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Restrictions on Biological Adaptation in Language Evolution

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Abstract

Language acquisition and processing are governed by genetic constraints. A crucial unresolved question is how far these genetic constraints have co-evolved with language, perhaps resulting in a highly specialized, and species-specific language "module"; and how far language acquisition and processing redeploy pre-existing cognitive machinery. Here, we aim to understand the circumstances under which genes encoding language-specific properties could co-evolve with language itself. We present a theoretical model, implemented in computer simulations, of key aspects of the interaction of genes and language. Our results show that genes for language could only have co-evolved with highly stable aspects of the linguistic environment: a fast changing linguistic environment does not provide a stable target for natural selection. Hence, a biological endowment cannot co-evolve with properties of language that began as learned cultural conventions, because cultural conventions change much more rapidly than genes. We argue that this rules out the possibility that arbitrary properties of language, including abstract syntactic principles governing phrase structure, case marking, and agreement, have been built into a "language module" by natural selection. The genetic basis of human language acquisition and processing did not co-evolve with language, but primarily pre-dates the emergence of language. As suggested by Darwin, the fit between language and its underlying mechanisms arose because language has evolved to fit the human brain, rather than the reverse.

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Introduction

The mechanisms involved in the acquisition and processing of language are closely intertwined with the structure of language itself. Children routinely acquire language with little intentional tutoring by their parents and, as adults, use language with minimal effort. Indeed, our unique and near universal capacity to acquire and use language has even been cited as one of eight key transitions in the evolution of life (1). These features of species specificity and species universality, combined with the intimate fit between language structure and the mechanisms by which it is acquired and used, point to substantial genetic constraints. However, the nature and origin of the genetic basis for language is the focus of much debate (2-4).

An influential line of thinking in the cognitive sciences suggests that the genes involved in language predetermine a highly specialized and species-specific language "module," (5) "instinct," (6) or "organ" (7). This module has been assumed to specify a number of domain-specific linguistic properties, including case marking, agreement, and conformity to highly abstract syntactic constraints, such as X-bar theory (8). While some have argued that the genes encoding a language module arose through a sudden 'catastrophic' genetic change (9), and others have remained agnostic (10), "the default prediction from a Darwinian perspective on human psychological abilities" (11: p. 16) is the adaptationist view: that genes for language co-evolved with human language itself for the purpose of communication (1, 8, 12-18).

A challenge for adaptationist accounts is to pinpoint an evolutionary mechanism by which a language module could become genetically encoded (19). The problem is that many of the linguistic properties purported to be in the language module are highly abstract and have no obvious functional basis—they cannot be explained in terms of communicative effectiveness or cognitive constraints—and have even been suggested to hinder communication (20). By analogy to the conventions of communication protocols between computers, it has been suggested that even completely arbitrary linguistic properties can have an adaptive value, if the same conventions are adopted by all the members of a speech community (12). That is, although any number of equally effective communicative "protocols" may serve equally well for communication, what matters is that everyone adopts the *same* set of culturally-mediated conventions.

The subsequent shift from initially learned linguistic conventions to genetically encoded principles, needed to evolve a language module, may appear to require Lamarckian inheritance. However, the Baldwin effect (21,22) provides a possible Darwinian solution to this challenge. Baldwin proposed that characteristics that are initially learned or developed over the life-span can become gradually encoded in the genome over many generations because organisms with a stronger predisposition to acquire a trait have a selective advantage. Over generations, the amount of environmental exposure required to develop the trait decreases, and eventually no environmental exposure may be needed: the trait is genetically encoded. A frequently cited example of the Baldwin effect is the development of calluses on the keels and sterna of ostriches (22). The proposal is that calluses are initially developed in response to abrasion where

the keel and sterna touch the ground during sitting. Natural selection then favored individuals that could develop calluses more rapidly, until callus development became triggered within the embryo, and could occur without environmental stimulation. We investigate the circumstances under which a similar evolutionary mechanism could genetically assimilate properties of language in a domain-specific module (1, 12, 13).

Simulation 1: Establishing the Baldwin Effect

We specify a model of the mutual influence of language and language genes. To give the best chance for the Baldwin effect to operate, we choose the simplest possible relation between language and genes (23). We consider a language governed by n principles, $P_1,\ldots,P_i\ldots,P_n$, which may potentially be encoded in n genes. Each principle has two variants, which we refer to as $+_L$ and $-_L$. The corresponding genes $G_1,\ldots,G_i\ldots,G_n$ have three alleles, $+_G$, $-_G$, $?_G$, two of which encode a bias towards learning, $+_L$ or $-_L$, the third being neutral.

In each generation, a population of N agents attempts to learn the principles using trial-and-error learning. Fitness is determined by the number of trials required to learn the principles. Only the f fittest agents reproduce, by sexual recombination and mutation. The genes of each "child" in the new generation are derived by randomly combining the genes of two "parents" chosen at random. Each gene has a probability m of mutation (i.e., a locus is randomly re-assigned to one of the alleles $+_G$, $-_G$, $?_G$). Thus, the population is genetically selected to be good at learning the principles of the language.

To see how the Baldwin effect might operate, suppose the ith principle in the language is $+_L$. Learners with the appropriately biased allele, $+_G$, in the ith position in the

genome will tend to learn more rapidly, and hence have a higher probability of reproducing. Thus, over generations, correctly biased alleles will predominate and learning time will approach 0: the principles of the language will have become genetically fixed. Fig. 1 illustrates that this process occurs robustly in Simulation 1. The Baldwin effect is stable across wide variations in the initial percentage of neutral alleles, group size, genome size, percentage of learners allowed to reproduce, and the strength of genetic bias. The Baldwin effect emerges reliably: neutral alleles are eliminated, and "correctly" biased alleles are established. Hence, aspects of language that are stable over evolutionary time can become genetically assimilated (13, 24)—in particular, if there is a cost associated with language learning (25, 26) and no conflicting functional pressures (27).

Simulation 2: Language Change Eliminates the Baldwin Effect

As with ostrich calluses, linguistic principles that initially arise after substantial interaction with the environment can, after a period of natural selection, develop with minimal environmental input. Yet the Baldwin effect typically applies in fixed environments (28), while simulations of biological co-evolution suggests that the Baldwin effect is eliminated in a changing environment (29). Indeed, while the stable physical environment of the ostrich provides a reliable adaptive advantage for calluses, the linguistic environment appears highly *un*stable. Arbitrary linguistic conventions, prior to genetic encoding, are, like other cultural phenomena, subject to rapid change, which is generally argued to be very much faster than genetic evolution (30-32). Indeed, in the modern era, many properties of language continue to change rapidly. For example, the

entire Indo-European language group, including Breton, Danish, Faroese, Gujarati, Hittite, Tadzik, and Waziri which exhibit huge variations in case systems, word order, and phonology, have diverged in just 10,000 years (33). Thus, the "environment" of linguistic conventions changes far more rapidly, and yields far greater diversity (34), than the typical properties of physical and biological environments to which organisms must adapt, and which typically jump between a relatively small number of states [depending on climatic variation, or the population size of key prey/predators (35)].

Can the Baldwin effect lead to the genetic assimilation of linguistic properties (such as agreement, case marking, and X-bar theory) that putatively began as cultural conventions? In particular, is this possible given that cultural conventions provide a rapidly "moving target" for any biological adaptation?

In a second set of simulations (Fig. 2A), we introduced language change [which might arise, e.g., via grammaticalization or language contact (36)], by modifying a randomly chosen principle, P_i, with probability l. Fig. 2B-F (upper panel) illustrates that the Baldwin effect is substantially reduced even when linguistic principles change at the same rate as the mutation rate, m, of the genes ($l=m$); Fig. 2B-F (lower panel) shows that when linguistic change is more rapid ($l=10m$), the Baldwin effect is eliminated. Hence, genetic commitment to specific principles is maladaptive when the language is a "moving target." The most successful learners end up with neutral alleles.

Simulation 3: Language-Gene Co-evolution

In Simulation 2, language change is governed by cultural forces, uninfluenced by genetic factors. Yet linguistic properties may also be influenced, in part, by genetic biases. If

linguistic principles co-evolve with genetic factors, this might potentially stabilize the linguistic principles, and provide a fixed target on which natural selection over language genes might operate [cf., Baldwinian niche construction (37)]. In a third set of simulations, we explore this by allowing the language at generation *t+1* to be determined by a combination of genes and language at the previous generation *t*. Specifically, for the *i*th principle at *t*+1, there is a probability *g* that this principle is determined by the genetic allele that is most prevalent at the *i*th location at *t*. Otherwise, with probability 1-*g*, the principle is not influenced by the genes, but is subject to linguistic change with probability *l*, as before. If *g* is 0, the results are as in Simulation 2. If *g* is 1, the language is "re-invented" afresh at each generation, to fit the genetic biases in the population. In the latter case, the Baldwin effect is not required to explain a putative language module, because the linguistic properties are already determined by pre-existing genes. The critical question is whether low values of *g* (i.e., moderate genetic influence) can lead to a "runaway" tendency for a certain set of principles to be adopted; and hence to provide a stable selectional pressure so that the Baldwin effect can operate. Fig. 3 shows that only when *g* is high (≈50%) does the Baldwin effect re-emerge. But this case seems implausible for most, if not all, arbitrary linguistic principles—if selection pressure on the relevant locus is mainly influenced by the genes, then this indicates that the principle has a strong pre-existing genetic basis.

Note, though, that if the genetic influence *g* is high enough to lead to the Baldwin effect (i.e., high enough to out-weigh the influence of language change), *g* is also high enough to determine language structure even in the absence of language-driven genetic

selection (this is illustrated in Fig. 4). Specifically, the degree and direction of language change is almost unaffected when language-specific genetic selection (implementing the Baldwin effect) is replaced by genetic drift. Thus, the language is determined by the genetic biases of the population at the outset, independent of selectional pressure from the language to the genome [cf. (38)]. The Baldwin effect serves merely to entrench existing genetic biases in the population.

Conclusion

Although our results show that the Baldwin effect may apply to functional properties of language (Simulation 1), it is unlikely to be the mechanism for genetic assimilation of arbitrary linguistic properties that began as learned cultural conventions (Simulations 2 and 3). Thus, a highly intricate and abstract language "module" (5), "instinct" (6) or "organ" (7) postulated to explain language acquisition (7, 39), language universals (7) and the species-specificity of human language (8) could not have arisen through biological adaptation. Indeed, this conclusion is reinforced by the observation that, had such adaptation occurred in the human lineage, these processes would have operated independently on modern human populations as they spread throughout Africa and the rest of the world during the last 100 kyr. If so, genetic populations should have co-evolved to their own language groups, leading to divergent and mutually incompatible language modules (40). Linguists have found no evidence for this (6). For example, native Australasian populations have been largely isolated for 50 kyr (31), but learn European languages readily.

To make the conditions for Baldwinian co-adaptation as favorable as possible, our model has embodied the simplest possible relationship between linguistic principles and language genes: one-to-one correspondence. The complexity of the actual many-to-many relationship between genes and behavior makes Baldwinian co-evolution even less plausible, because selectional pressures from the linguistic environment will be substantially diluted (41). Perhaps the best candidate for a "language gene" is *FOXP2* (42), damage to which has been associated with morpho-syntactic deficits (43). However, *FOXP2* damage leads to a broader developmental impairment in the form of oral-facial apraxia (44), of which linguistic difficulties may be a consequence. This gene is also associated with gut, lung and heart function (45). Hence any selectional pressures on *FOXP2* due to language processing would (a) not directly associate with a putative language module; and (b) compete with selectional pressures from essential biological processes. Indeed, current knowledge suggests that *FOXP2* is not tied to arbitrary properties of language but instead appears to influence domain-general procedural learning systems (46) and a down-stream gene, *CNTNAP2*, which in turn affects phonological short-term memory (47).

Although we have shown that arbitrary linguistic properties cannot be genetically encoded through adaptation, this does not preclude genetic adaptation to aspects of language held stable by functional pressures. For example, changes in the vocal apparatus may have arisen from functional pressures to produce more intelligible vocalization, although this point is controversial (48-50). Possible functional properties include duality of patterning (51): i.e., the presence of two levels of symbolic structure [a pool of

phonetic resources from which to compose word forms (52); an inventory of constructions from which to compose sentences (53)]; syntactic devices to express propositional attitudes (12); and, more controversially, recursion (8, 48, 54). It is possible, however, that culturally-mediated linguistic change may shield the relevant learning and processing mechanisms from adapting to selective pressures even from functional properties of language (55).

If a biologically specialized language module incorporating arbitrary constraints is ruled out, what explains the close fit between language and its underlying mechanisms? One possibility is that the properties of natural languages, while apparently capricious, arise from underlying functional considerations, i.e., the optimization of communication (56); or from non-communicative factors concerned with the optimization of the relationship between sound and meaning (20, 57). More generally, this suggests that the biology of language results primarily from exaptation, not adaptation (58-60). If so, language may to a large extent have been shaped by evolutionary processes of cultural transmission across generations of language learners (61-63). These processes include grammaticalization: the continual routinization, generalization and erosion that underlie historical patterns of language change (36). Importantly, such cultural evolution is constrained by properties of the human neural and perceptuo-motor systems, which themselves have a genetic basis largely predating the emergence of language (40).

Although our simulations indicate that some biological adaptations for functional aspects of language could have taken place, we suggest that the close fit between the structure of language and the mechanisms employed to acquire and use it primarily arose

because language has been shaped by the brain through cultural evolution. Indeed, the astonishing subtlety and diversity of patterns in human language (34) may for the most part result from the complex interaction of multiple constraints on cultural evolution, deriving from the nature of thought, the perceptuo-motor system, cognitive limitations on learning and processing, and pragmatic/communicative factors (40). Thus, as suggested by Darwin (64), the evolution of human language may be best understood in terms of cultural evolution, not biological adaptation.

Method

Set-up of simulations The simulations investigate the co-evolution of genes and language in a small population of hominids. At each generation, a population of N learners attempts to learn by trial-and-error a language governed by n principles, $P_1,\ldots,P_i\ldots,P_n$, each of which has two variations, $+_L$ and $-_L$. The corresponding genes $G_1,\ldots,G_i\ldots,G_n$ have three alleles, $+_G$, $-_G$, $?_G$, two of which encode a bias towards learning $+_L$ or $-_L$; the third is neutral. The simulations begin with x neutral alleles, where x varies between 0 and 100%; the remaining biased alleles are set randomly to $+_G$ or $-_G$. We also varied group size, N, between 24 and 250 (65); language/genome size, n, between 10 and 50; percentage of learners allowed to reproduce, f, between 26 and 74; strength of the bias, p, of $+_G/-_G$ alleles for sampling the corresponding $+_L/-_L$ principles during learning between 0.8 and 1. The mutation rate, m, was fixed to 0.01 throughout the simulations (this is rather high, from a biological point of view, but does not qualitatively effect the results). In Simulations 2 and 3, the rate of linguistic change l is either the same as the mutation rate (0.01) or a factor of ten larger (0.1). In Simulation 3, the genetic influence, g, on the language is set varied between 10% and 50%. Here and throughout, multiple runs with the same parameter values yield qualitatively similar results. Figs. 1-4 show typical, randomly chosen runs.

Learning by trial-and-error To produce the initial hypothesis concerning the target language, the learner first stochastically samples a set of candidate principles, according to its genetic biases. At locus i, a learner with a genetic bias $+_G$ will sample the $+_L$ variant of the ith principle with probability $p>0.5$ and will sample the $-_L$ variant with probability

1-*p*; and vice versa for a learner with genetic bias -$_G$. The neutral ?$_G$ allele samples +$_L$ and -$_L$ with equal probability. Initially, and throughout learning, as soon as a principle is guessed correctly, it is fixed. Learning involves sequentially resampling any incorrect candidate principles, according to the initial genetic bias, in a random order, until all principles match the target language. The fitness of the agent, and hence whether it reproduces, is determined by the total number of resamplings required for the agent to learn the entire language.

Speed of learning Learners with an allelic bias +$_G$ on the *i*th locus will rapidly fix the correct variation for this principle (with expected number of steps $1/p$); those with bias $-_G$ will typically require more samples (expectation: $1/1$-*p*); and those with variant ?$_G$ will be intermediate (expectation: $1/(1/2)=2$). Note that unless *p* is either 0 or 1, and incorrectly set, for some allele, all learners acquire the language eventually.

Reproduction by sexual recombination For all simulations presented here, sexual recombination involves randomly pairing "parent" agents, and then creating a "child" agent by, for each genetic locus, *i*, taking the allele at that locus, randomly from either parent. Mutation then occurs randomly for each gene, at each generation, with probability *m*. The reassignment takes the value +$_G$, $-_G$ and ?$_G$ with equal probability.

Acknowledgments

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Figure Legends

Fig. 1. Arbitrary linguistic principles can become genetically encoded via the Baldwin effect. (*A*) Influences across generations for language and genes. The principles, P_i, of the language, *L*, are indicated by lightly-colored yellow ($+_L$) and blue ($-_L$) squares. The corresponding biasing alleles, ($+_G$) and ($-_G$), are indicated by dark yellow and blue squares. Neutral alleles ($?_G$) are shown in green. For illustration, we show just five linguistic principles and the mean population values for genes. Here, *L*, is fixed across generations (purple double lines); *L* exerts selectional pressure on the genes, *G*, at each generation (pink arrows); and the genes of the fastest language learners are transmitted across generations (orange arrows), subject to sexual recombination and mutation. Panels (*B*)-(*F*) show the percentage of neutral alleles, plotted against number of generations, across variations in: (*B*) percentage of neutral alleles in the first generation, 0 (red), 50 (blue) and 100 (green); (*C*) group size, 24 (red), 100 (blue) and 250 (green); (*D*) genome size, 10 (red), 20 (blue) and 50 (green); (*E*) percentage of learners allowed to reproduce, 26 (red), 50 (blue) and 74 (green); (*F*) bias of $+_G/-_G$ alleles for sampling the corresponding $+_L/-_L$ principles during learning, 0.8 (red), 0.95 (blue) and 1 (green). The remaining parameters in these simulations take the default values indicated in blue.

Fig. 2. The effect of language change on the genetic encoding of arbitrary linguistic principles. (*A*) Influence across generations is as in Fig. 1, except that language also varies across generations (purple arrows). Panels (*B*)-(*F*) are as in Fig. 1, except that language changes at the same rate as genetic mutation, *l=m* (upper part of each panel), or

ten times faster, $l=10m$ (lower part). The Baldwin effect is substantially reduced in the first case, and is minimal in the second.

Fig. 3. Co-evolution of language and language genes. (*A*) Influence across generations as in Fig. 2, except that the genome, *G*, also influences the language, *L*, in the next generation (red arrows). Thus, *L* and *G* at generation *t*+1 are influenced by both *L* and *G* at generation *t*. (*B*) The percentage of neutral alleles plotted across generations. Default parameters are as in Figs. 1 and 2. Genetic influence, *g*, is varied between 10% (brown), 25% (orange), and 50% (purple). With high genetic influence, the Baldwin effect re-emerges.

Fig. 4. The shaping of language by genetic influence. (*A*) Influence across generations is as in Fig. 3, except that the genes are no longer influenced by language or any other selective pressure. The genes of randomly chosen agents are sexually recombined and mutated (orange dotted arrows), leading to genetic drift. (*B*) The percentage of $+_L$ principles in the language, as a function of number of generations. We determine the interaction between high population influence ($g = 50\%$) and prior genetic biases by varying the probability of allele reassignment during mutation. The neutral $?_G$ allele always has a probability of 1/3. The $+_G$ allele has a mutation reassignment probability of 2/3, 1/2 or 1/3. The initial proportion of $+_G$ alleles are set to be the same as the mutation reassignment probability, with the remaining parameters taking the default values from Fig. 1-3. There are three degrees of $+_G$ bias: strong (2/3, red line), medium (1/2, orange)

and no bias (1/3, blue). The prior genetic bias towards $+_G$ is reflected in the proportion of $+_L$ principles: Language becomes aligned with the genes, even when there is no selective feedback from language to genes. (*C*) The percentage of $+_G$ alleles in the population, plotted across generations. The genes follow a random walk, based on the reassignment probabilities for $+_G$. (*D*)-(*E*) For comparison, we re-ran these *B-C* simulations, but where selection for language is reintroduced (as in Fig. 3). Comparing *B-C* and *D-E*, we see that, with strong population influence, language simply converges on the initial genetic bias of the population ($+_G$), whether or not genes are selected for language (as in the Baldwin effect).

A

Genetic influence on language

Generations

Language   Genes

B

Percent Neutral Alleles

100
75
50
25
0

0   200   400   600   800   1000
Generations

A

Generations

Random
selection

Language   Genes

B   Random Selection   D   Selection for Language

Language   Language

Percent +L Principles

C   E

Genes   Genes

Percent +G Alleles

Generations   Generations