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The flight of the locus of selection: Some intricate relationships between evolutionary elements

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ABSTRACT

Selection has enriched our understanding of the world since it was first applied to the evolution of species. Selection stands as an alternative to essentialist thinking, as a generalized and multiply applicable concept, and as a causal explanation for current forms within biology and behavior. Attempts to describe selection processes in a generalizable way have provided clarity about their minimal elements, such as replicators and interactors. This paper discusses the interconnectedness among different levels of selection using evidence garnered from evolutionary biology, development, epigenetics, neuroscience, and behavior analysis. Currently, it appears that replicators and interactors may be more fluid than previously supposed and that selection for particular traits may rely on both multiple levels of interaction and multiple levels of replication. Replicators, interactors, and environment share influence on one another, and different replicators may exchange critical control over similar interactor variation as evolution proceeds. Our current understanding of selection continues to undergo revision, and reference to a number of disparate fields can help to account for the complexity of these processes. An understanding of their interconnectedness may help resolve some mysteries that develop in fields that exclusively focus on one or a few, such as the focused study of behavior.

1. Selection and teleonomy

In 1858, Charles Darwin proposed the evolution of species by natural selection and supported his thesis with extensive observation of multiple species (Darwin and Wallace, 1858). Since then, the theory of evolution via natural selection has revolutionized our understanding of causation in the natural world. Neither "billiard ball" causation, so well investigated in the realm of physics, nor the unaccountable idea of teleological, purpose-driven design could clearly account for the awesome specificity and complexity of living systems that were clearly fitted to the challenges that they encountered in their environments. Darwin discovered that the consequences of past organism-environment relations could account for the apparent preparedness of living systems. Monod (1974) referred to this selection-based preparedness as "teleonomic" rather than "teleological" to specify adaptation as free from causes in the future. In the course of enormous scientific progress since that time, selection-based causation has been extended to new paradigms in the biological and behavioral sciences. It has become clear that the teleonomies of living systems and their behavior are the product of multiple, interdependent processes of selection in biological, behavioral, cultural, and other frames. The consilience thereby implied between fields of study such as evolution and learning does not require specialists in any particular field to integrate their understanding into the larger picture. However, the benefits of such integration might be far-reaching.

1.1. The importance of integrated understanding to the study of behavior

Natural scientists that study behavioral processes constitute a different community from those that study cellular and molecular processes, from those that study evolutionary processes, and from those that study developmental processes. These disparate fields each understand selection-based causation as foundational to their subject matter and acknowledge that in the continuum of the natural world, the principles established in each field must meaningfully interrelate. The existence of bridge-fields such as evolutionary development (evo-devo) implicitly acknowledge this consilience. Yet acknowledgement does not equal specific understanding of the bridging concepts (Bechtel and Hamilton, 2007) between one's own field and those of related fields or of the mechanistic diversity, recursive nature, and common patterns among selection systems. This paper is meant to enable such an understanding and to enable its heuristic use in the study of behavior.

Misunderstandings about cross-field relationships can lead to unnecessary scientific effort, for example to decide between "alternate"

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theories that do not actually compete but rather focus on different loci of a particular phenomenon. A wide understanding of such loci makes this mistake less likely. The outdated argument between nature and nurture serves as a simple example. The environmentally contextual nature of processes at the genetic, molecular, cellular, and developmental levels, together with the genetic, cellular, molecular, and developmental influences on environmental sensitivity demonstrate the folly of attempting to define a strict environmental/genetic dichotomy. In another simple example, the fact that heredity can be achieved via non-genetic or multiply-caused mechanisms serves as a warning against the assignment of "genetic", base-pair sequence explanations for all traits that show consistent heritability along some environmental gradient. Following the presentation of details about different levels of selection, this paper will return to particular cross-field concepts related to the study of behavior that may be reimagined with reference to this material.

Selection as a complex process produces variant dynamics and outcomes in different frames and even in varying conditions of the same frame. Thus, though the reader is encouraged to explore parallels between systems of selection, s/he should also avoid the assumption that parallels of detail will emerge. In fact, the aspects of systems of selection that differ from one another may be as illuminating as those aspects that are shared.

Due to the breadth and complexity of the topics covered, this paper should not serve as a thorough review of any of its constituent subtopics, and the reader is encouraged to make use of deeper reviews if seeking full understanding.

1.2. A framework for evolutionary frameworks

Discussing selection-based causation over multiple levels of analysis depends on having a well-defined conceptual framework that can be applied generally. One such framework was provided by Hull (1980), who suggested the terms "replicator" and "interactor" as discipline-neutral terms for the players in selection-based evolution. Hull defines a replicator as "an entity that passes on its structure directly". He defines an interactor as a unit that "directly interacts as a cohesive whole with its environment in such a way that replication is differential". The same entity could be both interactor and replicator, but interactors could also be separate entities that form in a manner at least partially informed by replicators. Hull identified selection as "a process in which the differential extinction and proliferation of interactors cause the differential perpetuation of the replicators that produced them", which process is a function of environmental features confronting interactors: selection contingencies. The result of this selection process is a lineage that "changes indefinitely through time either in the same or an altered state as a result of replication" – that is, lineages are what evolve via iterative selection. As Hull points out, replicators and interactors function *in* selection processes, and lineages are the result *of* selection.

Variation is also required for perpetuation to be meaningfully differential, and some mechanism must continually maintain not only copy fidelity in replicators and heritability in interactors but also low-level copy error or variation. This can happen with greater or lesser fidelity for different interactors and replicators, as I will discuss. Thus, the minimal pattern of a system of selection is variation, interaction, and differential replication that results in change or maintenance over time of lineages. This paradigm not only identifies important roles that might be filled by different variables in a selectionist framework, but also serves as a proposal for the minimal structures required for selection-based adaptation. It also enables the analogous comparison between the general processes that work across different selection frames rather than merely metaphorical comparison between those unique physical forms that carry out these functions in particular frames.

The following sections will introduce the reader to various entities that can serve as interactors in different selection frameworks, then to various entities that can serve as replicators, then to the interrelatedness of various interactors, replicators and selection contingencies. Subsequent sections will demonstrate how, within a single evolutionary lineage, the replication and interaction function can jump between entities, levels of analysis, and even selection frames. The final section will elaborate about the implications of this general understanding for basic and applied behavioral science.

2. Interactors

Analytical units similar to the interactor existed before and after Hull's idea; even Darwin chose the organism (a classic interactor) as the unit for his theories despite the fact that his postulated "gemmule" could also be construed as an early conceptualization of replication (Darwin, 1859, 1868). Hull's interactor, though, is useful in delineating evolutionary processes in a general framework, particularly in comparison with similar and popular but less precisely defined concepts such as reproducer (Szathmáry and Smith, 1997) and vehicle (Dawkins, 1982; Hull, 1988). Hull emphasizes the role of the interactor in the evolutionary process as separate in kind from that of the replicator rather than attempting to reduce the former to the latter as other prominent biologists have done. Dawkins for example framed interactors (which he calls vehicles) as a mere extension of replicators with comparatively little evolutionary importance (Dawkins 2016, 1986). Though replicators hold an undeniably critical role in the evolutionary process, evolution results from mutual influence between the entities rather than from the linear influence of one upon the other (Gould and Lloyd, 1999), as will be explained in detail below.

2.1. Organisms as interactors

In Hull's conceptualization, the organism is the prototypical interactor. The organism interacts with the environment as a cohesive whole to reproduce with greater or lesser success, which causes replication or non-replication of all replicators grouped within each organism and not just those that contributed directly to the fitness of the organism. For example, if an organism reproduces, all genes in the genome of that organism will generally be selected with equal probability; even in sexual reproduction the genes will be shuffled randomly. Thus, a gene that may have a neutral or negative influence on fitness will have an equal chance at replication compared to genes that conferred the requisite fitness that enabled reproduction in the first place. If organisms act as interactors in this way, it predicts that fitness-unrelated genes can be amplified via within-organism association with fitness-related genes. In fact, such evolution does occur. Over time and even as multiple organism-interactors shuffle their genes via genetic recombination and crossover, genetic "hitchhiking" of nonadaptive genes occurs, sometimes simply because the genes are located on the same chromosome as an adaptive gene or in close enough proximity that crossover is unlikely to separate them. The increase of genes in a population via association with other genes is called *genetic draft* (not to be confused with *genetic drift*) and demonstrates the necessity of interaction to fully explain both differential replication and the evolution of lineages (Neher, 2013). A gene is not selected only on the basis of its own function; it is sometimes selected by virtue of its place in the interacting organism.

Even genes that are selected via a direct contribution to fitness do so in a manner contextually dependent on other genes in that organism. For example, the adaptive contributions of genes coding for multiple proteins in the same cellular signaling pathway are interdependent. Selection of a gene will depend on not only its own contribution to fitness but on consistent within-organism grouping with other genes that make the contribution possible. The organism is thus not just the vehicle that the genes ride in; it is an internally interdependent unit in the evolutionary process.

2.2. Grouped-organisms interactors

Groups of organisms as interactors were postulated early on as a potential evolutionary unit (Darwin, 1888). Of course, the term "group" does not specify much except that more than one organism can be identified. "Group interactor" provides much better specificity; to qualify in this category, two or more organisms must function as a unit with heritable traits to interact with the environment in a manner that a single constituent organism would not, causing differential propagation of replicators.

Two approaches to group selection can be seen in *Multilevel Selection Theory*. In one approach, the interaction between the organisms of a group serves to select for aggregate trait frequencies among populations of organisms (called *MLS1 selection*). For example, one herd of elk may have an unusually high frequency of vigilance against predators and high social responsiveness to vigilant members. Members of this group (even those that are not vigilant or are less responsive) may have greater reproductive fitness compared to members of another group because of the group composition as well as the individual organisms' traits. This can result in more groups with high vigilance and responsiveness frequencies. In MLS1 group selection, adaptations can only manifest on the level of the organism: e.g. animals evolve to be both vigilant and socially responsive in part because of the group-level interaction of these traits (Okasha, 2005). In a second form of group selection, the interaction of a group propagates aspects of the group structure beyond mere frequencies (called *MLS2 selection*). MLS2 selection is measured by increase of frequency in group type rather than aggregate individual types (Okasha, 2005). This kind of selection opens the possibility for different specializations between members to produce internal organization and "harmony and coordination of … parts" (Gould and Lloyd, 1999). Thus, group structure as well as organism *phenotype* frequencies (observable organismal traits) can be reproduced (Gardner and Grafen, 2009). In both types of group selection, interaction occurs on the group level, but only in MLS2 selection can group-level adaptations appear. Groups such as bee hives, ant colonies, and family groups among birds show such specialization, with individual organisms taking on differential roles (Wilson, 1971; Gaston, 1978).

Opponents argue that group interactions can reduce to organismal interactions, and indeed the two levels of interaction are often redundant. Conspecifics (other organisms of the same species) are part of the environment of organisms, and adapting to the context of a group structure may confer a pattern of organismal fitness from which group patterns emerge. However, the total organismal fitness of a member of such a group as the elk in the example above remains critically dependent on the heritable reproduction of the group pattern, similarly to the contextual nature of a single gene that requires heritable cellular structure to function adaptively. This point becomes particularly salient when considering conditions where, unlike the vigilant herd example, organismal and group interaction have dissimilar effects. When group fitness comes at some cost to organism-level fitness of a subset of group members, between-group selection may come to oppose within-group selection. Biologists thought for some time that organism selection under such conditions must overwhelm group selection, but currently it is acknowledged by a growing contingent of scientists that group and individual contingences might reach an equilibrium (Wilson, 2015). Since organisms and groups have mutual influence on one another, and since organisms may have a greater or lesser ability to reproduce outside the context of the group, the causal contributions of individual vs. group interactors to selection can be complex and difficult to parse. Quantitative treatments using the two paradigms produce similar predictive validity (Okasha, 2016; Krupp, 2016). Group selection likely influences the evolution of behavior. Behaviorally important traits such as the evolved tendency toward conformity, even when it does not lead to individually adaptive patterns, may have developed via group contingencies.

Group interaction has often been dealt with in a manner that confuses interaction with replication. Group interaction does not necessarily require a particular pattern of replicator content among the members of the group; groups may or may not share genomes for example. However, any interactor including a group must show heredity of selected traits, and the most obvious mechanism for such heredity is shared genes or shared gene flow. Groups with shared genes like this are called supraorganisms (a.k.a. superorganisms). These can be groups that share a single genome and that work together for the reproduction of a single, common *germ line* (transgenerational reproductive cell lineage) such as ants and bees. They can be related groups that reproduce only with one another such as families, closely related subpopulations called *demes* or, in special cases, species (for more details about species as interactors see Lloyd and Gould (1993) and Goldberg et al. (2010)). The study of replicator content in groups has shed light particularly on the scenario mentioned above in which group interaction works against the organismal interaction of a subset of constituent organisms. Efforts to account for such phenomena led to theories such as kin selection, wherein individually maladaptive helpful behavior towards relatives may be directly selected by virtue of similar genomes (Frank, 2013). Quantitate treatment of social scenarios has also been well established in inclusive fitness and direct fitness theories (Queller, 2016). These theories may seem to oppose the idea of group selection by moving the locus of selection back to the gene or to the individual (and they are conceptualized as alternate rather than complimentary by many in the field (Wilson, 2012)), but in fact kin selection and inclusive fitness simply identify the locus of replication (shared genes) and analyze a lower level of interaction (interacting individuals rather than whole groups), respectively, rather than refuting the interactive role of the group itself. If group conditions did not heritably recur, the organismal interactor would not retain the same selection contingencies according to either of these paradigms. Although the controversy has not been settled, kin selection and inclusive fitness theories may be compatible with group selection paradigms both intuitively and mathematically (Wilson and Wilson, 2007; Eldakar and Wilson, 2011; Lehmann et al., 2007). Further, genetic relatedness isn't sufficient to establish group selection since interrelated animals may not form an interactor (Damuth, 1985), and although genetic relatedness may underlie group heredity the two remain separable (Goodnight, 2005). The problem with finding genetic replicators for all instances of group interaction may be related to the fact that shared genes do not constitute the only possible mechanism of group heredity. As I will highlight later, much recent work has established the potential for replication on levels other than the genetic sequence, and this may help account for apparent group-level (e.g. cultural) traits emerging from what appears to be insufficient shared replicator composition.

2.3. Other grouped interactors

Similarly to organisms, groups of cells can function as interactors. In many multicellular life forms, millions of cells interact with the environment as a single unit to cause differential propagation of only a few germ cells that derive a chance at multigenerational reproduction. Yet life on earth first evolved not as multicellular lineages but as single-celled lineages. Cooperation, then, occurred between cells before it was possible between multicellular animals. At some point, cells began forming colonies, working together, then taking on differential tasks within the conglomerate. The locus of interaction for one particular lineage moved entirely from the cell to the cell group. This evolution proceeded long ago, and today most cooperating cells are tightly integrated multicellular organisms that share genomes, akin to the supraorganisms of ant and bee colonies. Yet some single-celled life forms such as bacterial colonies also cooperate with one another, and even the cells of some multicellular forms of life, such as sponges, slime molds, and many plants retain functional flexibility and/or potential reproductive autonomy of *somatic* (within-organism) cell lines (Wilson and Sober, 1989; Buscema et al., 1980; Ganguly, 1960; Wang, 2015). In

these species, interaction can alternate between the cell level and cell group level as coordination emerges and fades. Sponges and slime molds constitute evolutionarily ancient examples that may be holdovers from the time on earth when multicellularity in animals was just evolving, before shared genomes and irreversible specialization developed and dominated in the realm of animal cell groups. Such interactors are susceptible to the same replicator-relatedness dynamics as are groups of multicellular animals (DeAngelo et al., 1990; Hudson et al., 2002).

Nor was multicellularity the first instance in which the locus of interaction moved toward a group of previously individual interactors. The field of abiogenesis (the study of the pre-evolutionary origins of life) currently holds that the first forms of life on the planet were self-replicating *RNA* (ribonucleic acid) molecules, potentially unbound by membranes (Joyce, 2002). Like *DNA* (deoxyribonucleic acid), RNA is composed of sequences of nucleotides that can replicate using themselves as a templates, however unlike DNA, RNA can form three-dimensional complexes with protein-like functionality (Gilbert, 1986). Thus, in the beginning the RNA molecule likely served as both replicator and interactor. At some point, groups of these molecules began to assemble into clusters isolated by a lipid membrane, and it became the cluster rather than the molecule that survived as a unit to reproduce. The replicating molecules could work together to form different interactive molecules that could take on differential roles. The membrane barrier compartmentalizing the activity of groups of molecules eventually defined the cell (Koonin and Martin, 2005). In other words, the locus of interaction switched from the individual molecule to a complex system of molecules grouped together into the cell in what might have been the first instance of group selection.

When a new level of interaction forms, it doesn't necessarily mean that the lower levels are entirely subsumed. Organisms within a group, for example, can still function as interactors so long as they reproduce. Further, even the molecule level can sometimes still interact in multicellular lineages because of the probabilistic disconnection of genes from one another that occurs in sexual recombination. This partial autonomy can encourage *intergenomic evolution*, in which genes located in separable regions of the genome coevolve due to their molecule–-molecule interactions (Rice and Holland, 1997; Blier et al., 2001). Still, interactors have become to a greater or lesser extent embedded in one another, as is illustrated in Fig. 1, which visualizes the recursive structure of selection on the molecular, cellular, organismal and group levels. In this paradigm, selection may occur along a component lineage within an interactor, forming greater interactor complexity as further structures are embedded (moving downward in the figure). In some multicellular organisms and some supraorganisms, this embedding is complete and the germ line isolated, which allows contingencies of selection between cells or organisms, respectively, to function only in the temporal course of the higher-level interactor, with iterative repetition across new generations of that interactor. Once this or any other mechanism of within-lifetime selection (*ontogenetic* selection) arises, its dynamics can undergo *phylogenetic* (across-generation) selection, either as a developmental sequence given heritably consistent ontogenetic selection contingencies, or as an evolved adaptability given variable ontogenetic selection contingencies.

The next section will introduce some ontogenetic selection paradigms in multicellular animals, many of which involve more complex interactors than single cells or molecules but which all govern the development of phenotypes. The reader is urged to remember the patterns highlighted through this summary of phylogenetic interaction and to note where those patterns repeat in ontogenetic selection processes and, particularly, in behavioral selection processes.

2.4. Ontogenetic interactors: selection within a lifetime

Ontogenetic selection processes evolve via phylogenetic selection. The physical replicative machinery and initial somatic interactors required for ontogenetic selection processes (such as the neuronal networks that may mechanistically support behavioral selection) have been developed via successive generations of phylogenetic selection. I will first discuss examples of ontogenetic selection in which ontogenetic contingencies are so invariant that they generally produce identical or near-identical results from animal to animal unless important variables are perturbed.

2.4.1. Selection during development

As animals develop from fertilized ovum into multicellular adult, many selection processes are vital for the emergence of physiology and anatomy. Selection-based cellular elimination via programmed cell death, for example, supports normal anatomical development (Sancho et al., 2013). For brevity, I will illustrate with only one detailed example: the development of the mammalian neuromuscular junction (NMJ), which is the connection between motor neurons and muscle fibers. For other examples, the reader is referred to the rich literature of developmental biology (e.g. Gilbert (2000)).

Early in mammalian development, fast-twitch muscle fibers are innervated by multiple nerve terminals (interactors), however only one nerve terminal will eventually remain connected to each fiber. The multiple-input stage is followed by a period of selective competition wherein the most active input that best induces depolarization of the muscle fiber will remain, while adjacent connections retract. The causes of retraction involve more than just the efficacy of the single connection; even relatively ineffective connections will remain unless a more successful input is nearby (Sanes and Lichtman, 1999). If the fiber loses this input later in life, axons will again grow out beyond the original synaptic site and re-establish a competitive selection scenario until the fiber is once again innervated by a single terminal (Darabid et al., 2014).

This kind of developmental selection generally occurs at restricted times of life and may cease at a particular equilibrium (for NMJ development, one single stable connection per fiber). Variation is produced briefly and selection proceeds through a few iterations only. However, other forms of ontogenetic selection may be temporally extended and more constantly iterative, providing the opportunity for ongoing adaptation.

2.4.2. Immunological selection

One such system of ongoing selection underlies the ability to defend against foreign pathogens including bacteria, viruses and parasites. Mammals are born with an innate immunity that is not sufficient to defend against the variety of pathogens that will be encountered. The adaptive immune system can recognize novel or recently evolved pathogens and build a defense against them. This ontogenetic adaptation works via targeted variation and somatic selection. One such mechanism involves *B cells* as interactors, which produce antibodies whose genes undergo random recombination. New B cells with recombined antibody genes are continually created during a lifetime, resulting in a steady stream of variant cells. These cells first undergo *negative selection*, or elimination of certain variants. The cells are exposed to the proteins that the body itself makes, and those that bind to these native factors are programmed for apoptosis or cell death. Those B cells that survive this negative selection process endure until such a time as a match for their antibody − some invader it can bind to − becomes attached and is endocytosed. The cell then takes the antibody together with the piece of the invader and presents it upon its cell surface in conjunction with a factor that can bind to a T cell receptor. Once that T cell receptor binds and recognizes that this B cell has "matched" to an invader, it induces the B cell to propagate, resulting in somatic *positive selection* (differential amplification of a variant) and further antibody refinement of activated B cells. These new daughter cells become primary fighters against the current infection and also propagate a memory cell line which survives after infection, ready to activate again (this time even faster) should a subsequent re-infection occur. Somatic

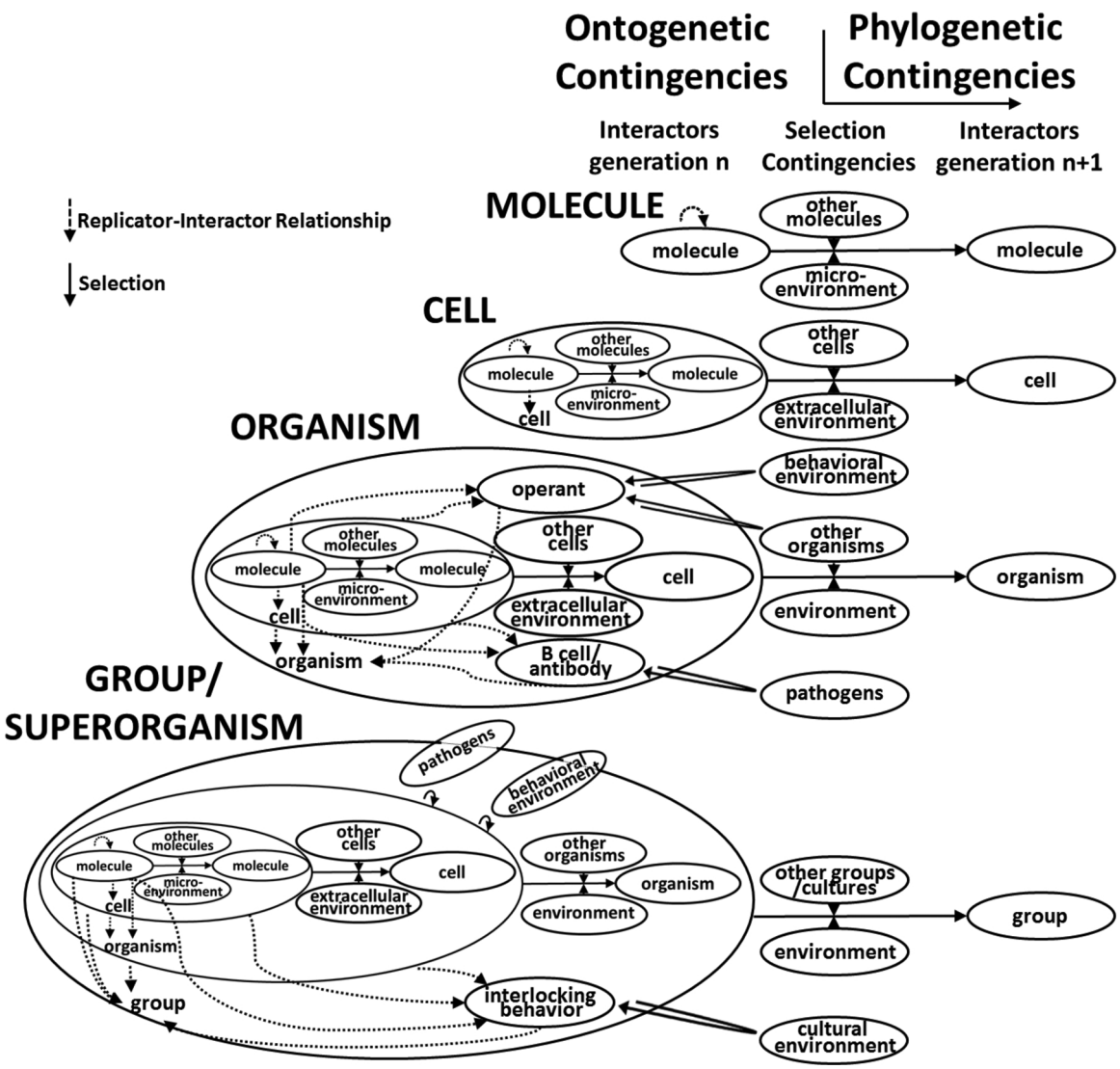

Fig. 1. A simplified schematic showing interactions between replicators, interactors and selection contingencies in lineages with primarily molecule, cell, organism, or group interaction. Dashed arrows indicate replicator-interactor relationships and solid arrows indicate selection contingencies. The right side of the figure constitutes phylogenetic contingencies, the left side ontogenetic contingencies. Note that previously phylogenetic units can become ontogenetic as they become nested within interactors. This schematic conveys several exemplar or potential scenarios and is not meant to be comprehensive.

selection has been well studied for many years, and this description only touches the surface of the topic. For greater detail, the reader is referred to the vast literature on adaptive immunity (e.g. Flajnik and Kasahara (2010)).

2.4.3. Behavioral and cultural selection

During their lifetimes, humans and other animals achieve varied and complex behavior adapted to changing conditions through another, complex ontogenetic selection process: behavioral selection. This process allows behavior as an interactor to be selected by the environmental consequences of that behavior (Skinner, 1984). When environmental consequences function to increase the frequency of a behavior, the positive selection process is called reinforcement. When they function to decrease the frequency of a behavior, the negative selection process is called punishment. For example, food typically reinforces behavior that produces it if the behaving animal has not recently eaten. Food selects or increases in frequency that subset of early, undifferentiated behavior that results in its ingestion (such as suckling on a nipple or grasping food and bringing it to the mouth). Stimuli that come to reliably predict opportunities to perform such behavior can also themselves become reinforcers, which can then shape problem-solving behavior that occurs long before final food acquisition such as walking toward fruit-bearing plants, stalking prey, etc. In this way, the animal learns to forage or hunt, manipulate food, etc. As food-producing behavior varies, selection for speed or efficacy or adaptation to minor changes in environmental conditions can occur. Many varied stimuli can act as reinforcers and punishers, opening the opportunity for the evolution of highly sophisticated behavior throughout a lifetime. The implications of behavioral selection extend even beyond motor behavior; the variation-and-selective-retention model has long been applied also to cognitive behavior and creative thought (Barrett, 2011; Simonton, 2010).

Behavior can also evolve on a higher level of interaction than individual behavior. When two or more organisms provide antecedent and consequent stimuli for one another they produce interlocking behavior (an interactor), and this behavioral cooperation can undergo selection. If two organisms work in a coordinated fashion, for example to gain food that they can both eat and that neither could obtain on their own, such interactions may themselves be selected in a behavioral version of group selection. The contingency in which coordinated behavior is selected as a unit by the environment is called a metacontingency (Glenn, 1988). As with complex behavior in behavioral

selection, the phenomena produced by metcontingencies can become increasingly complex. Such cultural selection may even constitute a developmental mechanism for phylogenetic group selection by producing interlocking, cooperative behavior that enables division of labor. Importantly, cultural evolution may proceed from replicators embedded in imitation, conformity, or tradition/teaching, which may have vastly different patterns of transmission and copy fidelity than genes. This point renders the study of cultural evolution into both a fascinating parallel and a nuanced divergence from the frame of biological groups (Campbell, 1965).

The emergence of cultural selection from the basic processes of behavioral selection could have proceeded in a manner similar to the emergence of group selection from organism selection. In other words, behavioral selection may have become embedded in cultural selection in a manner analogous to the embedding portrayed in Fig. 1, producing interdependence; embedded behavioral selection remains necessary for cultural forms to occur, and the selection of behavior depends on the context of the evolved culture.

Again, these cursory descriptions only touch the surface of a complex and dynamic field. As in previous sections, the reader is referred to a rich literature for further details (e.g. Skinner (1953), Glenn et al. (1992), Glenn (1991), Madden (2013)).

2.4.4. Neural selection

Within-lifetime selection processes also underlie the ability of the nervous system to organize itself and reorganize itself as a function of environments encountered, behavioral patterns developed, and even in response to injury. Selection on the neural level involves processes such as Hebbian plasticity. In Hebbian plasticity, neural connections are strengthened when neurons fire together – the presynaptic cell fires immediately before the postsynaptic cell – and weakened when they fire out of coordination (Markram et al., 2012; Song et al., 2000). Thus, connections in the brain that fire together functionally will be selected over those that don't. This could create a selection-based functional network. Feedback from the environment can add a layer of emphasis to this basic process by adding inputs that gate or differentially modulate synaptic changes based on reinforcing, discrepant, punishing and error-indicating stimuli (Pawlak et al., 2010; Pennartz, 1995). However, since it focuses on single, pre-existing synapses, this simple Hebbian model critically oversimplifies the process of neural selection. Neural selection deals not simply with a summation of synapses but rather with organized networks of variant cell types within variant brain structures forming reentrant and complex pathways that can be changed in structural and functional ways in response to selection contingencies. The network or cell group rather than the single synapse may be the focus of selection in such complex space. A great deal of brain structure and function develops via selection, and neural activity is partly induced and regulated by environmental stimulation, behavior and feedback.

Gerald Edelman (1987) presents a detailed theory of neural selection in his work "Neural Darwinism". According to Edelman, early in development, the brain is wired through selection processes involving guidance cues, cell surface interactions, migration, and neural apoptosis, resulting in a primary pattern of connectivity. Then, through lifelong iterative encounters with the environment, neuronal groups that underlie successful function are selected in a separate selection process. The diversity of connectivity patterns and reentrant pathways produce a neural *degeneracy* (where multiple, dissimilar components or pathways can perform similar or identical functions); different cell networks as interactors may be selected by the same contingency, producing a robustly adaptive brain that does not rely only on strict, linear wiring. Edelman's work is controversial in his field, and arguments exist as to the veracity of the concept of neural selection as well as the specifics of selection processes in the brain, including the existence and characterization of neural replicators. Edelman's theory, however, is not the only selectionist account of nervous system dynamics. Other work also indicates that selection processes may account for much of the adaptable wiring and rewiring of the brain during the lifetime, (e.g. Fernando et al., 2012; Kilgard 2012).

Though the processes of behavioral selection parallel (and may be mechanistically dependent on) those of neural selection, they are not identical. In behavioral selection, the selected interactor is the holistic response selected by environmental consequences; in late-stage neural selection, the interactors are likely neuronal groups and patterns of structural and functional connectivity that may be selected via environmental consequence or via other feedback loops. Although neural selection may be nested within and necessary for behavioral selection, the two processes as currently studied remain nonidentical.

2.4.5. The evolution of enveloped contingencies

Ontogenetic systems of selection such as those described above are integrated into phylogenetic evolution as depicted in Fig. 1. As interactors become more complex (lower in the figure), more and more ontogenetic selection frames and complex interactors emerge, enabling behavioral, immunological, and cultural selection. This fractal set of nested selection frames, termed *vicarious* selection by Campbell (1965), confers adaptability in fast-changing environments if at least one nested system of selection can respond along the temporal scale of environmental change. It also confers the ability to vary in response to a loss of environmental stability since ontogenetic selection may produce little variation until the environment changes.

The rise of some of these ontogenetic selection systems, such as the adaptive immune system, were enabled by the embedding of previous interactor entities into a larger interactor as shown in Fig. 1; for example, multicellularity introduced the opportunity for the evolution of a defense system based on somatic selection. The question of what conditions are necessary for the emergence of a new level of interaction remains speculative. One possibility involves a processes similar to that which supported the emergence of the first evolving molecules: self-organization coupled with self-replication. To expand on this point, let us consider the non-evolving domain. The exigencies of the physical world often impose "selection" contingencies on substrates that cannot replicate, which can result in order but not in evolution. Examples include crystal formation, wherein the internal selection contingencies imposed by the chemical regularities of interacting atoms produce ordered structures that do not evolve. Another frequently cited example is the orbits of planets around a star, which include only the limited set of initial particle trajectories that can produce a stable orbit. These kinds of "selection" differ fundamentally from those about which I've been speaking because they all lack a fundamental term: the subtly varying replicator. When principles of atomic interaction produced the first nucleotide sequences, the self-replication and mutation capacity of the resulting molecules produced a dramatically different potential than crystal formation had.

Interactors may sometimes become similarly organizationally integrated with one another. Cultural embedding provides an example. As pointed out elegantly by Campbell (1965), group and social behavioral organization may result from "internal" selection contingencies similar to those that order crystals: individual behavioral interactions and the principles that organize them. Of any group patterns thus formed, those that demonstrate self-propagation or heritability would then be available for external, cultural selection. Similarly, self-propagating patterns of multi-molecular, multicellular, or multi-organismal structures may have occasioned the advent of a new level of interaction, and the coalescence of the grouped unit itself may have been strengthened due to such selection.

2.5. Summary

Interaction can occur on multiple levels of analysis, from molecule to cell to organism to group, as illustrated in Fig. 1. Interactors on different levels can simultaneously function; for example organismal

interaction and group interaction may simultaneously govern the evolution of an elk lineage. Interactors such as the ant colony supraorganism may also completely envelop lower-level units such as the single ant organism. Interactors must have heritable traits, but that heritability doesn't automatically imply a particular replicator composition such as a shared genome. Selection can also occur within the lifetime of an interactor, but ontogenetic and phylogenetic interaction are not autonomous phenomena. The organismic machinery necessary for ontogenetic selection processes to occur − such as neurons or B cells − themselves evolved via phylogenetic selection and allow for vicarious, nested selection processes. Phylogenetic interactors are often dependent on ontogenetic selection for their developed form; neural anatomy, immunological responses, and behavioral repertoire are traits of phylogenetic interactors (organism) as well as ontogenetic lineages. Similarly, cultural interactors are dependent on behavioral lineages. These relationships pose interesting questions about replication since the heritability of interactor traits such as individual behavior or group coordination can rely on more than genetic sequence alone. I will next move to a discussion of replication in order to fully expand on this idea.

3. Replicators

DNA sequence remains the most obvious, well-characterized example of an evolutionary replicator, and it is the only one that remains uncontroversial today. However, the speculations of decades past that mere base pair sequencing could account for the heritability of an awesome array of complex traits seen in the living world has been largely discarded, and other, "*epigenetic*" replicators have been discovered. The term "epigenetics" has taken on a highly varied set of definitions. For the purpose of this paper that term will refer to any replicator that is not a base pair sequence of RNA or DNA but that is still transmitted via a molecular or subcellular process. Replicators may also exist at a higher level, but I will not refer to those potential forms of replication as epigenetics.

3.1. Epigenetics: ontogenetic and phylogenetic inheritance

In most multicellular animals, non-bacterial constituent cells share a single genome, yet they are morphologically and physiologically extremely variant on a scale comparable to between-species variation in single-celled organisms (Jablonka and Lamb, 2014). This cell-type morphology is heritable within cellular lineages and will be passed to somatic daughter cells via epigenetic rather than genetic replicators. Liver cells produce more liver cells, muscle cells produce more muscle cells, etc. Although these epigenetic replicators account for somatic heritability, they cannot themselves participate as replicators in the evolution of the species unless propagated through the germ line (reproductive cells) of the organism. Though it was long assumed otherwise, scientists now know that the mechanisms of somatic inheritance are not entirely restricted to somatic cells. Epigenetic signals can affect inheritance across the germ line, which means they may participate in the evolutionary process across lifetimes and not only within them. I will outline the four categories of epigenetic inheritance as reviewed by Jablonka and Lamb (2014) in order to provide examples.

3.1.1. Self-sustaining loops

When a cell divides, not only the genome but the content and structure of the cytosol is inherited by the daughter cells. If that cytosol contained the requisite signaling proteins for silencing or expressing genes, and particularly if those signaling proteins participate in positive-feedback loops where their presence leads to their continued maintenance, then a pattern of induced gene expression can be passed on to daughter cells. Inheritance via the cytosol can encompass more than what Jablonka and Lamb (2014) summarized. For example, the internal structuring of cytosolic factors in the fertilized ovum together with uneven cytosolic inheritance among daughter cells (each daughter cell inherits an asymmetric segment of the cytosol) has long been understood as a primary determinant in body axis development of *Drosophila* (Gilbert, 2000). It is now understood that similar mechanisms can also act as a substrate for intergenerational heritability, explaining the nonmendellian inheritance patterns of some traits such as the mating types, toxin production, and other traits of Paramecium (Preer, 2006).

3.1.2. Structural inheritance

Structural inheritance can encompass a wide array of phenomena in which some physical structure other than the gene in the parent cell serves as a template from which the development of the same structure in the daughter cell is derived. This form of inheritance can be seen in the ciliated cortex of paramecia; when the patterning is disrupted mechanically by inappropriately positioned grafts, the disruption is heritable (Beisson and Sonneborn, 1965). It also encompasses the realm of self-propagating proteins or prions. Prions are able to change the conformation of other proteins to replicate their own three-dimensional structure without changing the protein sequence. Prions are most famous for their ability to cause pathologies such as mad cow disease and tauopathies (Clavaguera et al., 2013; Prusiner, 1995), but some prions are part of the cellular and epigenetic inheritance landscape, too (Yool and Edmunds, 1998).

3.1.3. Chromatin marking

A gene's function can be regulated by other DNA sequences such as the gene's promoter region, its enhancer regions, and other cis- or trans-regulatory regions. But in addition to this sequence-based landscape, non-sequence-based modifications to the DNA molecule itself and to packaging structures related to DNA (which together is called *chromatin*) can participate in controlling gene expression. For example, methyl groups can be attached to cysteine residues of DNA, and heavily methylated regions of DNA tend to be transcriptionally silenced. Specific factors can copy methylation patterns to new DNA strands during replication, making such patterns heritable along the somatic cell line and the germ line (Trerotola et al., 2015). Gene expression can also be controlled via modifications of protein groups that package DNA called histones. These positively charged factors form a core around which the negative DNA molecule is wound. Chains of histones pull DNA together even more closely via histone–histone interactions. In order for a gene along the DNA sequence to be expressed, it and its regulatory regions must be sufficiently released from this packaging so that room can be provided for the transcription process to proceed. If the histone–histone interactions are tight or if DNA is bound tightly around the histone, the sequence is less accessible. Modifications of these histone groups can control this access. Methylation of the histones makes them more hydrophobic, which pulls them together. Acetylation and phosphorylation gives them negative charge, which loosens their interaction with the negatively charged DNA molecule. Histone modifications are far more complex than described here; other potential modifications include ubiquitylation, sumoylation and more. These modifications form a histone code, and importantly that code can be transmitted to both daughter cells during cell division, although the mechanism for replication is not yet well understood (Trerotola et al., 2015).

3.1.4. RNA interference

RNA interference works both apart from and in coordination with the chromatin marking mechanisms. In RNA interference, strangely formed or double-stranded RNA is recognized by a protein called Dicer, which cuts it up into small interfering RNA (siRNA) pieces. These pieces interact with the messenger RNA that shares their sequence, inducing the destruction of that mRNA and halting the translation of the gene. Sometimes, siRNA also affects the original DNA code from which the RNA came and can cause it to be silenced via the chromatin marking mechanisms outlined above, resulting in an induced, heritable silencing

of the gene. This system likely evolved as a defense against transposons and viruses: infectious bits of DNA or RNA that sought to usurp the machinery of the cell for their own replication and that frequently form double-stranded transcripts. Once this defense evolved, it was available to influence heritable replication and to take on other, non-defensive functions in the system. RNA interference now works as a vital part of the genome; double-stranded RNA naturally transcribed from endogenous stretches of DNA can participate in the normal regulation of gene expression.

Interfering RNA also has a remarkable ability that chromatin markers do not; it can be transmitted between cells. Though the mechanism is not yet fully understood, interfering RNA is often transferred over long distances within an organism, potentially inducing heritable change even across the germ line (Trerotola et al., 2015). This likely began, again, as a transmission of defense against infection to other cells in the organism. On an evolutionary level it critically opens the possibility that induced epigenetic changes can have both widespread somantic and germ line impact.

3.1.5. Epigenetic evolution

Since they can and do affect the germ line, do these epigenetic replicators in fact lead to heritable interactor traits that participate in evolutionary processes? That question requires more than restricted demonstrations of epigenetic inheritance and has not yet been answered. Still, it has been demonstrated that heritability of at least some phenotypes is epigenetic, and that induced traits can be fixed in a lineage via epigenetic inheritance. Examples of heritable epigenetic phenotypes range from plant morphology (Cubas et al., 1999) to mouse fur coloration (Morgan et al., 1999; Blewitt et al., 2006).

3.2. Cellular and supracellular replication

The genetic recombination that accompanies sexual reproduction places special importance and relative autonomy on the subcellular replicators. But consideration of the most common form of asexual life on the planet, bacteria, makes it difficult to draw the line of replication definitively at the sub-cellular level. When a bacteria cell divides, the result is two daughter cells with identical structure to the parent cell, including but not limited to genes. In this example, the cell itself can be conceptualized as the replicator of importance. Of course, the existence of cell replicators, just like epigenetic replicators, don't negate the importance of the gene replicator. In horizontal transfer between bacterial cells, the gene-replicator copies and transmits itself separately from the cell replicator in which it is normally nested. Thus, multiple coexisting replicators are available to participate in various evolutionary processes.

Replication may even occur on supra-cellular levels. I will summarize a few potential examples with the qualification that since the concept of supra-cellular replication is so new, developing, and controversial, these concepts should be taken as tentative.

3.2.1. Behavioral replication

Although behavioral selection as a process works within the lifetime of a single organism, rough patterns of behavior – even complex behavior – also repeat across generations of animals in an apparently heritable manner. What replicator may cause a fundamentally flexible process to produce heritable patterns across generations in this manner? A consistent environment may often shape consistent behavior, particularly where the reinforcement function (the ability of a particular stimulus to function as a reinforcer) is inherited via genetics or epigenetics. However, this heritable reinforcement function isn't always genetically determined. For example, transfer of materials from the maternal diet across the placenta, through the feces, or through nursing in mammals such as rabbits can determine the initial appetitive or aversive nature of food objects encountered in the environment (Bilkó et al., 1994). The inheritance of such food preferences is not determined by genes but by the diet of the mother, which was in turn induced by the grandmother's diet, etc. Thus, food preference shapes the feeding behavior that then establishes food preference and behavior in another animal. The behavior copies itself via the transmission of food particles, effectively taking the role of a replicator.

Behavior may also serve as a replicator copied via self-propagating environmental exposure. In other words, the behavior of a parent may guide offspring into a selecting environment that in turn shapes the same behavior in the offspring. For example, many species of birds have highly heritable patterns of migration. This pattern in whooping cranes is not transmitted genetically or epigenetically; proximity to natal group members reinforces chick actions that maintain it, and the resulting initial movement along the species-specific path shapes future migratory behavior in the youngsters (Mueller et al., 2013). Parental behavior thus induces the young to encounter an environment that in turn shapes behavior similar to their own. Similar effects can be seen in a variety of migrating animals (Scott et al., 2014).

In this example, the transmission of a behavioral replicator may require other crane-specific replicators such as those determining the reinforcers necessary to learn from the route once encountered, but such interdependence is not an atypical attribute of replicators. The hemoglobin gene, for example, cannot be replicated without a functioning DNA polymerase gene, which helps to form the complex that copies DNA.

In other examples, behavioral inheritance may be due to genes that directly influence both stimulus function and the environment. For example, the developing animal's genetically determined skeletal, muscular, and neural structure provide each species with unique behavioral contingencies such that particular sets of motions will result in pain, comfort, sensation, effective object manipulation, ingestion, etc. Behavioral contingencies determined by anatomical and physiological factors may even contribute to the development of cognitive behavior (Barrett, 2011). In mammals, anatomy can also determine a key learning environment for the next generation. The consistencies of the mammalian prenatal environment, determined by the mother's anatomy, can result in much of the stereotyped behavior of neonates. For example, shortly before birth, the amniotic bath changes in composition such that oral exposure becomes opioid-inducing. The baby develops suckling oral responses in this prenatal period potentially because of consequent, reinforcing access to these factors, and as a result becomes prepared to suck even without experience with milk as a consequence (Korthank and Robinson, 1998). The uterine wall also provides exponentially increasing resistance to limb flailing as the baby grows. At a particular point in development, it becomes extremely difficult to kick with multiple limbs but still relatively easy to kick with one. The baby learns to alternate limb motions, and thereby to emit alternating motions similar to walking upon birth (Brumley and Robinson, 2010). Thus the phylogenetic inheritance of behavioral structures, even when dependent on lower replicators, may involve an ontogenetic selection mechanism for the development of that behavior, just as the formation of heritable anatomical structure often involves selection-based developmental processes. Still, the key replicators necessary for the propagation of these particular behavioral structures are likely genetic rather than behavioral.

3.2.2. Microbiome replication

Many animals depend on bacteria residing in the gut to provide enzymes for digestion, the genes for which aren't included in their own genome. Each gut constitutes an ecosystem of various species living in various ratios in a complex web of interaction and competition (Human Microbiome Project Consortium, 2012). In humans, the varied content of this microbiome has been shown to have a vast array of effects on traits ranging from metabolism (Ley, 2010) to heart failure (Nagatomo and Tang, 2015) to immunity (Thaiss et al., 2016) and more. The microbiome is sensitive to environmental factors such as diet but is also largely heritable. In mammals, it is inherited through the maternal line,

partly through exposure to maternal flora during vaginal birth and breastfeeding (Bäckhed et al., 2015; Jost et al., 2014). A newborn's gut is virtually sterile and primed for colonization. The composition of earliest bacterial exposure will play a large part in determining lifelong microbiome phenotypes, which for females will in turn be passed on to offspring.

3.3. Summary

Replicators can be found on multiple levels of analysis within an interactor and can work together to produce interactors. Fig. 1 includes sample visualizations of this complex replication process from the simple scenario of a single self-replicating and interacting molecule to that of a supraorganism with many levels of replication contributing to each interactor. Although sometimes a single gene variant will cause a dramatic interactor variation, for example in sickle cell anemia (Rees et al., 2010), most phenotypic variation, even in largely genetically determined traits such as eye color, results from the joint contribution of multiple replicators (Liu et al., 2009). A particular gene may also have varied effects depending on other genes (Hoh and Ott, 2003). Non-genetic replicators add another layer of replicator complexity to this picture. Identical genomes can specify a variety of traits depending on epigenetic, developmental or even behavioral replicators (West-Eberhard, 1989). All of the replicators listed in this section can potentially serve as influences on behavior and may underlie the particular patterns of behavioral development seen in within or across lifetimes. Phylogenetic replicators may even emerge from ontogenetic processes, for example when behavior influences the development of its own structure in the next generation.

This section has not identified a replicator for every example of interaction outlined in part two because in some of those systems, such as in the behavioral selection system, a definitive replicator has not been identified (although neural replicators have been postulated). Selection, including natural selection, has often been initially described without commensurate identification of a replicator (Darwin, 1859).

With the multi-tiered nature of both interactor and replicator now established and summarized (Fig. 1), I will take up the mutual influence that these elements may have on one another and on the selecting environment.

4. Mutual influence between interactor and environment

4.1. Niche construction

Interactors evolve as a function of the environment, however sometimes the environment also changes as a function of interactors. *Niche construction* occurs when organisms affect the environment in a way that alters future selection contingencies for their own lineage or for other species. Beavers, for example, have evolved the tendency to dam rivers and thereby construct ponds or lakes that in turn serve as their selecting environment. This has led to the evolution of aquatic locomotion in beavers and has influenced the evolution of traits such as tolerance to thermal changes in other species (Skelly and Freidenburg, 2000). Niche construction may operate locally or broadly. The extreme oxygen production that occurred early in the history of life by newly evolving photosynthetic organisms, for example, broadly set the stage for the evolution of a new kind of life that could utilize oxygen (Laland and Boogert, 2010; Laland and Sterelny, 2006) and chased the remnants of oxygen-intolerant life to restricted environments. Niche construction may be seen as a system of "inheritance" since environmental changes can last long enough to affect the next generation. However, there are key differences between such "inheritance" and the heritable traits of interactors themselves. Niche construction may be an important process in the course of evolution, but constructed niches are not inherited in the same way as are the traits of an interactor. They constitute changes in the selecting environment, which is only indirectly related to the replicators or interactors undergoing selection.

4.2. Co-evolution, escalation and the red queen

Interactors can also serve as environment to other interactors. When two species affect the selection contingencies of one another, co-evolution results (Vermeij, 1994). Such relationships, for example those between flowering plants and bees, may be mutually beneficial. Nectar production induces bees to visit a flower and feed, thereby smearing pollen on their bodies. They then carry the pollen to another plant, enabling the sexual reproduction of the plant over distances. Plants and their specific insect and bird pollinators can co-evolve into greater and greater specificity. Some hummingbirds, for example, often have beaks that uniquely fit their co-evolved flower. The flower thus does not lose nectar to non-pollinators and the hummingbird has a monopoly on a food source (Altshuler and Clark, 2003). In this kind of mutualism, complimentary traits arise from co-evolution. Alternatively, co-evolution can induce an ever-escalating arms race when arising from parasitism or predation. A prey population may evolve better camouflage, predators may in turn evolve better eyesight, prey may in turn evolve better hiding strategies, etc.

4.3. Similar phenomena in behavioral and cultural selection

Concepts analogous to niche construction and co-evolution can be seen at other levels of analysis and may be general phenomena in selection-caused systems. For example, some behavior modifies the environment in such a way as to alter momentary behavioral contingencies, similarly to niche construction. In both paradigms, one interactor (response) alters the environment and thereby changes the probability of selection for itself and other interactors (the same response, different responses). This behavioral pattern has been studied well in self-control literature, e.g. Goldiamond (1965), and a complex set of such strategies was described in detail by B.F. Skinner in his recommendations for self-regulation in old age (Skinner and Vaughan, 1983). Examples can be found on the cultural level, too; for example, the behavioral interlocks in Amish communities result in the production of environmental infrastructure which in turn selects traditional individual behavior patterns. Amish communities construct old-fashioned buildings, roads, transportation and clothing and organize the community to expel objects that run on electricity or gasoline. These cultural interactors produce a behavioral environment that selects for hand-washing clothes, farming, caring for horses and chickens, and electricity-independent recreation.

The behavioral responses (interactors) of one organism can also constitute the selecting environment for those of another, resulting in behavioral co-evolution. This co-evolution may also either establish cooperative interlocks or an escalating arms race. Members of families, frequent competitors or close friends often develop audience-specific interlocking behavior such as inside jokes, gestures, strategies, etc. that co-evolved in a particular relationship via behavioral selection.

4.4. Summary

In the previous pages I have developed three main points. First, interaction and replication occur on many different interdependent levels, both within and across lifetimes. Second, interactors can proceed from multiple replicators. Third, the environment that selects interactors can in turn be shaped by interactors, and interactors of different lineages can provide selection contingencies for one another. I will now further elaborate on the shifting of interaction, replication, and environmental contingencies among physical entities in the course of a lineage.

5. The movement of the locus of interaction

As I've already stated, the locus of phylogenetic interaction for many individual lineages has changed during the course of evolution. In one of the earliest examples, interacting RNA evolved the capability to code for molecules made of amino acids rather than nucleotides: proteins (Gilbert, 1986). Protein could serve as a more versatile molecule than RNA, with differential folding and interaction capabilities providing a wider range of chemical function. Interaction continued to expand, proceeding from the molecular level to the cellular level, then to the multicellular level and potentially to the group level. Sometimes this movement involves overlapping active interactors (e.g. MLS1 group selection), sometimes a complete transfer of interaction to a new level (e.g. most multicellular animals), or the development of a new nested interactor (e.g. behavioral selection). Sometimes the order can reverse; slime molds retain their ability to move from cell-group to cell interactors and vice versa (Wang, 2015).

6. The movement of the selection contingency

The locus of environmental selection contingency can also change over the course of a lineage. This may happen when the physical environment shifts as with changing climate patterns, volcanic eruptions, niche construction etc. It can also change in a continual manner as it does in co-evolution and escalation. When selection contingencies undergo continual or self-reactive change, equilibrium may be impossible. The Red Queen Hypothesis states that this lack of equilibrium selects for mechanisms of constant variation such as sexual reproduction. In support of the hypothesis, the probability of extinction among species has been shown to be steady through time; longer evolutionary exposure to the environment doesn't seem to serve as a protective buffer against extinction as would be expected if the Red Queen Hypothesis were false (Van Valen, 1973). Similar co-evolutionary, shifting contingencies acting at the behavioral or cultural level might be expected to similarly preserve mechanisms of variation.

Changes in selection contingencies may also happen without any shift in the environment if a previously irrelevant or inaccessible feature of that environment comes into relevant contact with an interactor. For example, the scales of avian ancestors became increasingly branched and fluffy because of selection for increased thermal insulation. At some point, these scales (now feathers) reached a form that, for reasons that were coincidental and not directly selected, birds could use them to lift off the ground slightly while running. This exposed birds to a new selection contingency embedded in the unchanged environment: feathers providing better lift could now be selected for. This process, called exaptation (Gould and Vrba, 1982), has counterparts in other systems of selection. For example contingency adduction, in which a previously shaped response can be utilized in a new contingency, constitutes a behavioral analog to exaptation (Andronis et al., 1997). This term would apply to the sucking response first shaped by amniotic fluid access that later becomes shaped by milk. A narrower, special category of contingency adduction called the behavioral cusp is defined as "any behavior change that brings the organism's behavior into contact with new contingencies that have even more far-reaching consequences" (Rosales-Ruiz and Baer, 1997). In this scenario, the selection of a behavior (such as walking) brings the lineage into contact with new selection contingencies that then take over the evolution of many responses in the repertoire (such as door and cabinet opening, obstacle climbing, object carrying, etc.). In ethological (Bekoff and Allen, 1995) and sociocultural (Campbell, 1965) fields, similar transfer of function has been postulated as the mechanism for the evolution of specific rituals from behavior that previously functioned to fulfill different contingencies. For example, aggression rituals, which function to achieve a nonviolent social outcome, may have originally developed from the physical motions involved with attack, which functioned to injure the opponent.

7. The movement of the locus of replication

The replication function can also switch physical entities during the course of evolution; the transfer of replication from RNA to DNA provides the earliest known example. RNA is thought to have relinquished its replication function to DNA in most lineages because the latter serves as a more stable molecule and thus assures better replication fidelity (Reichard, 1993; Koonin and Martin, 2005). As evolution proceeded, the replication function became layered and diffuse through many other replicators. Thus, the locus of replication has also been mobile along extended lineages.

I've already summarized the potential for phylogenetic replicators to emerge from behavioral ontogenetic processes (e.g. behavioral replicators). Similar transfers can occur from other ontogenetic frames. For example, some adaptive immunological sequences selected within the lifetime of the animal may be transmitted to germ line cells via soma-to-germline feedback (Steele and Lloyd, 2015). As mentioned previously, induced epigenetic changes may also sometimes transfer to the germ line.

Replicator mobility seems to act differently from interactor mobility. In interactor mobility, new interactors may become layered and balanced with older ones (e.g. in simultaneous between-group and within-group selection) or may result in the complete transfer of an interaction role (e.g. from the cell to the organism or from the single fertile insect to the ant colony). In contrast, changes in the locus of replication seem to be additive in that they generally leave other replicators fully in place. Genes have not lost their replication function in the evolutionary process because of the evolution of epigenetics; instead layered replication simply provides for multiple sources of heritable interactor variation, and all layered replicators are selected at once.

7.1. Multiple replicators as a mechanism of balanced flexibility and stability

Both variation and high-fidelity replication are necessary for selection to occur. Yet one process comes at the expense of the other where only a single replicator determines a trait. Replication on multiple levels coupled with ontogenetic processes allows for both stability and flexibility in the same lineage. For example, epigenetic silencing of maladaptive genes can produce phenotypic change without permanent loss of the genetic code. Such silenced genes along with much of the noncoding genetic regions of the genome constitute hidden sources of potential variation (called *cryptic variation*) that can be induced to potentially heritable activity and influence in novel situations. Similarly, within-lifetime selection processes such as behavioral selection permit rapid adaptation to environmental perturbations without immediate loss of evolved capabilities in old and potentially recurring environments.

7.2. Switching replicators for the same trait

These replicator dynamics present an important potential: the same trait may evolve due to selection of different replicators at different points in the lineage. A trait selected ontogenetically may become selected phylogenetically, or an interaction that selects epigenes in the short term may select genes much later. This means, among other things, that phenotypic change can predate genetic change for the same trait.

Because of the complex causation I've been describing, phenotypes are often variable even given the same genotype. Across a particular range of environments, this variation is called a *reaction norm*. *Phenotypic adaptation* can occur in the space of the reaction norm without changes in genotype; operant selection could be considered a highly sophisticated example of phenotypic adaptation. However, when selection for particular phenotypes continues to occur in invariant ways, sometimes the reaction norm itself narrows onto that particular

phenotype. This can occur when selection of the lower-level replicator has "caught up" with phenotypic adaptation in a process called genetic assimilation. Thus, sometimes adaptation can actually precede the selection of genetic replicators that support that adaptation. This point has often been studied on a strictly biological level, but its implication for behavior is profound. If ontological processes can produce an adaptive behavioral structure that then becomes successively more broadly heritable between environments, it implies that the influence between evolution and learning may be bidirectional. The impact of behavioral and cultural changes on the evolution of a species has previously been considered in terms of niche construction and coevolution (e.g. Richerson et al., 2010). However, the process of genetic assimilation goes farther; the behavior need not alter the physical or social selecting environment in order to have some influence on the direction of evolution. Ontological process need only drive phenotypic exploration of the behavioral solution space, and when adaptations are encountered they may become fixed in the organismal lineage.

7.3. Examples of genetic assimilation of phenotypic adaptation

One of the early examples of genetic assimilation was discovered in fruit flies, whose normal wing structure could be perturbed by exposing developing larvae to heat. Heated animals sometimes produced a new "cross-veinless" wing structure. This trait never occurred in the unheated population. However, when researchers artificially selected among heated animals for the cross-veinless phenotype for a mere 23 generations, they discovered more changes than they expected. Flies started to develop cross-veinless wings without the heat. In these lineages, the reaction norm had narrowed; the cross-veinless phenotype had become genetically assimilated and would arise regardless of environmental context (Waddington, 1953).

Examples of genetic assimilation are most obvious in artificial selection contexts (Waddington, 1956; Moray and Connolly, 1963), however evidence has also been found in naturally evolving species (Adams and Huntingford, 2004; Aubret and Shine, 2009; Marks, 1989). These examples include assimilation of anatomy, physiology and behavior that first adapted phenotypically, then became fixed.

In genetic assimilation, the reaction norm narrows; however evolutionary processes can either narrow or widen reaction norms. *Genetic accommodation* refers to any such change in the reaction norm. An unstable environment should theoretically select for maintenance of a wide reaction norm whereas a highly consistent environment should result in assimilation. However, evidence indicates that this phenomenon is relative to the lineage in question. If a stable environment is newly encountered, it constitutes a variation for a colonizing species and may increase reaction norms in the short term. The body plan of pumpkinseed sunfish and the jaw size of the tiger snake, for example, both show developmental plasticity in response to rearing diet. That diet-induced influence (reaction norm) is larger in populations that were introduced recently in their evolutionary history to an environment with relatively stable dietary resources (Parsons and Robinson, 2006; Aubret et al., 2004; Crispo, 2007). In a laboratory example, selecting for survival in unusually high-salinity environments causes the saline-regulatory anal papillae of artificially selected fly lines to increase their developmental reaction norm (Waddington, 1959; Crispo, 2007). Thus, genetic assimilation may be preceded by a widening of the reaction norm given a new environmental challenge. One is struck by the similarity of this two-phase process to extinction-induced or novelty-related variation in behavioral processes, which are often followed by reinforcement-shaped selection of particular new variants.

Further examples of genetic accommodation and assimilation have been both demonstrated and suggested, though biologists still debate the issue. Many reviews provide rich perspectives on the current evidence (Pigliucci et al., 2006; Crispo, 2007; Pigliucci and Murren, 2003; Ehrenreich and Pfennig, 2015). Importantly, the definition of genetic assimilation and accommodation does not refer explicitly to genetics; rather, it refers to change in the reaction norm. Despite using the word "genetic", the term "genetic assimilation" does not specify what (if any) epigenes, genes, ontogenetic selection processes, or developmental processes have been affected to fix the reaction norm.

7.4. Is the replicator term necessary for selection to occur?

Phenotypic evolution can account for evolutionary changes without genetic change, and thus could be interpreted as evidence that the replicator term is not necessary for selection to occur. This view presupposes that genes or epigenes constitute the only relevant replicators for heritable phenotypic adaptation, which as this paper points out may be an unsafe assumption. Similarly, heritability that follows from polygenic inheritance and complex interactions between various replicators and environment, (e.g. Via and Lande, 1985), may also be interpreted as selection without a clear replicator simply because of the minimal and blurred role of individual replicative entities. However, it doesn't follow that replicators have no role to play simply because multiple replicators are necessary for trait inheritance or because the effects of those replicators remain (as always) environmentally dependent.

However, selection as a process critically requires mechanisms of variation, and these mechanisms need not themselves be selection-based. Since selection can only work on the current band of variation available in the population, nonselective random processes that influence either genetic or phenotypic variation can sometimes be required to account for evolution, particularly in small populations as in *genetic drift*. Such random processes may even expose an interactor to a new selection contingency (Lande, 1976). However, this nuance does not mean that evolution proceeds without replicators (or interactors for that matter). To so conclude would be a category error; variation, like replication, is embedded within the process of selection and they do not constitute a alternate processes.

7.5. Summary and reflections on behavior

Replicators on many different levels work together to produce interactors, and the relative contributions of different replicators can contribute to phenotypic plasticity or stability. Traits can evolve without changes in genetic code via a shift in the phenotypic frequencies along a reaction norm. Evolution of the reaction norm itself is called genetic accommodation, and phenotypically evolved traits can become fixed in the reaction norm in a process known as genetic assimilation.

Behavioral scientists should be aware that these dynamics can potentially affect their subject matter. When a particular behavior develops via ontogenetic selection and contacts a phylogenetic selection contingency, that behavior may become genetically assimilated across multiple generations. This doesn't mean that the behavior becomes inevitable (after all, reaction norms are measured under a naturally limited range of environmental conditions), nor that complex, species-typical action patterns don't involve operant behavioral selection. However, the functions of various stimuli in the behavioral selection process may shift in a manner that makes the behavior more likely to develop under a wider range of variation in the learning environment. This may even occur in captivity with animals that are constantly shaped to the same tasks or selectively bred for success at those tasks. Environmental sensitivity may also be exaggerated when individual animals encounter new environments or when a population from the wild is taken into artificial environments for multiple generations. Potential random changes could occur in individual data when small groups of animals are inbred. An understanding of the nuances potentially at play in the behavioral system as a result of the processes that I've described can heuristically lead to methodologically and theoretically thoughtful consideration of such variables in the investigation of basic behavioral processes.

I will finish by considering the implications of these points in the behavior of behavioral scientists, summarizing a few practical leads that might be drawn from these ideas.

8. Some implications for basic behavioral questions

Although behavioral science itself rightfully focuses on its own selection framework, an awareness of its role in associated selection frameworks could be useful in asking and answering important behavioral questions. For example, such awareness may lead to work that eliminates the apparent tension between the field of ethology, which has established that behavior can be phylogenetically selected, and the field of behavior analysis, which has shown that behavior can be ontogenetically selected, flexible and adaptive. In behavior analysis, particular species have shown the propensity for species-specific motion patterns to persist even in environments not artificially designed to reinforce them (Breland and Breland, 1961). Conversely, ethologists have found that the natural environment is often a necessary component for the expression of phylogenetically evolved behavior. This has led to the suggestion that evolution has created multiple separate phylogenetic behavioral processes, some of which produce species-typical behavioral patterns without any behavioral selection at all, and another, operant process that produces learned behaviors. Yet an examination of the larger framework of selection does not support such an assumption. What may be inherited (more parsimoniously than separate, autonomous processes) are the physiological and environmental conditions that produce either consistent or variant outcomes from ontogenetic behavioral selection. Since ontogenetic selection may be involved in the expression of any given phylogenetically selected behavior, interested researchers could focus a hybrid, interdisciplinary effort on elucidating the process by which such behavioral structures develop within as well as across lifetimes. Such an interdisciplinary approach could identify the necessary and sufficient ontogenetic variables that develop and maintain species-typical patterns or prevent their development. It could discover mechanisms for behavioral heritability and differential sensitivity to environmental contingencies. It would also eliminate the tendency to halt investigation by labelling a particular behavior as “hard-wired” without exploring potential mechanisms of such invariance. I do not suggest that some such efforts have not already taken place, particularly in behavioral proofs of principle that discover how to produce the behavioral characteristics of one species in a different species (e.g. Epstein’s insight experiments (Epstein et al., 1984)). However, unexplored opportunities exist to do so in a more explicit fashion that can complement rather than challenge work in other fields.

I will provide an example in the form of a thought experiment loosely based on real work in songbird vocalization. Suppose that the vocalizations of a songbird are reinforced by the attraction of a mate. This behavior may initially vary within a population if mates respond to many different songs. If however, mates respond preferentially to a particular song, the flexible behavioral selection process would negatively impact fitness because early variation no longer holds much advantage and could result in delayed reproductive success. Suppose a phenotypic variant emerges in which vocalizations are shaped by the similarity of auditory feedback to the song of a birds’ parent: by definition a successful song. Early unreinforced behaviors could occur without delaying successful mating. Such birds along with their nested replicators should be strongly selected for. Once the new reinforcement function is fixed in the population, animals may no longer individually respond as sensitively to variations in mate preference; even if mating as a reinforcer remains strong, the initial song variation has narrowed and subsequent shaping may be limited or slow. Still, the behavioral selection process has not been eliminated or rendered unnecessary. This idea is testable in individual circumstances; it predicts that song development can be still be ontogenetically controlled by response-produced auditory matching. Although this thought experiment is entirely speculative, it is inspired by research indicating that either feedback-shaped song matching or female preference can maintain the form of courtship song in various species (King et al., 2005; Johnson et al., 2002).

9. Some implications for behavioral technology

Unlike realms of basic inquiry, application seeks to control interconnected rather than isolated processes in living systems. An understanding of integrated frames of selection may therefore be most practically utilized in the form of application. I will suggest only a few of myriad possible examples. First, conservation efforts currently hold great importance because of the accelerated extinctions induced in recent geological history by human activity. For this reason, animals from captive breeding programs may be reintroduced into habitats where they were previously extinct. In these scenarios, the species have been temporarily removed from a natural environment that may have participated in the heritability of traits critical for species survival. Reintroduction efforts may be more successful if potentially disrupted traits can be probed, identified and re-established. For example, when the Sandhill Crane was re-introduced to Florida, it was quickly discovered that important species-typical patterns were absent, including migration. Eventually, the team figured out that they could induce a new lineage of this important and previously heritable behavior by training birds to follow small airplanes and then flying those planes along the historical migratory route (Mueller et al., 2013). Newly hatched sandhill cranes in the group required no such intervention; the lineage of behavioral replication was re-established. Similarly, retraining of foraging or predator defense patterns may be important to reintroduction efforts (Griffin et al., 2000). Chinook salmon, for example, showed better survival upon release when they first underwent predator avoidance training (Maynard et al., 2001).

These ideas may also be applicable in the teaching or training of domestic animals. If animals are selectively bred along a narrow spectrum of behavioral performance, it may affect heritable influences on stimulus function or physiology-related contingencies. Awareness of this potential may improve our ability to teach, train, and breed domestic animals in the healthiest ways. Exotic animal trainers may similarly benefit; for a long time this field has acknowledged the importance of natural history in understanding body language and species-typical reinforcers and punishers (Ramirez, 1999). However since phylogenetic selection does not prescribe that selected behavioral patterns will occur captive environments, trainers would do well to test species-specific assumptions and probe for conditions that could produce useful (or problematic) flexibility. Some work has already been done along these lines, such as the explicit training of animals born and reared in an artificial environments to behave similarity to their wild counterparts in order to benefit from enrichment and daily routines (Neto et al., 2016).

10. General summary

Selection as a system of ultimate causation has revolutionized our understanding of living systems. Replicators and interactors, the minimal entities necessary for selection-based adaptation, can each be found on multiple levels of analysis and in selection frames that account for the development of both ontogenetic and phylogenetic teleonomies. Replicators, interactors, and environmental contingencies influence one another in many ways, and these influences together with the movement of these functions from one physical entity to another help to account for the course of evolution. In a given lineage, different levels of interaction could simultaneously exist or could replace one another; conversely, replicator roles generally exist simultaneously in many layered entities. Layered replicators with varying degrees of stability acting simultaneously within and across lifetimes can conditionally provide both phenotypic flexibility and stability across a lineage.

I have utilized the generalized selection processes identified by Hull (1980) to organize different systems of selection into a common framework. I have drawn analogies from many systems of selection to the behavioral and cultural systems of selection. I have identified direct interactions within and between these frames. This framework provides a starting point from which to draw testable applications or basic questions about behavior in its wider context. This general awareness of the broader framework of selection can potentially enrich the efforts of behavioral scientists and place our work in its interconnected niche across the life sciences.

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