Domains of Interaction in Evolution
Transmission, Construction and Selection

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1 Biological Evolution 101

My intention in this short piece is to provide an introduction to some fundamental structures present in all biological processes, explain their importance in evolution, and then attempt to survey those correspondences with social processes and mechanisms of social change (A great deal in a small space implies compression). I also intend to be a little controversial! What I mean by biological processes, are such sets of interactions as metabolism, development, behavior and ecological interactions. What I mean by evolution is how these processes came into existence from different processes (often simpler ones), and how they are able to persist through time.

Evolutionary biology struggles with two empirical problems: firstly, accounting for the enormous diversity of sets of processes, and secondly, accounting for the enormous complexity of individual processes. Neither problem is solved, but greater progress has been made in our understanding of the former. Examples of the first are accounting for species diversity in a given area, or the expected distribution of alleles in a population. Examples of the second are the origins of sex and the metabolic origins of dominance. Some measure of our limited progress in the second can be gleaned by noting how controversial these areas remain; part of the explanation for this might be the ensemble averaging that comes for free in population genetics and ecology.

It is frequently stated that a necessary and sufficient condition for evolution is that populations should be variable, that this variability should be propagated with high fidelity from one generation to the next, and that this propagation is a function of properties of individuals correlated with properties of the totality of their environment. While these are necessary they are not sufficient. We should at least add that individuals be capable of growth or development. In contemporary jargon it is said that there exists a genotype to phenotype mapping.

2 Problems of causality in genotypes and phenotypes

Whether speaking of gemmules (Darwin), determinants (Weissmann), pangens (de Vries) or genes (Johannsen), there has been awareness that propagation between

generations of cells or multicells is mediated by something other than the whole organism. Moreover something smaller, and in some sense simpler. From Morgan (1930s) onwards, the gene has become the atomic unit of intergenerational transmission. Following Weissman, the genic complement of the mature individual is labeled the germ plasm; it is understood as particulate and combinatorial. The long-term persistence or *continuity* of the germ plasm was a defining property of life for physicists (Schroedinger 1944). In 1953 Watson and Crick provided the structure in which the gene was assumed to reside.

Having transmitted the genes (more on this later) we now need construct an organism capable of re-iterating the process. We need some mechanisms for constructing a phenotype from the genotype.

2.1 A digression on von Neumman, replication and robustness

Muller argued that self-replication was *the* defining characteristic of life, and that by extension, the most critical component of an evolutionary theory. Inspired by this definition of life mathematicians and physicists set about constructing minimal formal automata capable of self-replication (more recently Dennet has echoed this position by asserting that the replicator dynamic is "Darwin's dangerous idea" - a domain-independent algorithm of complexification). The discrete arena in which these explorations were made was with cellular automata. Von Neumann (1949) very soon recognized the limitations of this position " One of the difficulties in defining what one means by self-reproduction is that certain organizations, such as growing crystals, are self-reproductive by any naive definition of self-reproduction, yet nobody is willing to award them the distinction of being self-reproductive... "

Burk's (1970) summarized this position with a simple model "... Consider, for example, a two-state cellular system whose transition function takes a cell into state one when any of its neighbors is in state one. Define an automaton to be any area, even a single cell. A cell in state one then reproduces itself trivially in its neighboring cells... ". As this is uninformative, this lead to the statement that that which was replicated required at least some minimal level of "complexity".

Von Neumann's solution was "... A way around this difficulty is to say that self-reproduction includes the ability to undergo heritable mutation as well as the ability to make another organism like the original. And moreover that machines manage to construct other machines more "complex" that themselves, in an open-ended way with the potential for unbounded evolutionary growth of complexity. In other words Von Neumann stressed the need not for a universal replicator, but a *universal constructor* , *robust* against mutational perturbations. The notion of *open ended* and *robust* evolution continues to be a central research theme at SFI. It might also be worth noting at this point that replication need not imply perfect fidelity, as degeneracy in the construction process ensures a robust form of transmission. The high school example is that of translation of mRNA, which as a result of degenerate codon assignment allows different transcripts to encode identical polypeptides.

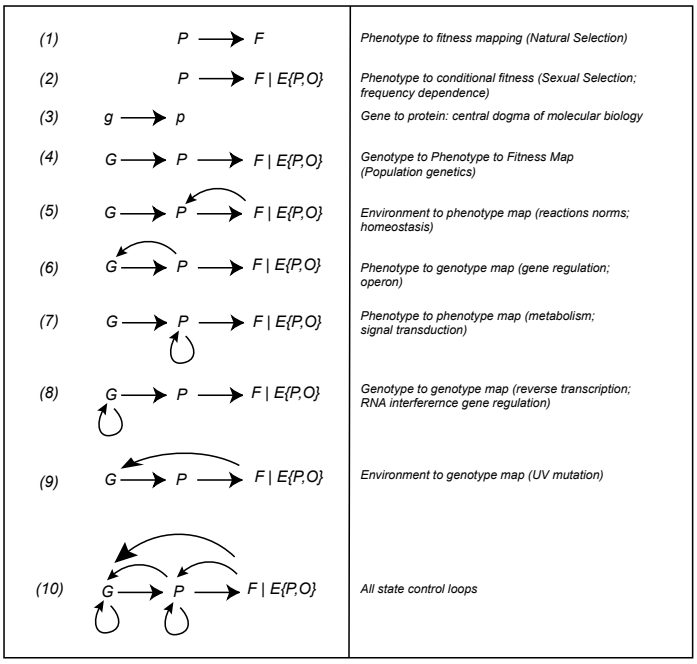
And construction is of course what the molecular machinery of the cell is capable of, as evidenced by viruses that use host cellular machine tools to reconstruct their own, and novel phenotypes, and the use of transgenically modified bacteria, to produce vast quantities of a desired protein, such as insulin.

2.2 Back to the G-P map

The picture of proteins derived irreversibly through the translation of ribonucleic-acid transcripts, became known as the central dogma of molecular biology. This provided the molecular explanation for Weissman's continuity of the germ plasm, and consolidated the picture of development as a function mapping an argument in one domain (genetic) onto its image in a (phenotypic) co-domain. It was deemed perfectly natural to expand both the genetic and protein sets to include all genes and all proteins, and thereby speak of the *genotype to phenotype* map. By identifying phenotypes with payoff or fitness values, and assuming this a total-function, then we have arrived at the *Genotype-Fitness* map, the basis of the familiar Wrightian picture of evolution as a trajectory of genotypes over an adaptive landscape.

2.3 Is there a G-P map?

Without covering all of the history, we might note (see table below) a series of modifications of the genotype, phenotype, and fitness relations, as conventionally depicted as mappings.



Figures (1) and (2) are pre-Mendelian. Figure (2) makes the obvious point that the fitness or payoff depends upon the configuration of the environment, and in particular, the behavior of other organisms. It is this very simple scheme that tempts us to make statements to the effect that the design of a structure can be understood solely in relation to requirements imposed by the environment. In other words, to think as engineers, and view biological structures as solutions to design problems we

might come to understand if we understand the environment (functional task) well enough. Sensorial organs (eyes, ears and noses) and locomotive appendages (legs, wings and fins) have proved well suited to this style of reasoning. Metabolism, signal transduction, immune responses, sexuality, cellular differentiation, behavior and senescence are all areas in which this approach has proved to be weak. As an obvious example consider sexuality, once erroneously thought of as a means of promoting adaptability, but now thought of as a solution to problems endogenous to biological processes: mutational buffering and parasite escape.

Figures (4-10) show that the number of causal connections between the three variables (only two are strictly observables: fitness remains enigmatic except in simple models), increases as we increase the realism of the model, until reaching a state (10), where speaking of mapping, at least in the mathematical sense, becomes problematic. A better term is a causal state, with which an observable can be correlated. Contemporary evolutionary biology must in some sense contend with this architecture. This is largely because these domains need to be understood in relation to *both* endogenous processes, arising out of domain-specific system dynamics, and exogenous function arising through interaction with the environment. This argument has several components: (1) to understand the range of possible solutions to a functional requirement we need some understanding of the endogenous systems dynamics (this means transition rules, stochastic effects, degeneracies); (2) the extent to which the endogenous dynamics are tunable through selection (role of energetic constraints, limitations from sampling etc); (3) The nature of the network of connections between domains (G, O, F, E) and the spatial and temporal scales of variable change.

2.4 Recovering a map through a separation of time scales

One criterion for extracting a mapping from the causal state is when there is a demonstrable separation of time scales. When metabolic interactions, or signal transduction, proceed faster than gene expression; or when development proceeds faster than frequency dependent payoffs. In these cases the arrow of interest in the figure can be interpreted as a first or second order rate constant, and the dynamics endogenous to a system simplified.

3 Heredity

As a result of the almost intractable complexity of the constructive processes giving rise to figure (10), in other words all those endogenous dynamic processes bearing on the phenotype and genotype, evolutionary biology has often accepted the simple scheme (4) and concentrated on the transmission of g (genes) from one generation to the next. In this way sophistication has grown in modeling mechanisms of inheritance, assuming a simple phenotype and environment. I am thinking of Mendelian segregation, dominance relations, imprinting theories of conditional gene activation etc. Theory in this field is thereby mostly concerned with the interaction between payoffs and transmission mechanisms. While this scheme is obviously very far from the reality of biology, I will argue that it is even further from the reality of the social sciences. And hence the use of population-based biological models as templates for investigating culture call for careful deliberation.

4 Building bridges from biology to human culture

There are broadly two ways in which biology might inform the social sciences. The first is to recognize that humans are indeed primates, and that there are biological foundations, shared with other species, from which we are derived. Thus the interest in genetics, cognitive science, psychology, ethology and biological anthropology. This I shall call the approach from shared biological substrate. The second is to search for general principles shared by all complex adaptive systems, irrespective of homology (e.g. general principles of signaling systems, scaling laws, computation and organization, network dynamics and robustness principles). Both are explored at SFI and elsewhere, whereas the latter is a more unique contribution of SFI-style science. I am one more interested in design principles than in building a theory of culture based on the biological substrate of kin selection and the like. A third approach attempts to straddle both substrate and culture-dynamics approaches, and consider the ties between the genetic and the cultural dynamics (Boyd and Richerson). However the cultural dynamics in these approaches have none of the constructive properties I am calling for, and stress *population thinking* at the cultural level combined with a catholic set of inheritance systems.

4.1 Biology to culture maps

Let's start with the most simple minded projection of biological principles onto culture that we can think of, translating term by term, and ignoring the rules of their respective grammars.

In this case we map directly from the heritable domain of the gene to the imitable domain of the strategy, and directly from the replicative domain of fitness to the score-able domain of payoffs. This is the formal framework of both population genetics and evolutionary game theory. We are in the business of tracking the history of the frequency of genes and strategies as they relate to constraints of transmission and payoff. These are very powerful approaches and they manage to do completely without phenotypes. Given that phenotypes are the structures whose complexity and diversity evolutionary theory was nominally invented to explain, what is going on? It is simply the separation of time scales mentioned above. If the mapping from G to P to F is well defined, then this is acceptable. And when dealing with g(gene) to p (protein) maps rather than G (genotype) to P (phenotype) maps, this can be the case. Most population genetics models that are quantitative models of empirical data, relate to genes and proteins, rarely to sets of genes and protein networks, and almost never anything above.

But what of models in the social sciences? The interpretation of G, P and F are all rather fuzzy categories. Clearly in game theory models, strategies must map to G as these are passed on, whereas P is not (most of the time). In biology E acts on G predominantly through P, whereas in social science, the claim seems to be that E can work directly on G. I think this is not the case. I want to claim that there is something peculiar about evolutionary game theory in social science, as it seems to confound the rules that are transmitted with the strategies that individuals adopt. In other words, construction rules are neglected. And whereas in biology this might be OK with g to p maps, it is probably not OK in biology with G to P connections or in the more complex social sciences.

4.2 The stained glass windows of the soul

Consider a gene contributing to eye color: this is not a gene FOR eye color. The diffraction of light through the iris is regulated by varying amounts of eumelanin (black brown) and pheomelanin (red yellow) produced by melanocytes. Variation in the expression of the Melanocortin-1 Receptor gene (MCIR), leads to variation in eye color. Now if selection should operate on the phenotype eye color, it does so by differentially acting on MCIR alleles. Genetic transcription of this gene is regulated by transcription factors activated through signal transduction pathways, responding to variation at other genetic loci. Eye color is a developmental construct, and selection, to change the frequency of eye color, must work on the encoded developmental strategy. Eye color is not inherited; MCIR alleles and their epistatic interaction partners are inherited. This indirection is of fundamental importance when considering the origin and persistence of complex structures.

Now consider a typical model in the application of game theory in social science. The strategy (e.g. cooperation) is the gene (that which is transmitted and encodes) and the phenotype (that which develops and interacts), *simultaneously* the encoding and the complex trait. This is empirically and logically suspect, as a behavior is not cooperation (C), but something through course graining that sits in a set of variable behaviors, that we can tag as C with the suitable application of a payoff function. If we want to understand the diversity of C solutions we need to draw a distinction between the recipe and the dish (At this point I expect someone to get up and attack me!).

4.3 Endogenous domain dynamics

So far I have focused on a missing domain (P), now I want to say something about feedback within domains, and the importance of endogenous dynamics. In other words move on to figures (5-10). The buzzwords in mathematical biology have been for some time gene regulatory networks, pattern formation, community dynamics etc. All of these refer to patterns of interaction within the G, P or E domain. I think it safe to say that most ecologists would think an evolutionary theory useless if it did not take into account the rules of interaction among individuals or communities. Much of the recent progress in ecology has consisted in extending the complexity of endogenous systems dynamics to include stochastic effects, space and network structure. Evolutionary ecology is explicitly concerned with the interaction of the system dynamics with the mechanisms of strategy transmission and the problematic nature of payoffs. Similarly in the new field of *evo-devo*, the relationship between gene regulatory architectures is becoming coupled to modular recombination during evolution, to produce varied body plans.

I assume human culture at least as complex as cells, individuals and ecosystems, and yet this is rarely reflected in the theories. What are we to do?

5 A plan of action

What I want to argue for is the fusion of a new cultural dynamics with an evolutionary dynamics of culture, and not a collapsing of one upon the other. Much the same way that in biology we might study the dynamics of cellular processes and their

relation to the evolutionary dynamics of individuals, and predator prey dynamics or community dynamics in relation to the evolutionary dynamics of species. Here is a list of suggestions.

5.1 *Modus Tollens*

1. Identify exactly what is being transmitted between individuals. In other words identify the atomic units of cultural inheritance, rather than assume they are words or norms etc. 2. Study the rules of transmission, rather than borrow the asexual, non-recombinant unicell model from biology 3. Develop models specifying the endogenous construction rules connecting inherited units to behaviors. This includes the way in which the transmitted information interacts with its developmental context. 4. The above will require a better understanding of pattern formation, epistasis, computation etc, as well as paying more attention to the data bearing on the cognitive rules of learning, pattern formation etc. 5. The above imply the need to understand culture as something emergent, rather than the set of all strategies, and see it as much more than the sum of minds, and in all likelihood cybernetic (to include material culture). 5. Stop imposing a too simple notion of function on behavior. Function is really not a thing but a correlation between the Phenotype and the Environment. In biology adaptation also emerges at this boundary. These are tricky concepts, let's be more honest about this. 6. When using the word fitness be very explicit about its interpretation (payoff, life time reproductive success etc), and make quite clear that it is in all likelihood not fungible.

5.2 *Modus Ponens*

1. Recent progress in adaptive dynamics has made it possible to include ecological interactions, within an evolutionary dynamic. This is a literature worth reviewing. 2. Computational power allows us to explore axiomatically grounded agent-based approaches (think Lambda and Pi calculus), and specify rules of interactions that are transparent, non-arbitrary, and capable of being informed by good experimental data. 3. Areas such as linguistics have great data, and a strong formal tradition. This is a group that evolutionary biologists should be talking with (I and others are trying), and represents what we might call an interface-science. Others are anthropology, economics etc. 4. Quit imposing function on our system and explore the consequences of the simplest possible rule systems which allow for the emergence of interesting behavior and the possibility of some analysis (e.g. econophysics). 5. Integrative science: workshops such as this one allow us to expose metaphor, and strive to uncover true regularities.