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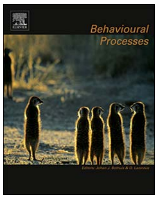

An integrated bayesian theory of phenotypic flexibility

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ABSTRACT

Phenotypic flexibility includes systems such as individual learning, social learning, and the adaptive immune system. Since the evolution of genes by natural selection is a relatively slow process, mechanisms of phenotypic flexibility are evolved to adapt to contingencies on the time scales ranging from a few hundred milliseconds (e.g. avoidance of immediate physical threats) to a few millennia (e.g. cultural adaptations to local environmental variation in the Holocene). Because environmental variation is non-stationary and fat tailed, systems of phenotypic flexibility sometimes have to be creative. They do this by means of random innovation, or exploration, and selective retention. The canonically rational way to deal with variable, uncertain environments is the Bayesian process of using new data to update priors based on past experience. Organic evolution updates the gene frequencies of populations based upon the fitness of alleles. Learning updates behavioral priors based upon the reinforcement of alternate behaviors. Genes and mechanisms of phenotypic flexibility are not isolated but richly interact. Classically, genes are said to code for the reinforcers that shape behavior in individual learning, for example. It is currently controversial whether or not these interactions include a role for the products phenotypic flexibility directly shaping selection on genes.

1. Introduction

The purpose of this paper is to try to think hard about the role of phenotypic flexibility in evolution. All organisms face the problem that environments are variable in space and time. Evolution by natural selection adapts to this variation by changing gene frequencies. Such changes often evolve rather rapidly in time and finely in space (Thompson, 2013). Nevertheless, even bacteria that can evolve very rapidly have inducible enzyme systems to respond to changes in their environments (Parke, 1975). Bacteria also have a form of adaptive immune system, CRISPR (Horvath and Barrangou, 2010). Eukaryotes have several well-known systems of phenotypic plasticity, including reinforcement based learning, social learning, the adaptive immune system, use-based adaptations of anatomy and physiology, and homeostatic systems designed to maintain key internal parameters constant in the face of environmental variation. Others are less well known or more controversial, including niche construction (Odling-Smee et al., 2003) and transgenerational epigenetic inheritance (Jablonka and Raz, 2009). Organisms also make use of commensals to adapt to variable conditions. For example, the microbiomes of larger animals flexibly extend the range of metabolic pathways that can be used in digestion by deploying bacterial enzymes to break down exotic foodstuffs. Japanese cuisine includes large amounts of seaweed and accordingly Japanese guts commonly harbor bacteria with enzymes that break down algal polysaccharides (Hehemann et al., 2010). Mechanisms of phenotypic flexibility appear to be costly adaptations to judge from the adaptive immune system (Lochmiller and Deerenberg, 2000) and the investment in brain size related to individual and social learning (Aiello and Wheeler, 1995; Reader and Laland, 2002).

Two important issues will be the focus of this paper. First, what is the general relationship between systems of phenotypic adaptation and the processes of genetic evolution? Second, how do the various forms of phenotypic flexibility relate to one another?

To answer these questions it is helpful to have a general theory of how organisms deal with environmental variability. My candidate for a foundation for such a theory is Bayes' Law. Inheritance systems like genes, epigenetic inheritance, and culture furnish prior hypotheses about what the world might be like. For example, a bacterium with a suite of inducible enzymes for carbohydrate metabolism has a suite of hypotheses about what the carbohydrates might be in its environment. The particular medium the bacterium finds itself in furnishes data about what the concentrations of various carbohydrates actually are. The bacterium produces the appropriate enzymes accordingly, updating the inherited hypotheses. As the external supplies of carbohydrates change, enzyme activity is upregulated and downregulated according to the ongoing stream of data from the environment. Updating is iterative. Multiple priors may need to be combined to make the best guess of the best behavior. A young subsistence farming family inherits genes related to digestive physiology and cultural skills and beliefs related to what plants and animals to grow under prevailing conditions. If the

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environment changes both sets of priors will be important. If the family's genes include a gene for adult lactase persistence, they have what amounts to a stronger prior to put more emphasis on dairying compared to a family where lactase is down-regulated at weaning. But a lactase-persistent family with no recent cultural history of dairying will in effect have a prior that dairying is not a feasible option despite having the biological capacity to digest milk sugar as adults. If this family has access to an extension course in dairying they may calculate that engaging in dairying is worth the risk.

The attraction of the Bayesian approach to building a theory phenotypic flexibility is that Bayes' Law is the canonically rational way to combine past experience with new data, *absent any limits on computation*. The concept of priors captures the idea that innate or earlier acquired knowledge should play a role in determining behavior while the concept of data captures the role that the current environment plays in determining behavior. The persistence of nature-nurture debates stems from a poor appreciation of Bayes' argument. Most commonly, we should expect past experience (nature) and the state of the current environment (nurture) to co-determine behavior. Bayesian models are flexible and can be applied to many concrete problems ranging from word learning to disease diagnosis (Perfors et al., 2011). Given the wide range of systems for phenotypic flexibility and the complex ways in which they interconnect, this flexibility is an advantage, but at the same time it highlights the deep similarity of all these systems.

The fact is that there are always limits to computational power. What might be called the Evolutionary Bayesian Hypothesis of phenotypic flexibility holds that adaptive evolutionary processes will favor systems of phenotypic flexibility that yield behaviors that are as close to the results of formal Bayesian analysis as the limits to biological computation permit. We expect that adaptive processes will construct the same general sorts of approximately Bayesian computational schemes as Bayesian statisticians devise to get around the limited computing power of modern computers. The Bayesian hypothesis thus asks two questions. How close to the Bayesian ideal do actual systems of phenotypic flexibility come? What algorithms actually implement the approximately Bayesian inference?

The competing hypothesis is that there are mechanisms of phenotypic flexibility that are non-Bayesian in lacking the structure of using data to update priors and yet are adaptive. Perhaps panic is an example. When confronted with a large, immediate threat, computational systems are unable to deploy priors and assess the incoming data in light of them. It may be adaptive to merely flee from the threat as rapidly as possible or lash out with any weapon or tool available if confined. The advice "don't panic" captures the idea that if time permits, decision-makers should do their best to approximate Bayes' Law rather than blindly flee or lash out.

Evolutionary biologists are engaged in a vigorous debate about the role that phenotypic flexibility plays in evolution. The Modern Synthesis made a sharp distinction between proximal and ultimate factors in evolution (Mayr, 1961). The ultimate causes of biological phenomena are natural selection acting on genetic variation. The products of ultimate causes include the highly diversified set of proximal causes that adapt the phenotype of organisms to local spatial and temporal variations in the environment. Mayr argued that ultimate causes were sovereign, leading to proximate causes but proximate factors having no effect on ultimate causes except through selection on the genes that control the proximate systems. Recently, old doubts about this dogma have been revived. Developmental systems theorists (Oyama et al., 2001) argue that developmental systems play a strong and innovative role in genetic evolution and call for an Extended Evolutionary Synthesis. A simple case is the Baldwin Effect (Baldwin, 1896). If a population of organisms migrates to a new environment, learning and other elements of phenotypic flexibility will adapt it to the change. Perhaps in the old environment most green fruit was unripe and inedible and most red fruit edible. In a new environment perhaps much green fruit is edible and most red fruit is poisonous. Learning will enable individuals to switch their diets but at some cost of initially suffering the ill effects of sampling poisonous red fruit and passing up nutritious green fruit. In the old environment, genotypes (priors) that favored eating red fruit and avoiding green fruit would have evolved in order to lessen the errors that young animals would make if they had to rely entirely on learning (new data). In the new environment selection will favor the opposite genetic prior among youngsters for the same reason. More generally the leading edge of adaptation may commonly not be natural selection on genes but mechanisms of phenotypic flexibility (Laland et al., 2011; Levis and Pfennig, 2016; West-Eberhard, 2005). Phenotypic flexibility essentially "proposes" adaptive innovations and if they are successful genes will "accommodate" them as in the red versus green fruit example. Often, learned behaviors will expose latent quantitative genetic variation to selection, leading selection to favor genes that generate the same phenotype as learning, but with less cost than pure learning. Human culture, because it comparatively rapidly evolves complex, radically innovative adaptations to new environments, is arguably an especially potent form of phenotypic influence on genetic evolution (Itan et al., 2009; Laland et al., 2010; Richerson et al., 2010). These ideas have met with considerable resistance; some evolutionary biologists defend the Modern Synthesis and the proximate-ultimate barrier along the lines of Mayr, (1961) original argument (Haig, 2007; Scott-Phillips et al., 2011).

Interactions among the various forms of phenotypic flexibility are much less well investigated, for example, interactions between niche construction, individual and social learning and genetic evolution. This is partly because very different disciplines attend to the different forms of phenotypic flexibility. Immunologists study the immune system, ecologists study niche construction, psychologists study learning and sometimes social learning, anthropologists study culture, anatomists and physiologists study the phenotypic adaptations of anatomy and physiology, and so forth. A famous paper by B.F. Skinner (1981) noted how a general model of selection by consequences points up a similarity between natural selection acting on genes and reinforcement acting on individual learning and culture. But he did not systematically explore the interactions between these three systems. For a contemporary version of this analysis see Baum (2017); Campbell (1965) used the term "vicarious selectors" for the factors that generate reinforcement. Selection acting on genes will generate reinforcers that in ancestral environment acted as priors to help organisms learn useful behaviors. For example, sweet taste is a positive reinforcer because hard working hunter-gathers and farmers were generally short of calories and sugar rich food items like fruit and honey helped balance their energy budgets. From a whole-organism perspective we might expect the various systems of phenotypic flexibility to be partly specialized but also tightly integrated. Most of the symptoms of illnesses, many generated by the interaction of the immune system with pathogens, are quite aversive. Thus individual and social learning will treat illness as data that might update priors, say about suitable locations for habitations (Whitehead and Richerson, 2009).

2. The nature of environmental variation

A common model of noisy environments is to assume that variation is normally distributed around some mean value. The normal distribution has only two parameters, the mean and the variance. More realistic distributions often require additional parameters that are a nuisance for theorists interested in analytical models where possible. However, most environmental time series are characterized by red ($1/f^{\theta}$) noise, where f is frequency and $\theta = 1$ is sometimes called pink noise and $\theta = 2$ is red noise proper (Whitehead and Richerson, 2009). Introducing such a troublesome complexity requires simulations to analyze. The "color" of noise is an analogy with the electromagnetic spectrum. In the case of red noise, as the frequency (f) decreases the amount of variation at that frequency increases. A Fourier transform is an algorithm that fits a continuum of sines and cosines to data in the

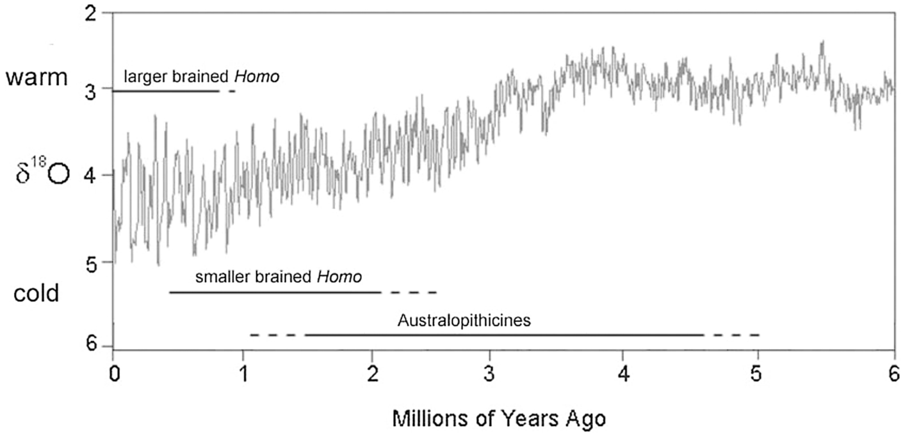

Fig. 1. Oxygen isotope estimates of the earth's temperature of the last 6 million years. The variations in this record reflect the small perturbations in the earth's heat budget due to variations in the earth's orbit. Up until about 3.5 million years ago (mya) the record is dominated by the precession of the equinoxes, a 21–23 thousand year quasi-cycle. From about 3.5 mya until 1 mya the dominant pattern is driven by the 41 thousand year cycle of the tilt of the earth's axis of rotation with respect to the sun. Finally, for the last million years the pattern is dominated by the ellipticity of the earth's orbit about the sun which has a 100 thousand year period. After Opdyke (1995) and Klein (2009). Note that in this time series the mean temperature is declining, the variance is increasing, and the pattern of autocorrelation (spectrum) is changing, making it non-stationary.

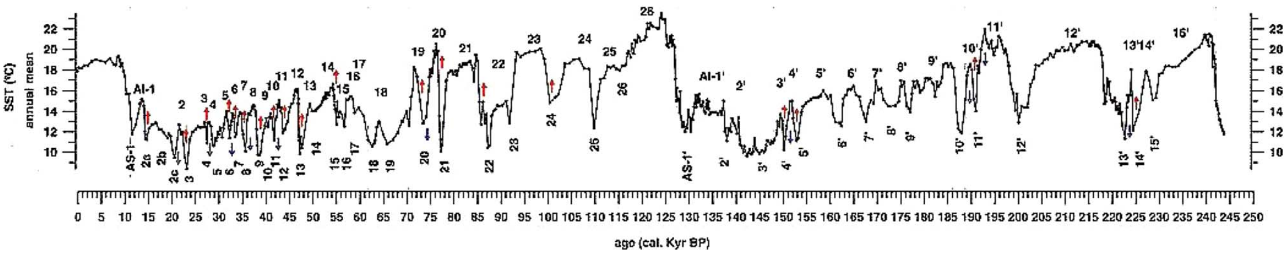

Fig. 2. Estimated sea-surface temperature from a core raised from the Mediterranean Sea covering the last two 100,000 year glacial cycles and a bit more. Notice the many abrupt warmings and coolings indicated by the red and black arrows, signatures of non-linear feedbacks in the climate systems. Notice also that these abrupt events are more common in the last ice age than in the 2nd to last, an example of non-stationarity. Note the stable Holocene climate at the far left compared to the hypervariable last ice age from 50 thousand to 11,000 years ago. From Martrat et al. (2004).

time domain to express the data in the frequency domain. A Fourier transform is the mathematical analog of a prism that decomposes a beam of light into its constituent colors. (Fourier transforms are the oldest of such techniques but not necessarily the best (Huang et al., 1998)). White noise is noise with the same amount of energy (variance) at all frequencies. Red noise is has disproportionate variation at low frequencies and blue noise would be a spectrum with disproportionate variation at high frequencies[1]. Spatial variation is also normally red (e.g. Powell et al., 1975). Compared to the normal distribution, red noise produces fat tailed distributions. Readers of Taleb (2010) best-seller *Black Swan* will have some intuition for red noise–big, somewhat rare, surprises are more common than we would expect from the normal distribution. In addition to exhibiting red spectra, real environmental noise is generally non-stationary and non-linear. Non-stationary time series are those whose statistical properties change over the series. In the real world, means change, variances change, and the spectrum changes. Non-linear time series have abrupt or spikey changes rather than smooth changes easily fit by sines and cosines. Fig. 1 illustrates a non-stationary time series and Fig. 2 one that is both non-stationary and non-linear.

For the last 65 million years, the earth has mostly gotten cooler, drier, and more variable (Zachos et al., 2001). Grasslands and deserts have expanded and forests have shrunk. Over this span of time the brains of many mammalian lineages have expanded (Jerison, 1973). In primates, the brain size of extant species is correlated with innovative behavior and social learning (Reader and Laland, 2002). Theoretical models suggest that individual and social learning are useful to cope with variation on time scales of less than a generation to some tens of generations (Boyd and Richerson, 1985; Whitehead, 2007). Larger brained birds and mammals can be introduced into novel habitats more successfully than smaller brained ones (Sol et al., 2008, 2005). With the partial exception of the latter part of the Pleistocene and the Holocene (Fig. 2), no evolution relevant environmental time series resolves the time scales at which mechanisms of phenotypic flexibility are most important. Given that nervous tissue has high energy demands we might expect brains to grow and shrink fairly rapidly under selection in response to the benefit/cost ratio of learning and social learning. Human brain size seems to have decreased appreciably in the stable Holocene relative to the unstable Pleistocene (Hawks, 2011). Brain size increases in the genus *Homo* parallel increases in millennial scale climate variation over the last eight 100,000 year glacial cycles (Richerson and Boyd, 2013). It is thus tempting to think that the increasing brain size of mammals over the last 65 million years is a proxy for increasing variation at the time scales relevant to individual and social learning.

Environmental variation is probably red and non-stationary out to the very long run. Geochemists describe ancient worlds that are startlingly different from the world of the last 65 million years, as variable as that has been (Ward and Kirschvink, 2015). For example, animal life as we know it only existed for about the last quarter of the earth's history, probably because the first three quarters of that history oxygen concentrations were very low. Evolution has no efficient way to adapt to rarer events, particularly abrupt changes (Whitehead and Richerson, 2009). Mechanisms of phenotypic flexibility can be tuned by evolution to cope with relatively high frequency variation and genetic evolution can track sufficiently slow low frequency change. Most evolving lineages eventually encounter large environmental changes to which they cannot adapt, and they go extinct, sometimes in cataclysmic mass extinction events.

For many environmental parameters we have very little information on what sorts of variation organisms face. The impacts of climate variation will be substantially filtered through their impact on paleo-ecological factors. Reconstructing paleo-ecologies in any detail is difficult (e.g. Saarinen et al., 2016). However, the impact of orbital and sub-orbital scale variation on vegetation is clear (González-Sampériz et al.,

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[1] For a good introduction to the concept of the color of noise see https://en.wikipedia.org/wiki/Colors_of_noise.

2010). Syntheses of existing and future data promise major advances in our understanding of paleoecology (Sánchez Goñi et al., 2017). For the purposes of this paper, the basic idea that organisms are forced to cope with a red noise environment will have to suffice.

3. Bayesian models to unify evolution and phenotypic flexibility

Psychologists and neurobiologists have used Bayes' principle as the foundation of a theory of how organisms should learn (e.g.Gopnik and Wellman, 2012; Perfors et al., 2011; Tenenbaum et al., 2006). Bayes' principle is the canonically rational method for combining constraints on learning with experience to probabilistically approach optimal behaviors in given environments. Artificial intelligence simulations and neurobiological experiments aim to understand the actual algorithms that people and other animals use to infer the structure of the world they must confront (e.g.Jadbabaie et al., 2012; Xu and Tenenbaum, 2007). Such algorithms most likely do not literally estimate the probabilities of data given hypotheses; this is a challenging task for computers in realistically complex cases. In practice, Approximate Bayesian Computation is often used to circumvent these difficulties (Lintusaari et al., 2017). Updating algorithms of mechanisms of phenotypic flexibility are likely to be only approximately Bayesian as well, but may be calibrated by evolution to simulate Bayes' principle fairly closely (Gopnik et al., 2017).

Tenenbaum and colleagues (Tenenbaum et al., 2011; Tervo et al., 2016) propose that humans and other animals use Hierarchical Bayesian Models to infer the structure of the world around them from sparse data. The idea is that, when learning, individuals can update hypotheses at several levels. A child learning the meanings of words is simultaneously learning the local meanings of words, potential cognates that can be used as priors in learning a related dialect or language, and high level principles of grammar from functional differences of classes of words like nouns and verbs. Different words have different functions in sentences and the meaning of sentences differ depending not just on the meaning of words but on the functional relationships between them. High level principles in turn can serve as hypotheses about the structure of analogous phenomena distantly related to the original phenomena that generated them. For example, complex skills and complex machines are a little like sentences with many sub-skills or parts that are functionally interrelated (Stout and Chaminade, 2012). A high-level grammatical construct can potentially be usefully applied to understanding any functional part-whole system. Similarly, high-level tree structures arise from folk biology and can be modified to classify artifacts. A structure for estimating the nature of an animal from the tracks that it leaves might be pressed into service for diagnosing diseases from their symptoms. Chains of cause and effect operating in time can also describe relations in space. Tenenbaum et al. (2011) speak of the "blessings of abstraction." Abstract knowledge is easier to acquire than the low-level details of learning a complex skill but is potentially a valuable starting point for beginning to master a new problem. Scientists often tout the virtues of simple general models along the same lines (Levins, 1966). Simple models often fit data fairly well and if not they are usually a good starting point for refinements.

Evolutionary processes bear a deep resemblance to Bayesian models of phenotypic flexibility (Suchow et al., 2017). The mathematical form of a simple selection model is very similar to Bayes Law:

$$p_i^{(t+1)} = \frac{f_i p_i^{(t)}}{\sum_j f_j p_j^{(t)}} \tag{1}$$

$$p(h_i d) = \frac{p(dh_i)p(h_i)}{\sum_j p(d|h_j)p(h_j)} \tag{2}$$

Eq. (1) represents the change in gene frequency over one generation, $p^{(t)} \to p^{(t+1)}$, given the fitness f_i of an allele i relative to the mean fitness of all alleles at the locus. Eq. (2) is Bayes' rule where the posterior probability of a hypothesis, $p(h_i|d)$ is the prior probability of the hypothesis, $p(h_i)$, times the probability of the data given the hypothesis, $p(d|h_i)$, divided by the sum of posterior probability of all hypotheses under consideration.

In the case of evolution, the iterative nature of updating is explicit. In Bayesian development applications iteration is often not explicit. The learner is conceived of as learning an approximation of a stable truth, such as the meaning of a word, from one episode of data acquisition. However, learning complex skills requires extended periods of time in which an apprentice uses iterated individual and social learning and other forms of phenotypic flexibility, such as muscle modification, to master a craft (Sterelny, 2012). The structure of the problems posed by variable environments for systems of phenotypic flexibility requires an iterated framework. In a red noise, non-stationary environment, change is ongoing. Small surprises (data outside the envelope of past experience) are routine and bigger surprises are not so rare. A history of reinforcement for a given behavior creates a current prior for that behavior. Ongoing use of the behavior results in a certain amount of reinforcement for that behavior, constituting new data on its efficacy. In a changing environment, previously positively reinforced behaviors may become unreinforced or punished, resulting in the more or less rapid extinction of that behavior. The existing titer of an antibody is the prior on the probability that antigens attacked by the antibody are a current threat. Absent the threat, new data cause the production of the antibody to be downregulated. The adaptive immune system generates random antibody configurations in low frequency. In a changed environment, a novel antigen is liable to be attacked by some rare random antibody. The immune system then ramps up production of that antibody, with luck before the individual is overwhelmed by the new threat. Cultural evolution is similarly iterated. Humans especially, but social animals in general, learn from each other. Rat pups when they leave the nest follow adults to sources of food, learning where to look for food and what foods to eat. Norway rats and black rats use very different updating rules. Norway rats have rather weak priors and readily adopt foods they smell on other rats, although they have a strong prior to be very cautious about novel foods they do not smell on other rats. Black rats acquire food preferences from their mothers and form very strong priors to eat what mom eats. In much of the developed world Norway rats have replaced black rats, perhaps because modern garbage dumps have more diverse and variable foods than historic ones, favoring the more frequently updated Norway rat social learning system (Chou et al., 2000).

Campbell (1960; 1965) proposed that, at bottom, all adaptive systems could be modelled as "blind variation and selective retention." Simonton (2010) reviews the last half century of work inspired by the 1960 paper. Campbell placed too much emphasis on the blind part of variation. Tenenbaum et al. (2011) argument that abstract models can help speed the search for solutions to novel problems is well taken. Wagner (2014) argues that biological systems have something like abstract knowledge that speeds the "arrival of the fittest." For example, at least some reasonably common enzyme variants well adapted to, say, cleave sucrose into two smaller digestible sugars, may have some activity in cleaving another indigestible sugar, maltose. If maltose becomes common relative to sucrose in an animal's diet, selection may favor mutations that optimize the sucrase locus for maltose digestion.

However, it will not do to underestimate the importance of random variation. Barrett (2012) used the norm of reaction concept as his basic model of phenotypic flexibility. A norm of reaction maps environmental cues onto phenotypic behavior. Barrett conceived of norms of reaction as genetically coded and shaped by natural selection but more generally they might also be influenced by a prior history of reinforcement or by socially transmitted priors. If norms of reaction are modelled as deterministic mappings, they underestimate the power of randomness in the face of red noise. Tooby and Cosmides (1990) insisted that adaptation can only reflect adaptations to past environments. Ironically, organic evolution depends upon random mutation and recombination to adapt fairly efficiently to novel environments. Adaptively shaped deterministic norms of reaction can only reflect data obtained in past environments, but red noise will often exhibit variation rare in the historical environment if not outside of the range of historical data entirely. The truly novel elements of an environment can be explored most efficiently with random modification of old priors or a random search for new hypotheses perhaps guided by abstract knowledge. Mechanisms of phenotypic flexibility essentially simulate the random mutation of organic evolution in this regard. Of course, mechanisms of phenotypic flexibility also depend upon priors shaped by past experience. What recommends the Bayesian approach is this mixing of past experience plus a little randomness, serving as priors, updated with current data sampled without bias.

4. Influences of subsystems on each other

Perreault et al. (2012) develop a Bayesian model of how individuals should update their behavior when they have access to two types of data, direct reinforcement, x, and observations of others' behavior, y, about whether the optimal current behavior is 1 or 2. The optimal estimate that behavior 1 is best is given by

$$p(1x, y) = \frac{p(x1)p(y1)}{p(x1)p(y1) + p(x2)p(y2)} \tag{3}$$

Perreault et al. (2012) show that this approach yields the same results as classical fitness optimizing models of the interaction of individual and social learning. This is an important insight because we might expect that priors and data from different modes of inheritance and different kinds of new data can usefully be combined. The design of organisms is rather modular, but the modules certainly communicate, uniting the parts into a system. A perfectly modular organism would have no need of nerves and hormones to communicate. Some colonial coelenterates and bryozoans approach this extreme. However, animals, even most plants, form well-integrated organisms with organs coordinated by extensive communication (Buss, 2014). The reasons for partial modularity are easy to imagine. The problem of adapting to diseases is rather different from learning a new behavior. Social learning can transmit priors across generations, a feature that is useful for adapting to variation with time scales longer than a generation. The advantages of specialization will drive the diversification of systems of phenotypic flexibility. But we might expect a good deal of systemic integration of parts in organisms' evolved approaches to phenotypic flexibility. In Perreault et al. (2012) example, if a particular important environmental variable is evolving moderately quickly on the generation-to-generation time scale, the most adaptive prior might be to weight direct reinforcement more or less equally with observations of other's behavior. A lifetime of finding direct reinforcement to be more accurate than tradition would cause parents to teach less traditional priors to their children. My father's oft-repeated opinion that "you can't stop the wheels of progress" might have been an example. See Giuliano and Nunn (2017) for an empirical study of how priors for the utility of tradition versus more rapid updating do vary as a function of environmental instability. In the rest of this section I outline some of the more obvious communication we might expect between subsystems.

4.1. *Genetic evolution*

In the classic Modern Synthesis model of phenotypic flexibility (Mayr, 1961), selection on genes leads to what amount to priors in the form of developmental switches that attend to environmental data during development or in the form of reinforcers that act as vicarious selectors of behavioral variation. As far as I am aware, no one doubts that this is part of the story. As we have already seen, Developmental Systems Theorists think that the Modern Synthesis story is quite incomplete and propose mechanisms such as genetic accommodation and social selection by which mechanisms of phenotypic flexibility can have effects on gene frequencies.

4.2. *Individual learning*

In reinforcement-based individual learning, learners have priors in the form of senses that act as reinforcers of behavior. A hungry Norway rat placed in a novel environment might have few or no priors for *where* food might be in the environment but it has an array of innate taste and odor priors for *what properties* suitable foods should have and not have. Rats also have the prior that novel foods are likely unsafe, no matter that they have a favorable taste and odor profile. As they search the environment for suitable foods they will find some that taste and smell like food. They sample them cautiously in small amounts and avoid them if they suffer ill effects. The eating of tasty food reinforces adding an item to the diet, but illness reinforces avoiding an item. A history of reinforcement leads to priors being heavily shaped by experience; rats rapidly learn where the food is in a novel environment. Perhaps no food turns out to have a highly favorable taste and odor profile, but some foods are sufficient for life and are eaten despite not being very tasty. In a constantly changing world the updating of the diet may continue for a lifetime, as it does in Norway rats. At the population level, genes for taste and odor priors are likely to vary. Some variants may inhibit the addition of a poorly tasting but nutritious food to the diet enough to have negative selective consequences, and other variants may discriminate enough in favor of the new food to have positive selective consequences. Eventually the evolution of taste and odor priors may accommodate to the new diet, strongly reinforcing consumption of the once barely edible food item. This mechanism, if it is commonly active, leads to the "flexibility first" argument of developmental systems theorists cited above.

4.3. *Cultural evolution*

In some ways culture acts much like genes, transmitting priors from one individual to another. In the classic studies of social learning in Norway rats pioneered by Galef (1996), rats have a genetic prior that any food odor they detect on another rat is safe to eat. In social learning Norway rats are as trusting as they are suspicious in individual learning. This system serves Norway rats very well. Because of individual neophobia only well tested novel foods enter the colony repertoire of foods, so any food being eaten in quantity by another rat is very likely to be safe. Even sick rats are trusted by the social learning system. Modern rat control strategies depend on slow acting poisons that subvert the neophobia defense. Rat populations in turn evolve genetic defenses against such poisons. The rat catcher's work is never done (Pelz et al., 2015).

Experiments with human children show that even at very young ages, children have sophisticated innate or earlier-learned priors that they use to evaluate new social information. For example, compared to apes, children "overimitate," copying functionally irrelevant parts of demonstrated actions as well as relevant ones (Whiten et al., 2009). However, children are very sensitive to normative information in the degree to which they overimitate (Keupp et al., 2013). If demonstrators suggest that the action demonstrated is part of a ritual or the rules to a game, most children imitate arbitrary parts of the demonstration rather exactly compared to if they are led to believe it is just a simple matter of a mundane functional performance (Herrmann et al., 2013). There are often good reasons to have a prior in favor of imitating people that are like oneself because similar people are likely to share ecological or social circumstances (McElreath et al., 2003). Cues such as race and dialect activate this prior, but interestingly accent trumps race (Kinzler et al., 2009). This makes sense because before long-distance transport few people would be exposed to different races, but socially or ecologically different neighbors would likely have different accents, even if not very different physically.

Social learning can modify, reinforce, or contradict genetically inherited priors. Human cuisines are largely an example of modification

(Aldridge et al., 2009; Anderson, 2014; Mennella, 2014; Rozin, 1996). Children inherit evolved genetic preferences for sweet and salty foods and an aversion to bitter tasting foods. Like rat pups, human children are neophobic. Sweet, fatty, umami and salty tastes are a prior to eat energy and protein rich foods that were scarce in pre-modern environments relative to the needs of hard working people. Bitter tasting plants are often toxic. Hence children prefer diets rich in sugar, fat, and salt, and light on vegetables. Today, food manufacturers produce inexpensive foods that reflect these innate preferences, leading to their reinforcement and to an obesity epidemic. Many human cuisines based on agricultural products were dominated by bland starchy staples that are decidedly less attractive than cleverly formulated modern snacks. Experimental studies show that children's food choices are influenced by social learning (Addessi et al., 2005), and children usually end up preferring familiar foods that they grew up eating. "Comfort foods" seem to function as signals of social belonging and people can get very attached to customary cuisines (Troisi and Gabriel, 2011; Wyke and Landman, 1997).

Many of the world's cuisines have a large place for capsicum peppers and other hot spices that activate pain sensors, so children have a strong innate prior that they are to be avoided. Yet such spices have anti-microbial and antioxidant properties (Billing and Sherman, 1998; Nascimento et al., 2014). Children, particularly in countries where hot peppers are regularly used in the cuisine, begin to acquire a preference for them in middle childhood (Rozin and Schiller, 1980) despite finding them aversive at younger ages. In this case, cultural and perhaps individually learned priors come to dominate genetic ones.

Boyd and Richerson (1985: Ch. 4) showed that culturally inheriting priors is adaptive in environments that vary at intermediate time scales. Considering vertical transmission from parents to offspring, environments that vary rapidly on the generational time scale cannot support cultural adaptions because parental environments do not predict offspring environments. Individual phenotypic flexibility is one solution for such variation. At very long time scales genetic evolution and geographic range changes (niche chasing) adequately track environmental change. As argued in the section above on the nature of environmental variation, the increasing tempo of high frequency climate variation at millennial and sub-millennial scales is a plausible environmental driver of the evolution of our costly system of cultural evolution. Cultures can change substantially on the century to multi-millennial time scales. Genes are more conservative. The human cultural system is quite flexible however. Horizontal transmission can spread both advantageous cultural variants and pathological memes on microbial time scales (Perreault, 2012). Humans have a range of social learning strategies that they can deploy, and these vary substantially from individual to individual in laboratory social learning experiments (Efferson et al., 2007; McElreath et al., 2008; Mesoudi, 2011). Whitehead (2007) simulation study generally found that multiple social learning strategies persisted indefinitely in red noise environments. The cause(s) of this variation are poorly understood. Quite likely cultures vary in their social learning strategies (Giuliano and Nunn, 2017; Glowacki and Molleman, 2017).

The most controversial claims about the role of social learning come from the case of culture in humans. The evolutionary study of human culture is a fairly large field (Henrich, 2016; Richerson and Christiansen, 2013). There have been two views about the basic relationship between genes and culture. Wilson (1978) argued that natural selection on genes resulted in an evolved human nature that ultimately controls cultural evolution. Richerson and Boyd (1978) introduced a game theoretic model in which genes and culture were like a mutualism between two species with partly cooperative and partly conflicting interests in the behavior of their common phenotype. For example, in humans culture may maintain much more variation between groups than genes can, making humans subject to group selection at the level of social groups (Boyd and Richerson, 1982). Such selection could result in cooperation including cooperation to punish individuals who fail to follow cultural rules. The innate components of the psychology that underpins our life in large, cooperative groups could have evolved under culture-led gene-culture coevolution (Richerson and Boyd, 1999). Punishment, for example, in humans is generally regulated by formal laws and informal traditions and customs. Punishment exerts social selection on genes as when incarceration, status reduction, or exile reduce the opportunity to reproduce, hence exerting selection against genes that increase the risk of punishment. A considerable body of empirical evidence is consistent with this hypothesis (Richerson et al., 2016). Thus, gene-based priors may often express the imperatives of cultural evolution rather than simply the action of natural selection on genetic variation.

Convincing examples of culture-driven gene-culture coevolution exist (Itan et al., 2009), and there is fair evidence that a great many more exist in the form of selective sweeps that accompany events like the transition to agricultural diets and sedentary life beginning about 11 thousand years ago (Hawks et al., 2007; Laland et al., 2010; Ross and Richerson, 2014). However, some influential evolutionary biologists and evolutionary social psychologists follow variants of Wilson's human nature argument and dismiss the possibility that culture can play a fundamental role in human evolution (André and Morin, 2011; Richerson, in press; Scott-Phillips et al., 2011). Some authors seem go so far as to rule out any significant role for culture or even individual learning in favor of genetically inherited but environmentally contingent cognitive rules (Morin, 2016; Tooby, 2015). Brown and Richerson (2014) attempt to clarify the issues involved. Many people think that in humans genes and culture are more or less equally important in the evolutionary process (Barrett, 2015; Henrich, 2016). Still other commentators argue that genes code only for rather general purpose behavioral primitives and even early emerging skills like imitation that seem to be innate specializations (Carey, 2009; Csibra and Gergely, 2011) are actually themselves cultural acquisitions (Heyes, 2016).

Disagreements on this topic persist for a simple reason. Although sequencing techniques allow us to "read" genomes in great detail, most genes are connected to phenotypes by complex networks of genes and complex developmental and regulatory circuits. The functional annotations of most genes are based on the pathological effects of rare mutants which are only a very general clue as to the function of normal alleles. The genes for which we can so far give clear evolved-functional accounts are limited to cases like skin pigmentation where few genes are involved, developmental pathways are simple, and fitness effects are large. For example, Northern Europeans came under strong selection for pale complexions as they came to depend very heavily on plant based diets poor in vitamin D (Jablonski and Chaplin, 2010; Wilde et al., 2014). The photosynthetic production of vitamin D in the skin is limited in the relatively low UV environment of the region. Earlier hunting and gathering Europeans were light brown in color but had ample vitamin D in their diets. Early farming pioneers to Northern Europe were rather brown people from Southwest Asia. Very pale complexions favoring photosynthetic vitamin D production only evolved in Northern Europe in the last 6 thousand years. Most genes and most culture are entangled in complex causal pathways built by gene-culture coevolution on the multigenerational time scale and by codevelopment on the individual lifetime scale. Thus determined skeptics of any persuasion can entertain doubts about gene-culture interactions. I think the evidence we do have points to highly interactive gene-culture coevolution in humans but there is much we do not yet know.

What about culture in other species? The last 25 years have been a golden age of for the study of non-human social learning (Heyes and Galef, 1996; Hoppitt and Laland, 2013). Comparative studies of apes, humans and other species such as fish show that humans are more skilled social learners than any other species but diverse species including other apes, and crow and parrot birds, and toothed whales stand out as skilled social learners (Heyes and Galef, 1996; Hoppitt and Laland, 2013; Tomasello et al., 2005; Whiten et al., 2011). Simpler systems of often rather specialized social learning are very widespread in social animals (Laland, 2017). It might be possible to dismiss simple social learning as non-adaptive semi-parasitic information scrounging (Rogers, 1989), but excellent cases have been made that many social learning systems are indeed adaptive (Whitehead and Rendell, 2015; Laland, 2017: chapter 4). As with the Baldwin effect in the case of individual learning, a strong case can be made that, although the human case is probably extreme, social learning is likely to have a coevolutionary impact on genetic evolution in many species. For example, orca matrilineages often have distinctive culturally transmitted feeding strategies. Whitehead (2017) and Whitehead et al., (2017) argue that the low mitochondrial genetic diversity of orcas and other toothed whales can be explained by successive matrilines discovering innovative feeding strategies that allow them to expand into a number of daughter lineages. As with the serial founder effect, the terminal living lineages will have lost diversity.

4.4. Other systems

Other systems of phenotypic flexibility are generically similar to learning and social learning. They all follow the pattern of inheriting priors genetically or culturally and using new data encountered during development to update them, and the pattern of interaction among systems. This essay is not the place to review them in detail but some issues are interesting and controversial.

4.4.1. Adaptive immune system

The vertebrate immune system is complex and heavily researched (Parham, 2014). It is divided into the innate subsystem and the adaptive sub-system. The innate system is heterogeneous, comprising such things as mechanical defenses to pathogen invasion like the skin as well as killer cells that attack invading bacteria or viruses. Some are targeted by natural selection on pathogens common in local environments, such as the many blood cell polymorphisms that protect tropical and subtropical people against malaria (Kwiatkowski, 2005). Selection for resistance to *Plasmodium falciparum* and *P. vivax* has been very strong in many human populations since the origin of agriculture around 10,000 years ago. This has given rise to many hemoglobin variants and other blood cell variants that confer resistance, the most famous of which is the sickle cell hemoglobin gene which protects heterozygotes from malaria but causes an often very severe hemolytic disease in homozygotes. Many of the blood cell polymorphisms are associated with costly diseases, a testimony to the even higher cost of malaria infections. They are, in effect, priors that malaria is an important environmental threat, but they cannot be updated quickly when their carriers live in malaria free environments. Other aspects of the innate and adaptive immune systems are also important in resistance to malaria (Gazzinelli et al., 2014).

The adaptive immune system is based on the hypervariability of immunoglobin genes. Recombination and mutation of this gene complex produces millions of variants each potentially binding a different antigen. If a particular antigen is present in the body any cells with antibodies that bind the new antigen are stimulated to reproduce, leading to the evolution of a population of cells capable of suppressing the parasite that is the source of the antigen challenge. Memory cells maintain a certain level of antibodies for a certain amount of time after the infection is cleared. In some cases, a lifelong immunity results.

Thus, the adaptive immune system, like individual learning, has inherited priors and a blind variation and selective retention system mechanism that uses data about the current environment to update those priors. The random variation in the adaptive immune system is necessary because infectious microbes and other parasites are actively evolving to evade the innate immune system. Given the short generation of the parasites, they can generally evolve faster than host genomes. The adaptive immune system responds on the same time scale as the microbes, usually more or less successfully defending the host.

Given the aforementioned high cost of immune system responses, it is no mystery why suffering from infections is aversive. Illness is data used in updating priors from the learning and social learning systems. All else equal, organisms will avoid behaviors that make them sick. Every culture seems to include a traditional medical system and such systems are efficacious enough to serve as a basis for drug development (Fabricant and Farnsworth, 2001). The most famous example is the Nobel Prize awarded in 2015 to Youyou Tu who developed artemisinin as an antimalarial drug based on its use in Chinese traditional medicine. Huffman (2008) reviews the evidence that apes and other animals have medical traditions based on individual and social learning. Successful medical treatments will in turn affect the evolution of both parasites and hosts.

In addition to the resource-based cost of the immune system, all vicarious selector-based mechanisms will generate a certain frequency of errors. This may result in random variation in the data an organism is responding to (Boyd and Richerson, 1989), from outdated priors such as tastes driving excessive consumption of nutrient-dense foods in modern environments, or from mutant priors in the adaptive immune system that lead to autoimmune diseases.

4.4.2. Developmental circuits and epigenetic inheritance

During the development of multi-cellular organisms complex regulatory circuits turn protein producing genes on and off in an intricately choreographed sequence to create the tissues and organs of the body (Schwarzer and Spitz, 2014). We have already met a very simple example in the regulatory gene prior that shuts down lactase production in mammals after weaning since most mammals will never need lactase again. Damaging this regulatory gene causes adult lactase persistence, a trait selected for in dairying populations (Tishkoff et al., 2007). In addition, developmental circuits often take environmental variation as data, apparently based on sophisticated priors. Kuzawa (2005) reviews data on the effect of maternal nutrition on birthweight and associated life history variables. Babies born to mothers with limited food intake are small but they also acquire a thrifty phenotype that can reproduce with more limited food intake than larger mothers. Even if well fed, small mothers tend to have small babies. Remarkably, there is evidence that this phenotypic adjustment integrates over generations via epigenetic inheritance. Food intake has noisy short-term variation and, probably, stronger variation, including at scales of multiple generations. A system that was sensitive to the longer time scale and that largely filtered out generation-to-generation variation would the most adaptive. Once again evolution could tune an approximately Bayesian system by adjusting the strength of priors to efficiently respond to the scale of variation in food supply that dominates the environmental data. Thus, developmental plasticity can apparently behave a lot like cultural inheritance, updating priors on a generation by generation iterative sampling of environmental data.

Developmental systems theorists argue that such transgenerational epigenetic inheritance systems are many, ubiquitous and important to phenotypic adaptation and to evolutionary processes (Jablonka and Raz, 2009; Uller, 2008). Other authors are considerably more guarded, particularly as it concerns the importance of transgenerational epigenetic inheritance in vertebrates (Grossniklaus et al., 2013; Heard and Martienssen, 2014; van Otterdijk and Michels, 2016). Much is still rather uncertain (Nestler, 2016). Most likely, the case for an Extended Evolutionary Synthesis to replace the Modern Synthesis in evolutionary biology will turn on whether transgenerational epigenetic inheritance of priors actually has an important role in the creation of adaptive variation in many if not all species (Laland et al., 2015) or whether such effects turn out to be relatively unimportant and rather constrained by priors that evolve directly by natural selection on DNA variation (Wray et al., 2014).

4.4.3. Symbionts and domesticates

All organisms live in a community of other species. Some community members are such close associates that we call them symbionts if they have a neutral or positive effect on the host and pathogens if their effect is negative. For example, all animals play host to a microbiome comprised of many species of bacteria, archaea, fungi and viruses. My discussion below is based on recent reviews by Knight et al. (2017) and Sommer and Bäckhed (2013). Recent advances in gene sequencing and parallel advances in data analysis techniques are making these complex ecosystems a hot topic for research. The voluminous and diverse gut microbiome has attracted the most interest. This microbiome seems to have, adding up all the genes an individual harbors, about 100X as many symbiont genomes as the host genome. Since microbes have a much larger range of metabolic systems than animals, it is not surprising that many species are symbiotic, producing metabolic products that the host body uses. In effect, harboring a microbiota enlarges the number of prior hypotheses about useful nutrients an organism can test against environmental data. Many of these microbes are specialized at the level of host species as well, suggesting a long history of coevolution between hosts and microbiota. The gut microbiome is very responsive to diet, health, age, and many other factors, responding, apparently adaptively, to environmental variation. Some variant gut microbiome communities are stable but others are pathological such as ones dominated by *Clostridium difficile*. *C. difficile* infections often occur after treatment with broad spectrum antibiotics that destroy the normal gut microbiome. The gut flora and its host are apparently coevolved with one another as the guts of mice raised without a gut microbiome develop abnormally. Genetic developmental priors require data from the microbiome to give rise to functional gut structures. Human infants normally acquire their initial microbiome as the pass through their mother's vagina during birth. The mother's microbiome changes considerably in the third trimester, suggesting that mothers are adapted to deliver the appropriate microbial priors to the infant at birth that are adapted to its initial all-milk diet and an untrained immune system. Infants delivered by cesarean section are slow to develop a normal microbiome and often suffer ill effects as a consequence. Microbiome composition is correlated with a large number of other host traits including non-obvious ones such as mental health. The causal pathways behind these statistical associations are mostly currently unknown.

The immune system of hosts appears to play a very important role in managing the microbiome. My discussion is based on recent reviews by Arnolds and Lozupone (2016) and by Kurilshikov et al, (2017). In the first place the immune system has a problem similar to self-non-self recognition. It needs to acquire priors to respond aggressively to pathogens but mildly, if at all, to symbionts. Pathogens will often cause inflammation, and the destruction of pathogens due to inflammation is one of the priors of the innate immune system that triggers the adaptive immune system to mount antibody defenses. Symbionts that do not cause reactions of the innate immune system seem to be recognized as safe. As with autoimmune diseases and reactions to harmless antigens such as pollen proteins, developmental abnormalities can cause this system to fail. We can imagine that the immune system has a sophisticated set of innate priors and updating mechanisms designed to monitor and manage the gut microbiome in a quite detailed way, but that speculation seems to be at least a little way beyond the current scientific frontier.

Macroscopic domesticates are rather similar to the commensal microbiota in terms of their evolutionary and phenotypic interactions with their hosts. Indeed some of our domesticates, besides our microbiota, are themselves microbes, such as the yeasts used in baking and brewing. Bio-archaeologists are very interested in the coevolution of humans and our domesticates. One of the interesting innovations in this field and related historical disciplines is the use of approximately Bayesian model-fitting algorithms (Bouckaert et al., 2012; Gerbault et al., 2014) that may provide clues to how Bayesian updating is implemented in systems of phenotypic flexibility.

4.4.4. *Homeostatic physiology*

We cannot sense many homeostatic processes, such as the regulation of blood pressure and blood glucose, without special instruments. However, many of our senses are heavily influenced by homeostatic physiology. Dysregulation gives rise to sensations of heat and cold, hunger, thirst and pain. To be deprived of breath is terrifying. Disruption of social bonds leads to fear and anger. Restoration of homeostasis leads to pleasant feelings of satiation, satisfaction, and well-being. The brain stem emotional circuits are reasonably well understood and are ancient shared features of the vertebrates (Panksepp and Biven, 2012). The emotional circuits directly trigger innate behavioral responses like panic and affiliative behavior, and they are also potent reinforcers active in shaping behavioral and cultural priors. Panksepp and Biven note that the brainstem emotion circuits do not just project into the cortical regions of the brain, they also receive projections from the cortex. Thus learning and culture readily update emotional priors. We have met the example of capcaicin switching from causing pain to causing pleasure in habitual users. Evans-Pritchard's (1940) ethnography of the pastoral Nuer of the southern Sudan notes their indifference to pain and discomfort. PTSD apparently includes an addiction-like preoccupation with stimulating elements of their combat experience in many cases (Campbell et al., 2016; Solursh, 1989). Nisbett and Cohen (1996) give a very impressive multimethod analysis of how the Southern US culture of honor works by manipulating hormone titers that are associated with fear and anger. These examples suggest that even very basic innate priors can be updated by personal experience or culture priors acting as data for genetic ones.

4.4.5. *Niche construction*

Odling-Smee et al., (2003) have argued that the inheritance of constructed niches is a third form of inheritance alongside culture and genes. The trouble is that ecological inheritance is in the first instance a material inheritance but not an easily replicated information-rich form of inheritance like genes, culture, and quite possibly epigenetic inheritance systems. Engelberg and Boyarsky (1979) characterize information in terms of the cost of replication. Genes have some matter and energy costs to replication, but a single male ejaculate or clutch of codfish eggs can have millions of copies of the parent's genome. These are information-rich structures. Niche constructions like beaver dams are massive in terms of energy and matter compared to a sperm. Because genes can be replicated cheaply, the genes leading to good beaver dams can increase in frequency fairly quickly. Take farms as a cultural example. Farms are hard work to pioneer and the modifications pioneers make are often quite valuable. Pioneer American farmers in the 18th and 19th centuries had quite large families, most of whom learned the skills to be farmers. However, only one, typically the eldest son, inherited the farm itself. If the others wished to be farmers, they had to homestead new wild land and put in the huge labor and capital investment to make it into a farm perhaps with some assistance in the form of a monetary inheritance, a bit of spare equipment or livestock, or a dowry (Judge and Blaffer Hrdy, 1992). The farm does contain some information about farming in the layout of fields and in any seeds or tools a purchaser might inherit. But it is unlikely that a naïve buyer could use this information alone to become a successful farmer. Nelson and Winter (1982) thought hard about this question in their discussion of the routines of firms. They did not completely discount the physical structure of the firm in contributing to the transmission of routines but thought that more information rich cultural transmission in the form of instruction, examples, policy and procedure manuals, shop manuals, and the like are much more important.

This objection aside, Odling-Smee and colleagues are correct that niche constructing organisms modify their environments thereby affecting genetic evolution, and providing priors and data to other modes of phenotypic flexibility. In humans, much culturally directed behavior results in niche construction. For example, humans use clothing and shelter to spread far outside the latitudes that are congenial to our basically ape physiology.

5. Summary and conclusion

The main argument of this paper is that evolution by natural selection is a Bayesian process in the sense that a populations' genes are priors about what sort of environment the population is likely to face. Gene polymorphisms represent different hypotheses. Mutations represent rare random novel hypotheses that are usually unsupported by environmental data, but occasionally receive support because they explore a new bit of design space relevant to a changing environment. The survival of and reproduction of genetic variants represents the serial updating of genetic priors. This secondary-school level account of evolution oversimplifies evolutionary and developmental processes. The complexity I have paid attention to here is the role of mechanisms of phenotypic flexibility. Environmental variability generally will favor updating systems that use environmental data to update priors as in the inducible enzymes of bacteria. In red noise environments, organisms will experience some environmental variation that is outside the range experienced in the ancestral past. Here systems of phenotypic flexibility build systems that, in effect, simulate organic evolution's acquisition of useful new hypotheses on a faster time scale. There are priors that reflect past evolution or past experience and a partly random system that can generate novel hypotheses to test. The selective processes in such systems are often higher level priors that apply rather generally across many behaviors. It is better to be well fed than starving or poisoned, though just what counts as food or poison can vary in different environments. The different systems of phenotypic flexibility are specialized in terms of the kind of data they transduce and in the way iterative updating takes place. The adaptive immune system is sensitive to microbes and other antigen-presenting threats, threats that can vary on short time and space scales due to the rapid evolution of microbes. The innate immune system adapts to threats that change over evolutionary time scales and to large scale geographical variation in threats like malaria. Individual learning is useful to adapt behavior to individual-level variation at short time and space scales. Social learning is useful to adapt to time and space scales that are intermediate between those for which genetic evolution and individual learning are useful. The different systems interact intensively. A given prior may contribute to the evaluation of many hypotheses as in the high-level general reinforcers. The data generated by different systems will often be combined to revise a given prior, as when data from own experience and from cultural practices are combined to select a tool or technique. Over the whole life course, all the systems of phenotypic flexibility will interact in what we might term a co-developing adaptive complex.

The most controversial proposal on the table is that systems of phenotypic flexibility play a leading role in evolution. The phenotypic-flexibility-first hypothesis holds that phenotypic adaptations are regularly or even always the innovative edge with genetic change accommodating the phenotypic innovations (West-Eberhard, 2005). The basic mechanisms that can link phenotypic flexibility to selection on genes are well known in the form artificial selection and sexual and other forms of social selection. I think that the evidence that cultural variation has played a leading role in human genetic evolution is strong. There is provocative evidence for other systems. Whether all this amounts to the need for an Extended Evolutionary Syntheses to replace the Modern Synthesis is partly a semantic issue. I think that the scientific issue is whether flexibility-first is pervasive in biological systems or a series of relatively uncommon special cases that can be assimilated to the Modern Synthesis theoretical system without root-and-branch reformulations of models.

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