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A general account of selection: Biology, immunology, and behavior

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Abstract: Authors frequently refer to gene-based selection in biological evolution, the reaction of the immune system to antigens, and operant learning as exemplifying selection processes in the same sense of this term. However, as obvious as this claim may seem on the surface, setting out an account of "selection" that is general enough to incorporate all three of these processes without becoming so general as to be vacuous is far from easy. In this target article, we set out such a general account of selection to see how well it accommodates these very different sorts of selection. The three fundamental elements of this account are replication, variation, and environmental interaction. For selection to occur, these three processes must be related in a very specific way. In particular, replication must alternate with environmental interaction so that any changes that occur in replication are passed on differentially because of environmental interaction.

One of the main differences among the three sorts of selection that we investigate concerns the role of organisms. In traditional biological evolution, organisms play a central role with respect to environmental interaction. Although environmental interaction can occur at other levels of the organizational hierarchy, organisms are the primary focus of environmental interaction. In the functioning of the immune system, organisms function as containers. The interactions that result in selection of antibodies during a lifetime are between entities (antibodies and antigens) contained within the organism. Resulting changes in the immune system of one organism are not passed on to later organisms. Nor are changes in operant behavior resulting from behavioral selection passed on to later organisms. But operant behavior is not contained in the organism because most of the interactions that lead to differential replication include parts of the world outside the organism. Changes in the organism's nervous system are the effects of those interactions. The role of genes also varies in these three systems. Biological evolution is gene-based (i.e., genes are the primary replicators). Genes play very different roles in operant behavior and the immune system. However, in all three systems, iteration is central. All three selection processes are also incredibly wasteful and inefficient. They can generate complexity and novelty primarily because they are so wasteful and inefficient.

Keywords: evolution; immunology; interaction; operant behavior; operant learning; replication; selection; variation

1. Introduction

What was so radical about Darwin's theory of evolution? In the following quotation, B. F. Skinner (1974, pp. 40–41; see also *BBS* special issue) dismisses the usual answers given to this question and suggests a nonstandard answer of his own:

> Darwin's theory of natural selection came very late in the history of thought. Was it delayed because it opposed revealed truth, because it was an entirely new subject in the history of science, because it was characteristic only of living things, or because it dealt with purpose and final causes without postulating an act of creation? I think not. Darwin discovered the role of selection, a kind of causality very different from the push-pull mechanisms of science up to that time.

Although we think that the late appearance of selection on the intellectual scene no doubt had numerous causes, we agree with Skinner that part of the answer is surely the counter-intuitive kind of causality exhibited in selection processes. Push-pull causation does seem "natural" to us. So does functional organization. But the action of selection processes does not. This fact about how people in the West think is reflected in natural languages. Finding terms to describe selection processes that do not have all sorts of inappropriate connotations is not easy.

Numerous biologists and philosophers of biology have presented analyses of gene-based selection in biological evolution (e.g., Dawkins 1976; Hull 1980; Lewontin 1970; Lloyd 1988; Sober 1984; Sober & Wilson 1998; Vrba & Gould 1986), but relatively few have tried to present a general account of selection to see which processes in addition to gene-based biological evolution are genuine selection processes and which are not. (The chief exception is Darden & Cain 1989.) Are selection processes sufficiently different from other sorts of causal processes to warrant a separate analysis? The sort of selection that goes on in biological evolution is surely an instance of selection, but how about other putative examples of selection, for example, the reaction of the immune system to antigens, oper-

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ant learning, the development of the central nervous system, and even conceptual change itself (Cziko 1995)?

In this target article we provide a general account of selection. The chief danger of such general analyses is that they can be either too broad or too narrow. If the account is too broad, then everything becomes a selection process – including crystal formation and balls rolling down inclined planes. We have no objection to anyone attempting to present general accounts of more global phenomena, such as the persistence of patterns, but we limit ourselves just to selection processes. The other danger is to make the analysis so narrow that each putative type of selection becomes unique. For example, the genotype/phenotype distinction plays a central role in gene-based selection in biology. Is this role common to all selection processes or unique to selection at the biological level? Being able to distinguish self from nonself is crucial in the immune system. Is the self-nonself distinction also important in other sorts of selection? In operant learning, selection occurs only with respect to sequences of environmental interaction rather than with respect to numerous concurrent alternatives. Is this difference sufficient to disqualify it as a case of selection?

Such questions cannot be answered a priori. We have to try various analyses and see how they turn out. Our goal is to see if selection processes can be construed usefully as a special sort of causal process. The success of such an analysis will be determined by the use that those scientists working on various sorts of selection can make of it. If they find that our analysis helps them to understand the sort of selection they are studying more clearly, then it has succeeded; if not, then it has failed (for a defense of the method of abstraction, see Darden & Cain 1989). Even though causation is absolutely central to our understanding of selection, we do not attempt to present a general analysis of causation in this paper. In the past, some of the disputes that have arisen with respect to selection actually turn on different views of causation (e.g., Brandon 1982; 1990; Brandon et al. 1994; Glymour 1999; Sober 1984; 1992; van der Steen 1996). Ideally, we should include an analysis of causation alongside selection. However, every analysis must stop somewhere. Not all of the substantive terms used in an analysis can be analyzed. We do not present an analysis of causation in this paper because the literature is too vast and the alternatives too various. For better or for worse, in this paper we depend on the reader's largely tacit understanding of this extremely basic notion. The most that we can do in the space of a single paper is to point out when different notions of causation have caused problems, as in the instance cited above.

The three authors of this target article come from three very different backgrounds. David Hull (1980; 1987) emphasizes his work on gene-based selection in biological evolution, treating selection as an alternation between replication and environmental interaction. Sigrid Glenn contributes her work on operant learning as a selection process (Glenn 1991; Glenn & Field 1994; Glenn & Madden 1995). Rod Langman (Langman 1989; Langman & Cohn 1996) adds his extensive theoretical analysis of the immune system. In this paper we strive to pool our conceptual resources to produce a general account of selection adequate for the three sorts of selection under investigation – gene-based selection in biological evolution, the reaction of the immune system to antigens, and operant learning.

We do not offer an analysis of three other possible examples of selection – the development of the central nervous system, social learning, and conceptual change. We do not include an extensive discussion of neuronal development because the empirical facts remain too controversial (Edelman 1987; Quartz & Sejnowski 1997). Once neurophysiologists have worked out the basic structure of neuronal development, we will be in a position to evaluate this process to see if it can legitimately count as a selection process. If it fits our analysis, well and good. If not, then either neuronal development is not a selection process or our analysis is deficient. The second example of a putative selection process that we do not discuss in this paper is social learning, even though social learning is one of the most commonly cited examples of a selection process. Instead we limit ourselves to individual learning as a selection process. The strategy that we have adopted is to deal with the simplest cases first. Once we understand the most unproblematic instances of selection, we can then turn to the more difficult cases. The same justification applies to conceptual change, including conceptual change in science. Is the process that has allowed the three of us to understand selection in biological evolution, immunological reactions, and operant learning a selection process? Is conceptual change a selection process? Although we find the construal of conceptual change as a selection process fascinating, we do not discuss it in this paper (see Laland et al., 2000).

From the start, we have to register one warning: None of us claims to present the standard interpretation of the processes with which we are dealing, mainly because no such standard interpretation exists in any of the three cases that we investigate. For example, numerous objections have been raised to neo-Darwinian versions of evolutionary theory, especially the heavy emphasis that is placed on genes and the cavalier attitude frequently exhibited toward the environment. With a few noteworthy exceptions (e.g., Brandon 1990), the environment is treated as an unarticu-

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lated background against which selection operates. With respect to the immune system, considerable disagreement exists concerning the mechanism that allows the immune system to react selectively against nonself but not self components (e.g., Silverstein & Rose 1997). Numerous versions of learning theory can be found in psychology. Even if one limits oneself just to operant learning, disagreements exist. Can the stimulus that functions with respect to behavior be inside as well as outside the organism?

As much as scientists strive to reduce the amount of disagreement in science, they never come close to succeeding, and if science itself is a selection process, they cannot. In this article we could not examine all versions of all of the theories that we treat. We had to select one from each of the domains. The issue is whether this version and others like it can be properly construed as selection processes, not whether we accept the reader's preferred version. This caveat applies with special force to theories of operant behavior. Some psychologists reject such theories out of hand. Others have strong preferences for one version over all others. In this paper we cannot answer the objections that have been raised to any of the three broad ranges of theories we discuss. Instead, for the purposes of this paper, we accept their overall adequacy and proceed from there to decide whether or not they exemplify a particular sort of process – selection. The point of this paper is not the choice of the one and only correct version of any of the theories that we treat. It is to discover if theories of this type can be construed as exemplifying selection processes.

Yet another problem that we confronted in writing this collaborative article is that the three of us used very different terms to describe what we took to be the same sort of process. From the outset, we had to reduce differences that were mainly terminological – a task that turned out to be much more difficult than we had anticipated and not fully completed even now. One danger was allowing the process of biological evolution to play too large a role in our undertaking. Because selection processes were first worked out in gene-based biological evolution, the temptation is to take it as standard and compare other candidates to it, but such a strategy would be biased. Historical precedence does not guarantee conceptual priority. In this paper we need to investigate each candidate in its own right, rather than taking gene-based selection in biological evolution as the standard by which all other putative examples of selection processes are to be evaluated. Even the use of the phrase "selection in gene-based biological evolution" is misleading. Both the functioning of the immune system and operant behavior are to some extent gene based and biological. However, they also include processes that are not gene based in this narrow sense. But for want of a better name, we retain the phrase "gene-based selection in biology."

2. A brief characterization of selection

Several authors have attempted to characterize selection in as brief a fashion as possible. For example, Campbell (1974) describes selection as a function of blind variation and selective retention, while Plotkin (1994, p. 84) characterizes it as a matter of generation, testing, and regeneration. The trouble with these characterizations is that they are too brief. If one wants to understand selection, a sentence or two, no matter how succinct, will not do. Understanding space and time requires more than looking up these terms in a dictionary or in a physics text. Instead one must learn the relevant physics. Similarly, anyone who wants a deep understanding of selection has to study this phenomenon. Just inspection of a brief characterization of the process will not do. This much being said, we define selection as *repeated cycles of replication, variation, and environmental interaction so structured that environmental interaction causes replication to be differential.* The net effect is the evolution of the lineages produced by this process. Each word in this definition needs careful explication. The message is not to be found in the preceding brief characterization of selection but in the ensuing discussion.

2.1. Variation

Variation is sometimes considered part of the selection process (Darden & Cain 1989), sometimes as a precondition for selection processes (Hull 1980). Either way, variation is absolutely essential for the operation of selection processes. If there is no variation, then there are no alternatives to select among. However, the characterization of the variation that functions in selection processes has been one of the most contentious topics in the literature – and the most frustrating. It seems that no adjective exists in the English language that accurately reflects the sort of variation that occurs in selection processes. Is this variation blind, chance, random, nonprescient, nondirected, nonteleological, unforesighted, or what?

First and foremost, the variations that function in selection processes of all sorts are caused – *totally* caused. No one writing in this literature feels inclined to introduce miracles in their descriptions of variation. The task is to describe the sorts of causes that produce this variation. When advocates of selection say that the variations that are operative in selection are "blind," they cannot possibly be using this term in a *literal* sense, as if some variations can see and others cannot. They must mean it in some metaphorical sense. When they term variations "chance" or "random," they cannot be using these terms as they are defined in mathematics. The requirements specified in these definitions are so rigorous that few, if any, natural phenomena can meet them.

Evolutionary biologists are well aware of the various factors that cause mutations. They are also aware that these mutations frequently depart from anything that might be termed "pure randomness." In fact, in many cases the very biologists who insist that the variations that function in selection processes are random are the ones who discovered these departures from randomness in the first place. For example, mutations that produce melanic forms crop up in certain groups of organisms with a greater frequency than the laws of chance would allow. On certain chromosomes, hot spots exist that exhibit extremely high rates of mutation. For example, whole segments of immunoglobulin genes have bursts of mutation 10^6-fold greater than average (see Dawkins 1996, pp. 80–82 and Pennisi 1998, for additional examples). The other adjectives used to modify variation arise in the context of selection in conceptual change, but no one thinks that people, including scientists, are prescient. People may try to anticipate the future, and we can even predict the future in some cases, but no one is literally prescient.

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Confusion in these matters stems in large part from the legacy of the early days of evolutionary biology, in particular the controversy between the Darwinians and Lamarckians. Critics of Darwinian versions of evolutionary theory tend to term any departures from the simplest forms of inheritance "Lamarckian." To be sure, numerous forms of nonstandard inheritance have been discovered over the years (Crow 1999). The issue is whether or not any of these fascinating forms of inheritance are in any significant sense "Lamarckian." The distinction between Darwinian and Lamarckian inheritance depends on the distinction between genotype and phenotype. According to the inheritance of acquired characteristics, the environment modifies the phenotype of an organism so that it is better adapted to the environmental factors that produced this phenotypic change in the first place – better adapted than those organisms that were not modified in this way. This phenotypic change is then transmitted somehow to the genetic material so that it is passed on in reproduction. Thus, according to this view, species can rapidly adapt to environmental change. In Darwinian evolution inherited variations are random with respect to (i.e., independent of) the effects that they produce, while in Lamarckian evolution they are not.

Both aspects of the preceding discussion need emphasizing. First, in Lamarckian evolution, the phenotypic change that results must make the organism better able to cope with the environmental factor that produced the phenotypic change in the first place. They must be adaptations. Exposing the skin to increased sunlight causes it to darken so that the organism is better able to withstand increased sunlight. Second, in Lamarckian evolution the phenotypic change must be transmitted to the *hereditary material* so that it can be passed on *genetically.* A mother dog giving fleas to her puppies is not an instance of the *inheritance* of acquired characteristics because it is not an instance of inheritance in the sense required by Lamarckian inheritance. Biologists do not have a corner on the term inheritance. Other workers can and do use it in a variety of other senses. Our discussion, however, concerns Lamarckian inheritance as a biological phenomenon (for a sampling of the recent literature on Lamarckian forms of inheritance, see Andersson et al. 1998; Benson 1997; Jablonka & Lamb 1995; Lenski & Mittler 1993; MacPhee & Ambrose 1996; Peck & Eyre-Walker 1998; Rosenberg et al. 1995).

In sum, statements about the sorts of variation that function in selection processes need not include any reference to their being blind, random, or what have you. All of the terms that have been used to modify variation are extremely misleading. Hence, we see no reason to put any adjective before variation in our definition of selection. Our analysis concerns only those instances in which variations occur, without regard to their eventual contributions to fitness in biological evolution or some corresponding circumlocution with respect to the immune system and operant behavior. In this target article we deal with natural selection as it functions in Darwinian evolution today. Darwin himself included Lamarckian forms of inheritance in his theory, but Darwinians today do not. Darwinian evolution is currently limited to Darwinian (or Weismannian) inheritance. If Lamarckian forms of inheritance turn out to exist, we have no doubt that these mechanisms will be promptly incorporated into the Darwinian theory the way that neutral mutations were.

2.2. Replication

Replication is the second important notion in our brief characterization of selection, and it poses as broad a spectrum of problems as does variation. Replication contains two elements; iteration (or repetition or recursion, depending on one's terminological preferences) and information. Early on, Dawkins (1976) published a highly influential general account of selection that emphasized the role of replicators. They are the entities whose structure contains the information that is passed on differentially in selection. The structure of replicators counts as information in the sense that it codes for the character of the individuals (or vehicles) that the replicators produce. The only variations in the structure of replicators that matter are those that modify the relevant vehicles. These vehicles then interact with one or more local environmental conditions. Some of these variants survive to replicate, and the process begins again. That is why Plotkin (1994, p. 84) in his analysis of selection emphasizes generation and *re*generation. However, sequential replication is not enough. Variants must be linked to proliferation so that at any one time numerous alternatives are available for selection. At the very least, the frequency of replicators must change sequentially through time.

The only feature of the analysis of selection-type theories provided by Darden and Cain (1989, p. 110) with which we disagree is the demotion of iteration to an ancillary feature of selection. For them, selection is essentially a one-shot deal that can be, but need not be, repeated. They replace iteration with such evaluative notions as benefitting and suffering: "Several types of effects result from the differential interactions. In the short range, individuals benefit and suffer." Although they realize that such terms as benefit and suffer sound anthropomorphic and value-laden, they have to introduce them because they do not treat iteration as central to selection. A single cycle of replication and environmental interaction would fulfill the requirements of their analysis, just so long as it hurt or helped the relevant individuals.

In our analysis we avoid the use of such problematic notions as *benefit* because of the central role of iteration. If some characteristic is increasing in frequency, then it is very likely (though not necessarily) doing some good. It is better adapted to its environment than other variants. According to our account, Darden and Cain's single-cycle analysis of selection is (at most) a limiting case of our account (see section 4.3 under "big-bang" for further discussion). One reason why we prefer no mention of benefit and harm in our general account of selection is that their elimination from explanations of biological adaptations was one of Darwin's major achievements. We are not inclined to reintroduce such notions at this late date if we can avoid it. Iteration has problems of its own (e.g., how to keep "survival of the fittest" from degenerating into a tautology), but these problems can be handled with only a modicum of care and effort (see Lipton & Thompson 1988).

Replication is inherently a copying process. Successive variations must in some sense be retained and then passed on. In many earlier definitions of selection, all that is required is heritability, not genealogical inheritance. As Thompson (1994, p. 638) observes with respect to gene-based selection in biological evolution, natural selection "does not require genes or even direct descendants; all it re-

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quires is that the presence of a configuration of elements in one generation makes more likely the presence of the same configuration in the next generation." We agree with Thompson as far as genes are concerned but draw the line at descent. In biological evolution, replication is accomplished by molecules of DNA splitting and the missing nucleotides being filled in so that the information contained in the resulting molecules is retained. This is one way for replication to occur, but it is only one way. If splitting and reassembly is considered to be essential to all selection processes, then only gene-based selection in biological evolution and the functioning of the immune system count as selection processes. We think that this restriction is too narrow. A variety of mechanisms exist that can have the same effect as splitting and reassembly.

We have taken the opposite tack with respect to descent. Mechanisms other than modification through descent could serve the function that descent does. However, thus far, descent is the only mechanism that has evolved to produce the correlations necessary for selection. A more general analysis than ours might be couched in terms of retention of pattern or configuration from one generation to the next. However, in the absence of replication, the notion of *generation* becomes extremely problematic. In our analysis, we emphasize the mechanisms that produce evolutionary change, not just correlations. The preceding discussion is just one instance of the problems that arise in conceptual analysis. Is our analysis too broad or too narrow? Others might well make decisions different from ours, decisions that might have considerable merit.

In our analysis, the first component of replication is iteration (or repetition or recursion). The second is information. As Williams (1992, p. 11) points out, structure is necessary for selection, but structure alone is not good enough. Some of this structure must count as information. With respect to gene-based selection in biological evolution, "A gene is not a DNA molecule; it is the transcribable information coded in the molecule." DNA exhibits numerous structural elements. For example, it forms a double helix, and the bonds that connect the two bases that make up each of the rungs of the DNA ladder are easier to sever than those that connect successive nucleotides. With respect to gene-based selection in biological evolution, the preceding features of the DNA molecules count as structure but not as information. Of course, DNA itself had to evolve via selection. DNA molecules are adapted to replicate. The features of DNA molecules that allow them to replicate were selected for in the origin of life (Küppers 1990). But these features of DNA molecules do not "code for" anything.

In gene-based selection in biological evolution, much of the relevant information is comprised of the linear sequence of bases in molecules of DNA. Unfortunately, in spite of the massive amount of work done by a variety of scholars on explicating the notion of information, none of the suggestions made thus far is adequate to distinguish information as it functions in selection processes from other sorts of structure. For example, physicists treat any structure as "information." The information contained in a double helix is no different in kind from that exhibited in the linear sequence of bases. As helpful as the work of Dretske (1981) and Küppers (1990) may be in other respects, it cannot be used to distinguish the special sort of structure exhibited by sequences of base pairs in molecules of DNA from structure as such. Nor is it adequate to make this crucial distinction with respect to the immune system and learning. The one bright spot on the horizon is that several biologists, such as John Maynard Smith (2000), and philosophers of biology such as Peter Godfrey-Smith are currently working on the problem. Progress may be forthcoming. If we are to have an adequate conception of selection, progress in our understanding of information *must* be forthcoming. In the case of causation, the problem is that too many different analyses of causation exist, some adequate for certain causal situations, others adequate for others. In the case of information, the problem is that too few analyses of information exist, and none of them is adequate for understanding selection processes. In writing this article, we were presented with two choices: register this major deficiency in our understanding of selection and move on or present from scratch an analysis of information that is up to the task. We decided on the first alternative. We hope that others will eventually come to adopt the second alternative (for a critical evaluation of the recent literature on information theory, see Sarkar 1996 and Harms 1998).

2.3. Environmental interaction

Dawkins (1976) placed considerable importance on the notion of replication. It is the primary explanatory concept in his analysis of selection. Many critics think that Dawkins places too much emphasis on replication as if it were sufficient for selection. They also raise the issue of the problematic character of information, as we have. Dawkins also introduced a second notion, that of a *vehicle.* According to Dawkins, replicators replicate themselves (homocatalysis). In addition, they produce vehicles (heterocatalysis). Replicators do more than just cause or produce vehicles; they *code* for them. For Dawkins the relation between replicators and vehicles is that of development. A third major criticism of Dawkins's view of biological evolution turns on the relation that he sets out between replicators and vehicles. Replicators not only code for their vehicles but also ride around in and steer them. Vehicles are nothing but survival machines, lumbering robots controlled by the replicators that produced them.

To begin with, Dawkins's vehicles of selection have to be distinguished from Campbell's (1979) physical vehicles. For Campbell, *vehicle* refers to the material basis or carrier of information; for example, molecules of DNA that incorporate information in the order of base-pairs, the paper on which books are printed, the plastic that was once used for phonograph records, and the chips in electronic computers. Clearly, Dawkins means something else by vehicle. Most narrowly, he means the organisms produced by genomes. Needless to say, this narrow notion immediately raises the nature-nurture issue. In what sense does a genome code for an organism? A genome all by itself never produced anything (Marx 1995). Genomes plus numerous other factors produce organisms. However, according to the standard framework, both genes and environmental conditions *cause* traits, but only genes *code* for them. Of course, the metaphor of genes coding for traits remains as problematic as ever. We are well aware that sketches of several alternatives to the traditional gene-based view of biological evolution exist. Our concern in this article is to provide an account of selection adequate for the traditional view, not to answer every objection raised to the traditional view.

If the general analysis presented in this article is to be ap-

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plied to specific instances, the terms used in this analysis must be operationalized. Environmental interaction must cause replication to be differential. For example, drift is differential perpetuation without environmental interaction. In this connection, selection *for* is often distinguished from selection *of*. A gene contributes to the development of a trait that interacts with the organism's environment so that this gene replicates more profusely than the genes of conspecifics that lack this gene and trait. This gene is being selected for this ability. A second gene adjacent to the first gene may piggyback on it. Because this second gene does not interact in the relevant sense with its environment, it is not part of the cause of this increase in frequency. As we construe selection, development is only one of the causal relations that can exist between what Dawkins terms replicators and vehicles. For a truly general account of selection, a much broader relation is necessary. The relation must be causal, but it need not be developmental. Numerous other processes are also operative. For example, molecules of DNA interact with their environments to replicate themselves, but this process does not involve anything like ontogenetic development in the production of vehicles.

If the distinction between replication and environmental interaction does anything, it goes a long way in resolving the levels of selection controversy. When Dawkins (1994) says that genes are the units of selection, he means replication. Genes are the primary units of replication and hence selection. When others such as Mayr (1997) say that organisms are the primary focus of selection, they mean environmental interaction. In gene-based biological evolution, organisms are the primary units of environmental interaction and hence selection. To be sure, both replication and environmental interaction are necessary for selection, but we do not think that either is sufficient by itself. Both are needed for selection to occur. As Lloyd (1988) has pointed out, the levels of selection controversy concerns environmental interaction, not replication. Entities from molecules of DNA, cells and organisms to colonies, demes, and possibly entire species interact with ever more inclusive environments in ways that bias replication. Selection involves two processes, not one. There are units of *replication* and units of *environmental interaction*, but there are units of *selection* only in a highly derived sense, in the same derived sense that IQ is a measure of intelligence (Heschel 1994; Hull 1980).

3. Selection in biological evolution

The highly general characterization of selection set out in the preceding pages applies in a straightforward way to selection in gene-based biological evolution. In each case the sort of selection that population biologists study can be seen to be a special case of the more general analysis of selection provided in this article (for a recent criticism of analyzing selection in terms of replication, see Griesemer 1999).

3.1. Mutation and recombination

In gene-based biological evolution, the sources of variation are point mutations and recombination. Point mutations result in a single nucleotide being changed. Recombination results from the reorganization of the linear structure of DNA. As it happens, recombination produces most of the variation that is actually operative in biological evolution. The linear sequence of nucleotides in DNA provides the information necessary for the production of proteins. Any rearrangement of these orderly nucleotide sequences stands a chance of changing the genetic information encoded in its DNA and possibly the phenotype of the organism as well. The causes of variation in the genetic material are important. The effects that genes have on the phenotype of an organism are equally important. In selection, genetic variations must result not only in phenotypic variations, but also these differences must affect the individual with respect to survival and/or reproduction.

At one time, biologists believed that the vast majority of mutations result in a decrease in proliferation, while only a small percentage increase proliferation or do not affect it at all (but see Peck & Eyre-Walker 1998). Mutations can fail to affect proliferation in two ways: either they have no phenotypic effects or else the phenotypic effects make no difference to survival and/or reproduction. Once biologists had more direct access to the genetic material, they discovered all sorts of unexpected things about it. They found that most of the genetic material has no apparent function. Perhaps it did in the past, perhaps its current functions have yet to be discovered, but right now most of the genetic material does not seem to do much of anything. In part as a result of the former finding, it turns out that most mutations are selectively neutral (i.e., as a result of environmental interaction, they neither increase nor decrease in frequency), while some are selected against (they decrease in frequency because of environmental interaction), and only a small percentage are actually selected for (they increase in frequency because of environmental interaction). As important as Kimura's (1983) work has proven to be, his claim that changes in our beliefs about the relative frequencies of these three types of mutation requires a new theory of evolution has not been widely accepted (see Brookfield 1995).

3.2. Replication

What are the primary replicators in biological evolution? Genes, larger chunks of the genetic material, and sometimes even entire chromosomes can function in replication. Replication at higher levels of organization may also occur, but the more inclusive the entity, the harder it is for the requirements of replication to be met. The important point is that once the notion of replication has been distinguished clearly from environmental interaction and selection, this question (and it is an empirical question) can be answered more definitively. What are the entities that interact with the environment in ways that result in differential replication? Everything from genes, cells, and organisms to hives, demes, and possibly entire species. Environmental interaction wanders up and down the organizational hierarchy, while replication is largely limited to the genetic material. In some circles the view that genes are the primary replicators and that environmental interaction occurs at a variety of levels is considered radical – possibly true but still in need of extensive elaboration and corroboration. In other circles, it is considered to be the received view that needs to be replaced by a more sophisticated theory.

Needless to say, wide agreement does not exist about the character of this more sophisticated view. As is usually the case in such disputes, one side parodies the other. For example, certain critics of the received view treat replication

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as a nonsense notion, as if replication is supposed to occur in the absence of any and all environmental contributions, but even the most rabid gene replicationist knows all of this. Quite obviously, replication requires all sorts of environmental inputs, including energy and the relevant enzymes (Marx 1995). Traditional versions of neo-Darwinian theory have enough faults without inventing irrelevant parodies. Perhaps evolutionary biologists have not spent enough time attempting to integrate development into evolutionary theory, but they are well aware of its existence and the need for such an integration (e.g., Davidson et al. 1995). Perhaps an adequate theory of evolution will require the sort of fundamental revisions that some critics of the received view suggest (e.g., Griesemer 1998; Jablonka & Lamb 1995), but evolutionary biologists are likely to be swayed more by positive contributions than by continued criticism.

3.3. Environmental interaction

In the traditional view of biological evolution, the primary means of recording (or retaining) and passing on variation is via genes. That is why we have been terming selection in biological evolution *gene-based.* Then these genetic variants must interact either directly or indirectly with the environment so that in the last analysis replication is *differential.* Some replicates are more likely to be passed on than others. In addition to replicating, genes also code for phenotypes, and these phenotypes can be exhibited at various levels in the organizational hierarchy from genes, cells, and organisms to colonies, populations, and possibly entire species. Genes interact with their cellular environments, but they also interact with increasingly more complex environments via their surrogates. The *fit* between these phenotypes and their environments determines which genes get passed on and which not. In more general terms, the information contained in replicators gets passed on differentially because of how successfully they or their products interact with their respective environments (Brandon 1982).

What are the entities that function in environmental interaction? Can we get along just with the notion of phenotypic effect, regardless of these effects being bundled together into organisms? As strange as it might sound, genes themselves exhibit adaptations. The most obvious thing about DNA is that it is adapted to replicate. During periods of replication, genes interact with their immediate environments. They could not replicate without appropriate environmental contributions. Organisms exhibit phenotypic traits in the most obvious sense. Some organisms in a species have split telsa; others do not. At the other extreme, even species exhibit phenotypic traits. For example, the peripheries of the ranges of some species are highly convoluted. If speciation usually occurs at the peripheries of these ranges, then such convolutions, if they are heritable, might count as adaptations for increased rates of speciation. Some authors complain that requiring adaptations for selection, including species selection, is too restrictive. A more general notion is required, the sort of general characterization that we have provided (Gould & Lloyd, forthcoming; Lloyd 1988; Sober & Wilson 1998; Wilson & Sober 1994).

Much of the discussion of selection in the recent literature has concerned replication, but environmental interaction is at least as important in selection as is replication. The strongest feature of Darden and Cain's (1989) analysis of selection is the emphasis that they place on environmental interaction. As they put it, "individuals must be in an environment with critical factors that provide a context for the ensuing interaction" (Darden & Cain 1989, p. 110). The debate that Dawkins's *The selfish gene* (1976) elicited was generated in large measure by an ambiguous use of the term *selection* in the literature. One side of the dispute conflates replication with selection, while the other side conflates interaction with selection. Dawkins argues at great length that in biological evolution the relevant replicators are genes and only genes. Replication is certainly necessary for selection as it occurs in biological contexts, but it is not *sufficient.* Replication and variation in the absence of environmental interaction results in drift, and as important as drift may be in the evolutionary process, it is not a consequence of selection (Donoghue 1990). Selection requires an interaction of some sort between the environment and the replicating entity.

Dawkins's opponents have countered that organisms are the primary focus of selection. They, not genes, are the units of selection. Just as Dawkins, early on in the controversy, too often elided from replication to selection, his critics tended to equate selection with environmental interaction. As in the case of replication, environmental interaction is necessary but not sufficient for selection. Without replication, iteration is impossible, and in the absence of iteration, selection could not be cumulative. Selection is the result of differential replication *caused* by environmental interaction. Once again, selection is two processes, not one. It is the alternation of replication and interaction with the occasional introduction of variation.

3.4. The environment

Of all the terms in the preceding characterization of selection, *replication* has received the greatest attention. However, the most difficult notion is that of the environment. Not until Antonovics et al. (1988) has it received the analytic attention that it deserves (see also Brandon 1990). These authors distinguish between three different sorts of environment – the external, ecological, and selective environments. The external environment is the "sum total of the factors, both biotic and physical, external to the organism that influence its survival and reproduction" (Brandon 1990, p. 47). The ecological environment of an organism is composed of "those features of the external environment that affect the organism's contributions to population growth." (Brandon 1990, p. 49). Finally, the selective environment is an area (or population) that is "homogenous with respect to the relative fitness of a set of competing types" (Brandon 1990, p. 69).

One problem with respect to selective environments is whether or not to include other organisms, including conspecifics, as part of the selective environment. Such decisions have effects, for example, on how one handles cases of density dependent population regulation (Brandon 1990, p. 65). Wilson and Sober (1994, p. 641) see this issue as clearly distinguishing their views from those of Dawkins. No sooner did Dawkins introduce the notion of a *vehicle* in his account of selection than he began to undermine it. "I coined the term 'vehicle' not to praise it but to bury it" (Dawkins 1994, p. 617). According to Dawkins (1994, p. 617), "Natural selection favors replicators that prosper in their environment. The environment of a replicator in-

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cludes the outside world, but it also includes most importantly, other replicators, other genes in the same organism and in different organisms, and their phenotypic products." Wilson and Sober (1994, p. 641) respond that Dawkins's goal of reconceptualizing vehicles of selection as part of the external environment (in Brandon's sense) reveals a deep contradiction in the gene-centered view of selection. Clearly the notion of environmental interaction deserves at least as much attention as replication.

4. Somatic selection in the immune system

More than a million different antibodies are needed to provide sufficient protection against the huge number of pathogens a host may encounter during its lifetime. Antibodies are protective because they act as markers that signal the recruitment of powerful biodestructive cells and enzymes which then destroy the pathogen and stop it from overgrowing the host. Managing to make sure that none of the millions of antibodies recognize any part of the host is obviously essential. If there were only a few antibody specificities, and a correspondingly small number of genes encoding these antibodies, then any rare cases of self-recognition might reasonably result in the destruction of that rare organism – this is an example of germline selection. When the rate of evolution of the pathogen (often hours) is much faster than the rate of evolution of the host (often months to decades), then the host genome cannot carry the millions of different genes needed to track the millions of different mutations in the pathogens. Moreover, among the millions of different antibodies, some will inevitably recognize a self-component of the host and have the potential to destroy the host. What makes the immune system special is that it is able to select on the specificity of each antibody and eliminate the deleterious antiself before it can actually kill the host. Because each different antibody is expressed in a different cell with a correspondingly different set of genes that encode that antibody, the immune system is able to select on the cell in order to eliminate these antiself antibodies instead of having to eliminate the whole organism. This form of cellular selection on genetic variants is an example of somatic selection. In immunology it is common to refer to the germline as the genetic material that is selected upon when individuals are replicated and to distinguish this from the soma where the genetic material of individual cells can be varied and selected upon as cells are replicated. While the factual basis for phenomena discussed here can be found in any modern textbook of immunology, the conceptual analysis should not, however, be taken as representing the standard view of the immune system.

4.1. Variation: The origins of antibody diversity

The genetic basis of antibody diversity is partly due to the presence of several different, normally inherited genes and partly due to mutations that occur in these genes when they are expressed in the soma as antibody-producing B cells. Extensive genetic and sequencing studies can be summarized along the following lines. The antibody molecule is made up of two different polypeptides, the L (light) and H (heavy) chains, which are encoded at two different genetic loci. The particular specificity of an antibody is determined by roughly equal contributions from the L and H chains. The part of each chain that is primarily concerned with antibody specificity is called the V (variable) region and the remainder the C (constant) region. Each region is encoded as a separate gene segment, and there are about 100 V-L and 100 V-H gene segments but only one C-L and one C-H segment. A series of gene fusions permanently changes the chromosomes in B cells and results in the joining of any one of the 100 V segments with the single C segment to produce a single V-C gene that encodes the complete L or H polypeptide. The 100 different L chains and 100 different H chains form random pairs and 10,000 corresponding different specificities. The gene fusions are arranged in such a way that joining errors are maximized. Consequently, few B cells are actually able to produce two L or two H chains. In other words, the B cell is made functionally haploid so that each B cell expresses only one kind of L-H pair and, therefore, one specificity. Of course, the level of waste is relatively high as 70 to 90% of B cells that attempt to produce antibodies fail and are eliminated.

Throughout the life of an organism, the B cell population is undergoing constant renewal, and this renewal requires the mechanism for eliminating potentially self-reactive B cells to operate continuously throughout life. Controversy surrounds the details of this mechanism of self-nonself discrimination, but the exact nature of this mechanism is unimportant here. The result in any case has to be that individual B cells can be somatically selected according to the particular antigens that react with their receptors. The result is a means of selecting against B cells that can react with self components and neutral selection on B cells with specificities that do not react with self components. However, when a B cell that has not reacted with self is subsequently confronted by the particular pathogen with which it can react, then the B cell is strongly selected for, and, so long as antigen persists, the cells undergo many rounds of mutation and division while secreting huge amounts of their antibodies. The negative selection pressure imposed by self components is constant (self is constant). During the many rounds of cell division that occur when a B cell is under selection by nonself antigens, mutations are introduced in the V segments of the L and H genes that, by chance, affect specificity. These mutations are so important that a special mechanism operates over the V gene segment and is able to introduce single base changes at the rate of 10^{-3} per base pair per generation; in contrast, the normal rate of mutation of around 10^{-9} per base pair per generation operates on the C segments. Some mutations in V segments are neutral and do not affect specificity, others destroy function, and a few change specificity and improve the ability of the antibody to react with its antigen at much lower concentrations than were present when the B cell was initially selected.

A brief comment on some terms and concepts might be helpful. Antigens are the parts of the pathogen that react with antibodies. Usually the term *paratope* is used to describe the part of the antibody that binds the antigen, and the term *epitope* is used to describe the site on the antigen that reacts with the paratopic part of the antibody. A complex pathogen, such as a bacterium, can expose many epitopes and induce the production of many paratopes, including mutant forms of the initially selected paratopes. The actual B cells that are selected by a particular antigen

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will depend on the concentration of the antigen and the affinity of the B cell receptor for that antigen. As a result, some B cells will respond only at high antigen concentrations while others will respond only at low antigen concentrations.

4.2. The replication-variation-interaction sequence

The course of events following infection by a pathogen begins with a small inoculum of dividing pathogens. Initially they are at too low a concentration to cause the selection of any B cells. Then, after some time, the number of pathogens increases to reach a concentration that can induce an antibody response. The responding B cells proceed to divide and secrete huge amounts of antibody. As the antibody diffuses into the body fluids, it binds to the pathogen and so marks it for destruction. Providing enough antibody is present to halt the growth of the pathogen, then the immune system will have protected the host. As the numbers of pathogen decrease, the concentration of antigen driving the division and mutation of B cells also decreases with the net result that only those B cells able to respond to the lowest concentrations of antigen will remain dividing. In this case somatic selection for variant antibody genes allows some B cells to divide more often than others. During this period, B cells will have behaved in a manner very similar to the pathogenic organisms. Each will have also undergone mutation, and those pathogens that could render their antigens unrecognizable by the immune system will have been at a powerful selective advantage, whereas those B cells that could track the antigenic changes will help protect the host.

In summary, the B cell component of the immune system illustrates two levels of somatic selection. First, the steady flow of new B cells that can react against self components are deleted. Then, when nonself antigen happens to enter the host, those B cells that can react with the pathogen are selected to undergo many rounds of cell division and mutation with repeated selection for those B cells that continue to react with an ever decreasing concentration of antigen. This process is termed *affinity maturation.* When viewed in the context of the presence of environmental selection pressures (either self or nonself antigens), the individual B cells of the organism undergo a process that is indistinguishable from what is normally thought of as classical gene-based biological evolution of organisms, even though these B cells are not able to behave in all the ways often expected of an organism.

In terms of the overall effectiveness of the immune response, the affinity maturation process is of marginal significance because it occurs after the pathogen has been eliminated and can therefore only act during subsequent reinfections. The two significant selection processes occur first at the level of sorting the stream of new B cells into specificities that are either self (to be eliminated) and nonself (to be kept) and second at the level of amplifying only those B cells with specificities that react with the pathogen that suddenly and unexpectedly appears. The strict notion of serial rounds of replication, variation, and interaction applies only to the small component of affinity maturation in the overall immune response. However, it would be difficult to argue that the immune system does not undergo somatic evolution as a parallel to classical gene-based evolution found in the pathogens.

4.3. Somatic selection versus germline selection

Another important aspect of somatic B cell evolution is whether its origin as a part of the developmental program of the host is sufficient to disqualify this process as an example of selection. Included in this question is the inability of the immune system to continue evolving when the host dies. When the host dies of starvation or from being eaten by a tiger, it does not mean that the host's immune system is defective; it just happens to stop evolving because of some unselectable cosmic catastrophe. It seems unnecessarily restrictive to say that selection has to continue for some arbitrary period of time. To be able to show that somatic selection in the immune system stops for some reason other than a failure of the immune system is sufficient to conclude that a process of selection has been at work.

Many similarities exist between somatic evolution in the immune system and the functioning of the nervous system. In contrast to the detailed knowledge of the molecular and genetic structures and functions of the immune system, much less is known about the nervous system, and as our analysis of operant learning will clearly illustrate, even in a well-defined behavioral domain, the relevant molecular and genetic factors are almost unknown. Nonetheless, Edelman's ideas on immunology and neurobiology are sufficiently interesting to warrant comment. As a leading figure in the early years of modern immunology, Edelman was a strong proponent of what has come to be termed the "big-bang" version of the generation of antibody diversity. In particular, he postulated a somatic genetic recombination mechanism that could generate a huge number of variants without having to resort to point mutations, which he thought to be rare and to occur throughout the genome (Gally & Edelman 1972). This initial burst of genetic diversification dispersed the variants in different B cells, which were then subject to selection with respect to self and nonself reactivity. Further genetic diversification after infection and antigen selection was thought to be minimal because it might include the introduction of new specificities able to react with and destroy self – the host.

Under big-bang models, all of the diversity of the immune system is generated early in ontogeny, driven largely by the need to eliminate antiself at an early stage in order to leave the remaining anti-nonself repertoire large and readily induced. This conceptual framework of big-bang diversification in the immune system can be found in Edelman's later work on neuronal development (Edelman 1987). Two points need to be made regarding big-bang and the immune system. First, big-bang diversification necessarily includes all the waste in all of the possible lineages selectable by antigen. Second, Darwinism, if it exists, must surely be more than selection from an unimaginably huge pile of possibilities. As we argue here, Darwinism involves multiple cycles of selection; that is, interaction, mutation, and replication. Big-bang requires only one round of mutation and selection, followed by continuous selection.While big-bang is difficult to justify when the immune system is constantly being regenerated; this is less of a difficulty in the case of the brain, where little cell division occurs once it reaches its adult size.

The purpose for raising the big-bang principle is to emphasize that it denies the kind of serial selection we propose here for the three systems under investigation. Empirical

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observations notwithstanding, an a priori case can be made that if all possible variants are generated during big-bang and if the fraction of all possible variants used during the lifetime of an individual is very small, then the waste generated by unused variants is prohibitively large. *By generating variants among only those cells (or neural connections or organisms) that are already responsive to the selection pressure, waste in the production of unresponsive variants is limited and is not spread among all possible cells in all possible lineages. However, when variation is restricted to those entities responding to a selection pressure, each intermediate variant in a lineage must be individually selectable. If not, then the lineage would become extinct while waiting for a second or third variant to occur.*

One underappreciated selection pressure is the relative levels of waste, especially when evaluating probable versus improbable lineages. Before accepting models of neural behavior based on big-bang diversification followed by somatic selection, it would seem prudent to consider possible alternative models based on serial selection, because the level of waste in the latter is likely to be substantially less than in the former case. The widely quoted work of Hinton and Nowlan (1987) provides another illustration of somatic selection gone awry. Their assumption of 20 switches in a neural network, each individually inactive, but providing a strong selective advantage when correctly coupled, is close to impossible. It may be true that given this impossible starting condition a form of somatic selection might be envisaged that is capable of selecting the right combination of switches, and that eventually a germline selection for the switches all being in the right configuration is favored because the right combination is always found quickly. However, the exercise is rendered moot because the initial assumption is, at best, implausible. There is simply no remote likelihood of 20 gene duplication and mutation steps occurring in the absence of selection of the intermediates (i.e., the intermediate switches from 1 to 19 are individually unselectable).

4.4. Population-level selection in the immune system

Several mechanisms have evolved to produce the massive amount of variation necessary to make the immune system work. The genes that code for antibodies have developed a variety of mechanisms needed to rapidly diversify a relatively small number of germline genes in a large somatic population of B cells. Central to these mechanisms is the generation of a functionally haploid genome in the region encoding antibody specificity. The introduction of mutational variants in these haploid specificity regions creates a population of different B cells, which are then subject to further individual selection by antigen. The immune system also exhibits a very different kind of variation that is uniquely expressed at the level of populations of host organisms. This variation is confined to the 2–4 genes that determine what is termed the major histocompatibility complex (MHC) – the locus primarily responsible for the extreme difficulty in transplanting tissues. The MHC genes exist as a large number of alleles (~100) that are found at roughly equal frequency in the interbreeding population. Although there are roughly the same number of alleles of the hemoglobin genes, all but a few alleles are at a such a low frequency that they can be accounted for by mutation alone. To explain the roughly equal frequency of so many alleles at the MHC locus requires postulating a selection process that operates at the level of the genes in individuals and at the level of gene expression in the population.

The exact nature of the selection pressure operating on the MHC genes is not well known, but one compelling, illustrative explanation depends on the role that these genes play in immune protection against viral infections. In order for the immune system to respond appropriately to events occurring inside a virally infected cell (and without cracking the cell open to peek inside), the immune system uses the MHC genes to provide means for transporting intracellular peptides to the surface of the cell. Once these peptides are displayed on the cell surface, a special type of antigen-specific cell (the T cell) is able to bind specifically to the peptide under the right conditions and decide what to do. It works as follows: each MHC allele picks up a slightly different peptide fragment and presents it to the immune system. Viruses may well be selected for if they have mutations that disable or block the peptide binding site on the MHC and so stop the immune system from detecting presence of intracellular virus. To combat this occurrence, the host has at least two, and sometimes four MHC genes, each with a different peptide binding specificity. If all individuals in the population had the same alleles with the same peptide binding specificity, then, as the virus moved from one individual to the next, it could keep on evolving to defeat the MHC system. However, if the population possesses a large number of different alleles, then when the virus moves from one host to the next, all the selection in the previous host is canceled because the new host has new peptide binding rules determined by the new MHC alleles. Thus, each allele functions perfectly well in an individual, but selection on the virus extends over many individuals at the population level. One result of this process is the large number of alleles in the population. This situation can be contrasted with the large number of antibody specificities needed per individual. The polymorphism of the MHC locus provides a particularly clear example of selection on alleles of genes that must occur at the population level while still being executed at the level of the individual organism.

4.5. Serial somatic selection: The immune system is one example

In this brief overview of the immune system, we have extracted four examples of selection. (1) In the case of the 100 germline encoded V-segments at the L and H chain loci, these segments can produce 10,000 different LH pairs with different antibody binding sites. The selection that maintains these segments as different V segments is 100 pathogens that would be a threat if it were not for the specificities of these 100 unique LH pairs; the 9,900 other combinations are unselected as particular specificities and represent a very small form of big-bang. (2) Among the unselected LH combinations and point mutants of these segments (up to one million of them), some are able to recognize self components of the host and have the potential to kill the host instead of the pathogen; these specificities are selected against by killing the cell that makes that specificity of antibody before the antibody is secreted and can kill the host. (3) Some specificities are able to recognize antigens of the pathogens, and the B cells that make these specificities are induced to proliferate and mutate so as to

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produce new specificities that function better (at lower concentrations of antigen) than others. This example of somatic selection is also an example of serial selection of the type that forms lineages akin to those found in the serial selection processes of the evolution of organisms. (4) In another domain of immune system function, MHC molecules play a critical role in allowing the immune system to be informed of the presence of pathogens located inside the cells of the host.

Although each individual organism (host) has 2–4 different MHC genes, in the population of organisms, there are 50–100 alleles, all at roughly the same frequency; and this implies that selection, which must occur in individuals and their genomes, is via a selection pressure that only affects individuals because they are in a particular population (i.e., the selection pressure is particular to the population an individual is a member of). This process is strictly germline selection, not somatic selection, and is sometimes referred to as group selection. If we take a hard position on selection processes and require repeated rounds of replication, variation, and interaction, then the immune system offers one example of selection in affinity maturation. However, taken together, the other two examples of somatic selection also seem to simulate all of the features we might expect of serial germline selection in classical gene-based organisms.

5. Operant selection

In one sense, all the behavior of organisms is the result of natural selection; in another sense, none of the behavior of organisms can be attributed to natural selection. The first statement follows from the fact that natural selection accounts for the range of behavioral potentialities characteristic of the organisms in any particular lineage and also for the processes that account for behavioral content that is uniquely suited to circumstances arising during an organism's lifetime. The second follows from the fact that processes other than natural selection are always involved when behavioral content actually appears in the behavior stream of a living organism. Between these two extremes lies the vast domain where behavioral scientists toil. Although no serious student of science would likely subscribe categorically to either of the two extremes, behavioral scientists with differing interests focus their attention on different segments of the continuum and tend to characterize those with interests elsewhere on the continuum as occupying one or the other of the extremes. Full scientific understanding of behavioral phenomena will require understanding the full range of behavior from one end of the continuum to the other.

5.1. Operant behavior

When the behavior in which scientists are interested changes in content, often dramatically, during an organism's lifetime, one might say that those scientists are interested in the behavior of behavior. While such a locution sounds odd, a cursory look at how *behavior* is used in science reveals that scientists discuss the behavior of volcanoes, proteins, hurricanes, immune systems, and the like. When change in the phenomena of interest is the object of scientific study, the scientists are said to be studying the behavior of the phenomena. If the phenomena of interest under investigation are the activities of organisms, and those phenomena are themselves exemplified by change, then behavior change or the behavior of behavior is the object of scientific study.

The behavior changes of interest here are changes in behavior that occur during a single lifetime. The topic under discussion is further narrowed to those changes in behavior that result from a selection process that is conceptually parallel to the natural selection of organismic characteristics across generations of organisms. Most of the scientists studying this type of behavior designate it as operant behavior, and they designate changes in operant behavior of a particular organism *operant learning*. Traditionally, operant behavior has been defined as behavior that operates on the environment and changes over time (in form, organization, or relations to the antecedent environment) as a function of its consequences. From the present perspective, "its consequences" is a shorthand way of saying the "goodness of fit between the behavior and consequent changes in the environment." In short, the particular operant behaviors that emerge and change during the lifetime of individual organisms are the results of "a second kind of selection" – a process that itself is the historical result of the "first kind of selection" (natural selection) (Skinner 1981, p. 501).

Many questions arise from a selectionist characterization of operant learning. How does operant learning fit into what we know about the evolution of species by natural selection? How does this second kind of selection differ from selection processes that result in the origin (and history) of the species? What are the units of selection in operant selection? In this paper, we address these issues briefly. We readily acknowledge that a complete explanation of operant behavior will involve processes other than operant selection, just as organic evolution involves processes other than natural selection. We also acknowledge that not all behavior is operant behavior and, hence, that no discussion of operant learning will answer, or even address, all the questions and issues pertaining to the range of phenomena in the domain of behavior.

We have chosen to focus here on operant learning for both conceptual and practical reasons. Operant processes are known to occur in several phyla, suggesting that their origin reaches deeply into the history of life on earth. Second, and paradoxically, operant selection seems particularly relevant to humans (Schwartz 1974, p. 196). Hominid anatomical features such as opposable thumbs, highly developed cortex, and vocal apparatus may have coevolved with increasing susceptibility to operant selection. On the practical side, thousands of experiments have yielded a large and complex literature from which to draw, some even conceptualizing results in selectionist terms (e.g., Staddon & Simmelhag 1971). Unfortunately, we can only draw upon an extremely limited part of that literature and will selectively attend to work that clarifies the theoretical perspective presented here. Finally, operant behavior is the area of interest of one of the authors. In the same way that "a zoologist may specialize on vertebrates without denying the existence of invertebrates" (Dawkins 1983, p. 405), we do not deny the existence or importance of behavior that is not operant.

Operant behavior, like biological evolution, is one of those simple topics that are widely, persistently, and sometimes perversely misconstrued or misrepresented (Todd & Morris 1992), thus guaranteeing that at least some readers

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will find what follows to be at odds with conceptions colored by such misrepresentation. In an attempt to preclude excessive cognitive dissonance, we begin with a few general points that we think critical to understanding the theoretical perspective presented next.

5.2. Adaptation and complexity

In the larger context of biological evolution, an organism's operant behavior has the biological function of interfacing between the organism and its world. An analysis of operant selection requires allowing the organism that behaves to recede to the conceptual background and making the interface itself the object of investigation. This change of perspective amounts to a figure-ground reversal from that which is apparent in direct perception. The new figure is by its nature difficult to see due to the temporal character of its structure. But behavior has structure of its own. It is made up of parts and wholes, which are parts of more inclusive wholes, and those parts have functions, as do the wholes. An operant repertoire is made up of interrelated behavioral lineages, each having its origin at a different time in the history of the organism, and each having its own history. As in the case of the evolution of life on earth, understanding the process requires focusing on particular lineages. Each behavioral lineage evolves in relation to its local environment, and changes in one lineage can impact other lineages in the organism's repertoire. A particular operant repertoire generally becomes, over time, increasingly complex in terms of the number of lineages it comprises, the complexity of its component interactors, and the historical and ecological relations among them.

The processes by which operant adaptation occurs are viewed here as analogous to the processes by which biological evolution occurs. Specifically, operant selection (in concert with other processes) adapts organismic activity over time to fit the environment in which it occurs. If the environment moves out from under the behavior slowly enough, the behavior may be able to adapt to the changing environment. If the environment changes too rapidly, the behavior may be extinguished. As in the evolution of species, operant behavior fits the present environment because of past selection and not because of any future state of affairs. Further, operant behavior that is well adapted to its environment may not contribute to the survival of the organism that is behaving. For example, behavior that is well adapted for producing drug-induced euphoria may result in premature death of the organism. Operant processes work the same way regardless of whether particular behaviors are conducive to survival of the behaving organism. So far as survival and reproduction are concerned, operant behavior is a very sharp two-edged sword.

Gene-based selection is studied in bacteria and fruit flies as exemplars of a process assumed to account for all species. Similarly, operant selection often is studied in lever presses and key pecks as exemplars of a process that has been shown to operate with respect to more complex behavioral units. Most readers of this paper will readily accept the proposition that a single set of processes accounts for the structural and functional complexity of primates as well as bacteria. Although we trust they can entertain the analogous possibility that a single set of processes can account for structure and function of behavior far more complex than lever presses and key pecks, they may draw their dividing line between operant behavior and higher behavior wherever they please.

In the next sections, we provide examples of the ways in which operant selection results in behavior change. The theoretical language used to describe the process is the language we suggest for a general analysis of selection rather than the language used by the original researchers. We readily admit that we are viewing operant behavior from a nontraditional perspective and regret that some parts of the analysis are somewhat speculative. Evolutionary biologists had to develop evolutionary theory for decades in the absence of an adequate theory of heredity. Even after development of Mendelian genetics was under way, considerable time elapsed before these two groups of scientists were able to see how the theories could be combined into a single coherent theory. Operant researchers and neurophysiologists are in a comparable position today. Neural mechanisms are not well understood by most operant researchers and the ways in which operant behavior changes as it undergoes environmental selection are not well understood by most neuroscientists. Although experimental evidence supports a selectionist interpretation of operant behavior and its neural underpinnings, theoretical revision is likely to be required. On the positive side, massive experimental evidence supports a selectionist interpretation of operant learning, and attempts to relate the findings to one another are increasing in number.

5.3. Operant interactors and the behavioral environment

The relation between responses and consequent stimulation (environment) is the area where most operant researchers have focused attention. In operant selection, the primary role of entities traditionally identified as responses is that of "interactor," the unit that "interacts as a cohesive whole with its environment in such a way that this interaction causes replication to be differential" (Hull 1989, p. 96). Although the most obvious entities functioning as interactors in behavioral selection are responses, some interactors in operant selection cannot easily be conceptualized as responses. For example, a group of responses may function as a cohesive whole in operant selection. The members of the group may be homogeneous, such as a burst of lever presses that interacts as a cohesive whole with its environment; or an interactor may be a cohesive whole made up of many different and functionally related parts, as in baking a cake or driving to work. Although the interactors that experimentalists work with in operant laboratories are often lever presses and key pecks, applied behavior analysts have demonstrated in hundreds of studies that operant selection occurs at many levels of behavioral complexity. To assist the reader in relating the ideas presented here to previously acquired concepts, we will use *responses* for interactors that can easily be conceptualized as responses and use the more technically correct term *interactors* when the events are less easily conceptualized as responses in the traditional sense.

In operant theory, activity designated as a response does not require a stimulus (Skinner 1953, p. 64). Beginning with the assumption that a particular response occurs because it is elicited by another particular event is neither necessary nor helpful. Most operant responses are functionally related to stimulating events, but those relations are exceed-

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ingly complex. For purposes of exposition, we will concentrate on the least complex of operant lineages – those that might be compared to prokaryote lineages in biological evolution (Glenn & Madden 1995). Traditionally, these operant lineages have been called response classes, but that terminology raises the same conceptual difficulties that arose from calling a species a class of organisms (Glenn et al. 1992). Both *response lineages* and *response classes,* however, imply that operant responses are parts of a population and the characteristics of a population of interactors are the focus of our interest.

When operant behavior is seen as the figure, against organism as ground, the elements involved in selection processes are analogous to (not the same as) those involved in gene-based biological evolution. In operant selection, the interaction step involves a relation between responses (interactors) in an operant lineage and changes in stimulation (consequences) that follow those responses. In the simplest example of operant selection, some relations between behavior and consequent stimulation have the effect of increasing the frequency of responses in the lineage to which the response belonged. This effect is called reinforcement. Other relations result in a decrease in the frequency of responses in that lineage. Depending on the nature of the change in stimulation, this effect is called either extinction or punishment.

The selecting environment (consequent stimulus changes) is a subset of a larger domain of events in the physical world that have function with respect to interactors (responses) in a particular operant lineage. The full range of environmental events having function with respect to the behavior of a particular organism (including events having discriminative, conditional, or motivating functions) is that organism's *behavioral environment.* Any behavioral environment is a subset of a still larger domain that comprises the environment as it is often construed – the physical world (including that part of it deemed social). These different uses of the word *environment* often go unrecognized and are the source of much confusion in the behavioral sciences, as they have been in the biological sciences (see Brandon 1990, Ch. 2).

The facts underlying the points in the previous paragraph are incontrovertible, but the conceptual language calls for further explication. The relation viewed here as the interaction step in operant selection must itself be related to the concepts of variation, replication, and retention in operant behavior as one exemplar of our general analysis of selection. In the following sections, those concepts will be discussed in the context of further discussion of the ways in which an organism's operant behavior changes over time.

5.4. Response frequency in operant lineages

One of the earliest and most productive tools of operant researchers was the cumulative record. Although a record depicts only a small amount of information about each response recorded, it captures a critical feature of evolutionary processes – the frequency at which responses in a lineage appear over time. The responses depicted in the record are those that satisfy the contingencies of selection designed by the experimenter. By changing the selection requirements, researchers bring about changes in the frequency, distribution in time, and selectable properties of responses in an operant lineage. Such changes were the initial subject of research on schedules of reinforcement (Ferster & Skinner 1957). Each schedule specifies a particular kind of selection contingency and consequently each results in its own characteristic response distribution.

Although the selection process works at the level of single organisms and results in historical changes in operant lineages in that organism's behavioral repertoire, a schedule of reinforcement produces its characteristic distribution in different operant lineages of particular organisms, across organisms of a single species, and across species on planet Earth. The striking similarities in distributions of responses on a particular schedule in different operant lineages may be viewed as behavioral heteroplasties. That is, selecting environments having particular features result in operant lineages having characteristic distributions. The distributions arise again and again when the selecting contingencies are repeated.

5.5. Selectable properties of operant responses

Just as all organisms have in common certain properties, all operant responses have in common certain properties. Common properties of organisms include length, width, height, and body mass. Gilbert (1958) identified the "fundamental dimensions" of operant behavior but, from the present perspective, he did not distinguish unequivocally between properties of operant responses and properties of operant lineages. Responses are components of individual lineages in operant selection, just as organisms are components of individual species (and lineages) in natural selection. Some demonstrated selectable properties of operant responses are duration, latency (interresponse time), force, form, direction, and relation to antecedent events. Because the level at which change is measured in selection processes is the population level, the properties of any one response are of little theoretical interest. Evolution occurs at the level of lineages and is measured in terms of response rate (frequency) or frequency of trait values in particular populations of operant responses.

Blough (1963) provided a graphic picture of change in an operant lineage as a function of change in selection contingency. In Figure 1, the response property of interresponse times (IRT) of successive responses in a pigeon's keypecking operant are represented by the height of a dot on the ordinate of the graph. Time is represented on the abscissa. In the first 20 min, while a peck produced food only at the end of 4-min intervals (VI 4-min schedule of reinforcement), the distribution of IRT values in the population of responses was stable. The response population characterizing the lineage at that time shows a good deal of variation in IRT values (0.1 − 6.0 sec) , with a clustering of IRT values around 0.4 sec. The vertical dashed line shows where the schedule changed to FR 30 (every 30th peck followed by food). During the next 100+ min, IRT values underwent a transition in which more and more IRT values clustered around 0.4 sec, although variants continued to appear through the whole range of IRT values. Such a change in population values in an operant lineage is conceptually equivalent to the often cited change in coloration of successive generations of English moths undergoing anagenesis after industrialization. There are differences, however. First, the moth population (as in all sexually reproducing organisms) was distributed in space at any particular time and it extended in time across generations of moths. The

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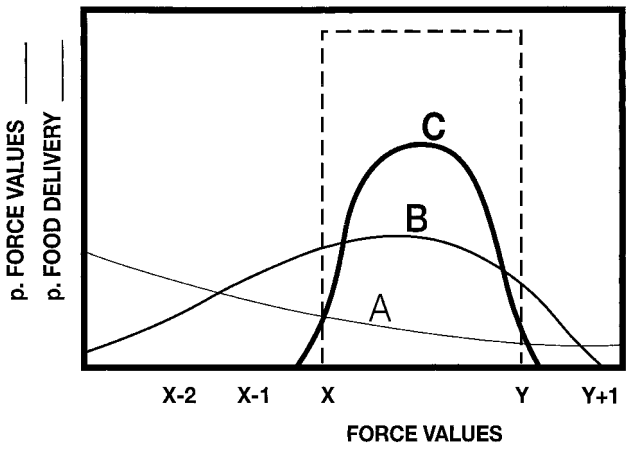


Figure 1. Depiction of changes in IRT values as a function of change in schedule of reinforcement. Please see text for discussion. From D.S. Blough (1963), Interresponse time as a function of continuous variables: A new method and some data, *Journal of the Experimental Analysis of Behavior,* 6:237–47. Reprinted with permission of the Society for the Experimental Analysis of Behavior.

distribution of IRT values in successive populations of pecks occurs only in the time dimension because organisms cannot press a lever more than once at a given time. Second, the trait of interest (IRT) in the operant lineage appears to have a broad range of values, whereas the trait of interest (color) in the biological population appears to have a small number of discrete values. So at the resolution of human observation, responses appear to vary continuously at least in some of their dimensions.

The formal and temporal properties of responses in an operant lineage may vary widely within a population or they may vary within a narrow range of values. IRTs varied by a factor of 60 in Blough's data (Fig. 1). This suggests that IRT may not have been the target of selection. That is, IRTs were not the property (response trait) on which food was contingent but rather they changed along with other properties that were the target of selection. This distinction pertains to that between selection of multi-dimensional interactors and selection for their particular properties (traits) (cf. Glenn & Madden 1995; Sober 1984).

Although operant researchers have not traditionally presented data to demonstrate selection for particular properties, Catania (1973) depicted how response populations at successive times could be depicted to demonstrate the effects of selection for response properties of specified values. An adaptation of his graph for present purposes is shown in Figure 2. The changing frequency of force values in a lineage of lever presses is depicted as it was observed in three populations of responses measured at successive time segments. Force values are fairly evenly distributed in the initial population of responses (A), representing a hypothetical population during a period of time before selection for specified force values. Curve B represents a distribution of force values in a later population, after implementation of a selection contingency in which food pellets follow lever presses having force values between *x* and y (and not otherwise). The dashed line represents the probability of consequent food for presses having force values between *x* and *y.* The distribution of force values in the B population shows the effects of the contingency of selection on the operant lineage. Population C shows the effect of further selection for forces between *x* and *y.* In the B and C populations, those responses falling outside the *x-y* force range represent unreinforced responses (and would not appear in a cumulative record). Although successive populations in this graphical depiction of operant selection are increasingly composed of responses adapted to the selecting environment, variants that fail to meet the selection contingency continue to appear in the lineage, at varying frequencies at different times during the history of the lineage.

5.6. Variation in operant selection

Each interactor in operant selection has many properties and each property occurs at various values in responses forming a lineage. Rarely will two or more responses in a lineage be alike in all respects. Interactors with fewer components are likely to resemble each other more closely than will interactors having many parts, each of which can vary along many dimensions. When a behavioral interactor interfaces with the consequent environment, replication of all of its properties either increases or decreases in probability. However, only some of the interactor's properties may be required for an adequate fit with the selecting environment (e.g., the force values in the preceding example). Those properties are differentially perpetuated in the population maintained by the current selection contingency.

Interactors in an operant lineage can be selected on the basis of the property of varying from their predecessor(s). Page and Neuringer (1985) performed a series of experiments in which a sequence of eight pecks, distributed across two keys, was required to differ in their pattern from (1) the previous sequence (Lag 1), (2) the five previous sequences (Lag 5), and (3) other previous sequences up to 50 (Lag 50). In the present context, each sequence of eight pecks is conceptualized as an interactor that either did or did not meet the requirements of the selecting environment. Selection was for interactors with a sequence of parts that differed from the sequence of parts of the interactor's immediate predecessor and for its last 5, 10, 15, 25, and then 50 predecessors. The eight-peck sequences showed variability in sequencing consistent with the selection contingencies, whether the requirements were gradually increased or whether a Lag 50 was implemented immediately (with other, naive, experimental subjects.) Various control procedures demonstrated that interactor variation was it-

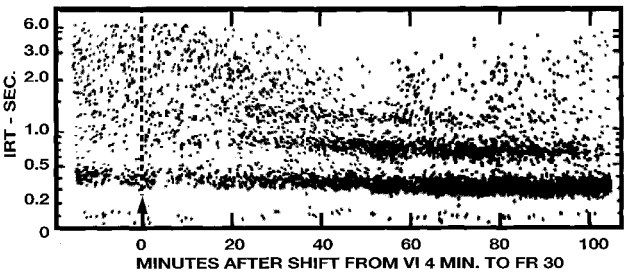


Figure 2. Hypothetical changes in populations of force values in a operant lineage undergoing selection for forces between *x* and *y.* Please see text for discussion. Adapted from A.C. Catania (1973), The concept of the operant in the analysis of behavior, *Behaviorism,* 1:103–16. Reprinted with permission of Cambridge Center for Behavioral Studies.

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self being selected. In further experiments, the authors provided strong evidence that the variability observed was most likely randomly generated ("the pigeons behaved as a quasi-random generator," Page & Neuringer 1985, p. 447) rather than the result of some kind of memory function.

There is a difference in variation as a dimension of behavior and measures such as duration or force as dimensions of behavior. *Variation* is a direct measure of a population (like frequency or rate at which interactors are generated), whereas *duration* and *force* are measures of individual members of a population, which can be represented statistically as measures of a population undergoing selection (as depicted in Fig. 2). Page and Neuringer (1985) concluded that variability in responses in an operant lineage is "a dimension of behavior much like other operant dimensions" (p. 450) in its susceptibility to selection. These and later experiments support that conclusion. And as in other kinds of selection, the susceptibility of variation to selection does not imply that selection is the *source* of the variation. Variants must occur before selection can operate. Page and Neuringer (1985) suggested that variation is an intrinsic property of operant behavior (i.e., has its origin in natural selection); operant selection can dial it up or down.

5.7. Origin of operant lineages

In one sense, as suggested earlier, all behavior has its origin in natural selection or, more proximally, the inherited behavior of individual organisms. In some cases, what is inherited has enough organization to be considered a behavioral lineage. For example, the pecking of pigeons is highly organized, in its formal properties as well as its relation to some properties of the environment, before operant selection begins to adapt features of an individual bird's pecking to local contingencies. Pigeons' pecking is a behavioral lineage that transcends the lifetime of individual pigeons. Its origin is in the history of the species. Operant modifications of the lineage during the lifetime of individual pigeons occur, but they are not encoded in the germline.

Some inherited behavior is not well organized with respect to its environment. Organisms of many species inherit a supply of "uncommitted behavior" (Skinner 1984, p. 219). It is the kind of activity seen when an organism is in an environment that contains few elements with which it has means of interacting. Such behavior is prominent in the repertoires of human infants and can be seen on occasion in human adults (e.g., profoundly retarded adults or adults submerged in water or isolated for a long time in an empty room). The supply of uncommitted behavior is primordial in phenotypic behavioral development. Operant lineages emerge from the primordial behavior of a particular organism when selection is contingent on particular properties of the primordial activity and, as a result, those properties begin to appear more frequently in the behavior stream. If the selection contingencies gradually tighten, a response lineage gradually forms out of the more or less random (or, at least, poorly organized) activity.

Although the emergence of organized activity from undifferentiated movements can be seen to occur in real time, it has been difficult to study it experimentally because of a lack of equipment that allows recording of both the behavior meeting the changing contingencies and the rest of the behavior in the subject's ongoing behavior stream. Pear and Legris (1987) were able to develop a computer program that continuously tracked the position of a pigeon's head. They specified an arbitrary response (not seen previously during extensive observation of the pigeon in the experimental setting) as the experimental target. The pigeon's head was to make contact with a 3-cm diameter virtual sphere at a particular location in the chamber. In addition to its precise spatial location, the form of response to be generated involved a dipping of the pigeon's head at that location. Beginning with a target virtual sphere that the pigeon's head would easily contact, the experimenters gradually increased the frequency of movements making contact with the sphere and then gradually reduced the size of the sphere. As a result of these changing selection contingencies, the movements acquired the target form and occurred in that form at the target location at high frequency. The interactor lineage that emerged in each of the three pigeons' repertoires was maintained by stable reinforcement contingencies thereafter.

Operant lineages that exist in behavioral repertoires do not all arise from primordial behavior. Many operant lineages come into being by the splitting and merging of previously existing lineages. The complex relations currently studied in operant laboratories involve the merging and splitting of operant lineages (see Sidman 1994, for history of one research program). Such behavioral complexity appears to require interactors that include stimulus parameters, interactors called "stimulus control operants" (Ray & Sidman 1970). The appearance of stimulus control operants in an operant repertoire has been likened to the appearance of eukaryotes in biological evolution (Glenn & Madden 1995). They allow for the grouping of responses into interactors having multiple parts and thus the evolution of behavioral complexity during the lifetime of one organism.

5.8. Replication and retention in operant selection

Selection is a two-step process. "A process is a selection process because of the interplay between replication and interaction" (Hull 1981, pp. 40–41). In operant selection, one step is the differential interaction between responses and consequent stimulation (environment) that "causes replication to be differential" (Hull 1989, p. 96). The other step is the differential replication of response characteristics in successive generations. Whether operant selection is a process that parallels natural selection and belongs to the class of theories sometimes called "Darwinian" depends on the requirements one makes of the replication process. If the environment must have multiple and differing copies of a replicator concurrently available for selection to occur, operant behavior seems definitionally excluded. However, there appears to be no reason to assume that all replication processes involve concurrently existing events or objects. All that may be required is a process that retains features of interactors (event or object) across generations in a lineage, with a mechanism of variation to introduce novelty. As we said earlier, successive variations must in some sense be retained and then passed on. This leads to questions regarding the site of retention of operant behavior and the mechanism by which passing on is accomplished.

So far as the material world is concerned, what is left after an operant interactor is gone is the central nervous system of the organism whose operant behavior is adapting to changing local contingencies. So the first step in operant se-

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lection occurs at the behavioral level (at the interface between organism and environment) and entails relations between interactors in a particular operant lineage and a selecting environment. And the second step occurs inside the organism at the neural level. Research on the biochemical mechanisms underlying learning and memory seeks to identify long-lasting changes that must occur in the strength of synapses as learning progresses and the learned behavior is maintained. "The range of possibilities for memory maintenance is large. None of the proposed models have been firmly excluded, and there seems to be no clear candidate" (Lisman & Fallon 1999, p. 340). Full understanding of operant selection requires understanding of the relation between the two steps in the selection process. Because one step occurs at the neurochemical level and the other at the behavioral level, such understanding necessarily entails synthesizing findings from research at these two levels. Unfortunately, researchers working on each of the two subprocesses, like geneticists and evolutionists before the modern synthesis, have little knowledge of one another's findings and often view with suspicion the conceptual framework of the other. There are exceptions. Donahoe and Palmer (1994) have begun to fashion a synthesis of biobehavioral processes in which they view neuroscientific findings in the context of a selectionist theory of learned behavior.

If the site of retention is the central nervous system of the learning organism, understanding of the mechanism(s) of retention will require investigation of changes in the properties (structural or functional) of neural activities as a function of differential interaction between responses and environment. In what follows, we draw on research that explicitly relates replication at the cellular level to operant processes. In a series of publications, Stein and his colleagues set out to assess Skinner's hypothesis that what constitutes a response at the behavioral level may not be that which is strengthened (i.e., replicated) in operant selection. Rather, a response's elements or atoms (i.e., characteristics or traits) are the units of behavior susceptible to operant selection (Skinner 1953, p. 94). No such element can be correlated with a unit of replication smaller than a single neuron, so Stein et al. used in vitro preparations in which single neurons were subjected to analog contingencies of operant selection. For example, Stein, Xue, and Belluzzi (1994) made micropressure administrations of dopamine contingent on spontaneous bursting frequencies of single neurons of a hippocampal slice. They demonstrated that bursting frequencies increased when dopamine (a chemical associated with the reinforcing effect of drugs) was administered contingent on bursting, the frequencies decreased when dopamine was not administered contingent on bursting, and bursting frequencies remained at or below baseline when they no longer administered dopamine independent of bursting (noncontingently).

In another study Stein and Belluzzi (1988) injected microadminstrations of dopamine immediately *after* a postsynaptic neuron was activated by a presynaptic neuron, with a resulting increase in the presynaptic neuron's ability to activate the postsynaptic neuron. Other experiments (Self & Stein 1992) showed that it was not simply the stimulation of cellular activity that explained the effects of the burst-contingent dopamine. As suggested by Donahoe and Palmer (1994, p. 56), the effects of consequent dopamine on the ability of one neuron to activate another "demonstrates that dopamine can modulate the activity produced by glutamate, which is the major excitatory transmitter at synapses in the cerebral cortex, including those in the frontal lobes."

The work of Stein and his colleagues has several implications relevant to our analysis. First, the in vitro preparation demonstrated that the unit of replication is likely to be only a very small part of "complex neuronal circuitry associated with the reinforced response" (Stein et al. 1994, p. 156). A second, related implication is that the combination of cell firings can differ from response to response in a succession of responses of a lineage. Similarly, the combination of genes can differ in a succession of organisms of a lineage. Third, the in vitro preparation removes the operant selection process from any experiential requirements. As Stein et al. (1994, p. 156) put the matter, "presumably, hippocampal slices do not experience 'highs'." Fourth, the replication required for retention of interactor properties in an operant lineage may not require (but would not preclude) retention of a string of chemicals (as in DNA) across successive generations. Retention of operant properties in a lineage may instead be characterized in terms of the probability of a neuron activating other neurons of a pathway resulting in effector activity ("synaptic efficacy"). In vitro cellular analogs of operant conditioning further suggest that contingent reinforcement modifies several dynamical properties of a multi-functional network associated with motor behavior and that antecedent stimulation is not required for operant learning (Nargeot et al. 1997; 1999). Presumably the mechanisms that account for retention of selected firing patterns will be found in the cellular chemistry of the modified network.

Differential interaction of responses and their consequent environments, then, has the effect of altering probabilities of the firing patterns of neurons. Differentially altered probabilities of events that pass on information (in this case, information coding for response properties) may be the hallmark of replication in selection processes. Unfortunately, we know little about the coding of information that is passed on. We hope others will fill this knowledge gap. In sum, differential interaction of operant responses and their consequent environments causes differential replication of the properties of interactors in operant lineages. Researchers who consider themselves to be working in two different scientific domains (the behavioral domain and the neuroscience domain) have studied, respectively, the two steps of operant selection: interaction and replication. If researchers in both domains were to approach their work from a selectionist perspective and seek to synthesize their findings, a unified biobehavioral science of operant behavior would appear possible.

6. Conclusion

The goal of this target article was to present a general account of selection that is adequate for three putative examples of selection processes – gene-based selection in biological evolution, the reaction of the immune system to antigens, and operant learning of individual organisms. After extensive reworking, sometimes generated by disagreements among the three authors of this paper, sometimes the result of two successive sets of referees' reports, we ended up defining selection in terms of repeated cycles of rep-

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lication, variation, and environmental interaction. These three processes must be so structured that environmental interaction causes replication to be differential.

All three systems include variation. However, the respective amount of variation differs from system to system. For example, point mutations are introduced into genomes at very low rates, but these rates must be too high for selection because mechanisms exist that repair them. Enzymes roam up and down strands of DNA, seeking out abnormalities and repairing them. However, in gene-based selection in biology most of the variation is introduced by recombination, not point mutations. Rates of variation are extremely high in the immune system. More than one mechanism exists to make sure that the variation needed for selection is present in ample amounts. How variable operant behavior is depends on how finely we analyze behaviors. The natural units of variation are less obvious with respect to behavior than with respect to the other two systems (Enç 1995).

The most fundamental distinction made in this paper is between passing on information via replication and the biasing of this replication because of environmental interaction. As we have argued at some length, selection is not a single process but composed of two processes – replication and environmental interaction. As a result, the issue of the levels at which selection occurs must be subdivided into two questions: at what levels does replication take place and at what levels does environmental interaction take place? These two questions elicit very different answers, depending on which of the three systems discussed in the paper is at issue. In gene-based selection in biological evolution, replication occurs primarily at the level of the genetic material, while environmental interaction takes place at a wide variety of levels, ranging from genes, cells, and organisms to kinship groups, demes, and possibly entire species. In the development of the immune system, gene-based selection in biological evolution plays the same role as in any other organismic system, but a second sort of selection also occurs. In somatic selection, those cells that specifically recognize a particular pathogen or foreign body respond and undergo extensive mutation and proliferation. Both replication and environmental interaction take place at the cellular level. In operant learning, the relation between an organism's responses and consequent stimulation causally affects the organism's central nervous system and subsequent behavior. The net effect is that some responses increase in frequency and others decrease.

Several problems arise in explicating the notion of replication. Even though the notion of information is fundamental to any account of replication, we do not provide such an analysis in this article. We anticipate that in the future, this need will be fulfilled. In replication the relevant information incorporated into the structure of replicators is passed on to successive generations of replicators. However, the mechanisms responsible for replication differ somewhat. Although the relevant replication in gene-based selection in biological evolution and the reaction of the immune system to antigens takes place at the genetic level, the details of these processes differ. In addition, the distinction between self and nonself that is fundamental in the immune system does not play a corresponding role in the other two selection processes. In gene-based selection in biological evolution and the reaction of the immune system to antigens, genes replicate by splitting and filling in the appropriate nucleotides. Replication takes place in operant learning at the level of neurological processes, the nature of which still remains largely unknown.

Another difference that emerged with respect to these three instances of selection is between linear sequences of replication and their cotemporal proliferation. In operant learning, organisms react to sequences of events that result in cumulative changes – behaviors are reinforced or extinguished. However, in the other two forms of selection, extensive concurrent variations are presented to the environment. Although we think that a multiplicity of cotemporal replicators massively enhances the strength of those selection processes that incorporate such multiplicity, sequences of replicators that do not proliferate in this way also count as instances of selection, at the very least as a limiting case.

Environmental interaction is also necessary for selection. Some entities must interact with their environments so that the replication processes associated with these interactions become differential. Just differential replication alone is not enough for selection, that is, if such processes as drift are to be distinguished from selection. As in the case of information, we were confronted by the problem of distinguishing causal processes from other sorts of processes. In the case of information, none of the current analyses of information make the distinction necessary for selection processes. In the case of causation, too many different analyses of causation exist, and none of them are totally superior to all others for all purposes. In all cases, however, selection consists of successive alternations of replication and environmental interaction.

The most common critical response to this paper will surely be that various authors prefer different versions of the three theories than those that we have investigated; for example, replication occurs at levels higher than the genetic material, the mechanism that we have sketched for distinguishing between self and nonself is inadequate, or they simply do not like operant psychology no matter how it is formulated. But these objections are peripheral to the goal of this article, which is to present a general account of selection that is adequate for the three sorts of theories that we have set out. Alternative versions of these three families of theories count against our analysis only if they cannot be characterized in terms of variation, replication, and environmental interaction.

If the preceding discussion has shown anything, it has been how counterintuitive selection processes actually are. The kind of causality involved in selection processes is, as Skinner (1974) noted, very different from our ordinary conceptions of causation. The two most striking features of selection processes are that they are both incredibly wasteful and yet able to produce genuine novelty and increased levels of organization. Given our ordinary notions, we might be led to ask how such wasteful systems can produce both novelty and increased organization. We suspect that selection processes are able to produce genuine novelty and organization only because they are so incredibly wasteful. The efficient production of novelty and order may not sound like an oxymoron, but we suspect that it is.

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Two stumbling blocks to a general account of selection: Replication and information

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Abstract: When one takes the evolution of operant behavior as prototype, one sees that the term replication is too tied to the peculiarities of genetic evolution. A more general term is recurrence. The important problem raised by recurrence is not "information" but relationship: deciding when two occurrences belong to the same lineage. That is solved by looking at common environmental effects.

The authors have made some good progress toward abstracting the concept of General Evolutionary Process. Particularly constructive are their emphasis on the iterative nature of the process of selection and their separation of interaction from replication. They are right also in their implication that part of the importance of this project lies in clarifying selection as a type of causality and as a valid basis for explanation (Baum & Heath 1992). The notion of operant behavior has been resisted by laypeople and scientists alike for the same reasons that evolutionary theory has been resisted: (1) an enormous preference for immediate ("push") causes, even if they have to be imagined, and (2) the implied rejection of cherished imaginary immediate causes such as agency, will, purpose, and intelligence. I suggest, however, that the authors might have made more progress had they considered operant evolution (i.e., shaping) as a prototype, instead of genetic evolution.

In attempting to abstract the General Evolutionary Process, no necessity requires that genetic evolution be taken as the prototype. It has the advantages that it is widely accepted among the scientific community and that its mechanisms are partially understood, but it has the disadvantage that its peculiarities are easily taken for necessary attributes. If instead one takes operant evolution as prototype, at least two issues are clarified: the term *replication* and the concept of *information.*

Replication. Even if it is true that DNA is in a sense "copied" (and the facts of recombination make this doubtful), in no useful sense are repeated occurrences of a behavioral pattern copies. If I brush my teeth every night before I go to bed, in no sense is my brushing one night a replica of my brushing the night before. Even though we are ignorant of the way the workings of the brain affect behavior, nothing we know suggests there might be a replica or representation of tooth-brushing somewhere in the brain either. Rather, as with other natural events, such as sunrise, hurricanes, birth, and death, the event occurs when the conditions are right (bedtime, bathroom, toothbrush, toothpaste, and so on). (The historical origins of tooth-brushing in a history of reinforcement and punishment – that is, by iterative selection – are another matter, of course.)

A more neutral term would be *recurrence,* meaning just "occurring again" or "turning up repeatedly." Replication would be just one type of recurrence – recurrence by copying. Other mechanisms of recurrence may be imagined; for operant behavior, we have the effects of context and cues known as stimulus control. Thus, one need not search for some sort of copying when talking about the recurrence of behavior. In particular, one need not talk nonsensically about things like "memes" "jumping from brain to brain" when talking about the spread of a behavioral pattern within a cultural group (Baum 2000; Dawkins 1989a).

Information. The authors assert, "the notion of 'information' is fundamental to any account of replication" and "In replication the relevant information incorporated into the structure of replicators is 'passed on' to successive generations of replicators." Even if these statements were true of replication, they are irrelevant to the more general idea of recurrence. The authors' reliance on the notion of replication leads them to misstate the important issue involved, which is about relationship. Even if the structure of replicators is passed on, for purposes of evolutionary theory, the problem is not pondering the "information incorporated"; the problem is deciding when two sequential occurrences belong to the same lineage.

The short answer would be that two occurrences belong to the same lineage if they share common ancestry (Ghiselin 1997). This, however, raises the question of defining "ancestry" in general terms. Here again, the example of operant evolution sheds light, because it leads us to see that "common ancestry" means common history of selection. My tooth-brushings Monday night and Tuesday night belong to the same lineage, not because of any "information incorporated," but because they may be attributed to a common origin – say, childhood training (i.e., exhortations from my parents and dentist, approval and disapproval, cavities or the lack of them, and so on; Baum 2000). The common origin and common history of selection, however, depend on common environmental effects. Both the origin of my tooth-brushing (exhortations, etc.) and the selection of my tooth-brushing depended on past effects of contributing to dental hygiene. Thus, common "ancestry" for behavior comes down to a common basis for selection or common history of environmental effects. In more abstract terms, two occurrences belong to the same lineage if they are attributed to the same history of interaction with the environment.

Dawkins (1989b) makes a parallel point for genetic evolution when he explains that the genes that promote dam-construction in beavers were selected by their effects on the beavers' environment. Indeed the genes "for" dam-construction are defined by those effects, for they are nowhere apparent in the structure of the beavers' DNA. Although the idea that genes influence behavior is widely accepted, the content of this idea differs little from the wide acceptance that the brain influences behavior. Almost nothing is known of how this occurs. If we had to rely on examination of structures in DNA or the brain to define the units of recurrence, we would be in deep trouble. But defining the recurring units in terms of their common environmental effects solves this otherwise intractable problem. Instead of "information," environmental effects turn out to be the key to defining lineages. I doubt one would recognize this without considering the evolution of behavior, whether across generations or within a lifetime.

Should we essentially ignore the role of stimuli in a general account of operant selection?

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Abstract: The selectionist account of behavior is actually a focused discussion of operant selection. To this end, the authors essentially exclude stimuli from their analysis. This exclusion is inconsistent with the importance placed on environmental interaction in their general account. Fur-

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ther, this exclusion limits the generality of their account by missing important sources of stimulus-elicited behavior (e.g., classical conditioning).

In the target article, Hull et al. clearly accomplish one of their goals. That is, they successfully describe individual, immune, and operant selection by repetition of three processes: variation, replication, and environmental interaction. Further, they provide the reader with much to think about. What is variation within each selection system? How does the repetition of the processes generate the variation and why is the variation differentially constrained? What retention mechanisms are required to retain past selected information for future selection? How can environmental interaction affect this retention?

Although I found the target article thought provoking, I was disappointed in the false advertisement of the title. I was expecting a "general account of selection" for behavior. However, they only provide a selection account of a narrowly defined sub-set of behavior. That is, the authors chose to emphasize "operant learning." I appreciate this choice given the daunting goal they set for themselves. Also, operant conditioning is a natural choice given that a selectionist framework has been more readily applied to research in that area. Perhaps my disappointment was greatest by their choice to seemingly ignore the role of stimuli in the generation of what the authors term "responses" or "interactors." This choice is made explicit when the authors state "In operant theory, activity designated as a 'response' does not require a stimulus." In the following paragraphs, I would like to note why I believe a general selectionist model of behavior (versus "operant theory") should take into consideration stimuli.

I will use the bar-press example employed by the authors as a starting point for discussing the importance of stimuli in a good selectionist model of behavior. The bar-press response would not be possible without the presence of the metal lever (bar) that extends into the experimental apparatus. This bar is a complex multi-modal stimulus likely including visual, olfactory, spatial, and tactile elements. One would be hard pressed to argue that the stimulus elements associated with the bar do not set the occasion, in part, for the response of depressing the lever. Other stimuli that will contribute to the pattern of responding include passage of time and environment (context).

Examination of response records of a well-trained animal on a schedule of reinforcement (e.g., FR) will reveal surprising variability in response generation. Such measures as initial response latency, inter-response time, and time to termination response will vary within a range (see Fig. 1 of target article). What are the sources of this variability? One answer to this question is that different inter-response times, and so on, were reinforced (i.e., selected by the consequence). Albeit likely, this answer is only partially satisfying. Direct observation of a rat receiving food reinforcement will reveal the development of interesting behaviors that compete with the bar-press response. These competing behaviors likely contribute to the variability. For instance, even though food has not been delivered, the rat will frequently move away from the bar and toward the food delivery area often sniffing, licking, and chewing the food cup or dipper entry. Clearly, this pattern of behaviors has been reinforced. However, why does this behavior occur in the food area and the not in the rear left corner of the apparatus? Those stimuli in the food delivery area are closely associated (temporally and spatially) with food.

Similarly, later in operant training sniffing, licking, and chewing type behaviors are also directed toward the bar. Students of operant conditioning will recognize this phenomenon as similar to that observed by Breland and Breland (1961). That is, stimuli closely associated with food (the metal bar in this example) will come to control food-related behaviors. These behaviors are conceptualized as evolutionarily selected response-tendencies to stimuli that have acquired motivational (appetitive in this example) value (e.g., Bolles 1975). To me, the present observations indicate that stimulus – outcome selection (Pavlovian/classical conditioning) likely occurs along with operant selection – regardless of the experimenter's intention. What sparse systematic research exists tends to support this notion. For example, Shapiro (1960) trained dogs to bar-press on a FI 2-min schedule of food reinforcement. In addition to measuring bar-pressing, the dogs were surgically prepared so that salivation could be monitored. As expected, bar-pressing was infrequent early in the interval and then increased as time to feed approached. Of interest to the present discussion was that salivation showed a similar pattern. This result has been taken to indicate that the cues associated with bar-pressing and passage of time acquire to ability to elicit salivation via stimulus-outcome selection (Donahoe & Palmer 1994; Kintsch & Witte 1962).

Hull et al. note the necessity of variability in a general selection account. I fully agree with this conclusion. One main point of the above discussion is to emphasize the importance of stimuli in the generation of variability. Responses do not occur in the absence of stimuli. Some of this stimulus-elicited variability is the result of "gene-based" selection; other variability is acquired during behavioral selection in the broad sense of the term. Along similar lines, the authors of the target article emphasize the importance of "environmental interaction" in a general account of selection. The very nature of the concept requires the object of selection ("responses" or "interactors" in the case of operant selection) to be affected by the stimulus conditions that define the environment. Indeed, in section 5.3, the authors briefly mention this fact by noting that "events" can have "discriminative, conditional, or motivating functions."

To close this commentary I would like to applaud the authors' emphasis on the importance of elucidating the physiological mechanism responsible for retention processes. This discussion would be further enriched by including stimuli into a selection account of behavior. This is especially true for stimulus-outcome selection (Pavlovian/classical conditioning). There are numerous well-studied in vivo and in vitro models of neural plasticity for classical conditioning (e.g., Boa et al. 1998; LeDoux 2000; Steinmetz 2000).

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A single-process learning theory

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Abstract: Many analogies exist between the process of evolution by natural selection and of learning by reinforcement and punishment. A full extension of the evolutionary analogy to learning to include analogues of the fitness, genotype, development, environmental influences, and phenotype concepts makes possible a single theory of the learning process able to encompass all of the elementary procedures known to yield learning.

The article by Hull et al. on the roles of variation, replication, and environmental interaction in selection processes hopefully will stand as a beacon in a long but only sporadically-connected line of works on the similarity between evolution by natural selection and certain physiological processes. The response to an article on learning and the evolutionary analogy I once wrote from my PhD. dissertation (Blute 1977, see http://cogprints.soton.ac.uk/) was either incomprehension or a rejection of all general learning theory including the utility of the analogy. Today, however, more psychologists understand evolutionary theory and they understand that something which evolves and is inherited necessarily also develops and functions physiologically.

Some elementary analogies between learning and evolution are obvious to those acquainted with both theories. Both are based on populational thinking. Reinforcement and punishment play the

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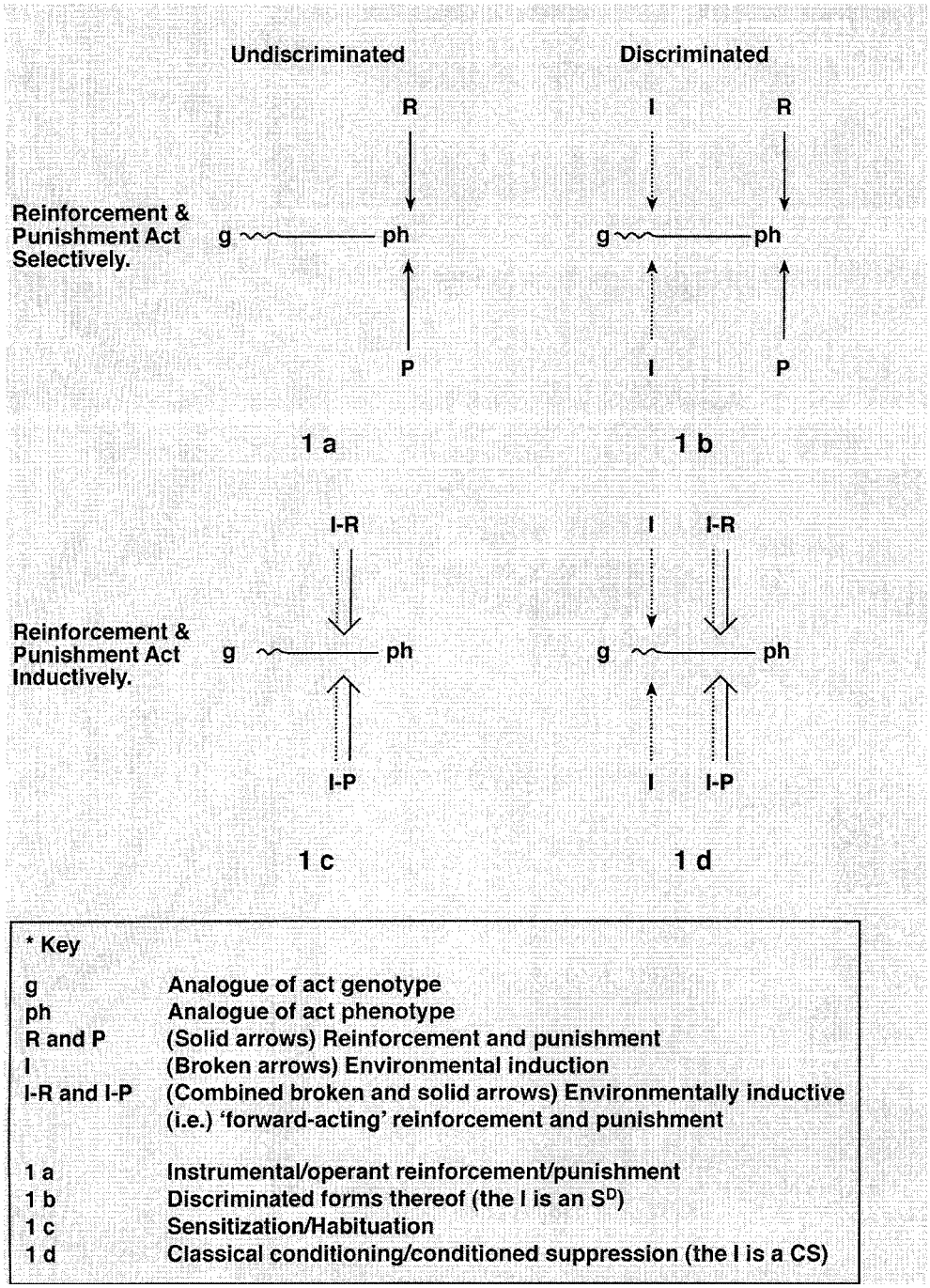


Figure 1 (Blute). Relationship among learning procedures as aspects of an evolution-like process.

role of natural selection in learning. Herrnstein's original matching law of learning is analogous to the Hardy-Weinberg equilibrium principle of population genetics – both being inertial principles similar to Newton's first law of motion. As he subsequently pointed out however, negative frequency-dependence also often obtains in both realms (Herrnstein 1989). Shaping, in which a new response group (e.g., stick-fetching in a dog) "evolves" from a previous one (e.g., orienting responses in the direction of the throw) is analogous to anagenesis (evolution in a lineage), whereas response differentiation in which more than one new response group (e.g., left and right turns in a t-maze) "evolve" from a previous one (e.g., milling around in the long arm of the maze) is analogous to cladogenesis (branching of a lineage). Although the themes of mutation and recombination are familiar, innovation by analogy in the ordinary language sense is widespread in both processes – in evolution genes are duplicated and diverge to function in new contexts. Herbert Simon (1962) was right that any selection process should show an equivalent of the biogenetic law. In learning, it is the gradient of reinforcement or of temporal discrimination (in frequency terms, the acceleration in responding within intervals on fixed interval schedules and, patterned differently, within the series of responses bounded by successive reinforcers on fixed ratio schedules). This "more change later than earlier within units subject to selection" principle may warrant a second look by psychologists now that biologists have found it does not apply to the very earliest stage (for a review see Hall 1997). The (limited) equifinality of learned acts (as first demonstrated by Tolman's place learning experiments) corresponds to regula-

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tive development, reaction norms or phenotypic plasticity in evolution demonstrating that in both processes it is "cognitive" programmes or algorithms that are learned/evolve including routines, sub-routines, sub-sub-routines, and so on. In evolution this is achieved through a complex hierarchy of controls on gene expression.

I agree with Hull et al. that the importance of the evolution-learning analogy – or as they prefer, of a general account (theory?) of selection processes – is its scientific usefulness. Biologists refer to the flip side of the coin of selection as adaptedness or fitness and distinguish between genotypes and phenotypes with a development process also subject to environmental influences linking the two. Learning theorists might find it useful to refer to the flip side of reinforcement as competence (already widespread in some literatures) – a competent act simply being one that has the properties required for reinforcement in a particular situation. (For a multitude of reasons what is biologically adaptive is not always competent and vice versa.) They may wish to consider the possibility that all acts possess an analogue of the genotype (e.g., endogenous firing of one or more pacemaker neurons or coupled groups, i.e., central pattern generating networks), development (initial firings activate other neurons), environmental influences (firings of later neurons in the pathway require "horizontal" environmental input as well), and phenotype (muscular or other act topography) distinctions. The same thing could be achieved without a sequence of cells if the initiating neuron(s) possessed an endogenous rhythm but also required particular synaptic input at a specific stage(s) of the cycle. In this view, a respondent is an act whose endogenous rhythm is rapid but which requires a highly specific set of inductive circumstances for its development – together creating an appearance of pure environmental elicitation. An operant is an act whose endogenous rhythm is slow but which is broadly tolerant of a diversity of inductive circumstances – together creating an appearance of pure voluntary emission. Given these distinctions, because in evolution environmentally-inductive ("forward-acting") influences may at the same time be selection pressures – permitting or preventing development from proceeding and altering its course in an adaptive or maladaptive direction (e.g., normal maternal influences versus thalidomide on the development of a mammalian foetus), a single-process learning theory capable of encompassing not only operant learning, but all the elementary procedures known to yield learning is possible (see Fig. 1).

A neural-network interpretation of selection in learning and behavior

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Abstract: In their account of learning and behavior, the authors define an interactor as emitted behavior that operates on the environment, which excludes Pavlovian learning. A unified neural-network account of the operant-Pavlovian dichotomy favors interpreting neurons as interactors and synaptic efficacies as replicators. The latter interpretation implies that single-synapse change is inherently Lamarckian.

In their account of learning and behavior, the authors define an interactor as emitted behavior that "operates on the environment." This definition excludes Pavlovian learning, where behavior is typically not emitted and does not operate on the environment. A more inclusive account arises from the neural-network model by Donahoe et al. (1993; for further research, see Burgos 1997; Burgos & Donahoe 2000; Donahoe & Burgos 1999; 2000; Donahoe & Palmer 1994; Donahoe et al. 1997). My use of this model here represents primarily a thought-experiment, interpretative tool, not a claim on the details of the structure and functioning of actual nervous systems. Following Dawkins (1982), "[t]hought experiments are not supposed to be realistic. They are supposed to clarify our thinking about reality" (p. 4). Although we care about neural plausibility, a proper theoretical understanding of the brain-behavior nexus lies in the future. Meanwhile, we can benefit from the precision allowed by artificial systems to "clarify our thinking" about selection in learning and behavior.

A unified selectionist account of Pavlovian and operant learning is possible only by viewing this dichotomy as nonfundamental. In our model, "Pavlovian" and "operant" name *procedures* that reveal different aspects of a single process ("conditioning"). Figure 1 shows a typical neural network. Circles represent abstract neurons (or neuronal microcircuits), whose states are represented by *activations.* Activation of input units (small circles) represents antecedent exteroceptive stimulation. Output activations represent behavior. Hidden elements mediate the environment-behavior relations. Lines represent preexisting synaptic connections (thin lines represent initially-weak plastic connections; thick lines represent maximally-strong, nonplastic connections). Connection strengths (efficacies) are represented by weights. An element's activation increases with its presynaptic activation(s) and/or corresponding weight(s). Activations and weights are real numbers from 0 to 1.

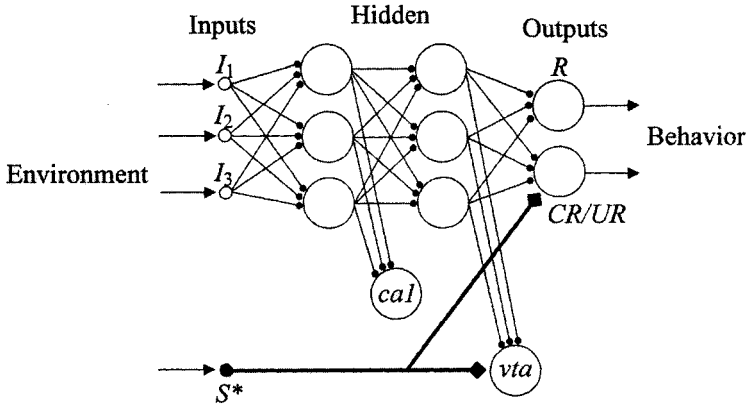


Figure 1 (Burgos). A typical neural network. Neurons are represented by circles and synaptic connections are represented by lines. See text for details.

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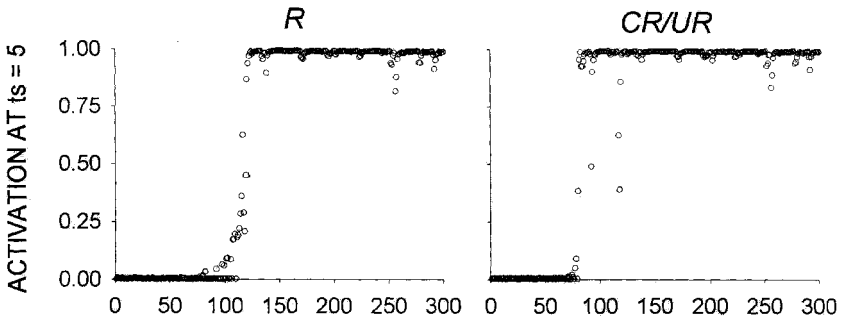


Figure 2 (Burgos). Activation of elements *R* and *CR* at ts = 5, as a function of trials.

Inputs consist of three primary-sensory (I) units and one unconditioned-reinforcement (*S**) unit. Above-zero *S** activations cause identical CR/UR activations that simulate unconditioned responses (*URs*). Above-zero I activations cause (through the hidden elements) CR/UR activations that simulate conditioned responses (CRs). The latter activations increase once the relevant weights have increased through reinforcement. This occurs according to a learning algorithm, defined as $w(i,j,t) = w(i,j,t-1) + \Delta w(i,j,t)$, where w denotes a weight, t denotes a moment in time, i denotes a presynaptic process, j denotes a postsynaptic process, and Δw denotes the magnitude of weight change. This magnitude increases with the pre- and postsynaptic activations, the temporal discrepancies in the activations of ca1 (cornu Ammonis 1) and vta (ventral tegmental area, which can also be unconditionally activated by *S**), and the total amount of weight available for allocation.

In contrast, *S** cannot activate *R*. Hence, no *R* activation is elicited, at least in the strict sense of the term (i.e., direct, unconditional, strong activation by *S**). However, *R*'s activation will depend on some antecedent stimulation, insofar as the relevant weights are larger than zero and *R* is in the pathway of any activated I unit(s). And at least one *I* unit must be activated, if we assume the network not to be in a vacuum. Spontaneous activation of *R* thus plays a minor functional role in simulations based on this model. Although isolated neural elements can be spontaneously activated, this capacity is rarely realized in output elements constituting a network that is supposedly immersed in an environment. Similarly, neurons that constitute in vitro neuronal circuits can be spontaneously activated (e.g., Stein et al. 1993). The present model, however, suggests that this capacity plays a minor functional role in learning-dependent neuronal changes in whole organisms that are immersed in an environment.

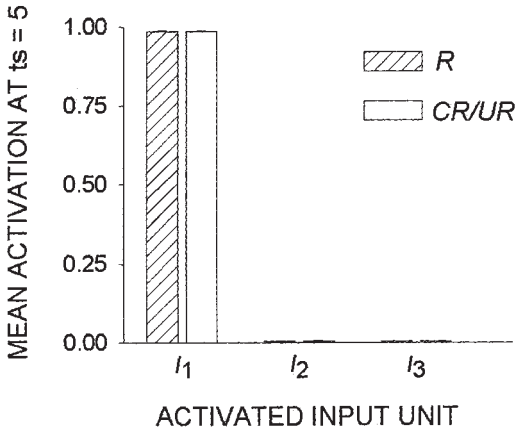


Figure 3 (Burgos). Mean activations of *R* and *CR/UR* at ts = 5 during extinction, where each input unit was activated for 25 trials. Activations of the different input units were randomly interspersed.

All initial weights were set to .01, allowing *I* activations to cause near- but above-zero initial activations in all elements. Training consisted in 300 trials where I_1 was maximally activated for 6 timesteps (ts). At ts = 6, *S** was also maximally activated (dominating over I_1 in activating CR/UR), if and only if *R*'s activation was above zero. Reinforcement caused large positive temporal discrepancies in the *vta* element's activation, which caused the relevant weights to increase across trials, allowing all element activations to increase to near-maximum levels. Figure 2 shows the activation increase for each output element, in the presence of I_1. In an extinction test, each input unit was activated for 25 trials (each unit's activations were randomly interspersed). Figure 3 shows that both output elements were strongly activated only by I_1.

In this simulation, responding to antecedent stimulation was the primary kind of environment-behavior interaction, which subsumed operating on the environment. In this view, interactors can be interpreted as the structures that implemented interaction. Interpreting output activations as interactors confuses function with structure, interacting with interactor. The present network implemented interactions involving direct activation by the environment (input units), direct activation of postsynaptic processes (input units and hidden elements), direct activation by presynaptic processes (hidden and output elements), and operating on the environment (*R* element). *R*, however, could have not operated on the environment, in which case an increase in its activation would have simulated a kind of superstitious learning (see Burgos 2000).

Regarding replicators, the authors' proposal favors an interpretation of weights as replicators. Learning in our simulations, however, does not require variation among initial weights. Synaptic-efficacy variation in real nervous systems thus may not be a condition for learning-dependent differential synaptic strengthening. In the simulation, efficacy variation was an outcome of, rather than a condition for learning. Synaptic efficacies thus may not be good candidates for replicators. Moreover, if replication is viewed as weight increase, the interpretation becomes more akin to Lamarckian evolution. Weight increase is a process of descent with modification, since weights at t are weights at $t-1$ modified by Δw. However, weights at $t-1$ are acquired properties. This outcome obtains in any cumulative conceptualization of synaptic change, which underlies most neural-network models. Either such conceptualization is fundamentally wrong, or single-synapse change is inherently Lamarckian. Still, learning at the network level involved selective change and stabilization of available connections for permitting certain input-output relations (cf. Changeux & Danchin 1976; Edelman 1987).

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Selection as a cause versus the causes of selection

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Abstract: Hull et al. rightly point out the special character of selection as a causal mode, but ironically they seem to force selection back into traditional causal modes by decomposing it into replication, variation, and environmental interaction. Many processes are selective, and a taxonomy of a broad range of kinds of selection may be preferable to narrowing the applicability of the term.

Imagine an astronomical variety of selection. A vast number of particles have scattered in the vicinity of a large planet, perhaps as a consequence of some catastrophic event such as a collision between a satellite and a body from outside the planetary system. Over time, many particles escape the planet or fall into it. Eventually those remaining form rings, like those of Saturn (other large planets in our solar system also have rings, though much more attenuated than Saturn's). Not being an astronomer, I make no claim for the accuracy or feasibility of my example, but formally similar examples can be hypothesized for other physical systems (e.g., in acoustics).

The planetary environment of our hypothetical example can be thought of as a selective system within which gravitational interactions select particles in certain orbits. Debates could then arise over whether the interactions among particles should be conceived of as engendering a sort of group selection, or whether the system selected particles or orbits (in the sense that some orbits reproduce themselves whereas others do not). Analogies might be drawn between the interactions in that variety of selection and those between each organism and the other members of its own species. In our hypothetical case and in natural phylogenic selection as it has unfolded on our planet, the other members of the population constitute part of the environments of each of its members.

This example includes replication in the cyclic character of the orbits within the rings, variation in the starting population of particles and those that may later enter the system, and environmental interactions of the particles interact with each other and with the gravitational field of the planet they circle. Yet I suspect that Hull et al. would object to this example of selection as relevant to their case, which they seem to want to limit to selection as it operates in living systems. Their introduction explicitly excludes from the category of selection processes "more global phenomena, such as the persistence of patterns." Yet what are genes and antibodies and operants if not persistent patterns?

If I misunderstand, I hope Hull et al. will clarify their point. If not, then I must argue against the restrictive view, because the properties of one selective system can sometimes provide useful suggestions about what to look for in others. Much can be learned from explicitly comparing the properties of operant selection and phylogenic selection (e.g., Catania 1973; 1978; 1987; 1995; Catania & Harnad 1984; 1988; Skinner 1953; 1981; Smith 1986; Sober 1984). For example, both artificial and natural selection played important roles in the history of Darwin's account, and both have parallels in operant selection, which can occur in either artificial environments or natural ones. As for Darwin, the controversy lies not with the artificial cases but rather with the extent to which operant selection occurs in natural environments (my bet is that the issue will eventually be resolved in the same way as Darwin's, in favor of a ubiquitous role for operant selection within natural ontogenic environments).

The organismic focus of Hull et al. leads them to assert that operant behavior selected in the interactions of an organism with its environment cannot be passed on to other organisms. Yet patterns of behavior are selected as they are passed from one organism to another. Darwin acknowledged such selection when he discussed language evolution (Darwin 1871; cf. Catania 1994). Dawkins (1976) dubbed such patterns memes, and Blackmore (1999) explored the implications of what has come to be called memetics or memetic selection. That vocabulary has been passed on (has itself become a meme). In Dawkins and in Blackmore, one challenge is to say what is selected in memetic selection. Skinner (1981) addressed similar issues in his concept of cultural selection; in his account what is selected is behavior. The treatment by Hull et al. of selection as it operates in immunological systems is welcome, but their explicit omission of memetic or cultural selection is simply baffling.

In their introduction, Hull et al. rightly identify selection as a counter-intuitive causal mode. Populations remaining after long periods of selection may include few if any traces of those that did not survive. Yet the irony is that Hull et al.'s objective seems to be to force selection into more traditional causal modes. Their descriptions of the components of selection characterize continuities as discrete steps. For example, in defining selection they speak of "repeated cycles of replication, variation, and environmental interaction," when each component exists at every instant in their systems. In phylogenic selection, for example, some organisms are reproducing while others are dying and the environmental interaction is continuous. They continue with "environmental interaction causes replication to be differential," and though here they speak in traditional causal terms, I take it as significant that they rarely speak of selection itself as causal.

Instead of trying to break selection down into components, I would prefer to start with systems of selection in the broadest possible terms, and then to move toward a taxonomy of selective systems with the sorting depending on a number of different dimensions. The structural properties of both what is selected and what does the selecting are obvious candidates, especially because selection is most likely to be misunderstood when either or both are not explicitly identified. For example, in discussing operant selection as the selection of responses by environmental contingencies, students often take a long time to appreciate how different this formulation is from one that says the organism selects the responses it makes in a given environment.

Another dimension along which systems might be classified is mechanism, illustrated by Hull et al.'s appeal to neural processes in operant selection as an analog of genetic processes in phylogenic selection. Yet Darwinian natural selection was identified before its mechanisms were elucidated in genetic terms in the modern synthesis (Bowler 1983; Mayr 1982), just as operant selection is readily identifiable even though its neural basis has yet to be determined. Immunological systems are clearly selective, but Hull et al. have not helped me to see how immunological systems should be related to other varieties of selection in living systems. My guess is that structural criteria for distinguishing among systems of selection such as locus (e.g., within species, within the bodies of individual organisms, within environments during individual lifetimes, within cultures) will eventually turn out to be less important than functional ones such as whether selection operates relative to the mean for a population or on the basis of absolute features (the former but not the latter guarantees continued evolution).

Does terminology from biology work in the realm of operant behaviour?

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Abstract: The authors note that theoretical revision is likely to be required to refine and clarify a general theory of selection so that it is capable of accounting for selection processes across different subject matters. Some aspects of conceptual language from the biological realm work across all

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three domains. However, terminological revision may be required where concepts of "replication" and "information" pose particular problems for the general theory as that is applied to operant research.

The thrust of the target article, that the causal mode of selection operates in the three domains in question, is difficult to dispute since copious experimental evidence is available in all three areas. Although they came to the principle later than biologists, workers in the operant tradition have long argued that selection is the key causal mode in the shaping and maintenance of behaviour, and a detailed attempt to situate the operant tradition within a wider biological framework is a welcome one. To the larger community, operant research still tends to be viewed as a branch of the discipline of psychology, but the conceptual and experimental gap between the two is now so wide that this relationship is becoming ever less tenable. Research in the field of psychology continues to concentrate on brief episodes in the life of organisms, a type of research that obscures the selective effect of interaction between behaviour and the environmental effects it contacts.

Selection is indeed, as the authors note, an unusual form of causation. It became visible in the biological realm when evolution scientists demonstrated the effects over time of interaction between features of organisms and their environments, and it only became visible in the realm of behaviour when operant researchers developed methods for studying behaviour patterns over long periods of time. The authors note that theoretical revision is likely to be required and it may turn out to be required more by the realm of operant selection than by the other two domains under consideration because of the nature of the subject matter and the tendency to adopt biological language for a general theory of selection.

Perhaps because terminology such as population, replication, information, variation, lineage, and so on is encountered more frequently in the biological realm, it seems more comfortable when applied to that realm. It is not clear yet that all of the terminology from the biological domain adequately or correctly describes selection processes as they occur in the realm of operant behaviour. Some of the terminology works equally well across the three domains. "Variation" and "environmental interaction," for example, do the job of describing the subject matter in each of the three domains. "Population" is somewhat more problematic when applied to operant behaviour in that it clashes with the biological notion of "things" that make up a population. In the study of operant behaviour, as the authors note, the organism that behaves fades into the conceptual background and what is left, or what comes to the conceptual fore, is the relation between behaviour and the environment it contacts. "Population" in this case is said to be a population of acts, transient and ephemeral events rather than ontological constructs, and this can be difficult to conceptualise.

More problematic theoretical terms in the general account of selection are "replication" and its attendant notion of "passing on information." Again, these are not uncomfortable concepts when applied to the biological realm because in that realm it is structures that are replicated and that convey biological information from one generation to another. Trying to apply those terms to the selection of operant behaviour begs questions such as "what is it that is replicated?", "what is the information that is passed on?", and even "passed on from what to what, or from where to where?". These questions arise for the same reason as the discomfort over "populations of acts" arises; because what is replicated (or repeated) has a different ontological status from constructs in the biological realm.

It may be that the terminological revision required is as simple as replacing "replicated" with "repeated" which, in the operant realm, can convey the fact that acts are repeated without requiring the attendant notion of information carried from one generation to another. However, it may not be so simple if the processes involved in passing on information are to remain as key concepts in a general theory of selection.

Heeding Darwin but ignoring Bernard: External behaviors are not selected, internal goals are

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Abstract: Hull et al. see responses and properties of responses as units of selection in behavioral change. However, this perspective cannot account for goal-directed behavior in which organisms employ variable means to reliably attain intended consequences. An alternative perspective is offered in which the intended consequences (goals) of behavior serve as the units of selection in behavior change.

I applaud Hull et al.'s efforts to provide a general account of selection applicable to the adaptive complexity that arises in biological evolution, the immune response, and behavior. The recognition that processes of iterative variation and selection may be involved in these (and perhaps all broadly conceived) adaptive phenomena is, I believe, one of the key insights of the twentieth-century that will continue to pay significant dividends in many fields of science throughout the twenty-first century (see Cziko 1995).

Other than to remark that I found their discussion of biological evolution and immunology of considerable interest, I will leave it to experts in these fields to comment on these parts of Hull et al.'s article. Instead, I will limit my remarks to their application of selection to behavior.

While I am convinced that a selectionist perspective has much to offer our understanding of behavior and learning, there is a serious limitation in Hull et al.'s application of selectionism to behavior, namely, seeing responses and properties of responses as the units of selection. The problem with this conceptualization is that in a natural environment, behavior that is to be reliably successful in bringing about a certain consequence must vary in order to compensate for unpredictable environmental disturbances. This characteristic of living behavior was noted by James (1890) who described the striking "varying means" that humans and animals employ to "fixed ends" (p. 4). Many fascinating examples of variability in the goal-directed behavior of insects in natural settings can be found in Russell (1945). But the very use of the word "response" suggests that behavior does not and cannot vary in this way because it views behavior at the end of a one-way causal chain running from environmental input to computed behavioral output.

Take, for example, Hull et al.'s discussion of force as a property of operant response that is subject to selection. Their stated assumption is that rewarding responses with force values between x and y will increase the frequency of responses with these force values while unrewarded responses having values outside this range will decrease in frequency. Although Hull et al. present this as a hypothetical example, such experimental results have been obtained for the rat by Mitchell and Brener (1991) and Slifkin et al. (1995).

But a more interesting experiment would be to vary, for each lever press attempt, the amount of force needed to depress the lever (and thereby obtain food) from values below x through those above y. Although I know of no such experimental studies, observations of animals in natural settings suggests that a rat that had learned to depress a lever to obtain food would vary the force used accordingly, using only as much force as needed (or just a bit more), assuming that feedback was provided to the rat during the behavior (such as a click) indicating when enough force had been applied. In other words, the animal would vary the force of its responses from one lever press to the next to reliably produce a desired result. An analogous situation is presented to you every time you enter a building with a manually operated door as you apply as much force as necessary to open it within a certain amount of time (usually within a second or two). While you may occasionally

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find a door so easy to push or pull that you fling it open, or a door with so much resistance that your initial attempt fails budge it, for a very wide range of resistances the time taken to open a door shows little variability while the force you apply shows much more variability.

This variability of goal-directed behavior cannot be accounted for by any theory that sees specific responses or properties of responses as the unit of selection in learning. Fortunately, an alternative perspective exists for understanding behavior and its adaptive modification that takes into account the goal-directed and variable nature of behavior. This perspective began with French physiologist Claude Bernard, a contemporary of Darwin who understood physiological processes as an organism's means of maintaining a constant internal environment in spite of unpredictable and varied environmental conditions. It was further developed in turn by Cannon's (1939) notion of homeostasis; the cyberneticians Rosenblueth et al. (1943; see also Wiener 1961); and most recently by Powers and his associates (see Cziko 2000; Marken 1992; Powers 1973; 1989; 1992; 1997; Runkel 1990) who have developed, described, and applied what they term perceptual control theory (PCT) to model and explain purposeful behavior. PCT employs circular causality working in a closed loop to model behavior as the means by which an organism controls its environment (or more precisely, its perception of selected aspects of its environment), and not as responses or properties of responses that are caused by the environment (see Fig. 1).

While is impossible in the space allotted for this commentary to provide a useful introduction to PCT, I will mention that PCT provides both a working model of the variability and effectiveness of goal-directed behavior. It also provides a selectionist account of learning by employing circular causality and by regarding intended outcomes (perceptual reference levels) as the units of selection, not responses or properties of responses. I refer interested readers to Runkel (1990), Powers (1997), and Cziko (1995; 2000; both books accessible in entirety via my Website) for introductions to PCT and to Powers (1973; 1989; 1992) and Marken (1992) for more technical accounts and relevant experimental studies. Computer simulations and demonstrations are available on the Web at www.ed.uiuc.edu/csg.

Hull et al.'s article is a valuable contribution to a general selectionist conceptualization of adaptive processes. But in order to account for the goal-directed nature of animal and human behavior, the one-way causal view of stimulus-(cognition)-response must be replaced by circular causality in which organisms vary their behavior in order to control its consequences. Combining Bernard's lesson which that of Darwin's provides a new view of behavior in which internal goals are varied and selectively retained, not external behaviors.

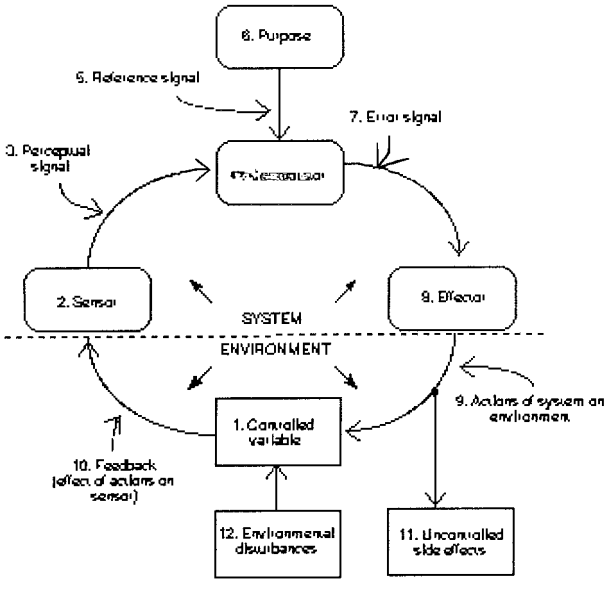


Figure 1 (Cziko). An elementary closed-loop control system that varies its output (behavior) in order to control its input (perception). Learning can as a result of variation and selection of the reference level (intended perception) by a higher-level control system.

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Selection and the unification of science

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Abstract: Selection in behavior analysis fits the criteria of replication, variation and interaction proposed by the authors except for information under replication. If information requires physical structure, behavior analysis does not fit that model because functional analysis may provide parallels between behavior, neurology, and biochemistry but not sequencing. The three sciences are not unified by the model but another is available.

These comments may apply only to the third of the three sciences represented in the target article, namely, operant behavior, conditioning, or learning otherwise known as the science of behavior. I do not consider myself sufficiently well-versed in either biology or immunology to contribute anything of importance to those fields although some of what I say may be relevant to them.

With respect to the question of whether operant conditioning can be considered to be a matter of selection, Glenn appears to have made a strong case at least with regard to the criteria of replication, variation, and interaction. However, it may not fare very well on the issue of information as a sub-category of replication.

I am reminded at this point of B. F. Skinner's (1974, p. 84; 1969, p. 106) statement that "a person is changed" when a behavior has been changed by its consequences. He did not go on to speculate about how the person might have been changed when the behavior was changed by those consequences. Indeed, he warned against such practices a number of different times but especially in "*Are theories of learning necessary*?" (cf., Skinner 1950).

Glenn suggests that the current research of Stein et al. (1994) as well as Stein and Belluzzi (1988) and Self and Stein (1992) may tell us how the person is changed when the behavior is changed by its consequences. However, their analyses still only demonstrate that, when behavior changes, other changes also occur at the neuronal and, perhaps, biochemical levels. To her credit, she does not suggest that those changes, in turn, explain the changes at the behavioral level although there may have been a temptation to do so which others may not resist.

That reductionism may lead to physics but it is not clear where we are to go from there except, perhaps, to "that's the way it is," or "it's the nature of the beast," or "God made it that way" since there are no sciences left after physics; metaphysics does not count (cf., Eacker 1983). It may be enough to show what is related to what at the behavioral level, and what is related to what at the neuronal level, and what is related to what at the biochemical level since that may be about as far as we can go with the analysis.

On the other hand, we could also just stay at the behavioral level because a functional analysis may tell us all we need to know about how to solve human behavioral problems. The so-called "complete picture" that is the hope of so many may be an unattainable myth since to have the "complete picture" may require that we leave behavior undisturbed, that is, not engage in either analysis or reductionism.

The upshot of this line of thinking is that, if selection involves

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replication, variation, and interaction and if replication in particular involves iteration or repetition as well as information then operant conditioning may not fit that model very well. It may not fit very well because, while we may be able to demonstrate a parallel between behavior change, neuronal change, and biochemical change, we may not be able to treat any of them as "information" if, as the authors contend, information requires physical structure. Behavioral science does not have a physical structure for information and may never have one since, as is so well known, the existence of things, and especially structures, is very difficult to establish (cf., Eacker 1975). In short, we may have correlation among those changes but not causation.

However, if a functional relationship between variables is about as close as we get to causation in the behavioral sciences (cf., Skinner 1953, p. 23), then we may speak of those relationships in causal terms. However, their sequencing may be difficult to establish unless we can show that the behavioral change occurs before, or after, the neuronal change and if before, or after, the biochemical change. At this point, it appears at least to me that they all occur at about the same time although I claim no expertise beyond the behavioral level.

From where I sit, it appears that a considerable amount of intellectual effort has gone into the writing of this article. The authors claim to have done so in order to determine whether it might be useful to construe selection as a special sort of causal process (without going into an analysis of causation). The larger context in which their article occurs may have to do with the unification of science.

Now I am also not well-versed in the literature on the unification of science but that is not going to stop me from saying something about it. When the methods of science are applied to human affairs (cf., Skinner 1953, p. 5), if they are (Skinner 1956), it may result in a science that is quite unlike any of the other sciences and especially if science is simply a matter of operant conditioning as proposed by Skinner in his case history.

On the other hand, if science is simply a matter of operant as well as respondent conditioning, then analyzing all of the sciences in those terms may help to facilitate the process of unification without reductionism although, so far, that does not appear to be what has happened for the science of psychology. There are those of us who still regard it as the science of behavior but, as is well known, psychology is far from unified on that point.

In conclusion, I applaud the authors for their efforts to establish a model for selection that might fit those sciences where selection is of interest. However, the fit is not complete for the science of behavior because there is no physical structure that corresponds to information. Therefore, the authors might look elsewhere and especially to the science of behavior if their larger aim is the unification of science or, at least, those sciences that they represent.

Selection: Unexplored and underexplored realms

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Abstract: A profound problem in viewing operant learning as selection appears to be the identification of replicators. Given the lack of consensus on what constitutes the appropriate unit of analysis for behavior, there may be multiple levels at which the metaphor of selection may be usefully applied. A final difficulty: The elements of selection in the evolution of species are objects. In behavior, they are events.

We welcome the notion of selection independent of its application. We agree that the measure of the applicability of any such notion is its usefulness.

Information theory. In their discussion of the need for an analysis of the relation "coding for," the authors state that the available work on information theory, including that of Dretske (1981), "cannot be used to distinguish" structures that code for some other structure and those that do not. In contrast, we anticipate that Dretske's (1981, Ch. 5) analysis of message versus channel will prove useful. According to Dretske's definition, the structure coding for an end structure is message while the double-helical structure, the type of chemical bonds, the means of gene transcription, and so on, are all channel with respect to phenotypic characteristics.

Those interested in what constitute the logical conditions on variation ("blind, chance," etc.), may wish to consider Peirce's (1898/1992, pp. 137–38) notion of "haphazard." Putnam (1992, pp. 62–67) speculates about this notion in terms of contemporary information theory.

Conditions for selection. Peirce (1931–1958, 6.15) appears to agree with the authors (and ourselves) that replication and interaction must be distinguished. He refers to two factors: heredity, which includes both replication and variation, and destruction, a separate process where "the amount of variation is absolutely limited in certain directions by the destruction of everything which reaches those limits."

Is some analogue to the genotype/phenotype distinction also necessary to selection? Even if the distinction is unnecessary to the selection process, selection systems that possess the distinction may be different in important ways from those that do not. A selectionist process may benefit by virtue of having separate containers for variation and interaction. Bateson (1972) finds two utilities.

First, the physical isolation of the genetic material prevents most somatic alterations from altering the genetic material. Thus, transmittable variations (either haphazard or "directed") rarely arise due to interaction. Barriers between variation and interaction make Lamarkian inheritance nearly impossible.

Second, Bateson argues that Lamarkian inheritance is self-terminating and thus destructive. Lamarkian inheritance would decrease variability by over-rapid adaptation. Approach to the physical limits of adaptation decreases variability ("ceiling effects"), which in turn curtails further selection and thus reduces later adaptability (in agreement with the authors' suggestion that "selection processes are able to produce genuine novelty and organization only because they are so incredibly wasteful"). Somatic adaptability within the life of one organism is also impaired by over-adaptation. As Peirce (1931–1958, 6.16) has it: "The Lamarckian theory only explains the development of characters for which individuals strive, while the Darwinian theory only explains the production of characters really beneficial to the race, though these may be fatal to individuals."

The conditions under which a selection process might be self-terminating in the absence of a barrier between variation and interaction or at the very least, more efficient in its presence, are severely underexplored. Computer simulations seem called for.

Mathematically, there is also an effect due to the incommensurability of genotypic coding and phenotypic realization. Small alterations in the genotype sometimes produce large changes in the phenotype; other large alterations in the genotype may produce no change at all. This range of outcomes for variations in genotype must have important effects in determining the impact of interaction. As an aside, we would emphasize, with Weiss (1924, p. 42–44; 1925, p. 55–56), that the same incommensurability found between genotype and phenotype exists between the units of motor activity (genotype) and the units of behavior viewed as action (phenotype). We find this important distinction remarkably underexplored.

Behavior as selection. Our own investigations of neural networks (Kemp & Eckerman, in press) based on the research of Stein and Belluzzi (1993) encourage us to share the authors' skepticism that all changes in behavioral propensities are due solely to changes in synaptic plasticity. We also suspect that "contingent re-

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inforcement modifies several dynamical properties" of real neural networks and hope that researchers in artificial neural networks will address additional sorts of neural plasticity.

Consideration of operant learning as a selection process involves the greatest speculation and most unknowns. Very little is now known about the behavioral implications of the facts of neuroscience, where replicators for behavioral selection are to be found. Also, the behavioral entities constituting the interactors are events, rather than objects. And, if the units of interaction in behavior are events, might not the units of replication and variation be also? Patterns of neural activity may be to behavior as the objects encoding genotype are to soma.

If the ultimate consideration of the applicability of a model is its usefulness, then it may be that operant learning might be modeled as a selection process at more than one level. Two (or more) sets of replicators and interactors might be independently identifiable and both make for useful models. Replicators might be synaptic efficiencies or neural activity or both.

Replication or reproduction?: Symbiogenesis as an alternative theory

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Abstract: This commentary takes issue with the idea that replication is a "fundamental element" in natural selection. Such an assumption is based on a traditional, mechanistic view of evolution. A symbiogenetic theory of evolution offers an alternative to traditional theories, emphasizing reproduction and qualitative development rather than replication and quantitative development. The issues raised by the symbiogenetic alternative may be extended to discussions of behavioral development.

In their target article outlining a general account of selection, Hull et al. suggest three "fundamental elements" in the process of natural selection: replication, variation, and environmental interaction. In this commentary I take issue with the assumption that replication is a fundamental element. A narrow focus on replication may bias a natural selection model toward a more mechanistic, nuclear vision of evolution (Margulis 1991) with limited explanatory power. This potential bias may extend to the authors' discussion of behavioral development through operant learning.

Replacing the concept of replication with reproduction (with replication understood as a necessary subset) might offer a model of natural selection that is both more inclusive and explanatory. Such a model would allow for both nuclear/Mendelian replication and cytoplasmic driven symbiogenesis to be integrated into a single "fundamental element." Symbiogenesis is, by its very nature, more concerned with reproduction (what Margulis defines as the *sine qua non* of the evolutionary process, 1970) than with continuous replication. It is a far more organic perspective (Margulis 1991), recognizing the necessity of qualitative change in the adaptive process.

Biologists recognize two possible mechanisms for evolutionary innovation. The first and more popular mechanism is the nuclear/genetic inheritance scheme based on a combination of Mendelian genetics and population based evolutionary theory (Margulis 1991). Essential genetic material is found in the nucleus of the cell. This genetic material represents heritable information that is transferred, through replication, from generation to generation. Opportunities for change in information are dependent on interaction between mutated genetic materials within the nucleus (variation) and the environment. Interaction leads to evolutionary conditions where the mutated genetic material will be replicated to a greater and greater degree (iteration).

A major difficulty with replication is that it offers little explanation as to how surviving species preserve reproductive integrity in the face of quickly changing environments. Replication is, as Hull et al. state, "inherently a copying process." Variation occurs primarily on the fringes, and selection through environmental interaction will generally be quantitative change over successive generations. As biologists from Kropotkin (1902) to Margulis (1991) have pointed out, this is an unrealistic explanation for speciation. Environmental circumstances often change qualitatively in relatively short periods of time while variation in genetic material occurs without relationship to consequence. The chance these genetic variations will address needs within a changing environment is at best fortuitous coincidence.

The concept of symbiogenesis stands as an alternative to the nuclear/genetic model. It suggests a second avenue for transmission of information between generations that is not dependent on genetic mutation. Cells incorporate alien organisms into their cytoplasm, integrating their current positive attributes (in relation to the environment) with those of the foreign organism (e.g., bacteria). Biology often has a bias in seeing foreign organisms as negative (Sapp 1991). This ignores the positive consequences of two very different organisms pooling their positive attributes in a changing environment in order to survive and reproduce.

Symbiogenesis has gained great credence in cellular biology but has so far had less of an impact on evolutionary theory. I offer three reasons why it should, and probably will, be taken far more seriously in the future. (1) Margulis (1970) offers a compelling argument for serial endosymbiosis theory (SET) in her exploration of the developmental relationships between the prokaryotes cell and the eukaryotic cell. Margulis uses the biological record to cast severe doubts on the traditional explanation of change as a result of genetic mutation. The development of eukaryotic cells through endosymbiosis is not only more elegant, but is a much better fit to current data. (2) In perhaps the only controlled experiment of speciation available (Dobzhansky's separation of one breed of fruit flies into two), the differentiation between hot breed and cold breed was actually caused by a parasite (i.e., symbiogenesis) (Margulis 1998). (3) Fossil patterns suggest that evolutionary development occurs in sudden bursts of activity (Eldredge 1991).

The same issues concerning replication are relevant to the learning/developmental processes. Operant conditioning falls into the same neo-Newtonian, mechanistic traps as the new synthesis in evolutionary theory. Hull et al. attempt to develop a similar type of nuclear model for exploration of behavior by having neurophysiologists play the role of Mendelian geneticists and operant learning theorists play the role of evolutionary theorists. It is difficult to know who might play the role of cellular biologists in this drama, so I will let the cellular biologists play it themselves. There is no doubt that replication works for the "least complex" lineages explored in the article. Traditional evolutionary theorists had little trouble outlining how small changes in the prokaryotes cell might have occurred. But it is impossible to explain how prokaryotes cells evolved into eukaryotic cells (without creating intermediary organisms out of thin air). The same I believe is true of learning and behavior. It is possible to show small changes in "least complex" lineages of behavior using traditional models of natural selection, but it is impossible to show how behavior becomes qualitatively more complex.

More organicist theories such as those offered by Piaget and Vygotsky (Glassman 2000) offer explanations for behavioral development that are more qualitative/stage based in nature (similar to the sudden bursts in the fossil record). In these types of scenarios there is still a nucleus (neural mechanisms), but there is also an important emphasis on the activity of the organism in the environment. Variations that allow humans to deal with fast paced, highly qualitative changes in their immediate ecology are usually dependent on reciprocal relationships between neural mechanisms and social/behavioral histories. Just as Margulis (1991) argued the impossibility of conceiving of a nucleus sans the foreign organisms that do or can exist in the cytoplasm, it is impossible to conceive of neural mechanisms outside of behaviors that do or can occur as a result of ongoing activity. A variation of SET may actu-

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ally be more persuasive in the study of behavior. Maynard Smith (1991) suggests the greatest question for symbiogenesis is whether transfer of cytoplasmic information is direct or indirect, a differentiation that has far less meaning in the serial development of behavior.

Hull et al. did leave a trap door in their discussion of operant learning, stating that this was simply the behavioral theory they found interesting. However by outlining a traditional, nuclear view of evolutionary development through natural selection they may have "stacked the deck." A behavioral theory then would have to come from a similar world hypothesis (as I believe operant conditioning does) to have merit in light of current evolutionary arguments. Both traditional evolutionary theory and operant conditioning dilute the importance of reproduction by emphasizing replication, limiting the explanatory powers of both theories.

The role of information and replication in selection processes

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Abstract: Hull et al. argue that information and replication are both essential ingredients in any selection process. But both information and replication are found in only some selection processes, and should not be included in abstract descriptions of selection intended to help researchers discover and describe selection processes in new domains.

Hull et al. have made a very valuable contribution to the project of understanding the nature and explanatory role of selection processes. However, I do not agree with their description of the essential ingredients in a selection process. Most important, they use the concept of *information* in a way that makes the problem of giving a general theory of selection harder than it has to be. I will also take issue with their discussion of "replicators."

As the authors say, understanding the theoretical role of the concept of information in biology has become a pressing problem. However, this is not a problem that has to be solved in order to achieve a general understanding of selection processes. That is because, despite what the authors claim, not all selection processes involve a role for informational properties. Or more precisely, not all selection processes involve informational properties in a nontrivial way. Any set of causal processes *can* be described in informational terms, but only a smaller set of processes are such that we *need* to use, or achieve real benefits from using, an information-theoretic framework to describe them. This smaller set of processes seems to include some, but not all, processes of selection. The reason for this lies in the nature of inheritance: some, but not all, processes of inheritance work via the transmission of information.

Although the informational description of genetic mechanisms has been taken too far in some recent discussions, there are features of actual genetic mechanisms that do seem to justify descriptions in terms of information and coding. Most generally, these descriptions are applicable because the set of molecules that do most of the "work" in essential biological processes – proteins – are generally not passed across the generations intact, but are specified by nucleic acid molecules that do cross the generational bottleneck. The role of DNA can be described as "informational" because of what it does *not* do, as much as because of what it does do. DNA's role in the cell is primarily the specification of protein structure and the regulation of this activity, as opposed to playing a more direct role in the processes that sustain life.

These features are found in genetic transmission, which is the most important kind of inheritance in inter-generational selection processes. But some *actual* forms of inheritance in inter-generational selection processes work differently, many *possible* forms of inheritance in inter-generational selection processes work differently, and we certainly have no reason to believe that all inheritance in other domains of selection works in this way. For an obvious contrast, consider sample-based inheritance systems. If a particular kind of structure must be passed across generations, one way to do it is to pass across some kind of coded specification of that structure, but another way is to pass a *sample* of the same structure that is needed (Godfrey-Smith 2000a; Sterelny 2000). We see this often in the transmission of gut symbionts from parent to offspring. We see something similar in the transmission of prions. And we can imagine alternative possible genetic systems in which samples of proteins rather than coded specifications of proteins are inherited. The fact that actual genetic systems work differently, specifying proteins with DNA, is a fact of great importance. But this fact, and the appeal of informational descriptions that derive from this fact, should not be seen as essential to all selection processes. Understanding why the actual world is dominated by one biological inheritance system rather than another is different from the task of understanding what is essential to and distinctive about selection processes themselves. As the authors say, if we built too many features of the "biological" (intergenerational and genetic) case into our general theory of selection, it becomes more difficult to recognize analogies between selection processes in different domains.

A similar point applies to the concept of a replicator. Like the concept of information, the concept of a replicator is useful and important, but replicators are not essential to all selection processes.

This idea is more controversial than my previous claim. It is very common to say that something must play the role of replicator in every selection process. But the usual requirements placed on something being a replicator are very stringent; they are more stringent than the requirement that the population in question contain some form of inheritance. For inheritance to exist, there has to be some way of reliably producing similarity (along some dimension) between relatives across generations, but this mechanism need not involve an underlying population of entities (like genes) that are copied in the wholesale and direct sense associated with the (Dawkins-Hull) concept of a replicator (see also Godfrey-Smith 2000b). And crucially, only inheritance of *some* kind is essential to the existence of a *selection* process. Selection requires no more than a systematic correlation between parent and offspring (in fact, selection could even happen if the correlations were only found between parent and grand-offspring, or a more distant relative). This point was made by Lewontin in his 1970 analysis of selection, and is also made clearly in a quote that the authors give from Thompson: natural selection "does not require genes or even direct descendants; all it requires is that the presence of a configuration of elements in one generation makes more likely the presence of the same configuration in the next generation" (1994, p. 638). Thompson is right, and Hull et al. do not give compelling reasons to give up on this insight. I should note here that the authors do express their rejection of Thompson's point with some caution; they realize the risk that they have thereby made their account too narrow. I suggest that this concern is justified; they have indeed made their specification of selection unnecessarily narrow.

Do operant behaviors replicate?

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Abstract: Operant conditioning is not a selection process. According to Hull et al., selection processes require entities that reproduce to form lineages. However, since operant behaviors do not reproduce, operant conditioning is not a selection process.

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Hull et al. offer an account of selection processes which, they claim, is general enough to subsume three possible examples of selection: natural selection, the immune system, and operant conditioning. This general analysis of selection is significant because it will clarify the idea of "conceptual selection" and may provide new insights into the metaphysics of selection processes. Hull's (1988) claim that conceptual change in science is driven by a selection process has been criticized on the grounds that theory change is very different from biological evolution. While Hull recognizes a number of differences between these processes, he argues that conceptual change is a selection process nonetheless. One way to resolve this dispute is to develop a *general* account of selection processes that abstracts away from the details of "gene-based biological selection." A similar process of abstraction has already proven very important in the units of selection debate. For example, Lloyd and Gould (1999) have recently argued that selection at different levels produces qualitatively different kinds of biological individuality. Thus, just as studies of higher-level selection have forced us to re-examine the relationships among adaptation, individuality, and natural selection, comparing natural selection to other possible selection processes may challenge our assessment of the conditions that are necessary for selection to occur. This commentary focuses on operant conditioning precisely because it challenges our current understanding of the relationship between selection, replication, and lineages.

Hull et al. argue that operant conditioning is a selection process. Even simple behaviors like lever presses vary (e.g., in the amount of force applied) and these variations can be shaped by operant conditioning. As we select for more forceful presses, the presses continue to vary in force, but the average lever press becomes more forceful. According to Hull et al., this process has the same causal structure as natural selection. Organisms vary in their traits and, because some variants are better adapted to their environment, the well-adapted variants increase in the population over time. Similarly, behavioral responses vary in their traits and, because some of these responses are better suited to the reinforcement schedule, those traits increase in frequency within the behavioral lineage.

According to Hull et al., selection requires "repeated cycles of replication, variation, and environmental interaction so structured that environmental interaction causes replication to be differential" (sect. 2). Does operant conditioning satisfy this analysis? Variation is not a problem: even simple behaviors like lever presses vary. Furthermore, the differential "fit" between variant behavioral responses and the environment influences the likelihood that those variants will be performed in the future. But even though operant conditioning involves variation and interaction, I maintain that (1) behavioral responses do not form lineages because (2) there is no genuine replication in operant conditioning. And if operant conditioning does not involve replication, then (according to Hull et al.'s analysis), it is not a selection process.

A lineage is a single, temporally continuous sequence of ancestors and descendants. The lineage is composed of short-lived entities which are related by descent (i.e., acts of reproduction). While genes, cells, organisms, and populations all form lineages, the reproductive acts which generate these lineages vary considerably: genes replicate; cells "reproduce" through mitosis; and sexual organisms reproduce through a more complex process of meiosis, sex, and development. (Because of these differences in mode of reproduction, these entities form different kinds of evolutionary trees, ranging from strictly branching to highly reticulated trees.) Given this standard account of lineages, operant behaviors form lineages only if operant behaviors "reproduce." Do lever presses reproduce? Is there a genuine pattern of descent so that one lever press gives rise to a later lever press? At best, this mode of reproduction is indirect. The indirectness of reproduction does not (by itself) force us to reject the idea that entities form a lineage. Sexual organisms form lineages in a relatively indirect way. A father directly contributes a haploid set of chromosomes, which then combines with maternal chromosomes, and this diploid genome then comes to influence the offspring's phenotype through a complex process of development. Thus, relatively indirect forms of reproduction are sufficient to generate lineages of sexual organisms. But part of the reason for treating this indirect relationship among organisms as reproduction is that the genes which contain information about the organismic phenotype reproduce in a fairly direct fashion. Because behavioral responses do not directly reproduce, they do not form true lineages unless we can identify some other entity (a replicator) which reproduces more directly.

What are the "replicators" in operant conditioning? Hull et al. acknowledge that we do not yet have an adequate answer to this question, but suggest that the replicators are the neurological structures that "code for" the structure of the behavior. Because there can be a long hiatus between operant behaviors, those behaviors form a temporally continuous lineage only if something else – presumably some neurological state – preserves the relevant information about the structure of the behavior during this hiatus. The problem, however, is that we have no evidence that this neurophysiological state actually *reproduces.* Rather, it would seem that past experience simply *modifies* an existing structure. Here is a rough analogy. Suppose that an organism produces a series of offspring, each one persisting for a brief time and then dying before the next is born. These offspring (which are roughly analogous to the sequence of operant behaviors) do not form a lineage. Without replicators – without replication of the structures that code for the behavior – operant behaviors do not form true lineages.

The analogy of the last paragraph is imprecise because it ignores the fact that operant conditioning involves a kind of "feedback" in which the environmental response to past lever presses changes lever pressing behavior. The behavioral repertoire not only changes, it evolves *so as to produce better "fit" between behavior and environment.* Even if we allow for this kind of "feedback," it is not clear that the behaviors form a true lineage. To incorporate this feature into our analogy, suppose that the mother stores sperm from a single mate and then, based on the performance of previous offspring, selects sperm to inseminate later offspring. Even if the sequence of offspring changes so that later offspring are better adapted to their environment, successive offspring form sibling pairs, not ancestor-descendant pairs. Without replication of the information that codes for the behavior, operant behaviors do not reproduce or form lineages in the required sense.

Faced with the difference between paradigm cases of natural selection and operant conditioning, two conclusions are possible. First, one might retain the analysis of selection (and replication) and conclude that operant conditioning is not a selection process. But if one were convinced that operant conditioning really *is* a selection process, one could infer that the present analyses of selection and/or replication are too restrictive. While I support the first interpretation, the issue is far from clear. By seriously confronting several causal processes that have been viewed as instances of selection, Hull et al. have forced us to re-examine fundamental metaphysical questions about the role of lineages in selection processes.

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Natural selection and metaphors of "selection"

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Abstract: Natural selection in the sense of Darwin always means physical propagation (positive case) or disappearance (negative case) of living or-

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ganisms due to differential reproduction. If one concentrates on this simple materialist principle, one arrives at a much better method of discerning true selection processes from largely nonrandom processes of internal rearrangement (somatic mutations) and reorganisation (operant learning).

In general, evolutionary biologists are used to speak of differential reproduction if they want to characterize what the authors of A General Account of Selection somewhat circumstantially describe as "replication, variation, and environmental interaction." In fact, every biologist can easily subscribe to the latter definition with the exception of the surprising, since unexplained neglect of the importance of neutral evolution its creators feel obliged to derive from it. They only remark that Kimura's (1982) at the time quite provoking work on genetic drift has not really been integrated into the modern neo-Darwinian synthesis. This is just not true if one considers the conceptual development of evolutionary theory during the last two decades (cf. Eigen 1987, p. 255). In fact, a quite recent article by Fontana and Schuster (1998) on the evolution of RNA molecules very convincingly demonstrates the fundamental role of intervening neutral phases of genetic change to allow the transition to more complex and thereafter often more robust levels of organization. And at a more general level this finding is closely related to the phenomenon of punctuated equilibrium where more or less long neutral phases of stasis alternate with rather short periods of rapid adaptive change (Elena et al. 1996; Gould & Eldredge 1977).

But this objection is still relatively minor compared with the impressive effort the authors have undertaken to elaborate the first sketch of a universally acceptable description of what can and should be understood by "selection." At the end of their considerations they come to a clear positive conclusion, that is in their view it is fully legitimate to speak of the presence of selection in all three investigated cases, that is, phylogeny, the development of immune reactions during ontogeny and, last but not least, operant learning. However, it is also possible to propose a somewhat different kind of analysis. For that purpose it is already sufficient to concentrate on criteria which could perhaps justify the rejection of the selection-hypothesis rather than merely looking out for many possible positive arguments, as has been done in the target article. To do so in the simplest form, we only need to formulate a combination of two criteria which should allow us to say no to the presence of true selection if only one of them applies:

Biologists in the tradition of Darwin should not speak of the coming into effect of selection if:

1. Already disappeared characters suddenly re-appear in subsequent generations (= false negative selection).

2. Characters which seem to have been positively "selected" during ontogeny never re-appear in subsequent generations (= false positive selection).

Usually, genes, genomes, characters, individuals, populations, and species never do come back once they have been negatively selected, and that is eliminated by Darwinian selection. This is the very simple, but fundamental essence of Darwin's idea of "natural selection" which, in that respect, was a clearly materialist one. Concerning biological evolution, there is only one apparent exception to that principle and that is the phenomenon of so-called back mutations which, however, are mainly caused by a special mechanism (DNA proof reading and mismatch repair, itself genetically evolved in most phyla) of compensating for all too high mutability rates and hence conserving the status quo. But already at all higher levels, characters once lost (let alone extinct species), have a zero chance of reappearing in the same form as before. This is also valid for those cases where a convergent selection pressure often leads to extremely similar, but nonetheless only functionally analogous characters in different species. The moment a concrete genetic lineage goes extinct, its unique combination of characters necessarily has to follow and this explains the basically historical, that is, irreversible nature of biological evolution.

What about so-called "somatic evolution" of the immune system with respect to these criteria? Clearly both possibilities from above can and will often occur. First, seemingly "new" antibodies must be expected to repeatedly arise in the next generation, but only because a specific pathogen did not occur in the previous one. Consequently, one and the same type of antibodies seem to appear and re-disappear completely independent of the succession of generations. But that means nothing other than that there was no real negative selection. Second, even if one specific type of antibodies has been "positively selected" during ontogeny, this does not preclude that true negative selection eliminates its carrier in the next generation (e.g., no offspring). Thus, "positive" here means only the environmentally induced production of those antibodies during a concrete life cycle, but not their real origin which must have to do with the genetical evolution of the preceding generations. This explains why today we speak of "adaptive" (cf. Encyclopedia 2000) instead of "acquired" immunity because it is more and more obvious that its acquisition has still to be done by conventional germ line evolution (Fearon & Locksley 1996).

The situation in operant learning – and, by the way, also in conceptual change in science and culture – is even more revealing. Cases 1 and 2 are in this view not rare exceptional cases, but rather they constitute more or less the rule. So it is striking that all kinds of typically human errors, from learning to ride a bike (e.g., certain over-reactions) to learning how to do higher mathematics (e.g., problems with Bayesian reasoning; Gigerenzer 2000) re-emerge in every new generation even though we should assume that they have been already definitely eliminated in the previous ones. The other case, the false positive selection (disappearance despite "positive" selection during ontogeny) is certainly more difficult to demonstrate, but nevertheless can be expected to occur all the time: A specific kind of solving a learning or more complex cognitive problem disappears in the next generation because its carriers, be it animals or humans, did not reproduce.

In conclusion, our considerations show (1) that true selection does only occur within real biological, that is, gene-based evolution within the germ line and (2) that both the immune system as well as the mechanisms of operant learning behavior are not independent ways of acquiring new characters, but quite to the contrary are themselves equally dependent on Weismannian genetic change. This however requires that, basically, the complete information necessary for ontogeny must already be present in the genome of the zygote, a view which, in the meantime, can be sustained for several reasons (Heschl 2000; for a discussion of "information" see Maynard Smith 2000). Both "new" antibodies and "new" variants of operant behavior are the result of already preadapted mechanisms which in some way have to "mimic" stochastic environmental influences, that is, random pathogen diversification in the first and unpredictable spatiotemporal contingencies in the latter case. This also explains why they look so incredibly "random," but they are definitely not examples of a separate evolution by variation and selection.

On the origins of complexity

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Abstract: Darwin's theory of natural selection is as applicable to the analysis of the behavior of organisms as it is to their origins. Skinner's theoretical writings have guided operant psychologists in this area. The behavioral account of selection by Donahoe and Palmer (1994) is positively compared to the points on operant selection made by Hull et al. The "general account of selection" was found to be useful.

We often marvel at the complex interrelations of the many species in this world and at their diversity. If we examine even the sim-

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plest of organisms we find an organization that appears the work of creative design. If we look at the "parts" of organisms and their behaviors we also see intricate complexities. Humans have been offering "explanations" of this complexity throughout most of their verbal history. Some explain creative design as the work of a "creator" (be that a deity or a conscious self). Others adopt an approach initiated by Darwin that replaces the "creator" with conditions for natural selection. Because this account postulates a different form of causality many have resisted it. Its applicability to areas other than explaining the origin of diverse species is not as well understood. The target article by Hull et al. is a good attempt to clarify the critical features of "selectionism" that are relevant across disciplines.

The characterization of selection as comprising "repeated cycles of replication, variation, and environmental interaction" which result in replications being "differential" is sufficiently general to apply across disciplines. It is the task of the discipline to operationalize each of the critical terms. The examples given from biology, immunology, and behavioral psychology were useful. Our backgrounds are in operant behavior so our focus is on the "Operant selection" sections.

Hull et al. suggest that matching processes can be found in disciplines applying selection theory to their complex subject matter. One could ask: "Are convincing matches required before a selection account is considered valid?" A syllogistic reasoning approach might answer "yes" but a pragmatic approach would not. It would assert that if the selection account leads to more effective research and applications (providing a better "fit") then each iteration of that account would evolve differentially to include increasing degrees of functional complexity. As a result the selection-based account would continue to evolve while other accounts might not.

As noted in the target article, the Radical Behaviorism of B. F. Skinner fully embraces selection as an explanatory mechanism. Skinner's writing on selection by consequences and the parallels to natural selection go back at least to 1953 (Skinner 1953). A collection of Skinner's papers on selection and evolution can be found in book form. (Skinner 1987). Operant behavior is defined by its effect on the environment and explained by pointing to its functional history of consequential effects. This is the core of selection theory. Even the capacity to be affected to different degrees by environmental changes as consequences and as discriminating antecedent events is "selected," albeit by natural selection (e.g., the behavior of many insects is less "operant" than that of humans).

The authors' account mostly matches other recent behavioral views of selection. For example, Donahoe and Palmer (1994) provided an overlapping list of three selection mechanisms. These are variation, selection, and retention. Let us look at how these two analyses compare.

Both sets emphasize the necessity of variation. In evolution this means intraspecies variability. In behavior this means response classes or behavioral lineages. Second, in both cases, variability enables the environment to act upon certain members of the class or species. Thus Donahoe and Palmer's "selection" is the "environmental interaction" of Hull, et al. At this point the two views are consistent. Where they differ is in the inclusion of the iterative, replicative nature of the process (the retention itself), and in the importance of a structure responsible for retention.

The replication of a selected behavior increases the probability of the occurrence of the selected behavior (or structure, or microorganism) and thus increases the probability that it will be selected again, so that the cycle can be repeated. The result is that particular outcomes will be differentially affected. This defines the reinforcement process. For most operant psychologists, this iterative process itself is sufficient evidence for retention. One does not need to ask where behavior is stored or how the effects of reinforcement are retained. Skinner (1974) suggests that the organism is "changed" and it is this "changed" organism that interacts with its future environments. Hull et al. do suggest that the retention of operant learning is the result of changes in the central nervous system but are unspecific. Donahoe et al. (1993) provide more detailed speculation as to the specific CNS structures involved in both respondent and operant conditioning.

In sum, the general model presented here is consistent with that put forward by other operant psychologists. At the beginning of their target article, Hull et al. provide Skinner's view that acceptance of Darwin's theory was delayed because selection required a new way of thinking. If one of the problems in the acceptance of operant learning is that, like Darwinism in its time, it requires a new way of thinking, then linking operant learning to the now accepted selection mechanisms of more fundamental sciences should help, and behavior analysis as a natural science should be advanced. It may indeed be further served by attempts like those of Donahoe et al. to locate in neurophysiology the equivalent of the gene, for it was the rediscovery years later of Mendel's work that contributed to the wider acceptance of Darwin's theory. While the work of the present authors to link operant learning to other sciences under the umbrella of selection may work, it may advance more rapidly with the determination of a retention mechanism.

During the preparation of this response, a radio spot noted the 170th Birthday celebration for Harriet. Harriet is a 170-year-old Galapagos Island tortoise that is considered to be the oldest living animal in captivity. Charles Darwin took Harriet from the Galapagos Islands when she was 5. This direct living link to Darwin, the individual most responsible for ingraining the approach of selectionism into modern science, reminds us of how young this approach is – it has occurred in the lifetime of a creature touched by Darwin. The tortoise also serves to remind us how slowly change moves in scientific thinking.

Sharing terms and concepts under the selectionist umbrella: Difficult but worthwhile

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Abstract: Comparing and sharing selectionist terms and concepts from disparate domains can aid understanding in each domain. But constraints of interpretive language will make this difficult – such as the bipolar constraint of interpretive language when addressed to intrinsically tripolar phenomena. Hull et al. acknowledge that some key terms in their account remain problematic; the term, "information," probably needs to be replaced.

It is a truism that learning to speak a second language results in an improved understanding of one's native language as well. Part of what comes through in Hull et al.'s target article, is the necessity, when sifting out the complementarities among their three disparate domains of phenomena and interpretation, of the authors' each re-sorting and clarifying their own concepts and terms. Examining the fruit of their remarkable collaborative effort from the viewpoint of a behavior analyst, I found benefits to be gained not only through the alliance of viewpoints commonly viewed as diverse, but also through the license it gives to re-examine our own terms and the relationships between our concepts.

For example, the terms, "response" and "to respond" often have struck me as carrying unfortunate baggage when labeling the focus of our study. They implicitly characterize the behaving organism as passively reacting to, rather than flexibly interacting with the events in its environment. Combined with the associationist metaphors of traditional learning theory, it plays into the pervasive mis-equating of behavioral psychology with "S-R psychology." The latter applies well enough to simple reflex reactions, but not to the phenomena that occupy most behavior analysts, whether basic or applied. Joining behavior analysis with its natural selectionist roots, rather than with the associationist roots of learning theory,

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invites us to consider alternative terms that might have equally broad but more appropriate generality. "Interactor," in place of operant response, seems to better convey the flavor of process as we understand it – although some of its variants may not work fluidly within our prose. "An interactant produces a consequence?" That will take some getting used to. "The child's interacting was reinforced?" The vernacular intrudes with a limited meaning, On the other hand: "The child's questioning, as an interactant, was reinforced." We may learn ways to do this.

There are subtle conflicts between the interpretive patterns that we inherit from ordinary language, and the patterns of best coherence within our interpretive systems. Behavior is (or should I say, interactants are) inherently tripolar; it involves the organism (represented in interpretation by its characteristics or internal processes), the organism's environment (including its past interactions within similar situations), and what the organism does – its behavior. In contrast, interpretive language, applied to behavior, is bipolar (noun-verb; agent-action; cause-effect; independent variable – dependent variable). Various interpretive assumptions and conventions follow from this bipolar/tripolar difference, and it also follows that any interpretive account of behavior is constrained into granting privileged interpretive status either to terms that characterize the organism and its inferred underlying processes, or to terms that characterize present and past environmental events. (I have spelled out these observations and their implications in detail elsewhere: Hineline 1980; 1990; 1992.)

The target article's distinction between replication and environmental interaction adds yet another twist to these conundrums. While I enthusiastically welcome this distinction for its making more tractable the issue of differing scales or levels simultaneously operative in related but distinct domains, I anticipate that we have yet to learn how to pull together environment-based and organism-based interpretive prose within a single paragraph without risking incoherence.

While the above concerns involve the internal coherence and compatibility of interpretive patterns, there also are, as the authors acknowledge, some unresolved problems regarding individual terms that are central to the discussion. Key among these is information. At one point, the linear sequence of nucleotides in DNA is described as providing the information necessary for the production of proteins. It seems to me that in a nontrivial sense, the linear sequence of nucleotides *is* the information. Perhaps there is a better way to say it than either of those two. Invoking the metaphor of recipe is a possibility, although genetic code is a bit more like recipe and cook combined, operative only in the context of a suitable kitchen, complete with utensils and supply cupboard. The recipe metaphor seems less apt for the selection process as embedded in operant behavior, however. Regarding the latter, it is suggested that: "Differentially altered probabilities of events that 'pass on information' (in this case, information coding for response properties) may be the hallmark of replication in selection processes." This characterization seems merely to obliquely acknowledge that particular response properties will change as a result of previous reinforcement. Nevertheless, one can hope that continuing collaborative efforts such as the one under consideration here will yield a unified conception of what is at issue when the term "information" is invoked, without propagating the vernacular baggage of that term, or perhaps by replacing the term itself.

Selection in operant learning may fit a general model

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Abstract: The generic account of selection proposed by Hull et al. readily fits operant learning where, by comparison with natural selection, the process is well understood but little is known about the mechanism. Objections within psychology, that operant learning ignores internal processes, fail to recognise the general significance of behaviour-environment interactions. Variation within operant response classes requires further investigation.

A feature of the Darwinian account of natural selection is that some individuals in a population produce more offspring than others. It is argued that this may, in turn, lead to characteristics of those more "successful" individuals occurring with higher frequency in later generations of that species. Once the possibility of such differential success is recognised, the question emerges as to whether a mechanism exists for the selective transmission of characteristics associated with differential success in producing offspring. Prior to the development of the science of genetics, discussion of the feasibility of Darwin's account was much concerned with evidence that differential reproductive success had indeed led to changes in the characteristics of species and to speciation.

There are strong parallels between the type of selection proposed by Darwin, and the process of selection by consequences proposed by Skinner (1938) that characterises operant learning. The central idea of operant learning is that some response variants may produce "better" outcomes than others, and in due course those response variants may become more frequent (in the population of response variants). At the same time, other variants may no longer appear or be extinguished, in the terminology used by behaviour analysts. This process would appear to be consistent with the general definition of selection offered by Hull et al.

In a further parallel with the case of natural selection, the question arises as to "what is learnt," or what mechanism exists for this selective change in the behavioural repertoire over time? As Hull et al. point out, recent research has begun (but only begun) to identify neural mechanisms that may have this role in operant learning. While evidence in this important area is novel and, as yet, uncertain there is much known of the effects of operant learning.

There is, then, a marked difference in the strength of the evidence available to sustain accounts of the mechanism on the one hand and the process on the other in the two cases. That is, the mechanism of genetics is increasingly well-understood and direct evidence of evolution is harder to assemble, while the neural basis of operant learning is poorly understood and there is much direct evidence of the process of operant learning changing human behaviour (see Leslie & O'Reilly 1999, for many examples). Darwinian natural selection is broadly accepted as the explanation of speciation and evolution even though it is hard to "see it working," but operant learning is not seen by the bulk of psychologists as central to the explanation of the complex changes over time that occur in human behaviour (although its importance for changes in the behaviour of nonhuman animals is acknowledged). In both cases, the mechanism and the process are quite different from each other, and bringing together the accounts of each is an interdisciplinary exercise.

The usual reason given for the downgrading of the role of operant learning in the modification of human behaviour is that it ignores the role of cognitive processes. However, this in some ways misses the point. As Hull et al. cogently point out, understanding selection of behaviour through interaction with the environment requires a shift in focus. Rather than being concerned, as traditional psychology was, with a person-centred account of internal processes, we are concerned with how the person's behaviour interacts with their environment. Whether or not we regard it as im-

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portant to specify internal, cognitive, processes as precursors to the behaviour shown by the person, the question remains as to whether operant learning can provide an adequate account of the interaction between their behaviour and the environment. We accept that there are limits to the malleability of human behaviour in this interaction and, for example, organic pathology may make certain types of behaviour unusually resistant to change, but, as Hull et al. remind us, we have evolved to be extremely sensitive to the environmental consequences of our behaviour because this characteristic is highly adaptive and evidence of this is all around us. For example, cognitive psychologists may choose to focus on internal processes said to precede utterances in conversation, but there is plenty of evidence of the crucial role of environmental consequences once the utterances occur.

There is one aspect of the account of variation and selection in operant learning by Hull et al. that may prove to be misleading, or at least address only part of the problem. They distinguish between responses and "interactors," on the grounds that the word "response" generally means a single piece of behaviour, while the unit of behaviour in operant learning is whatever interacts as a cohesive whole with the environment. They are correct to point out this difference between an operant response class (which interacts as a cohesive whole with the environment) and casual uses of the word "response." However, they go on to give details only of cases of operant response classes that are defined with reference to one of the formal properties, or dimensions, that response variants have. Important gains in conceptual clarity in operant learning have come through realising that operant response classes are functionally defined. This point, which originates with Skinner (1938) but has sometimes been ignored since, is crucial to understanding the lineage of an operant response class in human verbal behaviour. For example, under the contextual control of utterances with certain features we may when young use the word "car" to describe the family vehicle, and at a later date this response class may come to include "auto," "motor," and "station wagon." These new variants do not occur through variation in a formal property of the variant that was established earlier, but they may all become class members provided they all have the same function. This type of variation, which leads to formal diversity in the members of an operant response class, is not yet well understood in general terms.

This example from verbal behaviour points up one of the difficulties in completing an account of the process of operant learning that is as complete as that available in their other two examples. Nonetheless, the potential theoretical benefits of this exercise are great, as Hull et al. have illustrated.

Concerns of old, revisited

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Abstract: Commentaries surrounding Skinner (1984) were re-examined and applied to Hull et al. Hull et al. were found to address many of these concerns by paying attention to neuroscience, by providing some discussion of the origins of behavior, and by forwarding a deterministic account that may prove as revolutionary as that of Copernicus and Darwin.

Hull et al. replicate and extend a thesis originally forwarded by Skinner (1953) and revisited in various forms by Campbell (1960), Lewontin (1970), Dawkins (1976), Skinner (1981), and Dennett (1995). I will confine my comments to the replication (the similarities between gene-based natural selection and operant conditioning) because the extension to immunology is outside my area of expertise.

For this commentary, I returned to Skinner's (1981) "Selection by consequences" and the commentaries surrounding its republication in *Behavioral and Brain Sciences* in 1984. When some of the leading biologists, psychologists, and philosophers considered Skinner's proposal, several important critiques were raised, three of which I believe were frequent or important enough to discuss in light of Hull et al. First, Skinner fails to identify the behavioral unit of retention analogous to the gene and thereby ignores neurophysiology. Hull et al. hypothesize that behavioral consequences change the chemistry and firing patterns of the central nervous system, but identifying this neuro-chemical unit of retention will either be extraordinarily difficult or impossible (Uttal 1998).

Why was the gene so important in the acceptance of natural selection and why is it important to identify the behavioral gene? Before the "modern synthesis," Darwin's theory could not explain trait retention in sexually reproducing organisms. Without genetics, offspring would be an average of their parents, and the eventual population, uniformly gray. Darwin's theory was built on heritability; selection and speciation, and these could not be observed under laboratory conditions. Thus, physical evidence of a mechanism of retention was critical.

What of the behavioral gene? Is it also as critical to the advancement of a selectionist account of behavior? On the negative side, operant selection is rapid and has been observed in thousands of laboratory experiments. Thus, unlike Darwin's theory, operant selection is an established scientific principle. On the plus side, identifying a mechanism of retention would put to rest the criticism that operant selection suffers a circularity flaw. Most empirical attempts to resolve this problem have proven inadequate (e.g., Hull 1943; Miller 1951; Premack 1963) and although the account forwarded by Timberlake and Allison (1974) makes more accurate predictions, it appeals to hypothetical constructs (e.g., blisspoints) viewed as inadequate by those looking for physical mechanisms. The final resolution requires a neurochemical mechanism of retention be identified. I share the hopes of Hull et al. that identifying this mechanism may bring consilience to the biological and behavioral sciences.

The second critique of Skinner (1984) is that he ignores the origin of behavior and creativity. The biologists note that their understanding of selection has benefitted by studying the variables affecting genetic mutation. Hull et al. provide a brief summary of what is currently known about predictability and order in genetic mutations, how this variation is manifest in phenotypic variation, and how the natural environment selects for and against such properties. To be consistent with the hypothesized behavioral unit of selection would require an investigation of neurochemical "mutations" and their effects on operant characteristics that are in turn selected for (reinforcement) and against (punishment). Short of this, Hull et al. hypothesize that some "primordial behavior" is the product of natural selection and some operants owe their origin to old (i.e., previously reinforced) lineages. Examples of the latter have only begun to be studied in the extinction-induced resurgence literature (e.g., Epstein 1985).

What about creativity? Hull et al. show that numerous characteristics of operants can be selected for by reinforcement contingencies but they do not discuss creativity per se. Perhaps this is because arriving at a valid definition of creativity is difficult (Epstein 1996), or that creativity implies an autonomous creative mind. The biologist argues that the creativity seen in nature is the result of a mindless selection process. The Creative Agent is absent in this account and the outrage this omission sparked is still felt today. The human mind as an autonomous creative agent holds a more revered position in modern psychology and the position forwarded by Hull et al. is at odds with this notion.

The final critique of Skinner (1984) will probably be raised about Hull et al. as well. Essentially, this is the notion that the cause(s) of behavior must reside "in the head of the organism" rather than in the environment. What is meant by "in the head of the organism" varies considerably. Some commentators take odds with Skinner's black-box approach to the study of behavior. Skinner (1974), recognizing the limited measurement capability of

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physiologists of his time, focused his studies on environment-behavior relations and left the underlying neurophysiology for the neuroscientists. The position outlined by Hull et al. is much the same: the brain plays a critical role in behavior, but not much can now be said about that role.

Others looking for causes "in the head" of the organism are not referring to neurophysiology but to cognition. That cognition itself is selected by consequences is implicit in Skinner (1981; the laws of overt behavior were said to equally apply to covert behavior – cognition) and explicit in other selectionist accounts (e.g., Campbell 1960). Cognitive psychologists have always held that the neural mechanisms underlying cognitive processes will one day be identified by neuroscientists, and this process is apparently already underway. Hull et al. make no mention of cognitive psychology and so I note this omission in their coverage.

Those commentators appealing to the final meaning of "in the head" will be displeased with the position outlined by Hull et al. These critics suggest the organism is "self-regulating," which is to say autonomous of the environment said to shape and maintain behavior. Are these the last vestiges of what may be analogous to pre-Copernican, pre-Darwinian concepts of the relative importance of our position in the cosmos and our unique origin as a species? Just when I think this may be so, I talk with another modern psychologist who clings to the illusion of autonomy. It appears we have a long way to go before we look back at our pre-Skinnerian views of the importance of an inner causal agent and chuckle.

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Operant learning and selectionism: Risks and benefits of seeking interdisciplinary parallels

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Abstract: Seeking parallels among disciplines can have both risks and benefits. Finding parallels may be a vacuous exercise in categorization, generating no new insights. And pointing to analogous functions may cause us to treat them as homologous. Hull et al. have provided a basis for the generation of insights in different selectionist areas, without confusing analogy with homology.

People concerned with operant behavior are generally called behavior analysts, or informally, *Skinnerians.* And, though most of us are psychologists, we are somewhat outside the mainstream of psychology because of our reluctance to make much use of intervening variables, hypothetical constructs, reifications, or derogatorily, explanatory fictions, the fundamental building blocks of most psychology. So, we behavior analysts have taken great intellectual and perhaps spiritual comfort in finding a home on the selectionist continuum between Charles Darwin's evolutionary biology and Marvin Harris's cultural materialism (Harris argues that cultural practices survive if they contribute to the survival of the groups that practice them).

Most behavior analysts have found sufficient comfort based on the popular writing of Steven Jay Gould (1983) concerning biological evolution and Marvin Harris (1984)concerning cultural materialism, that we have not studied the technical writings in either field. Sigrid Glenn is among the few behavior analysts who are not so easily comforted; therefore, she has studied the selectionist continuum in greater depth and more thoughtfully than most of the rest of us behavior analysts. This sophistication is apparent in her contribution to the article on which I am commenting.

It is a fascinating exercise to find the parallels between one approach or discipline and another. And behavior analysts have been previously fascinated in that manner, as exemplified by our demonstrations of the parallels between Freudian psychoanalysis and Skinnerian behavior analysis or between economic analysis and behavior analysis, as well as between evolutionary biology and the acquisition and maintenance of response classes.

But this exercise in parallelisms is not without its risks. First, we risk not going far enough with the parallels, not exploiting them sufficiently. Given that we have demonstrated a parallel between two disciplines, what new insights does that give us into either discipline? Does the parallel suggest new causes or new functional relationships? Does it suggest new categories or classifications? Does it point to new dimensions of either the independent or dependent variables we have ignored or to dimensions we have considered important but now should recognize as trivial? The parallels drawn by Hull et al. do not give us these new insights into behavior analysis; but they may have delineated a model that may be productive of such insights in the future.

Second, there is the opposite risk, the risk of taking the parallels too literally. The authors attenuate this risk by saying, "The processes by which operant adaptation occurs are viewed here as analogous to the processes by which biological evolution occurs," and "When operant behavior is seen as the figure, against organism as ground, the elements involved in selection processes are analogous to (not the same as) those involved in gene-based biological evolution." Unfortunately, such disclaimers can be quickly forgotten, in the heat of intellectual discourse or in the heat of practical applications. The transient nature of disclaimers is frequently illustrated with disclaimers in the form of operational definitions; for example psychologists often operationally define "intelligence" as "the score obtained on an IQ test"; and then, within two or three sentences, they have reified intelligence into an internal, causal agent.

Not taking parallels too literally, I find it helpful to distinguish between "analogous" and a somewhat extended notion of "homologous," where two events are analogous, if they serve the same function of have the same effect; and where they are homologous, if they are based on the same underlying mechanisms or processes. For example, a rat's lever pressing increases in frequency, if that pressing is followed immediately by a drop of water, the directly underlying process being reinforcement. And our commentary writing might increase in frequency, if that writing is followed within a few months by publication, the directly underlying process being a rule-governed analog to reinforcement – it looks like simple reinforcement; it acts like simple reinforcement (same behavior-increasing function), but it is much more complex and requires sophisticated language skills (here I am not talking about the language skills needed for actually writing the commentary).

Similarly, the selectionism of operant learning is only analogous to the selectionism of evolutionary biology, it is not homologous; the behavioral and biological processes underlying operant learning differ from those underlying biological evolution. However, it makes sense that both biological evolution and operant learning should be selectionistic. Something like selectionism seems almost required, in both cases; otherwise, life would be much more chaotic than it is. But it also makes sense that these two examples of selectionism have evolved (been selected) as independent reactions to the demands of survival in the same environment. Biological evolution and operant learning have evolved analogously, not homologously.

In conclusion and as a behavior analyst, I think the authors have provided a valuable service in separating the essential features of a selectionist model (i.e., replication, variation, and environmental selection) from the unessential features of specific instances of that model (e.g., replication of organisms across space, in the case of biological evolution, vs. replication of responses across time, in the case of operant learning).

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How (and why) Darwinian selection restricts environmental feedback

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Abstract: Selectionist models date back to Empedocles in Ancient Greece. The novelty of Darwinian selection is that it is able to produce adaptively valuable things without being sensitive to adaptive value. Darwin achieved this result by a restriction of environmental feedback to the replicative process. Immune system selection definitely does not respect this restriction, and it is doubtful whether operant learning does.

Invest $100 in each of funds A and B. Both grow at a steady (possibly negative) rates, A faster than B. Your overall growth rate changes over time though the sub-rates stay steady: at first, the overall rate is the average of the two sub-rates, but it approaches the A-rate as A begins to dominate your account. The speed with which this change takes place is proportionate to the *variance* of the sub-rates. This is an instance of Li's theorem, itself a simplification of Fisher"s "fundamental theorem of natural selection" (Edwards 1994). Fisherian evolution is driven by differential growth rates, which can arise out of a great variety of causal arrangements. The fascinating question broached in the target article is: How many of these causal arrangements share the essential structure of natural selection?

Hull et al. claim that the kind of causation exhibited by selection is "counter-intuitive." This is very different from claiming that it is counter-intuitive to think that selection might be responsible for the complex adaptations displayed by natural organisms. In ancient Greece, Aristotle had already disputed a selectionist theory concerning adaptation, which he summarized as follows:

> Wherever all the parts came about just as they would have been if they had come to be for an end, such things survived, being organized spontaneously in a fitting way; whereas those which grew otherwise perished and continue to perish, as Empedocles says his "man-faced ox-progeny" did. (*Physics* II 8, 199a5–8 = Barnes 1984, p. 339)

Aristotle did not have any difficulty explicating selection itself; his difficulty lay rather in the use of an adaptation-blind process of generation to explain adaptation. Empedocles had no way of ensuring that selected traits were retained in the population. If his process of "spontaneous organization" (just a randomized assembly of organs) was really adaptation-blind – insensitive to the adaptive value of its products – we should expect monstrosities to be the norm rather than the exception.

Empedocles's omission is corrected by noting that if traits are inherited rather than assembled afresh in each generation, population proportions can be passed on. We need, then, to posit *two* processes. The first is replication-with-copying-errors, which accounts both for the retention of traits from one generation to the next, and for novelty. The second, which the authors call "environmental interaction" (EI) alters the proportions of pre-existent traits relative to the population. (The authors are right to say that the units-of-selection debate is greatly clarified by this idea.) EI is conservative in the sense that it produces nothing, old or new.

The core of Darwin's conceptual revolution lay in his thesis that (with some exceptions) EI has restricted feedback to replication. The altered proportions which are the "output" of interaction in one generation are passed on as the "input" for the next round of replication. (The authors are right to insist that selection is iterative; a one-stage "big-bang" process is "at most a limiting case.") Beyond this, EI cannot tamper with the productive process, for instance by replicating traits acquired during EI, or amplifying the replication of favoured traits. This is the restricted feedback condition.

Because environments are highly variable, there are exceedingly few traits or functions universally punished or universally rewarded by EI. In most cases, the process will result in an increase in complexity of organization as modifications accumulate, but precisely what functions will emerge is unpredictable. Darwinian adaptation is contingent upon circumstance. Call this the relativity condition.

Restricted feedback is central to the Darwinian idea that complex adaptation is achieved by an adaptation-blind process. Here's an example of a violation. The "Delta Rule""is used to train neural networks by comparing their output to the output desired by a programmer, and adjusting connection weights when their performance deviates. This process might be considered conservative because there is an element of randomness in network building – networks are not purpose-designed, their properties emerge. Nevertheless, Delta Rule training is the antithesis of Darwinian selection, because the process that generates output – the replication analogue – is adjusted in accordance with an above-the-fray evaluation of earlier outcomes. This violates both the conditions stated above. A similar observation tells against the authors' argument that Darwinian theory can accommodate Lamarckian inheritance. Only at the cost (which, admittedly, Darwin himself was sometimes prepared to assume) of abandoning the idea that EI is adaptation-blind, and reverting to an absolutist conception of adaptation.

Somatic selection in the immune system offends the Darwinian ideal in much the same way. Somatic selection is guided by system function, the toleration of self and the elimination of nonself. Thus: (1) B-cells are randomly generated, but are subject to "negative selection" when they react with self components. In this respect, immune selection is like *artificial* selection in which a breeder selects with the intention of achieving a particular result. Further: (2) B-cell replication is triggered by binding to antigen. Here the system is arranged to do what even breeders cannot do – to reach in and turn up the reproduction rate of favoured variants. Viewed close-up, the immune process displays a structural similarity with biological evolution. The appearance is misleading, not just because of the existence of a system function – biological evolution is unique in its lack of system function – but because "environmental" feedback directly affects replication rates.

Does operant learning violate Darwinism? Actually this question is more murky than might appear from the discussion of Delta Rule training above. Are different behaviours produced by different processes, none of which are affected, but some of which are eliminated, by environmental conditioning? Or is there a single behavior-production process that is modified in response to feedback? In my view, only the former would display appropriate independence, but the difference is probably notational, not empirical.

My point is that in Darwinian selection, environmental feedback is not allowed to modify replication, only its input. I emphasize the adaptation-blindness of natural selection, the difference between instruction and selection. The authors are more interested in the conservative character of selection, and whether "operant behaviour fits the present environment because of past selection and not because of any future state of affairs." No doubt, the choice between the two perspectives is ultimately context relative.

Evolution and operant behavior, metaphor or theory?

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Abstract: The idea that similar selective processes operate in gene-based evolution, immunology, and operant psychology provides an intuitively ap-

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pealing metaphor. This idea also isolates questions that operant psychologists should ask and makes some empirical predictions. However, the idea currently lacks the detail needed to precisely separate it from some plausible alternatives. This sort of thinking is the kind that operant psychologists should do if operant theorizing is to survive the competition among ideas.

We are behavior specialists. Therefore, we will comment only on the part of Hull et al.'s argument that applies to operant behavior.

The authors raise one of the most fundamental questions in operant psychology: Why do behaviors change in frequency as a result of their consequences? They propose an evolutionary metaphor. The animal varies its behavior. The behavior is selected for if it produces a positive consequence and selected against if it produces a negative consequence.

The idea that operant behavior is governed by a selective process similar to that seen in gene-based evolution has intuitive appeal. The authors describe Darwin as showing how biological adaptations can be explained by a mechanical historical process without appeal to a creator or to terms such as "benefit" or "harm." It seems reasonable to similarly describe operant psychology as showing that some behavior can be explained by a mechanical historical process without an appeal to future purpose or to terms such as "pleasure" or "pain."

The authors' more detailed view of selection as repeated cycles of replication, variation and environmental interaction also has intuitive appeal. Behavior exhibits a great deal of variability. Operant psychologists change the interaction between behavior and the environment (i.e., the consequences of a behavior) and they observe the effect on the replication of that behavior (i.e., on the future frequency of the behavior). The authors' division of selection into replication and environmental interaction seems particularly promising to us. It makes the role of the response clearer. As the authors argue, this distinction may also help to clarify the relationship between the study of behavior and neuroscience.

Although the present model provides an appealing metaphor, more specificity will be needed if it is to become an acceptable theory. The model does identify certain variables for study, as a theory should. As the authors note, to observe selective processes, psychologists should study the acquisition of behavior. Studying acquisition requires a shift from the current operant practice of examining performance primarily at asymptote (i.e., steady state behavior). To observe selective processes, psychologists should also study changes in behavior over time. This would return operant psychology to its earlier study of the distribution of behavior in time in contrast to the current practice of averaging behavior over large time units.

The model is also detailed enough to make some empirical predictions. For example, the authors argue that all reinforcement-sensitive properties of a reinforced response (e.g., its force, duration, latency) will replicate (increase in frequency), not just those properties on which the reinforcer actually depends. Eventually, however, selective pressure will maintain only those properties of the behavior that fit the environment. Although it is impossible to measure all characteristics of a behavior, existing technology would allow an experimenter to measure several aspects and to make the delivery of the reinforcer contingent on one of them. If the authors are correct, then all of the measured properties of a response that immediately precedes a reinforcer should be similar to the properties of the response that immediately follows the reinforcer. That is, the two responses should be similar in force, duration, latency, and so on. Responses that are separated by several reinforcers should continue to show a strong positive correlation on the dimension of the response that was actually reinforced (e.g., its duration). The similarity should grow weaker with greater separation for those characteristics of the response that were not reinforced (e.g., force, latency).

However, the model is not yet precise enough to allow its separation from some plausible alternatives. For example, reinforcers and punishers might directly modify or mold the characteristics of a single behavior, rather than select one behavior from among a number of alternatives. On the surface, one difference between this idea and the authors' model seems to be whether the environment chooses from among many behaviors or works directly on only one. The authors argue that operant behavior does not seem to involve the multiple copies of a response required by a selective model, but they conclude that selection may occur in the absence of multiple copies. More details about the operation of selection are needed before the selective model can be separated from this alternative view.

This criticism will not surprise the authors. They acknowledge early in the paper, that an analysis that is too broad will account for everything and an analysis that is too narrow will account for nothing. The problem is hitting exactly the right level of detail. We also acknowledge that our inability to make the needed predictions may be our failing rather than the authors. We lack the detailed understanding of evolution necessary for such predictions. A second paper that elaborated some specific empirical predictions of this model would be useful to us.

As a final comment, operant procedures have competed well in the marketplace of practical applications. Many scientists acknowledge that operant techniques provide useful baselines for assessing the effects of drugs, physiological manipulations, knockout genes, and so on. Most would also agree that operant techniques provide useful ways of dealing with many human behavioral problems (e.g., autism). In contrast, operant theorizing has not competed particularly well among scientific theories and some psychologists reject the idea that operant theorizing can contribute to an understanding of behavior. It seems to us that if operant theorizing is to survive, we must have more papers such as the present one in which operant psychologists work with scientists from other areas to address topics of mutual interest. Such interactions are exciting because, if the ideas are correct, then the detailed information that is available in one area of study can be used to quickly increase our understanding of another area of study. This sort of interaction could give operant principles the selective advantage necessary to become better accepted as powerful explanations for behavior.

Operant behavior and the thesis of "selection by consequences"

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Abstract: Behavioral theorists such as B. F. Skinner have argued that the thesis of selection by consequences applies to behavior just as much as to morphology. This commentary specifically examines certain respects in which the thesis of "selection by consequences" applies to the development of ontogenic operant behavior.

Operant behavior and the thesis of "selection by consequences." As the target article indicates, the behaviorist B. F. Skinner has sought to apply the thesis of "selection by consequences" as a causal mode to explain the development of certain forms of behavior. That is, just as interactions with the environment, most notably differential access to life maintaining resources and hence survival, are said to select from a population of organisms with varying body traits and characteristics, so also might interactions with the environment be said to select the behavior of organisms. For example, some instances of innate behavior, such as taxes, kineses, fixed action patterns, and reflexes might develop phylogenically from a population of such innate responses because they favor survival directly or because they are an incidental by-product of other features that have arisen through selection. This commentary specifically examines certain respects

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in which the thesis of "selection by consequences" applies to the development of ontogenic operant behavior.

In operant behavior, a response in the presence of an antecedent stimulus increases in frequency because of its prior consequences, which are called reinforcers. Reinforcers might be said to select a response from a prior population of otherwise "uncommitted" responses. Thus, a response occurs for some other reason first, and then becomes an operant.

Certainly an organism is more likely to survive if the consequences of its past responses affect its future behavior. In such an organism, the environmental consequences of its actions presumably change the state of its underlying neural systems, and the behavior becomes more frequent because those consequences produce those neural changes. Organisms whose nervous systems do change in this way flourish and reproduce. For example, the behavior of seeking food or avoiding predators might be strengthened, yielding obvious survival advantages. Again, the consequence does not always have to be directly related to a life-maintaining event, as in Clark Hull's drive-reduction, only to a process that proves valuable in some other sense and the susceptibility to reinforcement is passed on to future generations.

The suggested means of replication, retention, and transmission of operant behavior is "neural changes." The term is admittedly vague, but again there is a parallel with genetics. Prior to the discovery of DNA, the science of genetics was concerned with terminal relations. For example, Mendel was able to formulate quite accurate quantitative relations between parents and offspring of pea plants with respect to, say, smooth versus wrinkled skin. Mendel did not have any way of specifying the means by which those traits and characteristics were replicated, retained, and transmitted. That specification awaited the discovery of DNA and the grand synthesis. In any event, the discovery of DNA did not disprove Mendel's quantitative terminal relations, which remain as accurate as always.

Similarly, the science of behavior is concerned with terminal relations. It can formulate quantitative statements about the relation between certain environmental variables and resulting behavior, but not about any neurophysiological mechanisms according to which those terminal relations are replicated, retained, and transmitted. In that sense, a science of behavior awaits a contribution from neurology and behavioral neuroscience that is comparable to that which genetics awaited from biochemistry and DNA. Perhaps that contribution will involve synaptic mechanisms, long-term potentiation, or even adaptive neural networks. In any event, the contribution will not disprove the quantitative terminal relations developed in a science of behavior, which will remain as accurate as always.

Also interesting, although the development of morphology and innate behavior proceed according to Darwinian natural selection, the development of operant behavior might be said to proceed according to Lamarckian principles. That is, in the case of operant behavior, we are talking about the same response occurring more frequently in the same organism, rather than in future organisms. If we look to the nervous system that underlies operant behavior in that organism, we can see that a given set of neurons changes because of the environmental interaction called reinforcement. Those changes are then retained in the same neurons, such that the changed neurons contribute during the lifetime of the organism to future operant behavior of the new type. The neural system does not initially contain some mixture of unconditioned and already conditioned neurons (whatever "conditioned" will prove to mean), and the unconditioned neurons do not perish, to be replaced by new conditioned neurons by virtue of some interaction with the environment over time. Recall that the response occurred for some other reason first, meaning that the neurons were initially in the state appropriate to why the behavior was occurring. No neurons could be in the conditioned state initially because they had not yet experienced reinforcement.

In closing, perhaps the most important point to make about applying the thesis of selection by consequences to behavior is that it does away with the self as an initiator or creator. That is, selection as a causal mode in biology does away with purposeful "grand designs," but as Skinner cryptically points out, a comparable viewpoint regarding selection as a causal mode in the analysis of behavior has been a long time coming. The traditional view of the "free" individual initiating action according to mental or psychic processes still predominates. From Skinner's perspective, the strength of the traditional view is attributable to various mentalistic metaphors and myths that are cherished for extraneous and irrelevant reasons. Cognitive psychology and cognitive neuroscience are particularly pernicious examples to his way of thinking ("Cognitive science is most 'fertile' in breeding promises of great achievement. . . . The achievements have yet to be realized," Skinner 1988a, pp. 64–65). When Skinner (1988b) said that

> The explanatory terms which have been used for more than 2,000 years to explain human behavior are troublesome not because they raise questions about dimensions but because they assign the initiation of behavior to the person rather than to the person's genetic and behavioral history. . . . What causes trouble is the usurpation of the initiating role of the environment (p. 204)

he was suggesting nothing less than that the thesis of selection by consequences brings the interaction with the environment we call behavior squarely into the realm of science, as a subject matter in its own right.

A more pluralist typology of selection processes

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Abstract: Instead of using only one notion of selection I argue for a broader typology of different *types* of selection. Three such types are differentiated, namely simple one-step selection, iterated one-step selection, and multi-step selection. It is argued that this more general and more inclusive typology might face more effectively the possible challenges of a general account of selection.

As evolutionary approach is getting more and more popular in a large number of different disciplines, there is a strong need for a typology that would show what is similar and what is different in all these evolutionary models. The endeavor of the target article is of key importance in this respect, since it outlines a conceptual framework in which different selection processes of different research fields could be compared.

Hull et al. restrict themselves to three such phenomena though. Besides, they draw a strict division line between them and the rest of evolutionary processes, claiming that only these three processes count as selection processes. Even if they do not exclude the possibility of this list becoming longer in the future, they do not discuss any phenomena not fulfilling the criteria for being a selection process.

To put it differently, the target article focuses on what selection is, whereas I would like to address the differences between various forms of selection. According to the article a phenomenon is either selection or it is not. My question is about *what type* of selection it is.

I suspect the general line of critical responses to the target article will consist of arguments why one of the three processes does not fit the general definition of selection provided by the authors. The more pluralistic typology I tend to favor would offer a way of defense against these objections. Even if one of the processes covered by the authors turns out not to fulfill all the criteria of selection as defined in the article, it might fulfil a weaker criterion of selection.

At this point three types of selection processes has to be differentiated. I use the term *multi-step selection* as a synonym of what

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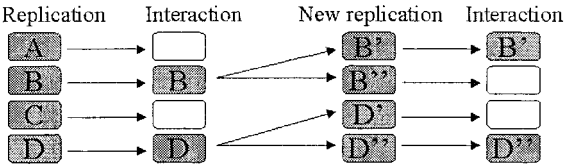


Figure 1 (Nanay). The replication-interaction cycles in multi-step selection processes.

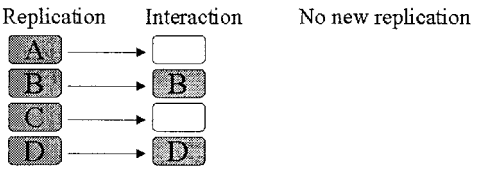


Figure 3 (Nanay). The one replication-interaction cycle in simple one-step selection processes.

the target article considers as selection proper, defining it as "repeated cycles of replication and environmental interaction so structured that environmental interaction causes replication to be differential." Besides multi-step selection, two further selection processes are included in the typology.

I call *iterated one-step selection* the process whereby there are repeated cycles of replication and environmental interaction, but where environmental interaction does not cause replication to be differential. In other words, environmental interaction does not influence the next replication. In the case of *simple one-step selection,* replication is followed by environmental interaction, but no new replication occurs after that: this is the end of the story.

The differences of these three processes are summarized in Figures 1, 2, and 3.

An important example of simple one-step selection is the development of the central nervous system (Adam 1998; Changeux 1985; Edelman 1987). From the initial variety of neural connections some are selected by environmental interaction, whereas other connections disappear during the development. No further replication takes place after the environmental interaction, since no (or very few) new neural connections are formed later.

It is more difficult to find a clear example of iterated one-step selection. Nevertheless, the otherwise highly controversial explanatory model of evolutionary epistemology seems to imply a version of such selection process, which can be examined without committing to the credibility of evolutionary epistemology as such (Campbell 1974; Popper 1972). According to this theory, all thinking processes can be characterized by repeated cycles of blind variation and selective retention. A variety of thoughts is produced continuously and blindly, but environmental interactions decide which thought will survive. These environmental interactions, however, do not have any impact on the next variation of thoughts (this characteristic of variation is dubbed as "blind" by Campbell).

Single one-step selection plays a central role in an earlier typology of selection processes outlined by Darden and Cain (1989). According to them, the criterion of selection is that replication is followed by environmental interaction, but this cycle need not be repeated. One-step selection is not only included in their typology, but it is also conceived as the paradigmatic case of all selection processes. The target article argues powerfully against the central role of single one-step processes in describing selection, but these arguments do not support their conclusion that "single cycle selection is (at most) a limiting case" of an account of selection processes. In my view, both one-step selection à la Darden and Cain and multi-step selection discussed in the target article could be included in the same typology as simple and more complex case of the same phenomenon.

It is important to point out that there is no strict boundary between the three types of selection processes outlined above. During the development of the central nervous system, for example, more selection takes place after the initial simple one-step selection, since some new neural connections are formed even in the adult brain. The distinction between iterated one-step selection and multi-step selection is even more difficult to draw. In the latter environmental interaction causes replication to be differential, whereas in the former it does not. This effect of environmental interaction on differential replication, however, is a matter of degree. The case in which environmental interaction has no influence on how differential the next replication will be, and the case in which it fully determines what replicators can and cannot replicate in the next generation (in gene-based biological selection for example) are just the two extremes. It can be argued that most cases of selection involving repeated cycles lie somewhere in between these two extremes. Operant conditioning might be an example for this.

If the difference between iterated one-step selection and multi-step selection is indeed a matter of degree, then a more pluralistic typology could provide the conceptual framework for examining both types of selection processes as well as the gradual transition between them. Even if this transition is not gradual, the pluralistic typology would provide a better framework for comparing multi-step selection with simpler selection processes. But if it is, the pluralist approach becomes probably the only way of defending the general project of accomplishing the endeavor of the article: of giving a general account of selection.

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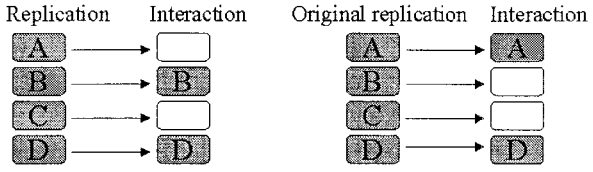


Figure 2 (Nanay). The replication-interaction cycles in iterated one-step selection processes.

"Which processes are selection processes?"

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Abstract: I argue that population-level selection does not necessarily have to be invoked to explain the polymorphism at the MHC locus. I argue that the authors' attempt to model operant conditioning in Darwinian terms faces a serious problem. Depending on how many operant responses we take to comprise a sequence, different conclusions about whether or not evolution is occurring in an operant lineage will be reached.

The authors' abstract characterisation of natural selection as a two-stage process involving differential replication caused by environmental interaction seems to me absolutely correct. I have one query concerning their account of selection in the immune system, one concerning their treatment of operant learning, and one general methodological remark.

In sections 4.4–4.5, the authors argue that a process of population-level or "group" selection must be invoked to explain the polymorphism at the MHC locus. I am unconvinced. An alternative explanation is that *frequency-dependent* selection is responsible for establishing and maintaining the polymorphism. The difference between these two explanations is as follows. In population-level/group selection, as these terms are usually used, two or more populations/groups must exist, which differ with re-

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spect to some group-level trait (e.g., the frequency of a particular allele), leading the groups to have different fitnesses. Frequency-dependent selection, by contrast, requires only a *single* population, with the fitness of any individual allele dependent on the number of copies of the allele in the population. Frequency-dependence is a standard way of explaining polymorphism *without* appealing to higher-level selection, and seems straightforwardly applicable to the alleles at the MHC locus coding for different peptide binding specificities. The fitness of any given allele will be low if many other individuals in the population possess it – as this raises the chance that the virus will have evolved a way to defeat the MHC system; conversely, a rare allele will have a fitness advantage. So instead of one allele spreading to fixation, a stable polymorphism is maintained.

It is possible that the authors understand the notion of group or population-level selection in such a way that every case of frequency-dependence counts as group or population-level selection. There is a tradition of thinking about higher-level selection in this way (e.g., Sober & Wilson 1998), but the majority of biologists agree with Maynard Smith (1976) that frequency-dependence and group selection are distinct causal processes. (See Okasha 2001 for discussion of this debate.) The authors need to clarify their stance on the issue of frequency-dependence.

The authors note a significant disanalogy between operant learning and standard examples of Darwinian selection. In the latter, the variants on which selection operates are present at the same time, while in the former the variants form a temporal sequence, each one existing for a moment before being replaced by another. The authors do not think this disqualifies operant learning from counting as a Darwinian process, but I am less sure. At the very least, it complicates matters considerably. The standard definition of evolution is "change in replicator frequency from one time to the next," but this definition cannot strictly apply unless the variant replicators do actually exist at the same time. To model operant learning as a selection process, the authors thus need a liberalised notion of evolution, presumably along the following lines. We consider a temporal sequence of (say) 10 operant responses and note the frequency of each type of response. We then look at a further 10 responses, and see if the response frequency has changed or not. If it has then evolution has occurred, if not. However, the results of this procedure depend crucially on how many responses we take to comprise the relevant sequence length. To see this, suppose there are just two types of response in a particular lineage, A and B. Suppose the first twenty responses in the lineage are as follows:

AABBA BAABB AAAAA BBBBB

If we take our sequence-length to be 10 responses long, we will conclude that no evolution has taken place, for the relative frequency of A and B is the same in the two successive 10-response sequences. But if we take our sequence-length to be five responses long, we will reach different conclusions. Relative to this way of dividing the lineage, no evolution has taken place between sequences 1 and 2, but between sequences 2 and 3 and sequences 3 and 4, evolution *has* occurred. Unless a principled way of choosing sequence length is specified, the occurrence or otherwise of evolutionary change is not a determinate matter. And even if this problem were solved, it is unclear how the crucial distinction between evolution by natural selection and evolution through random drift could be applied to changes in response frequency in operant lineages. For these reasons I am unconvinced by the authors' attempt to model operant learning as a Darwinian process.

The foregoing remarks notwithstanding, I agree with the authors that gene-based biological evolution may not be the only natural process which exhibits the abstract Darwinian structure. Certainly this cannot be assumed a priori. But simply because it is *possible* to model a process in Darwinian or quasi-Darwinian terms does not necessarily imply that it is *useful* to do so. Darwin himself invoked natural selection to explain the existence of adaptation in nature – a phenomenon which cries out for scientific explanation and was conspicuously lacking one until Darwin's own theory. But in many of the recent attempts to discern Darwinian processes at work in other domains, for example, in the realm of human culture, there is no comparable phenomenon which clearly requires, but totally lacks, a proper causal explanation (cf. Sterelny & Kitcher 1998, pp. 333–34). I would suggest that the same holds true of operant conditioning and the reaction of the immune system to antigens. This is not to cast doubt on the authors' careful, and very interesting, attempt to extend traditional Darwinian principles into these domains; it is rather to stress the uniqueness of the phenomenon which those principles were originally invented to explain.

Are theories of selection necessary?

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Abstract: This commentary is an attempt to sharpen some of the issues raised in the paper and thereby increase the generality of the proposal. Some implications of an exact definition of behavior for strategies of measurement and hence behavioral variability are discussed. The role of both respondent and operant behavior in natural selection is emphasized.

By attempting to offer an account of selection that is consistent across the diverse domains of biology, immunology, and behavior, Hull et al. would seem to answer this question in the negative. One theory should suffice, providing it is not so abstract and vague as to be largely useless. Theirs is a noble undertaking and will be a valuable addition to the reading list of every student of the natural science of behavior. My purpose in this commentary is to sharpen some of the issues and thereby, paradoxically, attempt to increase the generality of the formulation.

What is behavior? Johnston and Pennypacker (1980) proposed a definition of behavior that leads nicely to the conceptualizations in the present paper. Their definition reads "The behavior of an organism is that portion of the organism's interaction with its environment that is characterized by detectable displacement in space through time of some part of the organism and that results in a measurable change in at least one aspect of the environment" (p. 48). There are some implications of this definition that have immediate bearing on the Hull et al. position. First of all, the environment as used in this definition is not limited to that portion of the world external to the organism's skin; there is an internal environment as well. For consistency and completeness of the theory, selectionist principles must be equally valid in the internal environment.

Second, the definition embraces both operant and respondent behavior. It is important to note that operant behavior, as a subset of all behavior, is defined by its effect on the environment, not by its selecting consequences. Although the authors do not make this error, many readers may be confused on this point. More important, although respondent behavior at the individual level is clearly not modified by a process of selection as is operant behavior, it is intimately involved with that selection mechanism (contingencies of reinforcement). It may, from an evolutionary standpoint, be more fundamental in determining the adaptation and survival of the individual organism (see below).

Finally, this definition of behavior leads to the identification of the natural dimensions of behavior and from these to units of measurement similar to the c.g.s. system in the physical sciences. This practice serves to distinguish the science of behavior from the social sciences, placing it squarely in the company of the biological sciences. Some examples of these dimensions are furnished by Hull et al. and are requisite to any quantitative description of the dynamics of behavior undergoing modification by selection.

What is the nature of behavioral variability? The basic dimen-

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sions of behavior proposed by Johnston and Pennypacker (1980) are latency, duration, and countability. These are characteristic of every member of a response class. Secondary dimensions such as frequency and acceleration result readily from combinations of the basic dimensions. Variability, according to this view, is not a basic dimension of behavior. Rather, it is a characterization imposed by observers upon a collection of measures of individual responses. The assertion that variability is a fundamental dimension of behavior leads readily to the assertion that it is intrinsic (Sidman 1960), a status that forecloses the search for its determinants. It also leads readily to the practice of enlisting the variability as a basis for reifying suspected causes, as Quetelet did in the 1830s and for creating relative units of measurement as Fechner and Galton did late in the nineteenth century. From here, the regression to social science methodology is almost complete.

What is the role of behavior in evolution? Much of what I know about natural selection I learned from my lifelong friend and colleague, R. Bruce Masterton (Pennypacker 1999). In particular, he taught me that although the object of natural selection is usually the adult, the product of the process is another infant. Bruce was fond of quoting Gaylord Simpson (1949): "Hens are an egg's way of begetting another egg" (Masterton 1998).

Often overlooked by biologists and behavior analysts alike is the fact that very few infants reproduce. In order to transmit its genetic material into a subsequent generation, an organism must first adapt and survive to the point where it can reproduce. For most vertebrates, at least, a major modulus of this survival is behavior. Thus, natural selection occurs with respect to behavior in a fundamental sense. Those individuals who do not behave effectively are not around to beget new infants and whatever morphological or neurological characteristics they possessed that contributed to their demise will likewise not be reproduced. Selection occurs, therefore, with respect to those characteristics (morphological and physiological) than enable successful behavior. Any efforts to isolate those mechanisms and explicate their role in the selection of operant behavior must first deal with fact that they are themselves the products of selection. Candidates for inclusion in this universe are the sensory and physiological mechanisms that come into play when reinforcement occurs, when conditioned reinforcement (likely a respondent process) is developed and when certain stimuli become discriminative for the occurrence of operant behavior.

Do some or all of these mechanisms develop ontogenetically as a result of a selection mechanism as is proposed in the case of the immune system, or are they fully developed in the genotype, awaiting expression by the right combination of environmental events?

Conclusion. This theoretical formulation, as the authors point out, offers promise for meaningful interdisciplinary research. It suggests important research questions, such as what are the details of the change in the nervous system as an operant repertoire is acquired? What is the role of private verbal behavior in adaptation to a changing social environment? Questions as complex as these may now be addressed in a formidable multidisciplinary context. The rapprochement that Skinner anticipated in 1938 and 1950 may finally be at hand and it is to these authors' credit that they will have hastened its arrival.

Selection without multiple replicators?

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Abstract: Hull et al.'s construction of operant learning as an instance of selection gives rise to problems that weaken this application of selection theory beyond acceptable limits. We point out that most fundamental is a disregard for the need to include multiple concurrent replicators in any definition of selection and indicate how this problem may be solved.

Of the three phenomena considered in the target article, the analysis of operant learning is the most challenging and problematic. Some of the obstacles are due to our lack of knowledge. For example, it is not clear what would play the role of replicators in operant learning, or how feedback between environmental interaction and differential replication would be mediated. But an even more fundamental issue concerns the role of multiple concurrent replicators that differ in their replication rates. We will focus on this point because we believe it can be resolved in principle, without any need for further empirical studies of behavior or neurobiology.

The issue is raised by the authors in their introduction: "In operant learning, selection occurs only with respect to sequences of environmental interaction rather than with respect to numerous concurrent alternatives. Is this difference sufficient to disqualify it as a case of selection?"

The question would seem to be a matter of definition. In their general description of selection, the authors define it as "repeated cycles of replication, variation, and environmental interaction so structured that environmental interaction causes replication to be differential." The standard interpretation of the word "differential" is that in each cycle, multiple replicators differ in the extent to which they replicate. Hull et al. seem to concur with this interpretation, stating that, "variants must be linked to proliferation so that at any one time, numerous alternatives are available for selection."

Thus it would seem clear that selection requires multiple replicators by definition. When the authors discuss operant learning, however, they frame the theory in terms of a single replicator at any given time. They clearly recognize the problem this raises: "If the environment must have multiple and differing copies of a replicator concurrently available for selection to occur, operant behaviors seems definitionally excluded." However, they go on to conclude rather surprisingly that this requirement does not apply. They reason that, "there appears to be no reason to assume that all replication processes involve concurrently existing events or objects." That is certainly true, but replication is not the same as selection. With little further discussion of the problem, they decide in their conclusion that, "replicators that do not proliferate in this way also count as instances of selection."

This leaves unresolved contradictions between the authors' general definition of selection and their specific conclusion about operant learning. This new interpretation of selection also includes a much wider range of phenomena than the original definition. For example, imagine a bird that molts its feathers each year, then produces a new "generation" of feathers that is similar but not identical to the previous year's. This would fit the description of a process that retains features of an object across generations, with a mechanism of variation to introduce novelty. But surely we would not want to call this selection. Indeed this seems a clear example of the mere persistence of patterns, which the authors in their introduction explicitly exclude. Even if the sequence of plumages showed "improvement" over time by some criterion, it would not be by means of differential replication, and it would not be through a process of selection.

Is the definition of selection ultimately a matter of taste or semantics, with one answer being as good as another? It is not, in part because the theory of selection has developed beyond mere verbal argument. A tradition of rigorous mathematical description of the selection process in biology provides a foundation for developing a general theory. All formal representations of selection are quite explicit about the requirement for variation among multiple concurrent alternatives. This includes Fisher's "fundamental theorem of natural selection" (Fisher 1958), the Price equation (Price 1970), and the replicator equation (Schuster & Sigmund 1983). Here the role of variation is not just qualitative, but appears

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as a quantitative expression of the rate of change as a function of the genetic or phenotypic variance present at a given point in time.

To pursue the biological analogy, imagine an organism that consistently produces a single offspring and then dies. If we allow for heritability and mutation, and analyze this situation using the mathematics of selection theory we will inevitably conclude, quite correctly, that natural selection does not occur because there are no variants to select among. The lineage either persists or ends, but it will not generate adaptation. The same reasoning applies to operant behavior, for the same reasons.

Does this mean that the project of explaining operant learning as a selective process is doomed to failure? We do not believe so, because there is no need to envision the process as involving only a single replicator at any given time. If replicators consist of specific neural configurations that produce tendencies or proclivities for certain behaviors, it is not hard to imagine a population of such replicators that compete for the opportunity to be expressed as behaviors (interactors), and to be thereby strengthened or weakened according to their relative "success" (e.g., in eliciting positive affect). It is also not hard to envision that stronger neural configurations would be more likely both to persist and to spawn variants.

The formal structure of selection theory can be applied to such a scenario without any major conceptual obstacles. We could even envision the possibility that selection occurs among a set of concurrent alternatives previously generated in sequential order. Given a straightforward conceptual solution, what remains is only that the empirical aspects be clarified and tested. Indeed, a start has already been made on developing the quantitative selection theory developed in biology into a broader account of selection in general (Price 1995), and of learning in particular (Frank 1997). We think this approach holds considerable promise, and we urge the authors as well as other workers to forge ahead on this exciting endeavor.

Activity anorexia: Biological, behavioral, and neural levels of selection

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Abstract: Activity anorexia illustrates selection of behavior at the biological, behavioral, and neural levels. Based on evolutionary history, food depletion increases the reinforcement value of physical activity that, in turn, decreases the reinforcement effectiveness of eating – resulting in activity anorexia. Neural opiates participate in the selection of physical activity during periods of food depletion.

Selection of operant behavior by contingencies of reinforcement involves changes in an organism's neurophysiology and neurochemistry. Neural changes, in turn, participate as part of the contingencies of reinforcement selecting operant behavior. As Hull et al. point out, "the first step in operant selection occurs at the behavioral level [at the interface between the organism and environment] . . . And the second step occurs inside the organism at the neural level." The target article, therefore, raises the fascinating problem of the interplay of events outside and within the organism (see Skinner 1969, pp. 282–84 on behavior, and the nervous system). In this commentary, I explore the interrelationships between the biological, behavioral, and neural levels as illustrated by a biobehavioral analysis of activity anorexia.

In our laboratory, rats placed on food restriction and provided with a running wheel die of self-starvation (Epling et al. 1983; see also Routtenberg 1968). The wheel running of these animals increases exponentially over days. At the same time, the rats give up eating and their body weights plummet. Control animals, given the same food restriction but prevented from running, adapt to the reduced food supply and survive as healthy individuals. The laboratory model shows that food restriction induces physical activity that, in turn, suppresses eating. Epling and Pierce (1991) called this process activity anorexia.

A biobehavioral analysis of activity anorexia involves evolution and natural selection (i.e., the fit between phenotypes and environment as discussed by Hull et al.). For organisms faced with sporadic reductions in food supply (e.g., unpredictable famines), natural selection would have favored increased physical activity (see Mrosovsky & Barnes 1974 for cyclic reductions in food supply, hibernation, and anorexia; also see Mrosovsky & Sherry 1980 for a review of natural anorexias). That is, animals that traveled or migrated under conditions of food depletion contacted food, survived, and reproduced. Natural selection also would have favored anorexia during times of food-related travel. Under famine conditions, there would be a net negative energy balance between foraging for small, difficult to obtain food items and traveling to a more abundant food source. Animals that stopped to eat along the way would use up their energy stores and die. Those animals that gave up eating, and kept on going, often would have contacted a stable and abundant food source – increasing their reproductive success (see Epling & Pierce 1991; Pierce & Epling 1996).

For animals with this evolutionary history, we predicted that food depletion increases the reinforcement value of physical activity and that intense physical activity decreases the reinforcement effectiveness of eating. Pierce et al. (1986; Experiment 1) used male and female rats to test the reinforcement effectiveness of wheel running under different levels of food deprivation (see also Belke 1996). Animals were trained to press a retractable lever for 60 sec of wheel running. Next, we tested each animal at free feeding weight (100%) and at 75% of *ad libitum* weight on a progressive fixed-ratio schedule of reinforcement (an increasing fixed number of lever presses) for 60 sec of wheel running. The point at which the rats gave up lever pressing for an opportunity to run in a wheel was used as a measure of reinforcement effectiveness. Results indicated that, for each animal tested, wheel running sustained larger fixed ratios at 75% compared with 100% body weight. In terms of behavioral selection, our research shows that reductions in feeding enhanced the reinforcement value of physical activity. Additional research (Pierce et al. 1986; Exp. 2) indicates that increases in physical activity reduced the reinforcement value of eating (measured as the "give up" point on a progressive fixed ratio schedule of food reinforcement). These changes in reinforcement effectiveness insure that, during periods of food depletion, animals engage in physical activity rather than eating (i.e., activity anorexia).

At the neural level, endogenous opiates may function as part of the contingencies of food-related travel or physical activity. One possibility is that physical activity is partly maintained on a schedule of endogenous opiate release that requires more and more amounts of physical exertion (see Radosevich et al. 1989 on dose-response between intensity of physical activity and level of plasma β-endorphin). The endogenous reinforcement hypothesis suggests that injection of an opiate antagonist will decrease the level of wheel running of food-restricted animals. We tested the effects of the opiate antagonist, naloxone, on the wheel running of hungry rats (Pierce & Epling 1996). Rats were made hyperactive by restricting their feeding and providing a running wheel. Once wheel running stabilized, each rat was given injections of naloxone (50 mg/kg in saline) or saline (0.5 mL) on alternate days. Figure 1 shows wheel turns (1.1 meter per turn) for the one-hour period following the injection of the drug. For each animal, wheel running is reduced on days of naloxone compared with days of saline (control) injections. These findings suggest that the wheel running of food-restricted rats is increased by release of endogenous opiates – indicating that neurochemical changes are part of the contingencies regulating travel during periods of food depletion.

An important part of selection by consequences occurs during the lifetime of the individual. Our research on activity anorexia illustrates how environmental contingencies at the biological level (e.g., unpredictable food depletion) resulted in the motivational

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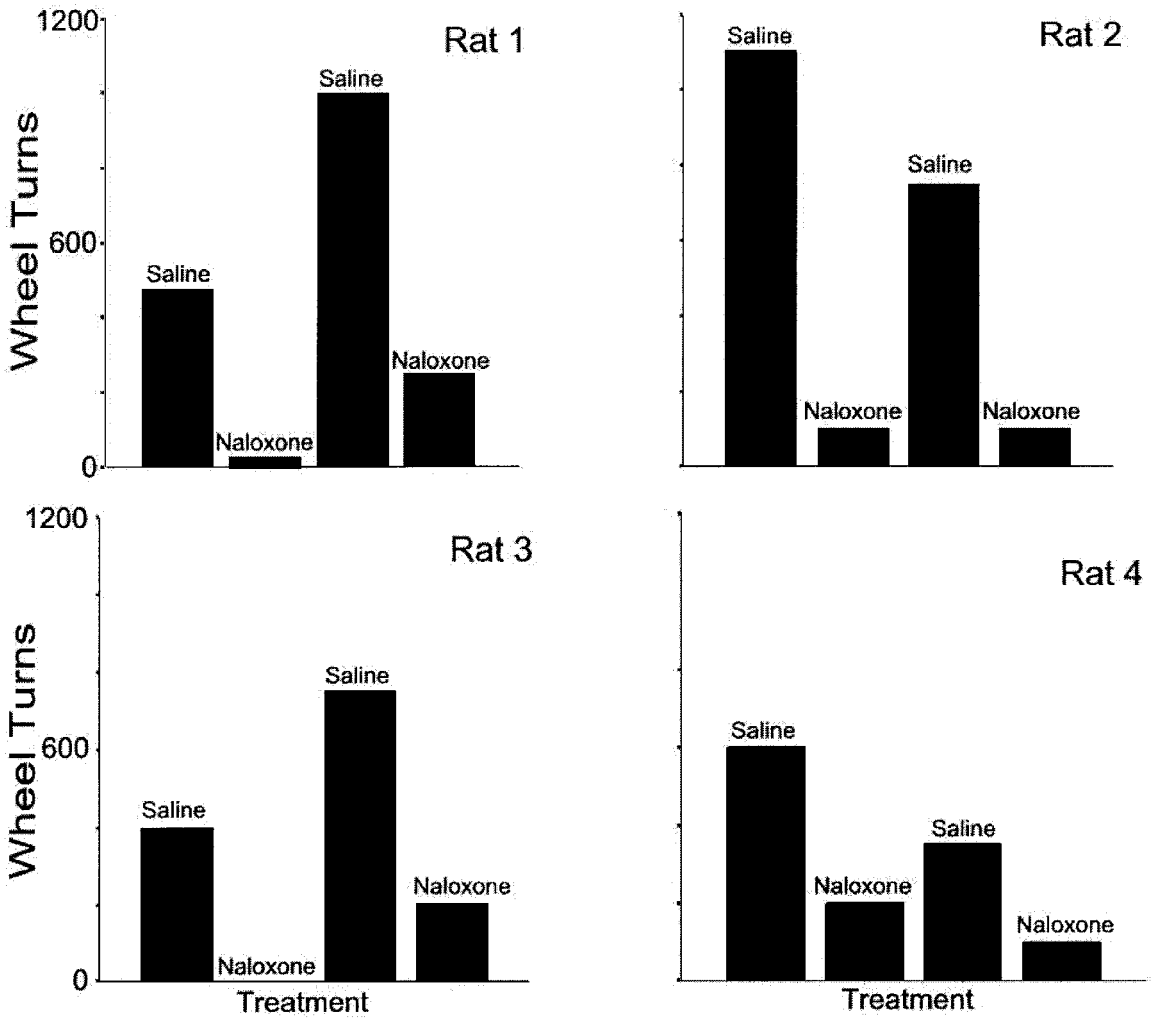


Figure 1 (Pierce). Number of wheel turns by four female rats in the first hour after injection of either saline (control) or naloxone (drug) on alternate days. The figure shows that, relative to saline treatment, naloxone consistently suppresses wheel running. The results suggest the involvement of endogenous opiates in the regulation of wheel running for food-restricted rats. Data are republished with permission from Pierce and Epling (1996, p. 73).

interrelations between feeding and physical activity during the lifetime of individual organisms. Changes in the reinforcement effectiveness of these behaviors during times of food depletion insure that animals travel when food is scarce and give up eating on a food-related trek. In addition, part of the food-travel contingencies involves neurochemical changes (e.g., release endogenous opiates) that participate in the regulation of food-seeking behavior. Overall, a biobehavioral analysis of activity anorexia supports the thesis of Hull et al. that behavioral and neural levels are interrelated in the selection of operant behavior and that behavioral selection ultimately arose from contingencies of survival or natural selection.

Replication in selective systems: Multiplicity of carriers, variation of information, iteration of encounters

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Abstract: An analysis of biological selection aimed at deriving a mechanism-independent definition removes Hull et al.'s obligatory requirement for replication of the carriers of information, under conditions, such as those obtaining in the nervous system, where the information content of a carrier can be modified without duplication by an amount controlled by the outcome of interactions with the environment.

Hull et al.'s reopening of the question of how to define selection in biology is very timely in view of our increasing awareness of the importance of selective processes in somatic as well as in evolutionary time frames. Unfortunately, rather than provide a principled analysis of the necessary and sufficient conditions for selection to take place, Hull et al. have given us an *ex cathedra* definition illuminated with examples. To illustrate how this approach might be strengthened, I will focus particularly on Hull et al.'s posited requirement for *replication.* By carefully considering the functional role of replication in the selection process, I arrive at a definition that is somewhat broader than that given in the target article.

Because I am going to explore whether replication is actually required in the variation-encounter scenario, I begin with a notion of selection, as the term is used in biology, that omits explicit mention of replication. According to this notion, the essence of selection is that a collection of variant elements of some kind interacts repeatedly with an environment in such a manner that some of the elements are favored over others. According to Hull et al., these

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elements can be genes in the case of biological evolution, B cells or T cells in the case of immunology, or "interactors" in the case of operant behavior.

Hull et al. quite correctly point out that selection must be cumulative in order to avoid a need for a prohibitively wasteful number of starting units. However, by failing to come to grips with the distinction between information and its material carrier(s), Hull et al. reject a whole class of possible selective systems with little or no replication that otherwise meet all the requirements.

What is absolutely indispensable for selection to be cumulative is that multiple units of information, embedded in encoded form in carriers of some kind, repeatedly encounter the environment, with the outcome of each encounter more favorable to some of the carriers than to others, depending on the information they contain. Between or during these encounters, the units of information must be subject to a process of blind variation. This variational process must be influenced by the results of the encounters in such a way that carriers containing the favored information and its variants constitute a larger portion of the population exposed to later encounters. The outcomes of successive encounter cycles may sometimes be more favorable to the variants than to their progenitors, allowing the population, over time, to respond in an ever more favorable manner to the environment.

Note, however, that although the total variation-encounter process must result in a new distribution of units with an increased probability of favorable encounters, there is no logical requirement that the number of units in the total population should increase. Indeed, even in the undisputedly selective case of biological evolution, total populations cannot increase as much as full exploitation of food resources occurs, rather, the distribution of variant forms in the population changes. Without an increase in the total population of units, the function of replication is reduced to providing a supply of variant units for new selection events. However, in a steady-state population, these new units can equally well come from variation in the information content of preexisting units, those that would be slated for death in the standard treatment of natural selection. Such variation might occur, for example, by changes in synaptic strength within groups of neurons.

Figure 1 illustrates one way that a population could increase its chances of favorable interactions with the environment via selection without replication of carrier units. In this example, selection influences the average magnitude, but not the direction, of the variation that occurs in each unit in each encounter cycle. (Compare Quartz & Sejnowski 1997.) Variation remains blind as to direction, but now the overall population moves toward the region of favorable encounter by modulating the amount of movement of each unit, rather than by eliminating some units and replicating others.

In the example illustrated, the states of the population in the schemes with and without replication are the same before and after the encounter-variation cycle, hence both schemes are equally admissible under any definition of selection that does not consider the internal mechanisms of the selection process. Such a definition would have the advantage of being applicable to analysis of a wider range of biological phenomena, including cases where mechanism has not been clearly established. The Hull et al. definition generally avoids reference to mechanism, but does incorporate it in the form of the explicit requirement for replication. However, under the analysis I have put forth, this requirement is unnecessary, and indeed, Hull et al.'s insistence on it appears quite arbitrary in this light. I suggest it should be eliminated, thereby providing an expanded definition of selection that is cleanly mechanism-free.

The broader definition permits a discussion of the possibility of selective mechanisms in the nervous system that are rejected a priori by the Hull et al. definition. Their treatment of this subject is somewhat inconsistent. In discussing development in the nervous system, they take the position that our knowledge of neural mechanisms is not yet sufficient to categorize such development as either selective or nonselective. At the same time, they consider the operation of the adult nervous system to be selective, at least in the operant conditioning case, with no more knowledge of mechanism than we have in the development case. In spite of their caveat about our poor knowledge of neural mechanisms, they venture, in their discussion of the immune system (sect. 4.3), to conclude by analogy that Edelman's (1987) proposals for selection in the nervous system are based on a "big bang" (noncumulative) mode of selection with the enormous waste that mechanism entails. I hope I have shown, however, that selection can occur cumulatively in collections of neurons or neuronal groups even in the absence of replication.

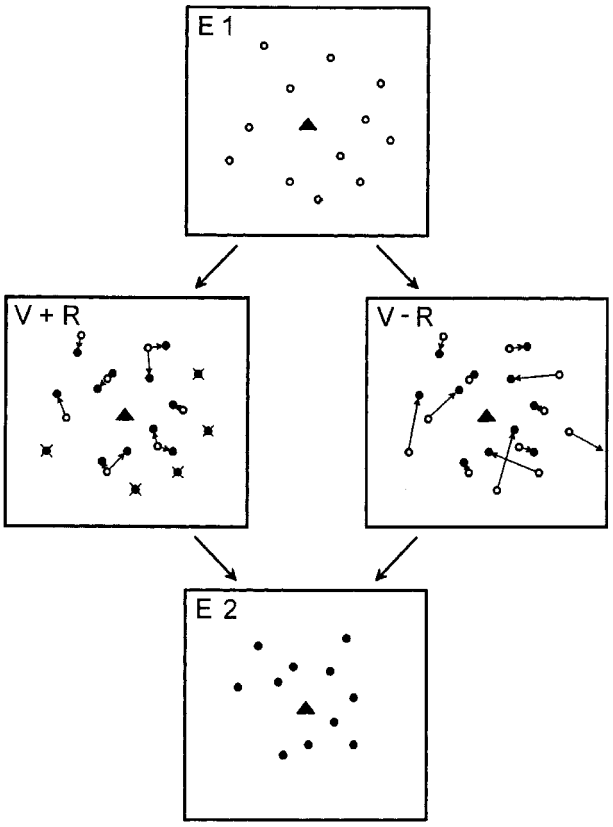


Figure 1 (Reeke). Selection by replicative and non-replicative mechanisms. Vertical and horizontal axes represent environmental parameters. Filled triangles indicate the values of these parameters in a particular environment. Information-carrying units are plotted as open circles at values of the parameters most favorable to their selection before encounter with the environment. Filled circles: after encounter. Arrows indicate changes in these units as a result of variation in their information content. E1: Situation before first encounter with the environment. V+R: Variation occurring during replication; units marked with an "x" are not selected and fail to replicate. V−R: Variation without replication; optimal interaction of units with the environment changes as a result of changes in their internal information content. E2: Situation before second encounter.

Variations and active versus reactive behavior as factors of the selection processes

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Abstract: The interaction of the organism with the environment requires not only reactive, but also active behavior (i.e., search activity) which helps

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subject to meet the challenge of the uncertainty of the environment. A positive feedback between active behavior and immune system makes the selection process effective.

The target article is an interesting attempt to compare some crucial features of the selection process in very different domains. Although the title of this article is very broad, the topic and the content of it is even broader and includes some suggestions very important for the methodology of science in general. Thus, by considering variations that function in selection process, the authors stress that all variations are caused by real events, and terms "chance" and "random" cannot be used in this context in the same way as they are defined in mathematics. "Few, if any, natural phenomena can meet these terms." I suppose that it is a very important statement especially being proposed by scientists from very different areas. I agree that the true random relationships are always artificial. An absorbtion of this point of view in science can help to approach even such an ambiguous and seemingly "nonscientific" topic as an anticipation of future events based on "insight" rather than on the probability forecast. If we agree that nature in general does not produce pure chance variations, then the topic of discussion around such phenomena will change. It will move from the dichotomy "causal versus non-causal relationships" to the more relevant dichotomy: causal relationships available for logical (monosemantic) analysis versus complicated causal relationships which can be grasped only by mean of the polysemantic way of thinking based on the right hemisphere activity (Rotenberg 1993a). Only the latter is responsible for the "irrational" anticipation of events, while these events are not random in any case. Such "irrational" anticipation may play an important role in the selection process on the behavioral level.

For me, the most interesting part of the target article is the consideration of operant behavior as a particular form of selection process. During the recent years, the development of biology was shifted almost exclusively toward molecular biology and the biology of genes. The environmental interactions of the organism were underestimated, although they are as important as genes for the selection. The behavior of the organism is only partly determined by the information fixed in genes. It depends also on the learning process.

I suggest that species with highly developed central nervous systems display general behavioral attitudes that are even more important for the environmental interactions than the particular operant behavior the authors are speaking about. By general attitudes I mean search activity versus renunciation of search (Rotenberg 1984; 1993b).

By search activity is understood activity designed to change the situation or the subject's attitude to it in the absence of a definite forecast of the results of such activity (i.e., in the case of pragmatic indefiniteness) but with constant monitoring of the results at all stages of activity. Obviously, search activity also covers partly operant behavior, because the main feature of operant behavior is the change over time as a function of its consequences (pragmatical outcome). However, according to that notion, operant behavior is totally determined by the requirements of the environment and has to fit the present requirements in order to be effective. The essence of search activity is different: the process of search by itself is more important than its pragmatical outcome (Rotenberg 1993; Rotenberg & Boucsein 1993). While operant behavior became senseless if the expected result is not achieved, search activity has a high value for the organism in any case and increases body resistance even if subject fails to achieve the desired goals (Rotenberg 1984). According to the authors of the target paper, operant behavior even being well adapted to the environment may not contribute to the survival of the organism that is behaving. Quite opposite, search activity in any form, including also those irrelevant to the environment, plays a crucial role in biological survival (Rotenberg 1994). I suggest that search activity is playing such a unique role in survival partly due to its very important contribution to the operant selection. Authors of the target paper emphasized that the mechanism of variation is present in the selection process in order to introduce novelty. However, on the behavioral level, variations by themselves (i.e., alternatives to select among) cannot serve this function without an inner mechanism which corresponds to the uncertainty of the environment and makes subject's behavior flexible in front of alternatives. Search activity represents such a mechanism helping subjects to meet the challenge of the uncertainty. If behavior only fits the present requirement of the environment, it seems to be not enough for adaptation. In order to master the changing environment, species with a highly developed central nervous system have to display not only reactive, but also active behavior. Search activity protects organism from rigid and stereotyped responses in the face of concurrently existing events and changing environment.

It is true that in operant learning, behavior is reinforced or extinguished according to the pragmatic outcome. However, even a very simple reinforcement like self-stimulation is a complex behavior which includes a choice in front of the uncertainty of the possible outcome, and consequently contains search activity (see Rotenberg 1984).

Finally, what is very interesting but unfortunately missed in the target paper, is an interrelationship between the two levels of selection: behavioral level (environmental interaction) and the biological level represented in the function of the immune system. Rates of variations are very high on both of these levels, and I suppose it is not unusual: a feedback exists between both levels. As we have shown (Rotenberg et al. 1996), any type of behavior which contains search activity enhances immune functions. For instance, the increase of the efficiency of the immune system was present even in cases when operant behavior was not effective and subjects failed in their attempts to solve tasks, however they did not give up and continued their efforts (Brosschot et al. 1992). I would suggest that the feedback between active behavior and immune system makes the selection processes on both levels less wasteful and inefficient than they may look on first glance.

Creativity as cognitive selection: The blind-variation and selective-retention model

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Abstract: Campbell (1960) proposed a "blind-variation and selective retention" model of creative cognition. Subsequent researchers have developed this BVSR model into a comprehensive theory of human creativity, one that recognizes that human creativity operates by more than one cognitive process. The question is then raised of how the BVSR model can be accommodated within the Hull et al. selectionist system.

Although I found the conceptual synthesis of Hull et al. quite thought-provoking, I regretted that they excluded from their synthesis a process that has great interest for psychologists – as well as considerable importance to the world at large. That process is creative cognition. Only a couple of decades after Charles Darwin published his *Origin of species*, William James (1880) first speculated that human creativity operated according to a Darwinian cycle of spontaneous variation and selective retention. This basic idea has been developed by many other subsequent thinkers, including both philosophers and psychologists, often under the guise of evolutionary epistemology (e.g., Popper 1979). Although Hull et al. specifically cite Campbell's (1974) contributions to evolutionary epistemology, I believe another contribution worth serious consideration is Campbell's (1960) article advocating a "blind-variation and selective-retention" (BVSR) model of the creative process. So important do I consider this latter essay that, when serving as the editor of the *Journal of Creative Behavior*, I made it a target article for "peer commentary" (Simonton 1998), with

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Cziko (1998), author of *Without miracles* (Cziko 1995), responding on Campbell's behalf (who was recently deceased). Moreover, for the past dozen years I have been actively engaged in developing my own elaborations and extensions of the BVSR model (e.g., Simonton 1988; 1997; 1999b). Most recently, my development of Campbell's theory has itself assumed the form of a target article subjected to peer commentary, albeit in *Psychological Inquiry* rather than *BBS* (Simonton 1999a). Another major contributor to these recent developments was Hans Eysenck (1993; 1995), who attempted to provide a personality basis for what he termed the "Campbell-Simonton model."

Hull et al. make it clear that the three processes they do discuss – evolution, immunology, and operant conditioning – often operate in rather distinct ways, notwithstanding the fact that they all can be subsumed under a single, generic selectionist scheme. By the same token, the creative process would be expected to depart from these other processes in certain details, despite its basic conformity to a selectionist framework. Indeed, one of the most fascinating aspects of creative cognition is that it is not one process but many. Although each works according to some kind of BVSR mechanism, the specifics of that mechanism can contrast greatly. Some of this complexity can be illustrated in the following three examples.

1. Creativity can certainly work in a fashion hardly distinguishable from operant conditioning. The individual may generate various permutations of established behaviors – often through playful manipulation of objects in the environment – and thereby encounter a combination of acquired behaviors that serves to solve some problem. Köhler (1925) provided a classic illustration of such behavioral insights in his observations of how Sultan was able to join two sticks to retrieve a banana placed just out of reach. Epstein (1990; 1991), a student of B. F. Skinner's, has proposed a model that explicates Sultan's insight behavior in totally operant terms. Furthermore, Epstein has shown that this model does an excellent job predicting insight behaviors in pigeons who are given problems comparable to those that Köhler provided his apes. Although one might be inclined to dismiss this behavioral BVSR as too primitive to support major acts of human creativity, this particular selectionist process actually has considerable importance. Kantorovich (1993), for instance, has argued that playful "tinkering" often provides the basis for breakthrough discoveries in science. Such unguided exploration and manipulation is a frequent source for serendipitous events that could not have been anticipated by logic or prior experience (Kantorovich & Ne'eman 1989).

2. For organisms with sufficient cognitive complexity, such as the human mind enjoys, the BVSR process can be rendered more efficient. As Campbell (1960) pointed out, both the generation of variations and the testing of those variations can occur covertly rather than overtly. Dennett (1995) styled creatures who can engage in this kind of problem solving "Popperian," in contrast to the "Skinnerian" creatures of the previous example. Such organisms take advantage of the fact that they contain internal representations of the external world, along with the representation of various ways of acting on that world. The internal representation, for instance, might be a "cognitive map" of the physical environment, which the organism can then use to conceive alternative routes should the normal pathway be obstructed. By engaging in such internalized "trial-and-error," the organism increases the odds that when it finally emits an overt behavior, that action will be successful. Of course, success is not guaranteed. One problem is that the representation of the external world may not be completely accurate. As a consequence, a failed action can also be taken as a test of the individual's representation, and thus indicative of a need for a change in that representation. That alteration may itself require the organism to engage in an overt BVSR process until that internal representation more closely approximates the environment. Once that adjustment is complete, the organism can return to the more efficient Popperian experience in which problems are solved through thinking rather than behaving.

3. Although there is no doubt that much creativity operates in the above manner – especially in everyday problem-solving situations – many of the more impressive acts of human creativity rely on a more sophisticated BVSR process. Human beings have minds that contain not just images of themselves and the outer world, but also cultural artifacts that can be used in lieu of those mental images. Those artifacts include language, logic, mathematics, graphics, symbols, and various tools and devices, whether mechanical or electronic. Dennett (1995) called creatures "Gregorian" who can exploit these means to problem solving, the only Gregorian creature we currently know of being *Homo sapiens.* Probably the supreme vehicle for this highly abstract form of BVSR is mathematics. Once a correspondence has been established between mathematical symbols and the external world, the symbols can undergo efficient manipulations to yield discoveries that then can be tested against the world, and new discoveries thus made. Sometimes these predictions will be derived in a systematic fashion from the mathematical representations, but other times the predictions will be the serendipitous result of playful tinkering with the abstractions and their connections.

The most common objections to Campbell's (1960) BVSR model of creativity is the hypothesized "blindness" of the process (e.g., Perking 1994; Sternberg 1998). Although it is obvious that the creator must engage in selective retention, the notion that the creator also must generate "blind" variations appears less so. This latter idea seems to run counter to the common assumption that creativity is a manifestation of intelligent behavior. The most prominent implementation of this assumption is the extensive attempts to write "discovery programs" that solve problems through well-defined logical processes (e.g., Langley et al. 1987). In the context of this work, Campbell's model may appear like another one of those over-extended analogies too frequently inspired by Darwinian theory. However, in my view these objections ignore the fact that the "blindness" of a variation procedure is not a discrete attribute. Rather it is a continuous quality that may range from totally constrained variation (e.g., random guess), with many intermediate levels of unguidedness (e.g., heuristic searches). The same, of course, holds for the variation processes that feed Darwinian evolution, which can range from completely constrained asexual reproduction to utterly random mutations, with numerous grades of genetic recombination falling between (depending on the degree of chromosomal linkage). Campbell's BVSR model merely maintains that as problems become increasingly novel and complex, the degree to which past solutions can effectively guide a creator becomes progressively reduced. Indeed, as serendipitous events indicate, often the solution to a problem may arise from domains where the creator least expects (e.g., Gutenburg's realization that the wine press solved his problem of how to mass-produce *The Bible*).

As I have documented at great length elsewhere (Simonton 1999b) the BVSR model helps account for many features of creativity that are otherwise difficult to explain. Examples include explanations for why highly creative individuals display a distinct cognitive incapacity to filter out "extraneous" information (Eysenck 1995), why performance on creativity tasks is enhanced when individuals are exposed to incongruous, unpredictable, or random stimuli (Finke et al. 1992; Rothenberg 1986), and why the computer programs that simulate most successfully human creativity invariably incorporate some procedure for producing "blind" variations, most often through a random-number generator (Boden 1991). Indeed, the most successful problem-solving computer programs to date, namely genetic algorithms and genetic programming, operate explicitly according to BVSR principles (e.g., Holland 1992; Koza 1994). The BVSR model has the additional advantage of integrating the psychology of creativity with other scientific domains with strong evolutionary foundations (Cziko 2000).

The last remark brings me back to the Hull et al. article. Given that they have attempted to integrate three distinct selectionist processes, how would they accommodate creative cognition in their conceptual scheme? Could they devise a single framework

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for the Skinnerian, Popperian, and Gregorian forms of creativity, or would these differ as much from each other as the three processes treated in their target article? And how would they use their scheme to incorporate yet a fourth kind of creativity that occurs when "Dawkinsian" creatures exchange "memes" back and forth, and thereby see their creative ideas develop in directions that nobody could have possibly anticipated? In a nutshell, I guess I'm asking Hull et al. to write an addendum on how creativity might fit within their selectionist system.

The causal crux of selection

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Abstract: Hull et al. make a direct connection between selection and replication. My view is that selection, at its causal crux, is not inherently connected to replication. I make plain the causal crux of selection, distinguishing it from replication. I discuss implications of my results for Hull et al.'s critique of Darden and Cain (1989).

To provide an accurate, general account of selection, the causal crux of selection must be laid bare. The causal crux of selection is the selective interaction, that stage of the adaptive process that has the propensity to produce downstream selection-type effects (in biological evolution, to which I will restrict myself, such effects are survival, reproduction, and lineage adaptation). Hull et al., in my view, do not make the causal crux of selection manifest in their general account. Indeed, Hull et al. bury the causal crux of selection within their conception of replication: Because Hull et al. demand that selection be tied, inextricably, to replication, they fail to adequately distinguish selection as a distinct stage in the adaptive process. To be sure, providing a general account of selection that does not allow spurious selection events requires that selection be tied to the appropriate downstream adaptive effects of interest. However, at the same time, the causal crux of selection must be made manifest in a way that does not confuse the fact that selection must be understood as a distinct stage in the adaptive process and not as embedded in other, contributing stages of it. In this vein, and in spite of Hull et al.'s criticism, I advocate Darden and Cain's (1989) abstraction of selection as a proper general account of selection. Indeed, I think Hull et al. are mistaken to criticize Darden and Cain's abstraction for its lack of a strict tie to replication: Darden and Cain are exactly correct to distinguish clearly, as a stage in the adaptive process, the causal crux of selection from its downstream effects. At the same time, however, Darden and Cain adequately tie selection to its downstream effects.

For Hull et al., replication involves, at its core, iteration and information (sect. 2.2, para. 1). Insofar as Hull et al. regard replication as an inherent element of selection, selection involves iteration and information. Selection so understood occurs again and again and carries information through the adaptive process. Replication, and replicators, were, originally, Dawkins's (1976) ideas. Dawkins's aim was to capture, in what he argued is the unit of selection, the cumulative nature of biological evolution, or what was, roughly, immortal in the evolutionary process (Dawkins 1976, pp. 34–36). Making the cumulative nature of biological evolution central, places the focus of an analysis of selection as a part of the process on the genetic material. After all, it is the genetic material, or the information encoded in it, that persists largely intact through the evolutionary process. Yet, selection is not cumulative; evolution is cumulative. Selection, as a factor in biological evolution, is the causal interaction that changes the chances of survival, reproduction (where replication becomes key), and lineage adaptation, that is, selection's downstream effects. Selection does not carry information through the evolutionary process. Rather, it is the causal factor that changes the chances that the downstream effects of selection will cause information, albeit modified, to persist over time. Selection and information are *not* inextricably tied together in the way that Hull et al. claim.

Given that selection can be disconnected from information in the way I have described, it stands to reason that it can also be disconnected from iteration. Consider the adaptive process stagewise: There are variants according to some property in an environment with critical factors. A selective event occurs based on the interaction between the variants and critical factor(s) in the environment. After the event, the relevant processes endemic to the individuals selected for or against, for example, those processes that are involved in survival, reproduction, and lineage adaptation, take over. That is, individuals, in biological evolution, for example, have increased or decreased chances to survive, reproduce, and adapt to their environments. Then, the process is back at variation, with selection occurring again, followed by selection's downstream effects. Iteration is appropriately demanded for the adaptive process of which selection is a stage. Iteration within the adaptive process is not inherently tied to selection. Indeed, selection that leads to the survival of an individual does not guarantee that the individual will reproduce. And so on. Because selection is not inextricably tied to iteration or information, it is a mistake to embed selection in replication. A more adequate understanding of selection is achieved by making plain the causal crux of selection as that distinct stage of the adaptive process with the propensity to cause downstream selection-type effects.

The characterization of selection that I have presented here follows closely Darden and Cain's (1989) abstraction of selection. Hull et al. reject Darden and Cain's abstraction because selection is not embedded within replication via an essential tie to iteration (sect. 2.2, para. 2–3). According to Hull et al., an unpalatable side effect in Darden and Cain's abstraction of not closely tying selection to iteration is the use of "benefit" and "suffer" to characterize selection's downstream effects. There is, in my view, nothing unpalatable here. In point of fact, "benefit" and "suffer" are idioms for referring to the downstream effects of selection. Elsewhere, and for general purposes similar to Darden and Cain's and Hull et al.'s, I have explicated the idioms as theoretical metaphors used as placeholders for the more specific downstream effects of the interaction of variants in an environment with critical factors (Skipper 1999). Darden and Cain's "benefit" and "suffer" is not problematic when understood in the way that I suggest.

Selection is best understood as a distinct stage of the broader adaptive process. As such, the adaptive process works almost precisely in the way that Darden and Cain describe: individuals vary according to some property (properties) and are in an environment with critical factors. The interactions of individuals in the environment with critical factors, that is, the causal crux of selection, has the propensity to produce downstream effects, for example, in biological evolution, survival, reproduction, and lineage adaptation.

Selection: Information and replication of the operant

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Abstract: Selection as a mode of causation is central to operant psychology. Response variation and selection by consequences corresponds to phenotypic variation, to differential survivability, and to reproduction of variants. In natural selection, genes code for phylogenic history but no analogous processes exists for coding behavioral history. Neuroscience suggests potential processes but the conceptual status of these events requires clarification.

> Change as change is mere flux and lapse, it insults intelligence. (Dewey 1990, p. 6)

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Modern scientists, as the inheritors of the legacy of Heraclitus, assume that change is ubiquitous and ultimate while permanence is appearance. As a consequence, interest in the evolutionary mechanisms transforming objects, organisms, behavior, and culture pervades philosophic and scientific discourse. One need only read the press to appreciate the continuing popular and scientific interest in the origins and processes transforming our solar system. In 1859, Darwin proposed natural selection as the mechanism of biologic transformation. Natural selection requires, at a minimum, phenotypic variation, heritable variants, and differential survivability and reproduction of variants. Differential survivability and reproduction of variants is termed fitness.

In their target article, Hull et al. define selection in the broader but parallel terms of replication, variation, and environmental interaction resulting in differential replication. Replication entails iteration and information. Iteration (a component of replication), variation, and environmental interaction are processes that can be found in operant behavior. The very process of reinforcement presupposes iteration as a defining feature. A tenet of operant theory, repeatedly demonstrated in the laboratory, is that consequences, reinforcers, alter response frequency, topography, and distribution. The Matching Law (Davison & McCarthy 1988) illustrates and quantifies differential replication. The Matching Law demonstrates that the relative distribution of alternative responses matches the relative allocation of reinforcers among the response alternatives (Davison & McCarthy 1988). Phylogenetically determined behavioral predispositions are the source of behavioral variants. Ethological releasing stimuli, reflex elicitation, and deprivation induce responses varying along important behavioral dimensions (Segal 1972). Differential reinforcement through a process of successive approximation shapes behavioral operant response classes from this population of behavioral variants.

While there is an obvious mechanism, genetic material, for the coding of phylogenic history and transmittal of information in biologic selection, there is no analogous process for operant behavior. As the organism is the locus of environmental action, it is reasonable to assume that the biologic functioning of the organism, particularly at the level of the neuron, will be altered by interaction with the environment (Feldman et al. 1997). The increasing sophistication of molecular biology and neuroscience is able to describe these changes and related mechanisms and may discover a process coding for the organism's behavioral history. For instance, in Aplysia the application of the modulatory neurotransmitter serotonin to one branch of the neuron during cell activation, subsequently amplifies the electrical response of that cell branch to stimulation (Bailey et al. 1997). This process can be viewed as coding for organism's interactions with the environment and, at the very least, may alter behavioral predispositions in similar contexts in the future. But as B. F. Skinner (1972) cautioned "we still have the task of accounting for the neural . . . event" (p. 71). Skinner's concern is not whether biologic events should be included in discourse about behavior. Rather, his concern is about the status of these events in scientific discourse. Skinner eschewed the mechanistic, chain mode of causal discourse (Alessi 1992; Chiesa 1992; 1994; Glenn et al. 1992). The mechanistic mode requires that causal relations be contiguous in time and space. Neurophysiological events would be viewed as the linking events occupying the temporal gap between environment and behavior. An alternative perspective, more congenial to Skinner's notions of selection, would view the same biologic events as among the multiple determinants of behavior. Implicit in this perspective is the behavioral analog of the biological dichotomy between genotypic and phenotypic expression. In the same way that genotype does not exclusively determine phenotype, behavioral history is not the exclusive determinant of response.

Radical behaviorism is the philosophic foundation of the study of operant behavior. Unlike the earlier behaviorisms of Pavlov, Watson, Hull, and Tolman, radical behaviorists abandoned the mechanistic, stimulus-response, mode of causal discourse for selection as the mode of causation. This approach has had far reaching and controversial implications for discourse about human behavior. Multiple causation is emphasized, causation over time is presupposed, and concepts of human agency have been eliminated. By any standard the adoption of selection on psychology has produced a revolutionary paradigm shift of scientific and cultural consequence (Kuhn 1962). Hull et al. have contributed by providing essential criteria for defining selection as a process and demonstrating its ubiquity.

Avoiding vicious circularity requires more than a modicum of care

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Abstract: Any general account of successful selection explanations must specify how they avoid being ad hoc or vacuous, hazards that arise from their recursive form.

Hull et al. are correct that Lipton and Thompson (1988) provides warrant for the belief that natural selection explanations are not necessarily circular. Indeed, Lipton and I argued that natural selection explanations are circular only to the extent that they are recursive. In a selection explanation, the explanandum (e.g., the properties of an organism) is explained by virtue of an explanans (e.g., selection for those properties) that refers to the explanandum. Strictly speaking, selection explanations are not circular so long as the frame of the explanans ("selection for") contains some information not provided in the explanandum. In general, recursive explanations have heuristic value in a science that is groping toward the discovery of the cause of some dramatic and well-defined phenomenon. For an example, one need only think of the medical search for the cause of AIDS. The infectious agent was known recursively as the human immunodeficiency virus (HIV) long before it could be identified as a particular structural entity.

However, I am not sure I agree with the target article authors that vicious circularity can avoid in natural selection explanations with only a "modicum" of care and effort. As Lipton and I point out, great care has to be taken in the deployment of recursive explanations to steer between the perils of ad hockery and vacuousness. Consider three explanations for the whiteness of a polar bear's fur:

(1) Because white bears have been selected for white fur.

(2) Because camouflaged bears have been selected for camouflage.

(3) Because disproportionately reproducing bears have been selected for disproportionate reproduction.

All three are equally recursive, but they are not equal in heuristic value. Neither (1) nor (3) offers any help in identifying the white bear's advantage, (1) because it applies only to white bears against a white background and (3) because it would be true of any creature no matter what its background. However, (2) is useful because it suggests a general class of causes to which having white fur against a white background belongs. Thus, avoidance of vicious circularity in a natural selection explanation is dependent on offering the right kind of description in the explanandum – not so narrow as to invite an ad hoc explanation, not so vague as to invite a vacuous one. Such descriptions require a precise understanding of the natural history of the creature whose existence and properties are to be explained by a selection theory.

This requirement has an important implication for Hull et al.'s project of providing a general of selection-type explanations. Any such *general* account must include a *general* description of the properties of the entities that are selected for, whether these entities be organisms, immune system elements, or habits. When we fail to include such descriptions in our selection-type theories, or when those descriptions are too specific or too vague, selection theories lose much of their heuristic value. Recursive theories with an

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inadequate specification of the explanandum are properly termed degenerate because their intellectual evolution has resulted in the loss of some crucial feature. For many years, I have inveighed against the degeneracy of sociobiological theory (Thompson 1982; 1987a; 1987b; 1993). Evolutionary psychology, by contrast, has struggled to shake loose this degenerate tradition by defining a priori the problems that human behavioral adaptations were designed to solve (Barkow et al. 1992; see also Cosmides & Tooby's remarkable "primer" on evolutionary psychology posted to the web at *www.psych.ucsb.edu/research/cep/primer.html*). Thus, by focusing on the specific demands of the human ancestral environment, evolutionary psychologists have been able to provide more heuristic selection explanations than their sociobiological predecessors.

Is operant selectionism coherent?

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Abstract: Hull et al.'s analysis of operant behavior in terms of interaction and replication does not seem consistent with a genuine selection model. The putative replicators do not replicate, and the overall process is more reminiscent of directed mutation than of natural selection. General analogies between natural selection and operant reinforcement are too superficial to be of much scientific use.

Analogies between operant reinforcement and natural selection have been around for decades (e.g., Postman 1974, p. 491), yet the vast majority of behavioral researchers still publish their findings without any reference to such analogies. Six years ago, Glenn and Madden (1995) could state that the serious work of explicating operant selection had "barely begun" (p. 249), and the target article reiterates their warning: Hull et al. acknowledge that their language is not that of the original researchers and that a selectionist perspective on operant reinforcement is not traditional (sect. 5.2). The effective novelty of Hull et al.'s approach to operant reinforcement raises a number of issues. How does the proposed framework compare to theories that make no appeal to biological analogies (e.g., Killeen 1995)? Do we understand operant reinforcement any better by calling it "selection"? Is our understanding of stimulus control improved by likening it to the "appearance of eukaryotes in biological evolution"?

As Hull et al. realize, such analogies will prove helpful only if they achieve the proper balance between generality and precision. At the most general lever, if selection is defined as any process in which *something* increases in frequency as a result of previous interactions, then operant reinforcement undoubtedly qualifies. Nobody who understands the definition of reinforcement could deny that it constitutes selection in this sense. But proponents of selection analogies surely expect more from their approach than an indisputable truth so devoid of implications for actual scientific research. Presumably, the promise of a fruitful analogy between natural selection and operant reinforcement is to identify some underlying similarity of process and to develop an empirical research program on that basis.

A major problem with this approach is that it is far from obvious that the proposed analogy characterizes a shared causal structure (cf., Darden & Cain 1989). Tonneau and Sokolowski (2000) have concluded that the selection analogy fails in this respect. The relevant arguments address many aspects of behavior and are too numerous to be reproduced here, but a few important points can be mentioned, if only briefly.

Trying to define a process of selection over the changing behavior of an individual overlooks the fundamental distinction between variation and transformation (Futuyma 1998, pp. 21–22). Hull et al.'s concept of evolution through variation and replication requires criteria for distinguishing variation from failure to reproduce, and reproduction from failure to vary. This logical requirement implies in turn that the temporal changes of an individual (as opposed to across-individual variation) are not the kind of variations appropriate to a selection process. Consider a pigeon preening its feathers and emitting a string of key pecks, for example. As the pigeon behaves, its body undergoes a continuous series of transformations. Does the transformation from preening to pecking document the death of preening followed by the birth of pecking, or the mutation of preening into pecking? Does the persistence of pecking document its successful reproduction or rather its failure to vary? Given these logical difficulties, Hull et al.'s claim to have identified "lineages" in operant behavior should be taken with caution.

Hull et al. might of course object that their treatment of operant reinforcement is not purely behavioral. Consistent with Hull's (1981) analysis of natural selection, Hull et al.'s operant selectionism indeed involves neural *replicators* as well as behavioral *interactors.* The inclusion of neural elements in the selection analogy, however, only creates more problems. First and foremost, the proposed replicators (such as synaptic connections) do not replicate. It is the frequency of the proposed interactors (responses or environment-behavior relations) that changes over time (sect. 5.4). The frequency of the "replicators" changes neither in space nor in time: A network of 10 nodes and 25 links before reinforcement will still compromise 10 nodes and 25 links afterward. Hull et al.'s model of reinforcement thus infringes on a fundamental requirement of selection processes: A genuine selection model should imply changes in the *numerical representation* of a feature in a population of replicators, and this change should be brought about by differential replication or conservation (as discussed in sect. 2.2).

In contrast, what plays the role of "replication" in Hull et al.'s framework is reminiscent of directed mutation: Hull et al. assume that operant reinforcement changes the state of some neural element or network, and that this modified state is thereafter retained across generations (see sect. 5.8). The non-Darwinian nature of the proposed process should be clear from the discussion of retention in the target article. In biological evolution, "retention" (that is, inheritance) retains features of replicators (and interactors) which selection does not change but *among which selection proceeds* (through differential reproduction, not permanent state changes). In Hull et al.'s analysis of operant reinforcement, however, the function of the retention mechanism is to retain *modified states* of alleged replicators which "selection" *changed* and among which no actual selection proceeds (since the proposed replicators do not replicate). The process is *selective* but is not one of *selection,* even by Hull et al.'s standards (sect. 2.2).

We do not believe that such discrepancies (and others as well: Tonneau & Sokolowski 2000) can be dismissed as trivial or irrelevant. Far from concerning implementation detains (like the nonexistence of sex among reinforced responses, for instance), they point to basic differences in the ways that operant reinforcement and natural selection are organized. Operant reinforcement mimics superficial aspects of natural selection through entirely different arrangements, hence general accounts of selection such as Hull et al.'s must fail in the case of operant behavior.

Glenn and her colleagues should be commended for trying to turn Skinner's (1981) selection analogy into a rigorous and productive approach. Too often in behavior analysis, the metaphor of selection has served as a rhetorical tool instead of a potential framework to be explored and evaluated in greater detail. We are afraid, however, that Hull et al.'s insistence on explicitness may have made the difficulties of operant selectionism more evident. Even if an example of replication can eventually be found in the nervous system at a molecular, biochemical level, the relevant happenings will be far removed from the evidence that suggested a selection process in the first place: response rate.

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Are units of retention necessary?

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Abstract: Hull et al. propose a set of features that can be used to identify explanatory systems as being selectionist. The commentary questions the necessity of identifying a unit of retention at the level of behavior-environment interactions. It is concluded that an answer to the question "Are units of retention necessary?" can only be given in light of a clear statement of the goals of a science of behavior.

The explicit goal of the target article by Hull et al. is to propose certain generic features of explanatory systems that will allow them to be identified as selection-based regardless of the "level" at which their analyses are carried out. Over the last 50–60 years, there have been several attempts to point out that the selection processes seen in natural selection are analogous to processes seen at other levels of analysis (e.g., Campbell 1960; Darden & Cain 1989; Lewontin 1970; Skinner 1953; 1981). The implicit and explicit goals of such comparisons have been to suggest that "selection by consequences" may be aptly described as a kind of causality or causal mode. The paper by Hull et al. continues in that tradition but makes two very important contributions. The first of these is the explicit attempt to identify generic features that must be present in any explanatory system for it to count as an instance of a selectionist process. The second important contribution arises from the fact that, for the first time, experts from three different areas, each capable of being described in selectionist terminology, have put their thoughts down together. The result is a scholarly treatise that is, at once, broad in its implication but specific in its focus and argument.

As immunology and evolutionary biology are outside my particular area of expertise, I will use the remainder of available space to discuss a particular aspect of the discussion on operant conditioning – namely, the discussion involving units of retention at the level of behavior-environment interactions.

One of the ways that the current contribution differs from previous attempts at conceptual integration (e.g., Skinner 1981) is in terms of an explicit treatment of the unit of retention within the context of operant conditioning. Hull et al. suggest, for example, that changes in the structure and function of parts of the central nervous system may be the units of retention – and, further, that these units may code for properties (or "traits") of the responses that enter into behavioral relations with the environment. Configurations of the central nervous system, then, would code for responses (defined in terms of operant contingencies) in a manner analogous to the relations between genes and phenotypes. Research guided by the selectionist perspective would seek to uncover relations between selection at the level of behavior-environment interactions and retention at the level of neurochemical interactions.

One wonders about the degree of correspondence that we are likely to find between regularities observed at the level of behavior-environment interactions and regularities observed at the level of neurochemical events, however. A seemingly large obstacle revolves around the fact the operants are defined, not with respect to any features of the response itself, but with respect to features of the environment. For example, a key peck may be defined as any response that closes a switch behind a key. Given the suggestion that, at the neurochemical level, the properties (traits) of a response are what is retained and the fact that, at the behavioral level, no specific properties are required in the definition of an operant, can we ever expect to find isomorphic physiological representations of operant organization? It is interesting, along these lines, to speculate whether earlier versions of behaviorism may actually have been better suited to guide the search for neurochemical units of retention in learning. Theories of reinforcement based on drive-reduction, for example, may have predicted a common neurochemical signature associated with a common event (e.g., drive reduction) at the time of reinforcement (Hull 1943).

This line of reasoning, however, reveals an interesting paradox: it was precisely the move away from theories of drive-reduction or principles of stop-action to functional definitions in terms of contingencies of reinforcement that gave Radical Behaviorism a clear advantage over earlier versions of behaviorism (Lee 1988). Identifying the unit of analysis as the observed functional relation between behavior and environment allows orderly relation to be observed and described at the level of behavior-environment interactions. The position that behavior is worthy of study in its own right is important because it is accurate description at the level of behavior-environment interactions that leads to practical prediction and control – the criteria by which the effectiveness of behavioral descriptions are judged. The wide array of applied endeavors under the umbrella of Behavior Analysis may be seen as evidence supporting this proposition. Do we stand to lose all that if an explicit focus on units of retention is added to the radical behaviorist position?

In the final analysis, conceptual reorganizations of the sort offered here will be selected or rejected on the basis of their utility in organizing or reorganizing the practices of researchers and practitioners in the various fields represented by the authors. Will a selectionist perspective of the sort offered by Hull et al. lead to new experimental questions and preparations in the operant conditioning laboratory? Will an emphasis on units of retention lead to a decreased reliance on histories of reinforcement? What, anyway, is the role of the historical perspective in the behaviorist approach to understanding behavior-environment interactions? Are units of retention necessary? Answers to these and other questions can only be provided in light of an understanding of the goals of a science of behavior. The target article is important, not only for the cross-disciplinary conceptual integration it is attempting but also because it raises extremely important questions within disciplinary boundaries.

Authors' Response

At last: Serious consideration

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Abstract: For a long time, several natural phenomena have been considered unproblematically selection processes in the same sense of "selection." In our target article we dealt with three of these phenomena: gene-based selection in biological evolution, the reaction of the immune system to antigens, and operant learning. We characterize selection in terms of three processes (variation, replication, and environmental interaction) resulting in the evolution of lineages via differential replication. Our commentators were largely supportive with respect to variation and environmental interaction but critical with respect to replication, in

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particular its appeal to information. With some reservations, our commentators think that our general analysis of selection may fit gene-based selection in biological evolution and the reaction of the immune system but not operant learning. If nothing else, this article shows that the notion of selection is not as straightforward as it may seem.

As might be expected, given the readership of *Behavioral and Brain Sciences*, most of the comments on our General Analysis of Selection concern operant learning. Gene-based selection in biological evolution and the reaction of the immune system to antigens fit the analysis that we provide quite well, perhaps because in formulating our analysis we were too biased toward gene-based selection and the immune system from the outset (**Baum, Chiesa**). Although we tried to present an analysis of "selection" that applied equally to all three areas, we may not have succeeded as well as we had hoped. All three phenomena exhibit peculiarities. For example, both the functioning of the immune system and operant learning take place within the life-span of single organisms. These two phenomena are part of ontogeny, not phylogeny, while gene-based selection in biology is not limited to single organisms. It is part of phylogeny. This is a clear difference, but is it sufficient to preclude the same analysis of "selection" being applicable to all three sorts of phenomena? In both gene-based selection in biology and the immune system, extensive concurrent variation exists. In the case of operant learning, little if any concurrent variation is present. Is this difference sufficient to preclude a single analysis applying to all three sorts of phenomena?

We think not; however, after puzzling over the objections raised by our commentators, we are forced to conclude that such differences seem more serious with respect to operant learning than with respect to the other two phenomena. Gene-based selection in biological evolution and the reaction of the immune system to antigens seem clear cases of selection, a few irritating dissimilarities notwithstanding. More serious doubt exists with respect to operant learning. If we have done nothing else in our target article, we have shown that the common belief that all three of these processes count as selection processes in the same sense of "selection" is more problematic than is commonly recognized.

In the face of such difficulties, three possibilities present themselves. We could simply conclude that operant learning is not a selection process, at least not in the same sense as the other two processes. Or we could make our analysis of "selection" broader so that operant learning is covered. Or, finally, we could construe operant learning in a way compatible with our analysis. Most of our commentators favor the second alternative, while we prefer a combination of the second and third alternatives. Because operant learning poses the most fundamental problems for our analysis, we hold off discussing it until the last sections of this response. We begin with more general criticisms and comments. But first we need to say a few words about the sort of thing that we are doing – conceptual analysis.

R1. General issues

R1.1. Conceptual analysis

Few problems in science are totally a matter of confused language, but the lack of conceptual clarity has on occasion produced long periods of unnecessary controversy. Selection provides special problems in this respect because in selection processes the cause seems to follow the effect. In this response, we attempt to produce a coherent set of terms that can be used in discussions of selection processes. At times scientists register irritation with respect to such efforts. They are "merely semantic." Happily all of our commentators see the point of conceptual analysis in science. As **Pepper & Knudsen** ask, "Is the definition of selection ultimately a matter of taste or semantics, with one answer being as good as another?" (para. 6). He answers unequivocally that it is not, in part because the "theory of selection has developed beyond mere verbal argument." We have some room for maneuvering, but in general, all three areas of science that we investigate have been highly developed. Each contains some claims that can be rejected or modified only for very good reasons. In developing a general analysis of selection, it is simply not true that anything goes.

As several of our commentators recognize, the most common problem in the production of conceptual analyses is making them either too broad or too narrow (**McSweeney & Aoyama, Pennypacker, Tonneau & Sokolowski**). Quite a few of the commentators on our article find the distinction between replication and environmental interaction extremely helpful (**Baum, Eckerman & Kemp, Hineline, Matthen, McSweeney & Aoyama, Okasha**, and **Skipper**). However, just as many find our analysis of "selection" too narrow because of the emphasis we place on replication and information. No one suggests that it is too broad. In addition, some of our commentators think that replication is part of selection but that we are mistaken in how we define "replication." Reference to iteration, repetition, and copying makes our analysis too narrow. It must be made broader to include mere reoccurrence, reappearance, persistent patterns, or correlations. Here, as elsewhere, we are not relying on the meanings of these terms in ordinary English but in the distinctions that they imply.

Our strategy in writing our target article was to start with fairly narrowly-defined terms and gradually expand our analysis. We picked gene-based selection in biology, the immune system, and operant learning because we thought that they were the least problematic examples of selection. Once we had developed a notion of "selection" and related terms adequate for these examples, we could then expand our analysis to more problematic cases, for example, neuronal development, social learning, and even memetic change. Darden and Cain (1989) included neuronal development instead of operant learning in their analysis. Numerous authors have treated social learning as a selection process, and recently the science of "memetics" has become quite popular (Aunger 2000; Hull 1988).

Catania finds this strategy baffling. He proposes to start with the broadest notions possible; for example, particles scattered in the vicinity of a large planet. As time goes by, some of these particles fly off into space, others fall to the planet's surface, while still others form rings around the planet. Catania proposes treating such gravitational processes as selection. We are just as baffled by Catania's strategy. If the different fates of these particles under the action of gravity count as "selection," then it is hard to see how any phenomena can be excluded. Several of our commentators have also urged us to expand our analysis to include other sorts of behavioral processes in addition to operant learning, such as stimulus-elicited behavior and goal-directed

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behavior (**Bevins, Blute, Burgos, Cziko**). Extending our analysis to cover additional sorts of behavioral processes is one thing; applying it to particles in space is quite another. **Reeke** complains that in our article we present an "ex cathedra definition illuminated with examples." (sect. 1). If he had been party to our deliberations that stretched over two years, he would have come to a quite different conclusion. Nor is the process complete. We have not been impervious to the objections raised by our commentators.

R1.2. Lineages and environmental interaction

The notion of lineages evolving under the influence of environmental interaction seems basically right to most commentators, while information, inheritance, and replication do not. Before turning to these latter problem cases, a few words need to be said about lineages and environmental interaction. Lineages are causally connected sequences of spatiotemporally continuous entities – in our target article lineages of organisms, lineages of acts (responses), and lineages of B cells. These lineages are what evolve. Inheritance as it functions in selection processes is more than just a sequence of events; for example, one billiard ball hitting another billiard ball, which hits another, and so. It is intimately tied to lineages. **Grantham** agrees that lineages are necessary for selection processes but argues that no such lineages are involved in operant learning because "there is no genuine replication in operant conditioning" (sect. 3). **Baum** agrees with the role of lineages in selection processes but thinks that our analysis of lineages is too closely tied to genetic replication. His alternative, which he takes to be adequate for operant learning, is that two sequential occurrences belong in the same lineage if they share a "common ancestry," and they share a common ancestry if they have a "common history of selection" or more generally, the "same history of interaction with the environment."

Dawkins (1976) emphasizes the notion of genetic replication – his selfish genes. In reading Dawkins, one gets the decided impression that genetic replication is all that really matters in selection. Even though he mentions "vehicles," he plays down their importance. In our article, we emphasize the role of environmental interaction because, without environmental interaction, changes in gene frequencies are the result of drift, not selection. For some reason, **Heschl** thinks that we have neglected the importance of Kimura's neutral theory of evolution and have denied that his contributions have been integrated into the modern neo-Darwinian synthesis. One of us has detailed at some length the integration of certain elements of Kimura's theory into present-day versions of evolutionary theory (Hull 1988). All we claim is that such modifications did not produce a "new" theory of evolution. Kimura's Neutral Theory did not replace the Synthetic Theory. After all, that is why it is termed "synthetic."

In general, however, our commentators agree with the emphasis that we place on environmental interaction. **Baum** finds the separation of interaction from replication to be particularly constructive. **Hineline** thinks that substituting "interactor" for "operant response" conveys the relational character of the learning process better than the traditional terminology. **McSweeney & Aoyama** see the division of selection into replication and environmental interaction particularly promising. **Okasha** agrees with our characterization of selection as a "two-stage process involving differential replication caused by environmental interaction" (para. 1), while **Skipper** argues that selective interaction is the "causal crux" of selection. Only **Glassman** rejects our undertaking in its entirety and seeks to replace it with his own symbiogenesis.

R.1.3. Replication and information

The three notions that provoked the most disagreement and controversy are replication, information, and inheritance. Several of our commentators argue that replication is too restricted a notion even for gene-based selection in biology and the immune system. It is certainly too restricted for operant learning. Still others want to jettison replication entirely. It is not only difficult to analyze but also beside the point. Still others argue that information has nothing to do with selection. Finally, several commentators discuss the role of inheritance, but too often what they reject with respect to replication and information sneaks back in under the guise of inheritance.

In our view, replication requires both information and inheritance. It is not enough for structure to be passed along; this structure must count as information. **Hineline** objects to our describing the linear sequence of nucleotides in DNA as "providing the information" necessary for the production of proteins. "It seems to me that in a nontrivial sense, the linear sequence of nucleotides *is* the information" (last para.). In this respect, we find ourselves in agreement with G. C. Williams (1986, pp. 116–17), when he emphasizes that phenotypes cannot count as "persistent entities subject to multigenerational evolutionary forces" because phenotypes "do not *persist*, they *recur*." But genes as physical bodies do not *persist* either. What really matters, according to Williams (1986, p. 121), is continuity of information. Nucleotides as material bodies come and go quite rapidly. Only information persists. But this difference between Hineline and the authors of this target article may be only apparent. **Reeke** acknowledges the importance of information in selection processes but chastises us for "failing to come to grips with the distinction between information and its material carrier(s)" (para. 2). Perhaps we did not "come to grips" with it sufficiently, but we explicitly acknowledged this distinction in our target article, citing yet another quotation from G. C. Williams.

In our target article we argue that reference to information is necessary in understanding selection processes. Many of our commentators disagree (**Baum, Chiesa, Eacker, Godfrey-Smith**, and **Hineline**). These authors think that reference to information in a general analysis of "selection" is not only unnecessary but pernicious by making operant learning look more different from gene-based selection in biology and the immune system than it has to. We also claim that none of the current analyses of information are good enough for the role that information plays in selection processes. In particular, it cannot distinguish between those traits that are "coded for" and those that are not. **Eckerman & Kemp** disagree. They think that Dretske's (1981) distinction between message and channel is good enough. "According to Dretske's definition, the structure coding for an end structure is message while the double-helical structure, the type of chemical bonds, the means of gene transcription, and so on, are all channel with respect to phenotypic characteristics" (sect. 1). Mahner and Bunge (1997) strongly disagree: classical information theory re-

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quires a coder, transmitter, receiver, decoder, and information channel in between, and they see none of these components in chemical systems. Godfrey-Smith (2000, p. 28) is a good deal more measured in his response. As he sees it, the concept of "genetic coding" is intended to distinguish between two sorts of causal pathways: those that lead from genes to traits and those that lead from non-genetic factors (including the environment) to traits. But on "any standard concept of information, both genetic and nongenetic factors can carry information about traits." Although Godfrey-Smith thinks that the notion of genetic coding does have a place with respect to the relation between genetic information and the primary structure of proteins, he thinks that this is the only legitimate use of this notion. In particular it should play no role in evolutionary theory. Thus, Godfrey-Smith disagrees with us about the role of information in selection processes but supports our claim about the inadequacy of standard concepts of information, including Dretske's concept.

We supplied no general analysis of "information" adequate for its role in selection processes. In the meantime, John Maynard Smith (2000) published a target article followed by responses from four philosophers – Sterelny (2000), Godfrey-Smith (2000), Sarkar (2000), and Winnie (2000). In his target article, Maynard Smith attempted to explain and justify the use of the information analogy in biology. These philosophers were not impressed. Part of the problem is differences in expository style between scientists and philosophers, but even if one is charitable in reading these papers, real differences persist. In the main, biologists are convinced that information is central to selection processes, even if philosophers do not think that any of the current general analyses of "information" capture these similarities. Philosophers tend to respond that, if the information analogy does not fit current analyses of "information," then the fault lies with the analogy. Winnie is the chief exception. He distinguishes between two general sorts of information theory – classical and algorithmic. In response to both Maynard Smith and his critics, Winnie (2000, p. 522) observes that what is missing is the "realization that this notion of 'information' is not that of Classical Information Theory, but Algorithmic Information Theory, and thus the search for strict analogues for 'messages,' 'channels,' and 'symbols' is unnecessary."

In sum, we agree that including fairly restrictive notions of replication and information in our analysis of "selection" raises the bar for operant learning. It may even be the case that we have raised the bar so high that operant learning cannot make it, but all suggestions for lowering the bar result in notions of "selection" that seem way too broad. If we made our definition as broad as many of our commentators would like, then operant learning poses no problems, but then just about any process turns out to count as a selection process.

R1.4. Information and inheritance

Several authors want to pry information, replication, and inheritance apart and restructure them. As **Godfrey-Smith** sees it, information and replication are found in some but not in all selection processes even if one limits oneself to biology. Of course, as he points out, any causal processes can be described in informational terms, but not all selection processes involve information in a non-trivial way. According to Godfrey-Smith, inheritance is also independent of information. Some but not all processes of inheritance work via the transmission of information. And finally, inheritance does not require even replication. To support these contentions, Godfrey-Smith introduces an example – gut symbionts. Micro-organisms commonly live in the gut of such ruminants as cattle and are passed on to successive generations of the ruminant independently of the ruminant's DNA. Godfrey-Smith takes this example to count against the belief that the only way that structure can be passed across generations is by the coded specification of that structure. Another way is by passing along a sample of that structure, and that is what Godfrey-Smith thinks happens in the case of gut symbionts.

This example warrants more attention. A calf does not receive the genes for its gut symbionts via its parents' DNA. It possesses no genes for generating these symbionts. Instead, it picks up these necessary symbionts by eating the fecal material of other cattle, most likely its mother, but treating such transmission as "inheritance" seems bizarre. In the case of the gut symbiont, the entire organism is ingested, but these organisms do not function as "samples" for the production of additional symbionts but reproduce in the usual way. From the start we have emphasized that genes are not the only replicators. If they were, operant learning could not possibly count as a selection process. The issue is the ubiquitous role that replication plays in selection. We do not see that Godfrey-Smith's example bears on this issue.

Godfrey-Smith goes on to reiterate arguments made by Lewontin (1970) and Thompson (1994) concerning the need for inheritance in selection processes. Godfrey-Smith argues that for "inheritance to exist, there has to be some way of reliably producing similarity (along some dimension) between relatives across generations, but this mechanism need not involve an underlying population of entities (like genes) that are copied in the wholesale and direct sense associated with the (Dawkins-Hull) concept of a replicator." Selection requires no more than a "systematic correlation between parent and offspring," or "parent and grandoffspring, or a more distant relative" (last para.). In biological evolution, the replication of genes is the primary, possibly exclusive, mechanism for the accomplishment of this task. But it need not be. Some other mechanism might be up to this task. So far the only mechanism that has evolved in biological evolution (save possibly prions) turns on the replication of genes. The replication of genes is surely not necessary for replication. The issue is whether replication itself is necessary. We think that it is.

Godfrey-Smith cites Thompson ((1994, p. 638) approvingly to the effect that natural selection "does not require genes or even direct descendants; all that it requires is that the presence of a configuration of elements in one generation makes more likely the presence of the same configuration in the next generation." Thompson's notion of selection is very weak. In addition to successive configurations of the same sort, all he requires is generations, but that puts a great deal of weight on the notion of "generations." A configuration occurs in Alpha Centauri; for example, two planets line up in syzygy with their star. A few Earth-years later, that same configuration occurs in our galaxy. Two of our planets line up in syzygy with our star. A split second later this configuration occurs in Alpha Centauri again. Why not count these successive configurations as fulfilling the re-

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quirements for selection? The answer is the absence of generations.

In addition to generations, **Godfrey-Smith** also requires relatives, parents, and offspring. If these notions are contained in the definition of "generation," then Thompson and Godfrey-Smith agree in their highly general analysis of selection; if not, they disagree. Right now the only hereditary relationship between parents and offspring is replication. Replication need not occur via DNA or RNA. How else? One might be able to come up with dozens of science fiction examples. The problem is finding a principled way in which to make these decisions – where to draw the line and on what basis. This problem is endemic to linguistic analysis.

Three notions have been suggested as being central to selection – *replication*, *information*, and *inheritance*. These three notions form eight possible permutations. We think that all three notions are part of selection. Others include inheritance and replication but not information. Still others argue that none of these elements are necessary for selection, and so on for all eight possible permutations. It would be unfair to say that the authors who hold these various positions have little in common save their conviction that their combination is the right combination. Nearly all the authors who discuss this issue express some doubt about the proper definition of "selection." No one has presented knockdown arguments for their preferred position. Instead, on these matters, readers will have to make up their own minds.

R1.5. Sequential iteration

Instead of postulating a single, fairly restrictive notion of selection, **Nanay** prefers three notions of selection that are collectively broader than our single notion – *simple one-step selection, iterated one-step selection,* and *multi-step selection*. Although Nanay thinks of his view as being "pluralistic," it is not pluralistic enough. He argues that multi-step selection is too narrow to apply to operant learning, but neither simple one-step selection nor iterated one-step selection is adequate to handle operant learning either. Hence, a fourth alternative is required. According to simple one-step selection, extensive variation exists. Nanay does not tell us the source of this variation. It may just exist (no explanation provided) or it may have been introduced gradually or quite abruptly. The environment then reduces the amount of this variation either gradually over time or in one fell swoop – but only once. The environmental interactions responsible for this reduction are adaptive. In some sense the items that remain are better than the original full array. Nanay thinks that the development of the central nervous system may well exemplify simple one-step selection. On rare occasions operant learning can also exemplify simple one-step selection. Allow a rat to respond only once and then kill it. Quite obviously, operant learning involves more than simple one-step selection. So do the other two.

In iterated one-step selection, there are repeated cycles of replication and environmental interaction, but these interactions do not cause replication to be differential. The initial story is the same as in simple one-step selection, but after the initial variation is winnowed down, the process is repeated. **Nanay** does not say very much about this process of iteration. After the initial variation is reduced, does it all spring back into existence again or must selection act solely on the variants that survived the initial selection process? On the former interpretation, iterated one-step selection is truly miraculous. On the latter interpretation, the initial variation is slowly used up. Nor does Nanay's reference to Campbell's evolutionary epistemology help in the least, and again the problem is the term "blind." Campbell thinks that initially, with respect to each organism, variation is roughly "blind," but as selection proceeds organisms accumulate the results of early selection. Hence, variation is decreasingly "blind" and increasingly constrained. Previous environmental interactions influence later interactions. We can overcome these constraints, but the process is extremely difficult (see **Cziko, Madden,** and **Simonton** as well as Heyes & Hull 2001). To make matters worse, iterated one-step selection is not good enough for operant learning. In operant learning later interactions are influenced by effects of earlier interactions. What is missing from iterated one-step selection is the periodic introduction of additional variation.

One objection that we have to **Nanay**'s pluralist analysis is that it is not sufficiently pluralist to handle operant learning. Perhaps multi-step selection is not good enough (we will address this issue shortly), but certainly Nanay's two additions do not help at all. A second objection to Nanay's two additional notions of selection is that they are parasitic on multi-step selection. Because multi-step selection exists, we recognize simple one-step selection and iterated one-step selection as separate causal processes, but if it did not, we would have no reason to distinguish these causal situations from any other. Even describing examples of these two putative selection processes cogently, leans heavily on the contrast with multi-step selection. What counts as one-step and iterated one-step selection? I make a copy of a letter and throw it away. Or I make a copy of a letter and throw the original away. Nothing more happens. We see no reason to term this change "selection." I make a copy of a letter, and throw the copy away. I make another copy of the letter and throw the original away. Then I make yet another copy of this letter and throw the copy away, and so on. Except for changes in the physical identity of these pieces of paper, no change occurs. Replication is not differential. Is this iterated one-step selection?

We acknowledge that the preceding examples seem strained, but that is because they are not presented in a real detailed context. In his warning about the dangers of recursive explanations, **Thompson** counsels that adequate attention must be paid to the details if vacuous or ad hoc explanations are to be avoided. For example, the most obvious explanation for polar bears having white fur is that it serves as camouflage. However, the more likely explanation is much more complicated and depends on the details of the makeup of polar bear fur. We urge a comparable warning for the examples used to evaluate various analyses of "selection" and related concepts.

R1.6. Group selection in the immune system

The problem of group selection is that it can be defined in various ways and made to look absurd or sensible accordingly. **Okasha** added the twist of frequency dependent selection. If there is a population with frequency distribution A and another with frequency distribution B and these are not the result of drift or bottlenecking, Okasha seems to suggest that the two distributions could have arisen via se-

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lection on individual organisms (required in frequency dependent selection) or via selection on the population (a usual requirement for group selection).

The MHC system poses a difficult problem for selection. To summarize briefly the key observations. Within a species, and in large measure between species, there is a common pool of 100 different genes that each act as a receptor for protein fragments found inside cells (some fragments are from the host, but the really important fragments are those derived from intracellular pathogens). An almost random sample of between 4 to 8 of these genes (usually referred to as alleles) is present in every individual. This sample is not quite random because it is arranged so that there are no repeats on the same gene. However, any sample of 4–8 taken from the pool of 100 functions equivalently in that each set of MHC alleles in a particular organism is able to recognize 99.9% of the protein fragments with which it will be presented. The net result when viewed from a population perspective is that each allele is expressed at roughly the same frequency. The MHC locus is the most polymorphic locus known. There are more alleles of hemoglobin, but there are a few common alleles and all the rest are at a frequency expected by mutation alone.

Any interpretation of this frequency distribution has to include the point made by **Okasha** that "the fitness of any given allele will be low if many other individuals in the population possess it." However, Okasha's statement that "conversely, a rare allele will have a fitness advantage" is not obvious in the case of MHC alleles. Consider sets of four alleles arranged so that each of the four recognizes a different quadrant of the protein universe, but each set represents a different way of slicing the protein universe into four pieces. So long as the protein universe is recognized by an individual organism, the MHC is capable of doing its job. The question now is what selection pressure would require all these different sets of four alleles if each performs an equivalent function? The answer, it seems, is tied to the vastly different rates of evolution of the pathogens (with generation times of the order of hours) and the rates of evolution of vertebrate hosts (with generational times of months to decades). Pathogens require a population of hosts for their survival, and this leads to a corresponding population level selection in the case of the MHC so that any selective advantage obtained by a viral pathogen that can avoid having its proteins recognized by the MHC alleles of one host is placed at no selective advantage when the virus moves to a new host which has an exceedingly low probability of having the same set of MHC alleles. It seems unlikely that a small set of MHC alleles could readily evolve into a large set under selection by an array of typical viruses because any new allele is effectively redundant and neutral in a given individual. A new allele is effective only if it is sparsely distributed in a population of individuals. Thus, the MHC alleles are more likely to have arisen as the immune system itself arose from some more restricted precursor, as new pathogens arose to challenge the emerging immune system. This scenario would be consistent with the striking similarity of MHC alleles in mice and men (similarity with respect to structure and function, not strict nucleotide sequences which necessarily drift).

Our emphasis in the case of MHC was on the necessity for selection to act on the population even though this requires selection on individuals. Whether this should be more properly termed frequency dependent selection remains unclear. Particular alleles are not under selection for their particulars. They are under selection because they are different and equivalent. Selection in this case cannot act to increase the frequency of a particular allele at the expense of any other allele that is less well adapted. Selection is for a dilution and dispersion of alleles in the population, not for any specific property that can be assigned to an individual. Different numbers of alleles and somewhat different distributions of alleles may occur in populations that are exposed to a few dominant pathogens that represent only a small fragment of the universe of proteins. Cheetahs, for example, have very few MHC alleles, but this is more likely a case of neutral drift in the absence of selection. Thus, there can be no selection in the population that raises or lowers the allele frequencies unless that selection results in the loss of a large fraction of that population. Although there are pathogens that have >70% lethality (e.g., plague, smallpox, and even now HIV), in no case is survival associated with particular MHC alleles, although these events can cause bottlenecking as an unselected byproduct. In the end, which name is given to the selection acting on MHC alleles is less important than recognizing that there can be selection pressures that act on populations as a whole.

Heschl raised some points regarding selection in antibodies; but, we suspect that these are simply misunderstandings. To clarify questions about variants that appear repeatedly in the antibody population, we point out that the appearance of a particular antibody specificity can be for any of three different pathways. First are the germline encoded specificities. There are around 100 genes for each of two polypeptide chains that combine as a combinatorial assortment to produce 10,000 different pairs and 10,000 corresponding antibody specificities in the B cell population. The B cell population contains about 10 million cells per gram of body weight (100 million in total for a mouse, 1 million million for a human) so that these germline encoded specificities will appear repeatedly in different parts of the immune system of one organism as well as being repeated in different individual organisms. Second are the specificities generated by a single mutation of the germline genes. In this case, the number of different possible single step mutations that can result in a change of antibody specificity is rather small (about 100 per antibody gene) so that in the large population of B cells each mutation will, by chance alone, occur repeatedly in the same individual as well as in different individuals. These single-step mutational changes will occur at a much lower frequency than germline-encoded specificities in the total B cell population. Finally, there are the vast numbers of multi-step mutations that in the absence of selection would be vanishingly rare. Thus, there are antibody specificities that arise at such a high frequency that they can be detected in the absence of selection, and others that require selection to attain functional levels.

R1.7. Entities, events, and units

As we treat selection in this paper, it is a process made up of two subsidiary processes that are related to each other in very precise ways. Selection consists in repeated cycles of replication, variation, and environmental interaction so structured that environmental interaction causes replication to be differential. The net effect is the evolution of lineages through time. Several things need to be noted about this

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characterization of selection. First, with the exception of lineages, no mention is made of any entities; only processes. However, reference to processes lead us quite naturally (if uncritically) to refer to entities functioning in these processes. For example, we postulate replicators as the entities that function in replication, interactors as the entities that function in environmental interaction, and lineages as the entities that evolve, but we do not postulate "selectors" as the entities that are selected, unless of course we are using this term ambiguously to refer to either replicators or interactors.

Evolutionary biologists do not recognize "selectors" as entities. How come? The reason for this reticence is that there are no units of selection, no entities independent of the other two processes. Selection is nothing but successive cycles of replication and environmental interaction. When biologists refer to units of selection, sometimes they mean units of replication and their accompanying entities – replicators; usually, however, they mean environmental interaction and their accompanying entities – interactors. At bottom, there are two primary processes and, if you will, entities that function in these processes, but there are units of selection only in a highly derived sense, postulated in order to function in various laws of population genetics. The concepts that population biologists devised to allow them to develop highly general laws about the evolutionary process are far from intuitively clear; for example, heritability and fitness. No one thinks that these units refer to entities. At bottom, the most fundamental contrast is between mechanisms and process laws.

Several commentators have remarked that if we couched our analysis in terms of events, not entities, we would be able to incorporate operant behavior in our analysis much more smoothly. For example, **Eckerman & Kemp** observe that the "behavioral entities constituting the interactors are events, rather than objects. And, if the units of interaction in behavior are events, might not the units of replication and variation be also?" (penultimate para.; see also **Chiesa**). Closely connected to disagreements about entities versus events is the idea of mechanisms. For a notion so central to science, until quite recently no one has spent much time trying to get clear about the relevant features of mechanisms. This omission has been rectified by Machamer et al. (2000). According to these authors, mechanisms are "composed of both entities (with their properties) and activities. Activities are the producers of change. Entities are the things that engage in activities" (Machamer et al. 2000, p. 3). As natural as reference to mechanisms is in certain areas of science, **Reeke** does not like mechanisms at all. His aim is to provide an "expanded definition of selection that is cleanly mechanism-free" (penultimate para.). At bottom, our analysis is couched in terms of processes – variation, replication, interaction, and evolution. Claims made about these processes can be couched in terms of entities or events as the occasion demands. In this respect we are dualists (see also Machamer et al. 2000).

Several commentators complained that operant learning did not possess the monolithic and discrete units found in gene-based selection in biology and the immune system. Operant learning cannot count as a selection process until psychologists produce units as unproblematic as genes. We think that these assumptions about "units" must come from our lack of knowledge about fields outside our own. The other guys' units always look less problematic than our own because we know so much more about our own. Several commentators refer to genes as the unproblematic units in gene-based selection in biology. For example, **Madden,** in his otherwise positive evaluation of our paper, remarks that "Skinner fails to identify the behavioral unit of retention analogous to the gene and thereby ignores neurophysiology. Hull et al. hypothesize that behavioral consequences change the chemistry and firing patters of the central nervous system, but identifying this neuro-chemical unit of retention will either be extraordinarily difficult or impossible" (para. 2).

For a short time, when Mendelian genes were first postulated, they were treated as if they were beads on a string. Rapidly, geneticists discovered that they were nothing of the sort. The story became even more complicated as we learned more about the molecular structure of the genetic material. Genes are anything but monolithic or discrete. Bits and pieces of genetic material are snipped out, some discarded, other bits pasted together with the aid of still other molecules. A single "gene" is actually a conglomerate of bits of genetic material gathered from various parts of the genome. The belief that unitary genes exist is commonly derided as "beanbag genetics." In fact, the processes of genetic replication and application are so complicated that it is a miracle that they ever work right even once, let alone almost all the time. The message is that **Madden** and others are right that neuro-chemical units may not be as discrete and monolithic as they would like, but the same can be said for other units, including genes. **Eckerman & Kemp** are also right that identifying the units of replication and interaction for behavior will be far from easy, but such difficulties are common in science.

R2. Behavioral ontology

Several commentators raised issues that pertain to the nature and measurement of behavioral phenomena. The ontological perspective explicit in the target article differs in some respects from the implicit ontology of most behavior analytic theory; and it differs radically from the prescientific ontology of our everyday experience of behavior. First, there is the issue of "what counts" as a single instance of operant behavior. We agree with **Eckerman & Kemp**'s distinction between a behavioral instance defined in terms of motor activity (however much of the organism is involved) and a behavioral instance viewed as an action. Operant interactors typically include specific organismic activity as well as parts of the world outside that activity (and often outside the organism). Even the "simple" acts we discussed (leverpresses and keypecks) necessarily include the operandum (lever or key) as part of each occurrence. Although **Bevins** preferred to focus on the lever's stimulating function, (the sight of it presumably functioning to evoke locomotion with respect to it and the feel of it presumably functioning to evoke downward pressure against it), the act of leverpressing entails both an organism's pressing movements and the lever pressed. As noted by **Pennypacker**, and also by **Vaidya**, the effect on the environment (e.g., displacement of the lever in space resulting in switch closure) qualifies as one of the defining features of an act's occurrence. In fact, variations of leverpressing that do not have the property of lever displacement do not get selected in the standard preparation. The effect (e.g., switch closure), as noted by Pennypacker, is not to be confused with the re-

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inforcers (e.g., food) that are the environmental selectors of the act. Traditionally known as "consequences," the reinforcers are not part of the act but are contingently related to the act. The contingency (fit) between interactor properties (including effect) and consequences (reinforcer deliveries) probabilistically determines features of interactors in the evolving or stable lineage.

Although **Pennypacker** supports a selectionist perspective of operant learning, his approach to variability differs from that of the target article. The difference rests on different ontological assumptions regarding the relation of operant instances to the unit of analysis (operant). Pennypacker's assumption is consistent with the time-honored ontology of an operant as comprising a *class* of individual responses (or acts). In this view, the relation of an operant (e.g., Rat A's leverpressing) to one of its individual acts is that of class to class member. The target article explicitly assumes a different kind of relation between an operant and its individual acts. Specifically, both an operant and the occurrences it comprises are individuals and the occurrences are *parts* of the more inclusive and evolving operant lineage. This perspective is consistent with the contemporary perspective of species as evolving individuals comprising organisms as their parts (Ghiselin 1974; Hull 1976). Anything that evolves must be localized in space and time; that is, it must have the ontological status of "individual" (Hull 1988). The theoretical terms in the target article for analogous relations between species and their organisms and between operants and their response instances were *lineages* and *interactors*. Pennypacker's concern regarding variability as a dimension of behavior stems from his ontology. Because only behavioral occurrences are individuals, only they can be directly measured. Since a class is not localized in space and time, it does not have a "basic dimension" (Pennypacker). However, if an operant (lineage of acts) is an individual, it is possible to measure it directly. Its rate is a property of the operant lineage as an individual, even though that measure may itself depend on the dimensions of its parts (just as the weight of an organism may itself depend on cellular water retention, fat composition, bone porosity, etc.). Similarly, variability among the individuals constituting an operant lineage can be measured directly and the variability viewed as a property of the particular operant lineage being measured (cf. Page & Neuringer 1984).

Because it is change over time in population characteristics of an evolving lineage that we are interested in, it is necessary to measure changes in evolving lineages over time. This bears on **Okasha**'s point regarding the need to segment time (in a principled way) to make possible any measure of evolution. Okasha sees this need as particularly problematic for operant selection, where multiple interactors (behavioral phenotypes) are not available at the same time for differential replication of selected characteristics in later interactors (phenotypes). However, what might "the same time" mean in the case of organic evolution? The facts of sexual reproduction have resulted in populations of genetically related organisms spread across space and overlapping one another in time. But evolution is measured in terms of changes in *successive* populations, so the "time" that is relevant to evolution is spread out over the *successive generations of interactors*. Where does one draw the line between a successor population and its progenitor population to determine if change has occurred? Not between one generation and the next (however generation boundaries are drawn) because evolution occurs across many successive generations. The principled answer we suggest is that if selection as the cause of evolution is being studied, then one looks not just at the lineage under investigation but also at its selecting environment to establish the boundaries of Time 1 and Time 2 populations. In operant experiments, the boundaries of populations are defined by the selection contingencies in effect during different time segments. Typically, when selection contingencies are stable, characteristics of the lineage reach a "steady state." A change in contingencies results in a transition phase and eventually another steady state (see Fig. 1 in target article for graphical depiction). This is rather like punctuated equilibrium in biology. In short, although evolution is often defined in terms of replicator frequency, as Okasha points out, selection requires that changes in replicators make a difference in the fit between interactors and their environments. Fortunately, the fit between interactors and their changing environments is exactly what can be directly studied in operant experimental work. As **Moore** points out, these "terminal relations" will not be changed when we understand the neurophysiological mechanisms that retain the structure of behavior/environment relations from one occurrence to the next.

Vaidya, whose response is generally favorable to our selectionist perspective of operant learning, asks "Are units of retention necessary?" – evoking recall of Skinner's famous 1950 article entitled "Are theories of learning necessary?" The 1950 article may be seen as a small part of Skinner's one long argument (beginning in the 1930s and continuing until his death in 1990): *for* a science of behavior/environment relations and *against* substituting guesses about the nervous system, the mind, or the conceptual nervous system for experimental analysis at the level of behavior/environment relations. Perhaps we should clarify that the target article is not meant to suggest a practice of substituting guesses about the nervous system for current or future knowledge of behavior/environment relations. It suggests that nervous system activities are part of the world to be explained by a selectionist theory of operant learning. The article provides a very sketchy outline for a synthetic theory of behavior and suggests that this synthetic theory belongs to a general class of theories in which variation, replication, and interaction account for novelty in the material universe in the absence of design. Again, how replication occurs in the behavioral case is as unknown now as knowledge of inheritance was in 1859, when the first theory of evolution by selection was put forth.

Are units of retention necessary? There may be disagreements regarding the nature of the units, but for a selectionist theory of learned behavior, the answer is likely "Yes." Nothing in the general account of the target article should be construed to suggest that learning can ever be accounted for solely in terms of what goes on in the nervous system. **Vaidya**'s concern that emphasis on units of retention could lead to decreased reliance on histories of reinforcement in the analysis of behavior is not entirely misplaced, however. The rapid development of molecular biology appears to have overshadowed all other aspects of evolutionary biology. The title of a recent book suggests the game is not over. *The triple helix: Gene, organism, environment* (Lewontin 2000) parallels quite nicely the perspective

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of the target article with regard to the interdependent roles of nervous system, acts, and selecting behavioral environment.

Malott wonders if the target article provides insight into new dimensions of behavior or environment or suggests new functional relations. **McSweeney & Aoyama** do formulate an experimental question they see arising from the selectionist account in the target article. They suggest making reinforcement contingent on one of several characteristics of successive operant interactors, measuring detailed changes in each successive response. It is not entirely clear to the authors that the formulation in the target article suggests the very next response after reinforcement would be maximally similar to the reinforced response, as suggested by McSweeney & Aoyama. Interactors in an existing lineage may vary as noise, adverse environments (for example, extinction), or reinforcement of variability itself (Neuringer et al. 2000). Any response that occurs is a member of a local population (part of the lineage) having more or less variation among its members. According to the target article, it is the population characteristics that change over time. Can the changes in the population be tracked on a response by response basis? That is an interesting possibility, but from the authors' perspective not a requirement for a populational approach.

McSweeney & Aoyama present an alternative hypothesis, to wit does the environment choose "from among many behaviors?" (para. 7). It is hard to answer this question because "many behaviors" can mean many different things. If the "many behaviors" all belong to the same operant lineage, we would say "yes, variations occur and only some are chosen." But there is another sense in which the environment may choose from many behaviors. This is the sense in which the environment chose mammals over dinosaurs in Earth's history. However, the principles of evolution don't work very well in this context. The "variation" between dinosaurs and mammals was not relevant to this differential selection. Nor did the interaction of dinosaurs with their environment differentially affect replication of mammal genes in any direct way.

R3. The biology of behavior

R3.1. External/internal behavioral environment

The research described by **Pierce** provides an excellent example of experimental work that demonstrates complex relations between actions altered by changing contingencies of reinforcement, the relative and changing values of different consequences as reinforcers, and neurochemical changes programmed by natural selection and activated by contingencies of reinforcement. Although Pierce's work implicitly supports our general account of selection, it does not directly address our thesis that selection is *repeated cycles of replication, variation, and environmental interaction so structured that environmental interaction causes replication to be differential*. Pierce's research does point up the artificiality of identifying the behaviorally functioning environment with only those events that occur outside the organism's skin. What counts as a behavioral environment is (1) any event (or relation between events) outside the behavioral unit to be explained (2) that has a functional (i.e., causal) relation to the unit to be explained.

R3.2. Neural subsystem in behavior/ environment relations

Burgos's Figure 1 can be read so that the neural network represents an environment/behavior sub-system of the more inclusive behavioral system generally designated as environment/behavior relations. Input neurons represent environmental events within the neural network, output neurons represent behavioral events within the neural network, and the hidden units a kind of sorting mechanism that links them in a variety of combinations depending on other factors within the nervous system, within other parts of the organism, and outside the organism. The sorting mechanism is the result of natural selection, but its current role in enhancing survival and reproduction of phenotypes (and thereby their genetically coded characteristics) is its organismic function of mediating relations between input and output neurons. The neural network in Burgos's Figure 1 is itself part of a larger behavioral unit that includes (1) events in the environment outside the neural network, represented by the word "environment" to the left of his network, and (2) the behavior that occurs at this time, represented by the word "behavior" to the right of his network. The "end points" of this larger system are generally the topic of interest to behavior analysts. As suggested by the word "environment" to the left of the network, activation of the network at any particular time depends on input from events outside the network (and usually outside the nervous system as well). Given the inputs from outside the network and the sorting, the *neural outputs are the initial phase of the behavioral event that occurs.*

In summary, neural networks are sub-systems of more inclusive systems described by the various environment/behavior relations constituting an organism's behavioral repertoire. The larger system includes more of the organism's body (e.g., receptors and effectors) as well as parts of the world outside the body (for example, the operandum and discriminative stimulus). Just as an organism's genes are part of the organism (phenotype) that interacts with the selecting environment, a response's neural components are part of the response (phenotype) that interacts with the selecting environment (cf. **Blute**). Which brings us to the issue of selection, because selection is that which explains the recurrence of, and changes over time in, particular environment/behavior relations that are observed.

What accounts for the origin and maintenance of the functional environment/behavior system as an evolving behavioral unit? Burgos's Figure 1 shows S*, representing unconditioned reinforcement, as an input to the system. Although the Figure generally suggests temporal relations among the elements of the neural network, input S* in its role as selecting environment is misplaced because the unconditioned reinforcer actually follows occurrences of the behavior in both natural and laboratory environments. While it surely is another input, this input has a different temporal relation to the behavior represented in the figure. This is important, because the function of S1 in the larger system is fundamentally different from the function of inputs represented by I1, I2, and I3. Specifically, I inputs function to evoke individual occurrences of the behavior. The function of S* is that of differentially selecting characteristics of some environment/behavior occurrences over others, thereby accounting for characteristics of the popu-

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lation of recurrences as they play out over time. It is possible that the mechanism underlying S*'s selective function entails the classical conditioning of Inputs 1–3 as CSs associated with the food deliveries. If so, Pavlovian conditioning is integrally involved in operant selection and its role may be something like that suggested by **Burgos**. Commentators **Bevins** and **Blute** also argue for a role for Pavlovian conditioning in behavioral selection processes, a possibility we are not opposed to but which would have taken the discussion in a direction different from our focus. In any case, distinguishing between the units affected by the environment when the environment is functioning as selector of population characteristics and the units affected by the environment as evoker of behavioral instances will likely remain important in a selectionist theory.

R4. Role of stimuli

Many commentators rightly pointed out that we neglected the role of stimuli in the process of operant selection. Reference to "stimuli" in the commentaries virtually always pertained to *antecedent* stimulation (not the stimulation involved in operant consequences). No doubt there is always a great deal of stimulation going on when behavioral events occur. The task of ascertaining the roles of stimuli in behavioral processes is made immensely difficult for students of learning by the fact that any class of stimulating events can and usually does have multiple concurrent behavioral functions. For example, the sound of the food dispenser may function concurrently as elicitor of conditioned salivation and conditioned reinforcer for lever presses. To make matters worse, a stimulus presentation may function one way at one time and another way shortly thereafter, depending on other events in the current causal nexus. (For example, a red traffic signal that usually evokes braking may evoke accelerating if no cars are in sight.) Rather than mislead readers of the target article by representing the functions of antecedent stimulation simplistically, the authors chose to consider the limiting case of operant selection – the case where no particular stimulation appears to be differentially correlated with instances of the operant. Although **Bevins** is probably right that "responses do not occur in the absence of stimuli" (penultimate para.), it does not follow that the stimulation occurring necessarily has a function with respect to any behavior that occurs. However, we agree with Bevins that the role of stimuli must be considered in a more comprehensive account of behavioral selection.

One reason any discussion of stimuli raises a host of complex issues is that antecedent stimuli have so many different functions with respect to operant behavior, for example, discriminative, conditional, motivative. One thing that they have in common, however, is that they are functions describing proximal causes of individual responses. Note that the behavioral unit being explained by the antecedent stimulus is a behavioral instance, or the occurrence of an act in an organism's behavior stream (e.g., Sam slammed on the brake when the traffic signal turned red). It is true that no scientific ascription of Sam's sudden braking to the change in traffic signal would be possible if the particular relation was not repeatable (and repeated). Nonrepeated causal relations between stimuli and responses no doubt often occur, but they are like mutations that did not "pay off" in the selection lottery. The causal relations relevant to a selectionist account are those that recur. A selectionist account of learning explains those systematic causal relations between stimulus events (and other current environmental conditions) and responses that reliably follow them. Selection is the ultimate cause of the functions of antecedent stimuli. That is, behavioral interactors are usually selected on the basis of their relation to specific current stimuli as well as on the basis of their own properties (see **Blute**'s Fig. 1 for schematic representation of this point).

As suggested above, the kinds of relations that can come to exist among stimuli (their presence, their properties, and relations among them) and behavioral interactors are many and often exceedingly complex. **Bevins** suggests that stimuli are important sources of variants among behavioral instances in a lineage. We agree. The classic case is seen in generalization gradients, where an organism trained to press a lever when a 500 Hz tone is on, but not when it is off, presses at varying frequencies when tones of 300, 400, 600, and 700 Hz are present. Because only the frequency of responses to the various novel stimuli is measured, little can be said about variation among their individual properties. However, it is known that a change in selection contingencies (for example, the 300 Hz tone becomes the stimulus that signals reinforcement for pressing) results in redistribution of the variants in terms of their frequency in the lineage.

R5. Replication in operant learning

Spiga, Moore, and **Leslie** agree in principle with our analysis and each offers a perspective on the issue that vexes many readers. What is the relation between neurochemical replication and behavioral interaction? Spiga considers the relation as causal, but the causation is like the non-exclusive causal relation between genotype and phenotype. Moore focuses on the response/consequence contingency (interactor/environment match) as the cause of changes in the state of the neural systems. Both of these suggested causal relations are consistent with the target article's account of selection as involving two related processes: (1) replication and (2) interaction with the environment that causes differential replication. Leslie points out that bringing together the mechanism and the process of operant learning is an interdisciplinary challenge. It is because operant psychologists currently know more about the process than neuroscientists know about the mechanism, that an integrative account such as in the target article has so many gaps.

Grantham raises interesting questions about what "descent" might mean in operant selection. Perhaps we can approach this problem by answering specific questions that might be raised about descent of any kind. Must a descendant phenotype in a lineage overlap in time at least one ancestor phenotype? Obviously not, in either the organic or the behavioral case. Must ancestor and descendant have some common features? Only if further selection depends on those features they have in common (which is almost always, perhaps always, the case). How are the features "passed on" from one phenotype to another? Clearly, some material must remain in existence between appearances of the ancestor and descendant phenotypes (fertilized egg, spores, DNA in organic case; neural pathways in behavioral case, etc.). How might this dormant "material" be activated? By the occurrence of some environmental event that

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previous selection has caused to have activating properties. Or perhaps by the passage of time if the material has endogenous cyclicity. How does the activation of the dormant material relate to the properties of the phenotype that appears? Not often in any 1:1 fashion but through a developmental process that is likely to be increasingly elaborate and dependent on additional "inputs" in the case of increasingly complex phenotypes.

Even if the unresolved issues regarding replication in operant selection are never resolved, **Grantham**'s conclusion, that if one retains a gene-based analysis of selection, then operant learning is not a selection process, is too far reaching. It is not necessarily the case that all selection processes work in similar ways, even though the goals of the target article were to examine the possibility of a general account that included operant learning. As **Hesse & Novak** ask: "Are convincing matches [in selection processes] required before a selection account is considered valid?" (para. 3). We see no requirement, only interesting possibilities.

R6. Conclusion

Evolutionary biologists and immunologists use the language of selection without any hesitation. However, as **Tonneau & Sokolowksi** observe, analogies between "operant reinforcement and natural selection have been around for decades." Yet the "vast majority of behavioral researchers still publish their findings without any reference to such analogies" (para. 1). Our analysis was designed in part to rectify this situation. We agree with **Malott** that, if our analysis is to become accepted, it must provide "new insights" into the phenomena under investigation (para. 4) or as **Vaidya** observer increased "utility in organizing or reorganizing the practices of researchers and practitioners in the various fields represented by the authors" (last para.). In our target article we tried to fulfill both requirements. In the final analysis, however, use will decide the fate of our General Analysis of Selection – or GAS for short.

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Letters "a" and "r" appearing before authors initials refer to target article and response, respectively.

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