methodological and inferential caveats that could cast doubts on the evolutionary import, perhaps even very reality, of selective associations. The caveats are not new, but tend to be overlooked. They can thus be taken as friendly reminders. They include the possibility of changes in an animal's attention and motivations, inadequate behavioral measurements, and effects of non-associative factors, and can interact in complex ways. Not just critics, the authors recommend valuable ways to tend to these caveats.

The papers attest to the rich tapestry that the topic of biological constraints on learning has become, showing that it still admits further analysis. They certainly do not exhaust it, let alone provide all the answers. Quite the contrary, they raise more questions, but this is the way of science. The papers thus leave many doors wide open for future research, giving a sense of never-ending adventure. Like previous efforts, then, they should be viewed only as steps towards, we hope, a better understanding of biological constraints on learning. They might suggest novel ways to address old issues, perhaps even raise new issues. To this extent, they will keep the tapestry growing ever richer, until the next reworking looms. To clarify the threads for that time, here are some thoughts on three conceptual issues that biological constraints raise.

Some Conceptual Issues

Just in case there was any doubt about the complexity of the topic, and given that the emphasis of the papers is on empirical, methodological, and theoretical issues, we finish by gathering three interrelated conceptual strands that are at loose ends: what is learning, what is a cause, and what is an explanation. The last two have been widely discussed in the philosophy of science. The following reflections will draw from such

discussions and apply them to the topic of biological constraints on learning, but in a preliminary, suggestive way, leaving most details out.

The general point will be that different concepts of learning, cause, and explanation might lead to different kinds and characterizations of biological constraints. There is little point in debating over different concepts. The position adopted here in this respect is *conceptual pluralism*, that having different concepts of something is fine, perhaps even inevitable and beneficial.

What is Learning?

The general issue of the role of concepts and definitions in empirical science has been discussed in the philosophy of science (e.g., Hempel, 1952). Also, conceptual questions of the form "What is *x*?" are quite common in science: What is matter? What is time? What is space? What is life? What is mind? Although they can be quite vexing, attempts to answer them can also be illuminating (e.g., Einstein's special theory of relativity was inspired by the question of what is simultaneity). Learning psychology need not be the exception to this. Of course, there are different concepts of learning, but then again, the idea is not to debate over but analyze them to see where they lead.

The question of what is learning, obviously, is not exclusive to the topic of biological constraints. However, the question brings some themes that seem specific to this topic. To mention just one, a reassessment of the generality of learning need not be restricted to laws and explanations, but can also include the very concept of learning. Just as a generalist view begins with a generalist concept, perhaps a biological view should also begin with a more biological concept. No concept of learning thus far has been defined explicitly in biological (i.e., evolutionary, adaptive terms. A more biological concept could help view learning more biologically.

Take Kimble's (1961) influential performance concept: "learning is a relatively permanent change in behavioral potentiality which occurs as a result of reinforced practice" (p. 6). A more biological concept would be the following (replacing the problematic *behavioral potentiality* with the less problematic *performance*): "Learning is a relatively permanent and *biologically constrained* change in performance that results from certain relations between stimuli and responses."

Other concepts emphasize mechanisms. For example, Domjan (2015b) defines learning thus: "Learning is an enduring change in the mechanisms of behavior involving specific stimuli and/or responses that results from prior experience with those or similar stimuli and responses" (p. 14). Talk of "*specific* stimuli and/or responses" gets a bit closer to a biological concept. However, stimuli and responses could be specific in ways other than biological (viz., having a particular spatiotemporal location, without any specifically biological connotation).

Here is a more explicitly biological concept that also emphasizes mechanisms of behavior: "Learning is a somewhat enduring and *biologically constrained* change in the mechanisms of behavior that results from certain relations between stimuli and

responses." Some might argue that biological constraints go without saying. Perhaps, but just as they say "out of sight, out of mind," one could also say "out of concept, out of mind." Excluding biological constraints from our concept of learning is likely to make us forget about biological considerations and fall back to a generalist view. Of course, outside of these definitions, a more detailed and precise characterization of what is a biological constraint would be needed.

The point, above and beyond the plurality of concepts of learning, is that biological constraints might be different things under different concepts of learning. One thing is biological constraints on performance changes, quite another biological constraints on changes in mechanisms underlying performance. Different kinds of biological constraints thus arise that may or may not go hand in hand. Empirical research would be needed to determine whether different kinds of biological constraints obtain and how they relate. A conceptual reflection on what is learning could thus inspire novel empirical research that might improve our understanding of biological constraints on learning.

What is a Cause?

The relevance of this issue here arises from Timberlake's (1990) claim that biological constraints "are not causal entities" (p. 52), whereas his proposed behavior systems are. His claim seems to be that biological constraints cannot be possibly causal entities *in principle*. He does not say exactly why this is a problem (perhaps he believes that only causes explain, that explanations can only be causal; but see later on explanation). Nor does he say what he means by *causal entity*; hence the present issue. Whether biological constraints (or anything else) are causes depends on what is a cause. Perhaps Timberlake believes there is a unique, universally accepted concept of a cause that applies to behavior systems but not biological constraints. However, this belief would be mistaken. Not only are there several current concepts of a cause, but some allow biological constraints to be causes.

One is found in probabilistic theories of causation (see Williamson, 2009). Intuitively, the core thesis of these theories is that causes raise the probabilities of their effects. More formally, let C be a cause of some effect E. On these theories, C is a cause of E just in case the conditional probability of E given C, or $p(E|C)$, is greater than the conditional probability of E given the absence of C ($\sim C$), or $p(E|\sim C)$. Succinctly, C is a cause of E if $p(E|C) > p(E|\sim C)$. There are several variations on this thesis, so probabilistic causation is a family of theories, rather than a single theory. They suffer from some shortcomings (viz., causes that lower probabilities, preemption, non-causes that raise probabilities), but thus far they have not proven insurmountable. Besides, these theories have several advantages that compensate for their shortcomings (the theories allow for imperfect regularities, the asymmetry of causal relations, and the screening-off of non-causes).

Probabilistic causation allows us to view biological constraints as raising the probability of certain behaviors, such as those observed in misbehavior and selective associations. Thus, nothing in probabilistic causation *logically* prevents viewing biological constraints as causes, even if hypothetically (probabilistic causation is a

metaphysical theory, and whether causes are hypothetical is irrelevant to the nature of causation). Whether and how such probability raising actually obtains remains to be empirically determined. But to illustrate its conceptual feasibility, here is a simple hypothetical example.

Take, for instance, raccoons that have been observed to misbehave after operant conditioning (i.e., perform certain responses that delay reinforcement; e.g., rubbing coins; see Breland & Breland, 1961). Despite being seemingly maladaptive, misbehavior could be hypothesized as an ultimate effect of a complex evolutionary *causal chain* where comparable behaviors (and their underlying mechanisms) conferred reproductive advantage to the raccoons' ancestors, under certain ancestral environmental conditions. The seemingly maladaptive character of misbehavior could be due to differences between environments where misbehavior has been observed and ancestral environments where similar behaviors were naturally selected for (see Note 2). Such evolutionary causal chain (or perhaps parts of it) would thus constitute biological constraints on present learning.

In terms of probabilistic causation, the ancestral behaviors and environmental conditions (A) could be hypothesized to raise the probability of occurrence of misbehavior (M) as observed in raccoons. One could thus hypothesize that $p(M|A) > p(M|{\sim}A)$. Again, this hypothesis needs to be empirically tested. The point is that probabilistic causation allows biological constraints to be causes of present behavior, at least conceptually.

Perhaps Timberlake (1990) meant to say that biological constraints cannot conceptually be *proximate* causes. Indeed, if biological constraints are ancestral, they cannot be proximate (relative to present animals). However, they could be *ultimate* or *distal* causes, which are not any less causal. Nothing in probabilistic causation restricts causes to proximate causes. Probabilistic causation thus applies equally well to ultimate causes. In the above hypothesis, then, A can be taken as distal[3].

There are other current accounts of the nature causation, like causes as INUS conditions (Mackie, 1965), counterfactual accounts (e.g., Lewis, 1973), and mark transmission (Salmon, 1977) and conserved quantity accounts (Dowe, 1992) of causal processes. It remains to be seen whether such accounts also conceptually allow biological constraints to be causes, and how. If they do, they might yield different ways in which biological constraints can be causal, insofar as each account provides a different insight into the nature of causation.

What is an Explanation?

[3]This clarification does not mean that distal causes influence present learning directly. Metaphysically, the hypothesis should be interpreted as referring only to *part* of reality. One could hypothesize about other parts, for instance, certain current brain mechanisms B as proximate causes of misbehavior that could *also* raise the probability of M. That is, one can hypothesize that $p(M|B) > p(M|{\sim}B)$, to assert proximate causation of M by B, and $p(B|A) > p(B|{\sim}A)$, to assert ultimate causation of B by A (which hypothesizes that present brain learning mechanisms too are biologically constrained by A). All these causal hypotheses are allowed by probabilistic causation and can be taken as complementary rather than mutually exclusive. It is an empirical matter whether and how they actually obtain and relate.

This issue arises from Damianopoulos' (1989) claim that inferences of biological constraints from evidence that has been obtained through certain experimental designs are not logically justified. The reason, he argues, is that these designs lack the proper controls, for which inferences from them do not satisfy a certain philosophical model of scientific explanation that he sees as being *widely accepted*, namely, the covering-law model (Hempel & Oppenheim, 1948). Only inferences of biological constraints on learning from designs with proper controls are valid in this model. It is not new to assert the invalidity of inferences from designs without proper controls (e.g., Linwick, Patterson, & Overmier, 1981). What is novel in Damianopoulos' (1989) analysis is the attempt to ground this assertion logically on a philosophical model of scientific explanation.

According to this model, the vast majority of scientific explanations can be construed as *deductive arguments* (suitably formalized in first-order predicate logic). The explanandum (that which is to be explained) would be the conclusion, often a statement of some particular observed phenomenon (e.g., "this iron bar expanded"). The explanans (that which is proposed to account for the explanandum) would be the premises. A premise must be either an *empirical* law (i.e., inductive generalization with a high confirmation degree; e.g., "all iron bars expand when heated"), or an empirical initial condition (a particular operation or treatment; "this iron bar was heated"). Nothing else can be part of an explanans in this model.

Damianopoulos' (1989) goal of assessing the validity of inferences of biological constraints from the evidence is laudable. He is also right that such inferences do not satisfy the covering-law model. However, the reason is not that they are made from evidence obtained without proper experimental controls. The reason is more fundamental: Biological constraints are an explanatory hypothesis, not an empirical law, initial condition, or particular observation. Consequently, they cannot be part of any scientific explanation in this model, whether in the explanans or as the explanadum. This outcome follows even if biological constraints are inferred from evidence obtained through proper experimental controls.

Moreover, the phenomena themselves from which biological constraints have been inferred are *exceptions* to certain empirical learning laws (e.g., "Law of Conditioning of Type S" and "Law of Conditioning of Type R"; see Skinner, 1938, pp. 18, 21). Therefore, not even these phenomena can be explained in terms of empirical laws using the covering-law model. The reason reveals a fundamental limitation of this model: Exceptions to an empirical law mean that it is not a *deterministic* law (which admits no exceptions), but *probabilistic* or *statistical* law (which admits exceptions), but the covering-law model was designed to be used with empirical laws that are deterministic, not probabilistic.

Hempel (1962) himself acknowledged this "fundamental difference" (p. 163) between deterministic and probabilistic empirical laws (for which he construed statistical explanations as *inductive* arguments, but this is another matter). Probabilistic laws are widespread in science (statistical thermodynamics, quantum mechanics, population genetics, and neurobiology). Learning and evolution by natural selection too are probabilistic in nature. Hence, deterministic explanations are not as

common in science as the covering-law model would have it. Even if they were, the model is still useless to logically characterize explanations of learning. Ultimately, then, the model's applicability proved too narrow. Consequently, it ceased to be widely accepted by philosophers of science at least two decades before Damianopoulos' (1989) paper[4].

All in all, contrary to his claim, the covering-law model is useless to assess the validity of inferences of biological constraints from the evidence. The model is useless even to logically reconstruct explanations of this evidence in terms of empirical laws of learning if these are probabilistic (which they probably are). Fortunately, alternative models have been proposed to better capture the logic of actual scientific explanatory practices. One that has become popular (but not universally accepted) among philosophers of science, and applies well to biological constraints, is the so-called "inference to the best explanation" proposed by Harman (1965).

According to this model, "our explanatory considerations guide our inferences" (Lipton, 2004, p. 1). That is to say, given some evidence, we tend to infer what, *if true*, *would* provide the best explanation of that evidence[5]. The best explanation (or "loveliest," as Lipton, 2004, liked to put it) among all available explanations is the most parsimonious, elegant, intuitive, and consistent with other evidence and our best available theories. Best explanations are thus highly context- and information-sensitive. Surely easier said than done, but no proponent of this model has said that it should be easy to apply.

Still, some applications are easier than others. Here is a relatively easy one: We explain smoke rising in the distance by inferring that it is due to a nearby fire, even if we do not see the fire. Other explanations are possible (e.g., an elaborate alien hologram), but the presence of a fire seems to be the best one. A more difficult application: We could explain someone wincing and saying "I have a headache" by inferring that this person is in pain. But a seemingly equally good explanation is that the person is feigning to have a headache. If the person is a complete stranger, then it will be difficult to decide which explanation is better, a real headache or feigning. If we have more information, then the decision might be easier. Perhaps we know this person to be a recurrent liar, in which case, the feigning explanation would be better (maybe we are watching a play where the person is an actor). Or perhaps we know this person to suffer from migraine, in which case the explanation of a real headache will be a better explanation. So, what is the best explanation strongly depends on the state of our knowledge at the time of the explanation.

This model applies nicely to biological constraints. They clearly are hypothetical explanatory inferences from certain evidence. They also seem to provide the best available explanation of phenomena like misbehavior and selective associations,

[4]Perhaps it was and still is "widely accepted" by experimental psychologists. If this is the case, so much the worse for them for choosing the wrong model.

[5]The precise logic of inference to the best explanation is still in debate. Some view it as a kind of inductive inference, others as a kind of deductive inference, and still others as a third kind of inference, different from induction and deduction. Consequently, whether an inference to the best explanation is "valid" cannot be unequivocally determined.

provided that they are real[6]. Non-adaptationist explanations, in terms of non-evolutionary influences, are possible (e.g., behavioral and brain features due to random mutations). But they are no better than biological constraints when it comes to consistency with the best theory currently available in biology, the theory of evolution by natural selection.

The risk of adaptationism here is high. But maybe the threat of adaptationism has been exaggerated. If adaptationist explanations like biological constraints are just working hypotheses, they pose no threat. Perhaps, then, biological constraints on learning should be treated as working hypotheses. The issue of whether biological constraints are good explanations of learning thus becomes a case of the issue of whether adaptationist explanations are better than non-adaptationist explanations, at the intersection of comparative and learning psychology.

There are other current models of scientific explanation that are worth examining to assess the explanatory role of biological constraints on learning. For example, Salmon (1971) proposed his statistical relevance model to capture probabilistic explanations (it can also capture deterministic explanations). The core thesis of this model is that explanations are not arguments, but statements of membership in classes that make a conditional-probabilistic difference. This model can be combined with probabilistic causation (see above), to obtain causal explanations that allow for the possibility that biological constraints on learning are probabilistic in nature[7]. In the unificationist model (e.g., Kitcher, 1989), a scientific explanation provides a unified account that allows understanding of a variety of phenomena. This model thus raises the question of the extent to which biological constraints provide a unified explanation that allows us to understand a variety of learning phenomena, not just misbehavior and selective associations, but also blocking, overshadowing, fixed-interval performance, and choice, among others.

Again, we have skipped most details, in the interest of space. Each issue admits much more detailed examination, but it is better left for future investigations. And there might be other conceptual issues (e.g., What is a law? What is a mechanism?). And, of course, much remains to be done empirically, methodologically, and

[6]The issue of whether the phenomena are real is different from the issue of whether inferring biological constraints from the phenomena is "valid" (whatever that means; see Note 5). One thing is to infer, say, selective associations from some evidence, quite another to infer biological constraints to explain selective associations. These are distinct tasks that could but need not go hand in hand. If indeed selective associations turn out to be unreal because of inadequate experimental controls, no explanation is called for, whether in terms of biological constraints or otherwise, as there would be no phenomenon to explain to begin with.

[7]Inference to the best explanation does not restrict explanations to causal explanations. Lipton (2004), for example, has objected to such a restriction by claiming that there still is no "fully adequate analysis of causation" (p. 31). He also claims that there can be non-causal explanations and they can be as explanatory as non-causal explanations (although this claim cannot be properly assessed without an adequate understanding of causation either). As said before, perhaps Timberlake (1990) believes that only causal entities explain (alternatively, that explanations are only causal). Therefore, the alleged non-causal status of biological constraints would be problematic because it would make them irremediably non-explanatory. This argument, however, can be refuted by claiming that explanations need not be causal (cf. Salmon, 1984).

theoretically. In sum, the topic promises to keep alive and well the intersection of comparative with learning psychology for a long time to come.

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