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Genomic Imprinting As a Window into Human Language Evolution

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Humans spend large portions of their time and energy talking to one another, yet it remains unclear whether this activity is primarily selfish or altruistic. Here, it is shown how parent-of-origin specific gene expression—or "genomic imprinting"—may provide an answer to this question. First, it is shown why, regarding language, only altruistic or selfish scenarios are expected. Second, it is pointed out that an individual's maternal-origin and paternal-origin genes may have different evolutionary interests regarding investment into language, and that this intragenomic conflict may drive genomic imprinting which—as the direction of imprint depends upon whether investment into language is relatively selfish or altruistic—may be used to discriminate between these two possibilities. Third, predictions concerning the impact of various mutations and epimutations at imprinted loci on language pathologies are derived. In doing so, a framework is developed that highlights avenues for using intragenomic conflicts to investigate the evolutionary drivers of language.

1. Introduction

We humans spend a large proportion of our time and energy communicating with each other.[1] We do this in a manner that is unique to us,[2] and use a learned code—language—that is distinct from all other natural signaling systems.[3] This behavior is crucial for the evolution of cumulative culture,[4] is important in humans' unusual ability to negotiate the division of labor,[5] and has been considered to constitute a major transition in evolution.[6] Over the past 30 years an abundance of literature has been generated on the evolutionary aspects of language[2,7–10] and, more recently, inroads have also been made into the genetic basis of language,[11] as well as the genetic changes associated with its evolution.[12]

However, the selection pressures that have honed our linguistic behavior remain almost entirely obscure.[13] One key unanswered question is whether our investment of time and energy into language is, overall, selfish or altruistic: that is, whether this behavior, and its requisite cognitive and anatomical machinery, provides a net benefit to the bearer at a cost to social partners, or the reverse. Typically, behavioral ecologists would seek to classify particular social behaviors either by directly measuring proxies of fitness or through phylogenetic comparisons.[14] But empirical fitness measurements present a challenge as language's fitness effects are highly dependent upon the type of social interaction within which it is employed (see **Table 1**), making it difficult to know whether all its relevant contexts had been correctly considered. Furthermore, the relative uniqueness of human communication limits the scope of traditional comparative tests. These issues and others have led some to describe the evolution of human language as "the hardest problem in science".[25]

Here we explore how the phenomenon of parent-of-origin specific gene expression, or "genomic imprinting",[26,27] may provide an alternative approach for determining whether human language is selfish or altruistic. First, we formally show that, at equilibrium, language can only be selfish or altruistic, and thus mutually beneficial or spiteful scenarios are not expected. Second, we show that an individual's maternal- and paternal-origin genes may have different evolutionary interests regarding the individual's investment into language, and that this intragenomic conflict may drive the evolution of genomic imprinting. We point out that as diametrically opposite patterns of imprinting are expected for language loci under selfish versus altruistic scenarios, this may provide fruitful avenues for empirically discriminating between these two possibilities. Third, we use these results to derive explicit predictions concerning the impact of a range of mutations and epimutations on language disorders (e.g., developmental language disorders), and how these effects manifest in parent-of-origin specific ways. This yields a conceptual framework that motivates future research activity on the social evolutionary drivers of human language and highlights those research avenues most urgently requiring further investigation.

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Table 1. Hypothesized functional uses of language.

Function	Summary	References	Social effect
Information sharing	Individuals give each other information about the environment	[15]	Altruistic
Information exchange	Individuals exchange information about the world with one another	[16]	Altruistic
Coordination	Individuals use language to coordinate joint activities, e.g. hunting/scavenging	[17]	Altruistic
Grooming and gossip	Individuals use language to facilitate group living, e.g. policing through gossip	[18]	Altruistic
Teaching	Individuals share information about tasks to decrease learning time	[19,20]	Altruistic
Manipulation	Individuals use language to disinform/manipulate others	[21,22]	Selfish
Alliance formation	Individuals use language to compete to form friendships/alliances	[23]	Selfish
Mate competition	Individuals use language to compete for mates	[24]	Selfish

These different functional uses have been proposed as potential ways that language level can feed back into fitness, and thus why language might have been selected for. Here we have assigned whether these hypotheses are marginally altruistic or selfish.

2. Is Language Selfish or Altruistic?

Natural selection adapts individuals to their environments, such that they appear designed to maximize their fitness. In social settings, the individual is adapted to maximize her "inclusive fitness", that is the total transmission of copies of her genes to the next generation.[28,29] She may achieve this either by increasing her own reproductive success (direct fitness) or alternatively by increasing the reproductive success of her genetic relatives, with whom she shares genes in common (indirect fitness). That is, genetic relatedness between social partners allows for the possibility of altruism: an individual may be favored to undertake a behavior that reduces her own reproductive success (by an amount C), so long as it gives a sufficiently large benefit (B) to social partners with whom she is sufficiently closely related (r, such that $rB > C$).[28,30]

Accordingly, when attempting to understand why humans invest the amount we do into language, we would expect that this, too, would have been shaped by natural selection to maximize inclusive fitness. Indeed, if our investment into language has been optimized by natural selection, we would expect it to have equilibrated at a level at which its direct and indirect fitness effects exactly cancel each other out (i.e., $rB = C$). Note that here the fitness effects are defined on the margin, i.e., they refer to the slope of fitness against trait value, rather than the absolute fitness consequences of the trait as a whole.[30] This admits two possibilities for the social consequences of language: investment into language is either altruistic ($C > 0$ and $B > 0$, with $rB = C$) or it is selfish ($C < 0$ and $B < 0$, with $rB = C$). Neither mutually beneficial ($C < 0$ and $B > 0$) nor mutually deleterious ($C > 0$ and $B < 0$) investment into language can be evolutionarily stable for any non-negative relatedness ($r \geq 0$). A full derivation of this point can be found in the Supporting Information.

For most communication systems the distinction between altruism and selfishness is fairly straightforward. For example, alarm calls in Belding's ground squirrels benefit other individuals at a risk to self, hence are altruistic.[31] Conversely, in a variety of bird species, begging signals benefit the signaler, while increasing risk of predation for the nest, hence are selfish.[32] However, language is used in a range of social interactions, some of which may appear relatively selfish and others relatively altruistic (Table 1). Consequently, the aggregate effect of language on social partners is not clear; indeed, this topic has been the source of much debate and confusion.[33] Discriminating between these two possibilities is key for our understanding of the evolution of language, including how and why language evolved, why it has the properties it does, and why it is used the way it is. One way to solve this problem would be to quantify and aggregate the different fitness effects of language. An alternative approach is to get natural selection to aggregate these fitness effects for us, and to record its findings in a readily readable form. We suggest that natural selection may have done just this, and, by acting differently on different elements of the genome, have left patterns of gene expression that can reveal the aggregate effects of language.

3. Inferring Selfishness versus Altruism from Imprinted Genes

3.1. Social Interactions Drive Intragenomic Conflicts

Above, we considered how natural selection shapes an individual's investment into language. This investment level however is determined by two sets of genes, one the individual inherits from her mother and the other from her father. Just as these parents may have different interests and thus come into conflict, so too may the two sets of genes that an individual receives from them. As David Haig has argued,[34] while maternal- and paternal-origin genes may be locked together in the same body, and thus share the same direct fitness, their different origins may mean they are differently related to the social partners around them and thus experience kin selection differently.

One reason that differences in relatedness between maternal- and paternal-origin genes may occur is sex-biased dispersal.[35] For example, if there is a biological, cultural, or other tendency for females to disperse to other groups before raising their own families and for males to remain in their natal group, then, on average, the children born within any particular group are likely to be more related to each other through their fathers than through their mothers. As a consequence of this, an individual's paternal-origin genes will be more closely related to the other individuals in the social group than are their maternal-origin genes. A similar result is also obtained if there

Box 1.
Relatedness through maternal-origin versus paternal-origin genes

For ancestral humans, it has been argued that, outwith the nuclear family, paternal-origin genes are likely to have been more related to social partners.[36] There are two main reasons for this: one is that humans are thought to have had predominantly female-biased dispersal, and secondly, that human males have a higher variance in reproductive success. Evidence for sex biases in these processes comes from three primary sources: phylogenetic, anthropological, and genetic.[36]

Firstly, our closest ancestors, the great apes, deviate from the typical male-biased dispersal of most mammals,[37] demonstrating a diversity of dispersal patterns.[38] Both bonobos and chimpanzees have strongly female-biased dispersal,[39,40] and in gorillas both sexes disperse,[41] with uncertainty about which sex disperses further.[42,43] As a consequence, it is thought likely that the last common ancestor of chimps and humans was either flexible in their dispersal patterns, like gorillas,[38] or had female-biased dispersal like the *Pan* clade.[44] Secondly, ethnographic studies of humans show that males have a higher reproductive skew,[45] and it was traditionally thought that humans were predominantly patrilocal too.[46] However, there is great diversity in current dispersal patterns,[47] and so anthropologists have been more equivocal on this second point, arguing that while agriculturalists are typically patrilocal,[48] nonagricultural societies—which are arguably the most appropriate to reconstruct ancestral humans—are predominantly bilocal.[48]

Finally, levels of genetic diversity on elements of the genome with different transmission patterns through males and females can be informative about sex biases in demographic processes.[49] These studies generally conclude that the effective population size of females is larger than males, and likely has been for most of human history, with the shift to agriculture associated with particularly extreme differences.[49] These extreme differences are thought to be influenced by both, a transition to patrilocality and an increase in the variance of male reproductive success.[49]

Collectively, these lines of evidence indicate that, for most of human history, there has been greater male variance in reproductive success, coupled with either equal or female-biased dispersal. The consequence of this is that, on average, paternal-origin genes are thought to have had a higher relatedness to social partners (although results for when maternal-origin genes have a higher relatedness can be found in the Supporting Information). This asymmetry in relatedness can then lead to intragenomic conflict—and the evolution of genomic imprinting—as described in the main text.

is greater male variance in reproductive success.[36] In humans, given the current knowledge of the combination of both dispersal patterns and variance in reproductive success, it appears that paternal-origin genes were, on average, more related to non-nuclear family social partners than were maternal-origin genes during the period that our linguistic behaviour was shaped by natural selection (see Box 1 and the Supporting Information for further details).

Differences in relatedness may mean that the maternal- and paternal-origin genes disagree about the phenotype the individual should express.[50] In particular, as relatedness provides the exchange rate between an individual's effect on their own fitness and on the fitness of others,[30] they will favor different levels of social traits. If relatedness is higher for paternal-origin genes, they will favor relatively altruistic behavior, while the maternal-origin genes will favor relatively selfish behavior. The direction of conflict over the phenotype between these two gene sets therefore depends on the marginal effect of that trait on social partners. Applying this to language specifically: if language is altruistic, then the paternal-origin genes will favor a larger language investment—in terms of the energy and resources allocated to language ability and activity—than will the maternal-origin genes. Conversely, if language is selfish, then the maternal-origin genes will favor a larger language investment than the paternal-origin genes. How this applies to some specific functions of language can be seen in Box 3.

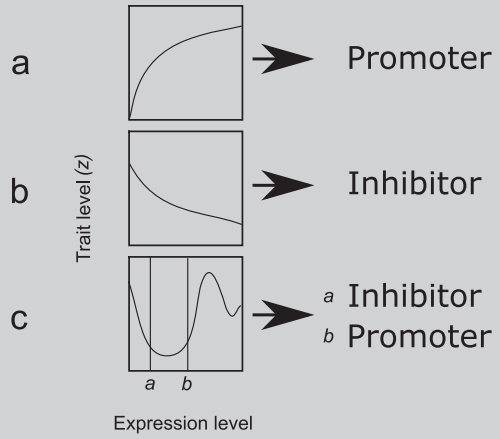
3.2. Intragenomic Conflicts Drive Genomic Imprinting

While we may talk about the different agendas of the maternal- and paternal-origin genes, these agendas are not directly visible. However, what makes this intragenomic conflict open to empirical investigation is that, according to the kinship theory of genomic imprinting,[34] these different agendas are expected to drive a difference in the expression of the two gene copies, culminating in one of the two genes being silenced.[26,27]

The reason for this is that while these gene sets have a different optimal level of language investment, they are assumed to determine that investment jointly through their combined expression levels, each gene being able to control its own expression and hence have an influence, but not full control, over the individual's overall investment into language. A gene, by modifying its level of expression based on its parent-of-origin, can push the total expression level, and thus investment, towards its personal optimum. However, each generation that gene will be pitted against its homologue from the other parent favored to do the opposite. Accordingly, over the duration of multiple generations, these two genes will try to push the total expression, and investment, in opposite directions. The conflict will escalate until the gene copy that desires the lower expression level cannot lower its expression any further—it is silenced. The higher expression copy can then set the trait level at its optimum, hence "winning" the conflict,

Box 2.
Promoters versus inhibitors

The kinship theory concerns conflict between an individual's maternal- and paternal-origin genes over the level of investment in a social trait. How that conflict over the trait then relates to conflict over the expression level at a specific locus depends on how expression from that locus affects the trait of interest, in our case language. Specifically, for the kinship theory, whether a gene is classified as a trait "promoter" or a trait "inhibitor" depends on how the marginal change in expression alters the trait of interest. If the marginal increase in expression increases the trait level then it would be classified as a promoter (panel a). Conversely, if a marginal increase in expression decreases the trait level then it would be classified as an inhibitor (panel b). Experimental manipulations have shown that the relationship between gene expression and traits can be complicated, and commonly do not show simple monotonic relationships.[57,58] In some cases, a marginal increase in the expression level may decrease the trait (panel c, expression-level *a*), but at higher expression levels may increase the trait (panel c, expression-level *b*). In such cases, whether this locus is a promoter or inhibitor will depend on the initial starting point of the conflict, most likely the optimal expression for the individual. Depending on that starting point, the locus may be classified as either a language inhibitor (panel c, expression-level *a*) or a language promoter (panel c, expression-level *b*). While direct experimental perturbation may not be possible to infer these relationships for language, associating natural variation in either copy number or expression level to the trait is an alternative way that this information about gene type could be gained.[59,60]



resulting in the locus being imprinted.[52] This outcome, whereby the gene copy that favors higher expression is expressed and the other silenced, has been termed the "loudest voice prevails" principle,[53] and has been demonstrated in both analytical models[52,54] and computer simulations.[55,56]

Which of the gene copies favors the higher expression level, and thus is expressed, depends then on the optimal level of language investment for that gene and also how expression from that locus affects language (see Box 2). For example, if increased expression from a particular locus leads to greater investment into language—a language "promoter" locus, sensu Úbeda and Gardner[36]—then the gene copy that favors the higher language level will also favor higher expression from that locus, and the gene copy that favors the lower level is predicted to fall silent. Conversely, if increased expression from a particular locus decreases language investment—a language 'inhibitor' locus—then the gene copy that favors lower language investment will favor higher expression from that locus, and the gene copy that favors the higher level is predicted to fall silent. When imprinted genes of different directions can interact, this may lead to further escalation of the conflict, potentially leading to greatly increased expressions from each locus.[61,62]

The kinship theory, then, combines information regarding the social trait type (i.e., selfishness verus altruism), relatedness asymmetries (i.e., higher via patriline versus matriline), and gene type (i.e., promoter versus inhibitor), to make a prediction about the direction of genomic imprinting at a particular locus (**Figure 1** and Box 3). Conventionally, this logic has been used to make sense of the presence and direction of imprinting at different loci, such as with the imprinted genes that affect seed size in angiosperms[63] and those that affect fetal growth in mammals.[64] Here in the case of language, there is scope to instead use the kinship theory together with patterns of imprinting, either already known or to be discovered, to make inferences about whether language is selfish or altruistic. In fact, we could use this same logic to infer any of the missing factors given a knowledge of the others.

3.3. Are There Imprinted Language Loci?

The kinship theory provides us with a method to link patterns of imprinting to the social effects of the traits they control. However, this is only possible if there are imprinted genes that affect the trait of interest. Given the potential for intragenomic conflict over language, it might be expected that all loci that affect language investment would become imprinted. But intragenomic conflict might not result in imprinting for several reasons, including: a gene lacking parent-of-origin information,[65] costs associated with imprinting,[66] or more general causes of imperfect adaptation.[56,67] Recent work indicates that there are only a few hundred imprinted genes in humans.[67] Thus, even if patterns of intragenomic conflict would be informative, without cases of imprinting, these would be less amenable to empirical investigation.

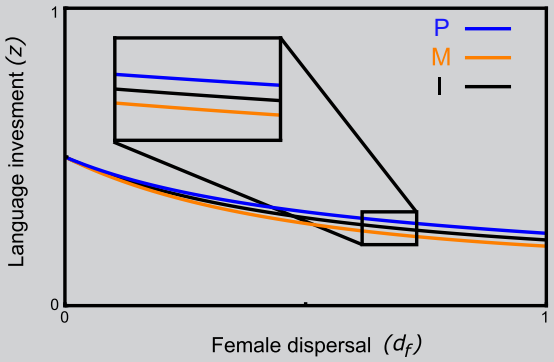
But despite the rarity of imprinting in the genome, there is evidence that some imprinted genes affect linguistic and communicative behavior.[68,69] While none of the genes that

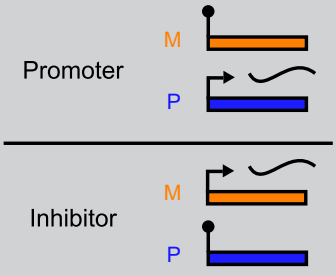
Box 3.
Mathematical models of language investment

Here we construct three different models of language function, in which individuals invest a portion of their resources into language x. Their probability of survival to adulthood $S(x, y)$ is modulated in different ways by their own investment x and the investment of social partners y. We analyze these three scenarios using the neighbor-modulated fitness approach of Taylor and Frank,[51] and identify the optimal language investment for: a maternal-origin gene (M), paternal-origin gene (P), and a gene ignorant of its origin (I). We then map the intragenomic conflict in these three scenarios into the patterns of gene expression, as predicted by the loudest-voice prevails principle[52,53] (full details in Supporting Information).

Cooperation| Language may be used to share information about the environment or about other group members or teach others new skills,

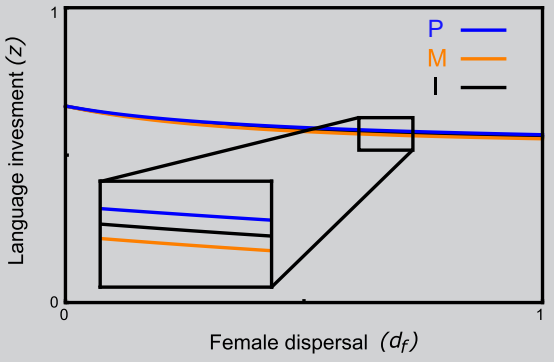
$$S(x, y) = y(1-x)$$

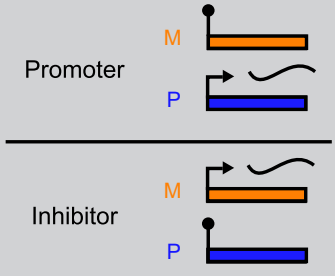




Coordination| Language may be used to coordinate actions and divide labor among group members. This may include tasks such as hunting or scavenging,

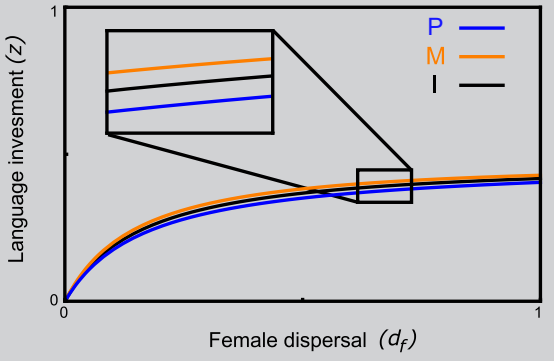
$$S(x, y) = xy(1-x)$$

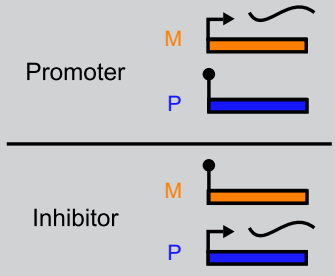




Competition| Language may be used to compete socially against other individuals for friendships, alliances, and mates,

$$S(x, y) = \frac{x}{y}(1-x)$$





have been robustly implicated in language-related disorders, e.g., *FOXP2* and *CNTNAP2*,[70] are known to be imprinted[71–73] (see the Supporting Information for a full list), there is evidence that others may be. One reason is the parent-of-origin effects identified in a number of genomic regions associated with language phenotypes, including significant paternal effects at 14q12,[74,75] suggestive maternal effects at 5p13,[75] and possible parent-of-origin effects in a chromosomal deletion in the 15q13.1–13.3 region, which might underlie different clinical manifestations for the same chromosomal rearrangement.[76] While such parent-of-origin effects can arise from processes other than imprinting,[77–79] it has been suggested that either

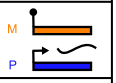
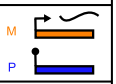
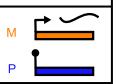
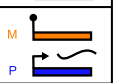
Type of social trait?	Gene type?	Predictions from the kinship theory
Altruistic	Promoter	Paternal Expression 
Altruistic	Inhibitor	Maternal Expression 
Selfish	Promoter	Maternal Expression 
Selfish	Inhibitor	Paternal Expression 

Figure 1. How social trait type and gene type together through the kinship theory make predictions about the direction of genomic imprinting. Here we have shown the cases where the paternal-origin has the higher relatedness. When the maternal-origin gene has a higher relatedness coefficient then the patterns of genomic imprinting are reversed.

imprinting or interactions with imprinted loci are the most likely explanations in these cases.[75]

A second source of evidence may be provided by language phenotypes associated with imprinting-related pathologies, because disorders with overlapping etiology can be indicative of shared pathways and genetic influences.[11] Angelman, Prader–Willi, and Beckwith–Wiedemann syndromes are disorders arising from imprinted regions,[80,81] and all three commonly demonstrate language deficits and speech problems.[82–84] In addition, mouse models of Angelman syndrome also demonstrate altered ultrasonic vocalizations,[85] indicating that there is nothing mechanical preventing something similar occurring in humans. Furthermore, autism spectrum disorder (ASD) and schizophrenia both have communication-related phenotypes.[86,87] Both have previously been linked to imprinting[88] and more recent empirical work strengthens this association.[89,90] Of particular interest for language is the *LRRC16A* gene, in which risk variants are maternally overtransmitted in cases of ASD.[89] It has been suggested that this gene may be associated with language deficits,[89] although in the original study it did not reach statistical significance.[74]

One point that emerges from the investigation of the genes that are known to underpin language is that it is likely that any gene affecting language also has some degree of pleiotropy with other behavioral or morphological phenotypes. This picture has also consistently emerged from genetic investigations into other human behaviors and psychiatric disorders.[91,92] Such pleiotropy may mean that any single gene may be involved in multiple social interactions simultaneously and contribute to fitness effects in potentially different directions. Thus, the imprinting status of any single imprinted gene could reflect selection pressures unrelated to language evolution, and potentially be misleading. So, while this method does not require that imprinted genes only affect language, it would require a comparison across multiple loci to cut through the statistical noise contributed by other phenotypes and to ascertain the overall selective pressures associated with language. Collectively, these lines of evidence, either through direct association with language-related phenotypes or through disorders manifesting associated language problems, suggest that at least a handful of genes associated with language-related phenotypes may be imprinted, enabling a test of selfishness versus altruism, across enough loci to give statistical significance to such a result.

4. Language Pathologies Provide Avenues for Empirical Testing

If there are genes whose evolution has been driven, at least in part, by selection pressures induced by language, then the above logic suggests that these should become imprinted. If, for some loci, this is the case, then it is expected to have important medical consequences, as imprinting alters both the frequency and the severity of mutations occurring at these loci.[93] These severe mutational effects are expected to be made more extreme still by interlocus conflict.[61,94] Furthermore, as these genes are expressed in a parent-of-origin manner, mutations to these genes will also have parent-of-origin effects. Previously, other imprinted genes have been implicated in a wide range of human pathologies, including growth and developmental disorders, cancers, and infertility.[93,95,96] The phenotypic consequences of mutations to imprinted genes therefore provide both a useful application of the theory to understand associated pathologies and also another avenue for empirical testing.

In particular, from predictions about patterns of imprinting under selfish and altruistic scenarios, we can make further predictions about when different molecular changes will have phenotypic effects, and, if so, in which direction they will pull the phenotype. In **Figure 2** (and in the Supporting Information), we consider both mutational and epimutational perturbations. Such perturbations may be either experimentally induced, e.g., in model organisms or cell cultures/organoids, or be naturally occurring variants in human populations. The phenotypic consequences of these mutations are then classified as either increasing the investment into language (hyperlingual), decreasing it (hypolingual), or having no effect (normal). The contrasting phenotypic consequences, and different parent-of-origin effects, for a gene deletion under selfish versus altruistic scenarios are given in Figure 2. The results for further mutations and epimutations are given in the Supporting Information.

While we can make predictions about the phenotypic effects of mutations, care is needed in mapping these to specific, known pathologies. One reason is that pathologies may arise from mutations that simultaneously affect multiple genes, for instance, many duplications or deletions.

This is particularly relevant for imprinted genes that are expected (and have been observed) to be located in clusters together.[97] Thus, while in principle a deletion or duplication of a single gene might in certain circumstances be expected to have no impact, mutational disruption of that gene might commonly be associated with simultaneous changes to other imprinted genes. Furthermore, for these predictions, the standard assumption of the kinship theory is that there is a monotonic relationship between expression level and phenotype, such that an increase or decrease in the amount of gene product from a locus will affect the phenotype in a consistent

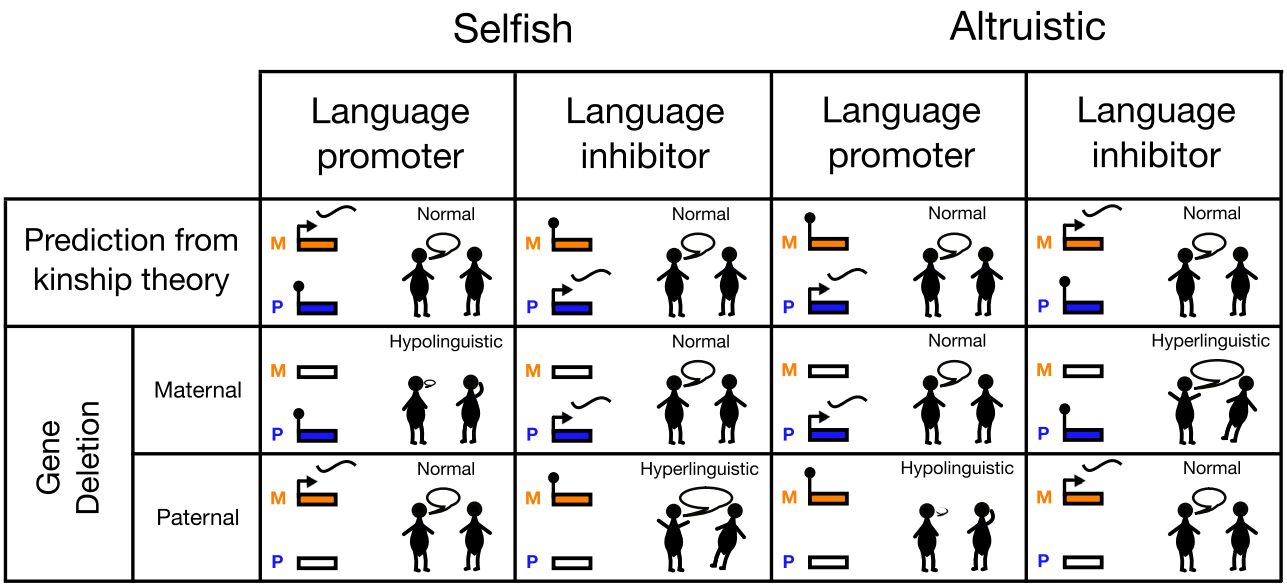


Figure 2. The gene expression patterns and phenotypic consequences of a gene deletion at an imprinted locus. This is done for a scenario where language is selfish or altruistic. A hyperlinguistic scenario is where those pathways which marginally increase language investment are overallocated, and a hypolinguistic scenario is where such pathways are underallocated. In both cases, the paternal-origin genes are assumed to be more related to social partners. Maternal-origin genes are colored orange and paternal-origin genes are blue.

manner. However, some genes have more complicated relationships between their expression levels and phenotypes,[57] and thus their effects will be less predictable, particularly with regard to extreme deviations. For instance, increasing the dosage of imprinted genes that marginally promote seed growth can, by increasing the rate of cellular division, actually make the resultant seed smaller.[98]

Nonetheless, previous examples have shown how certain pathologies associated with imprinted genes can be explained in the light of the kinship theory.[93] For example, Beckwith–Wiedemann syndrome (BWS) and Silver–Russell syndrome (SRS) are disorders that can be caused by opposite epimutations to the imprinted ICR1 region.[99] Normally, the paternal-origin copy of this region is methylated and the maternal-origin copy is not. The paternal methylation regulates the expression of two surrounding genes, *IGF2* and *H19*. Hypermethylation of this region produces two copies that have paternal-origin like methylation and expression patterns, causing BWS. Conversely, hypomethylation of the region results in two copies that have maternal-origin like methylation and expression patterns, causing SRS. In BWS, this misregulation causes overgrowth and increased risk of childhood tumors, while in SRS, there is severe intrauterine and postnatal growth retardation.[99] This pattern matches well with the expectations from the kinship theory, which predicts that, as paternal-origin genes favor higher growth in early life, an increased relative dosage of paternally expressed genes will result in overgrowth. In contrast, it is expected that, as maternal-origin genes favor greater restraint over growth, an increased relative dosage of maternally expressed genes will result in undergrowth.[64] We suggest that not only would we anticipate similar, reciprocal phenotypes for any imprinted related disorders associated with language, but, moreover, the different directions in which the two parental copies push the phenotype provide a further method to infer the agendas of these two gene copies, and thus the social effects of language.

5. A New Approach to Understanding the Social Evolutionary Drivers of Language

5.1. Using Intragenomic Conflicts to Understand Whole-Organism Adaptation

It is not clear whether human language is, on average, a selfish or altruistic trait. Here we have shown how intragenomic conflict and genomic imprinting can provide a new approach to tackling this problem, and thus provide a window into the evolutionary forces that have shaped human language. The intragenomic conflict between maternal- and paternal-origin genes offers such insights because it provides an unusually controlled natural experiment in which two gene copies value relatives differently and so push language investment in opposite directions. As the direction of this conflict is expected to manifest itself in a qualitative pattern of gene expression, genomic imprinting, it is also amenable to empirical investigation. Furthermore, empirical tests not only include the specific patterns of expression, but also the pathological consequences of mutations occurring at these loci. While here we have outlined how this approach may apply to language investment as a whole, one of the strengths of this framework is that the same logic could equally apply to distinct "language modules".

While this use of the kinship theory can be applied to many different social traits,[34] language provides a particularly useful application because, unlike other social traits such as sex allocation,[100] the selection pressures shaping this behavior

remain both obscure and highly debated.[13] This is because language affects the fitness of self and others through many different proximate mechanisms. These include not only the many different types of social interaction it mediates (see Table 1), but also the ways in which those fitness effects may be modulated by factors such as culture and social organization. Consequently, the aggregate effect of language on the fitness of self and social partners is not obvious. Yet, understanding this balance between direct and indirect fitness is key to understanding how selection has shaped our linguistic behavior over evolutionarily recent timescales. This knowledge may in turn provide others with further context in which to better understand selective scenarios surrounding the origin of language in our more distant evolutionary past. Although one way of tackling this problem would be to measure linguistic behavior[1,101–105] and link it to proxies of fitness,[106] aggregating these different uses to produce a proxy for total language investment and then linking this variation to variation in fitness would be technically challenging, and is unlikely to become easier over time.

An alternative approach, often used to tackle questions of biological adaptation, is the comparative method.[107] This has been very productive in determining the various genetic and morphological changes that have occurred in the human lineage, and in generating and testing potential explanations for them.[12,108] It can also be applied to better understand the aspects of language that are shared among ourselves and other species, including aspects of syntax,[109] speech production,[110] and turn taking.[111] However, the communicative flexibility of language enables humans to perform a range of different behaviors with it, and thus the balance of selection pressures that have shaped our level of investment are likely unique. This means that is not possible to quantitatively compare language between species, and link this to differences in relatedness, as one may be able do with other traits.[107,112] However, while we cannot make between-species comparisons, we can make comparisons between the investment strategies favored by different parts of the genome. Here we have focused on genomic imprinting and the intragenomic conflict between maternal- and paternal-origin genes, but other types of intragenomic conflict, such as that between sex chromosomes and autosomes or between nuclear and cytoplasmic genes, will also be shaped by language's effects at the level of the individual,[68] and thus provide further potential avenues for comparative investigation. These within-genome comparisons are not only useful when between-species comparisons are not possible, but offer arguably superior, more controlled, natural experiments.[113]

5.2. Assumptions and Further Questions

However, while the core logic of the kinship theory is by now well understood, its potential usefulness and appropriateness when applied to language rely on a number of assumptions that require further investigation. These include the assumptions that language is, at least in part, underpinned by imprinted genes, that the kinship theory provides the right way of thinking about why these genes are imprinted, and that our framework's molecular and demographic parameters can feasibly be empirically resolved to a degree that will enable clear-cut predictions to be made. While one could wait until there is a full understanding of these issues before attempting to develop theory on this topic, a more useful approach is to develop the theoretical and empirical research concurrently, as theory is most powerful when it is used to provide a priori predictions rather than simply post hoc explanations and when it is able to motivate and direct the empirical research along the most productive, hypothesis-driven avenues.[113–116] Thus, we believe it is worth exploring the predictions of current theory while we are at the cusp of attaining the requisite empirical data, rather than waiting until these empirical aspects are well understood.

Given the lack of imprinted genes currently known to affect language, it may seem as though this approach is unworkable. However, while much genetic and transcriptomic data have been collected, it has only been recently that we have started to gain a fuller understanding of the set of imprinted genes in humans.[67] This is partly due to the technical challenges in unambiguously determining imprinted gene expression and disentangling it from other phenomena.[117] Furthermore, imprinted genes can have complicated tissue-specific expression patterns, a feature that may mask their imprinting status. For instance, the imprinted gene *Grb10* is exclusively maternally expressed in the placenta, but then later becomes exclusively paternally expressed in the brain.[118] As a consequence, for many genes it remains unclear how extensive such parent-of-origin biases in expression may be.[119] Ongoing large-scale projects to map expression patterns in humans[120] offer opportunities to better understand the extent of both qualitatively and quantitatively imprinted genes in humans and their potentially tissue-specific behavior.

Even if we are close to a full understanding of the complement of imprinted genes in humans, it is not clear if and how expression from these loci affects language and communication. While the behavioral effects of some imprinted genes are starting to be dissected in more detail,[118,121,122] the phenotypic effects of many imprinted genes remain unclear, and thus it is not known what effect (if any) they may have on linguistic behavior. More generally, much still remains unknown about the genetic basis of linguistic behavior, and while it is known that certain aspects of the language phenotype are highly heritable,[123] the currently known variants can only explain a small portion of this.[124,125] Furthermore, the genome-wide association studies often used to identify new variants rarely incorporate parent-of-origin effects that may be required to identify the contributions of imprinted genes, although there are exceptions.[74,75] With improved statistical methods to discern different parental effects in association studies,[79,89] this may prove a fruitful avenue for investigation. Furthermore, many aspects of naturalistic linguistic behavior remain challenging to quantify, and consequently their genetic basis is almost entirely unknown. Thus, further investigation into both the phenotypic effects of known imprinted genes, as well as the incorporation of potential parent-of-origin effects into studies of complex traits, is required to better understand the potential contribution of imprinted genes to language.

If loci that are both imprinted and also affect language are known, it may still be that the kinship theory cannot be used to

infer selection pressures from their imprinting patterns. One reason is that imprinting may arise from processes other than intragenomic conflict.[126] If this is the case, then the direction of imprinting at a locus may not reflect intragenomic conflict, but instead some other selective pressure and thus inferences from the kinship theory would be misleading. While this is possible and further empirical and theoretical work is required to distinguish between these hypotheses,[127] we suggest that this is less likely. One reason is that for the imprinted genes that are well understood, the kinship theory is so far the best explanation.[128,129] In addition to having less restrictive assumptions, the kinship theory has been particularly successful in explaining many empirical patterns relating to imprinting, including the direction of imprinting, the reciprocal effects of imprinted genes, and the parent-of-origin effects on hybridization. Thus, at least currently, we would tentatively suggest that if imprinted genes are found, the kinship theory is the most likely causal explanation.

Additionally, even if imprinted genes affect language phenotypes, and indeed even if their imprinting arose due to intragenomic conflict, that does not necessarily mean that it was intragenomic conflict over language that specifically drove their parent-of-origin specific expression patterns. One reason, as mentioned above, is pleiotropy, and for this reason we suggest that studying aggregate effects across several loci may be more informative than studying individual genes in isolation. In addition to pleiotropy, while some studies indicate that imprinted genes can arise fairly rapidly in response to changes in the mating system,[130] it is not yet clear how, once imprinted genes have arisen, they may be constrained in their future evolution. It has been suggested that the imprinted genes that arose under the kinship theory would be particularly constrained due to their dosage-sensitive nature.[127] However, we currently lack formal models exploring these scenarios, and thus it is unclear to what extent the observed patterns of imprinted genes reflect current and recent intragenomic conflicts, and which others are "molecular fossils" from earlier conflicts. Thus, this potential confounding factor must be built into any analysis, and any potentially informative genes should be interpreted in a phylogenetic context as well.

Finally, our approach relies on an understanding of both patterns of relatedness (higher through patrilines versus matrilines) and the molecular biology of the genes in question (i.e., inhibitors versus promoters). While quantification of both of these factors is feasible in principle, there may currently remain ambiguity (and indeed controversy) about both the demographic parameters and also the molecular biology of specific genes. While ambiguity surrounding them does not invalidate the logic we have outlined, it does make conclusions stemming from it more ambiguous. Thus, greater work is needed to clarify these factors, and quantify the degree of uncertainty surrounding them. In particular, sex-biased demographic factors, such as dispersal, are known to vary across human societies.[47] Although we have suggested that under most scenarios paternal-origin genes are still likely to be more related to social partners (see Box 1), there may be cases where this does not hold. If so, then this opens up the potential for interesting comparative tests to be done between populations, although earlier caveats remain.

While links between the kinship theory and language evolution have been previously identified by a number of authors,[68,69,131] they typically have exclusively focused on interactions within the nuclear family and on language promoter loci. Perhaps owing to this, they have primarily focused on how genomic imprinting may have shaped the evolution of language, and thus how language itself may be an adaptation on the part of conflicting genes to divert contested resources from one social partner to another. In contrast, we have incorporated a more general set of social interactions, which can extend beyond the nuclear family (although the model could be parameterized in such a way to focus solely on this). Moreover, by considering both inhibitors and promoters, we would expect the evolutionary dynamics of language-promoter and -inhibitor loci to more or less balance out at the individual organism's optimum.[34,132] Accordingly, we emphasize that the most salient consequences of the intragenomic conflict lie in the patterning of the genome and in the maladaptive clinical pathologies associated with mutational and epimutational disruptions. Thus, we have instead argued that the logic of the kinship theory can be best used to generate strong empirical tests about the evolutionary pressures shaping language, rather than itself providing a new hypothesis for why language evolved.

6. Conclusions and Outlook

Over the next few years, it is likely that large RNA-sequencing projects will further underline the extent of genomic imprinting both in humans and other organisms. Moreover, new statistical techniques and larger datasets are likely to improve our understanding of the key loci that underpin human language adaptations. Here we have shown how these new data, when interpreted in the light of the kinship theory, can offer strikingly new avenues for tackling key problems concerning the evolution of language. Furthermore, we have shown how pathologies stemming from imprinted language loci can also be rationalized using this same logic, and thus be used as a further means of empirical testing. Finally, as the kinship theory is not exclusive to humans, these general methods may also be extended to investigate social evolutionary questions across a range of organisms in which genomic imprinting exists, including other mammals, arthropods, and angiosperms. More generally, we have highlighted how intragenomic conflicts offer relatively underexplored ways in which new molecular data may be leveraged to ask, and answer, fundamental questions about organismal adaptation.

Supporting Information

Supporting Information is available from the Wiley Online Library or from the author.

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Conflict of Interest

The authors declare no conflict of interest.

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Supporting Information

General analysis of language investment

The relative fitness $W(x, y, z)$ of an individual can be expressed as a function of their own investment x into language, their social partners' average investment y, and the population average investment z. We consider a locus which affects language investment, and denote by g the genic value of a gene drawn at random from the population at this locus. The condition for natural selection to favour an increase in the level of language investment is $\mathrm{d}W/\mathrm{d}g > 0$, where the derivative is evaluated at the population average genic value [1]. Using the chain rule, we can rewrite the LHS of this condition as:

$$\frac{\mathrm{d}W}{\mathrm{d}g} = \left(\frac{\partial W}{\partial x} \times \frac{\mathrm{d}x}{\mathrm{d}G} \times \frac{\mathrm{d}G}{\mathrm{d}g}\right) + \left(\frac{\partial W}{\partial y} \times \frac{\mathrm{d}y}{\mathrm{d}G'} \times \frac{\mathrm{d}G'}{\mathrm{d}g}\right) \quad (1)$$

where $G = \tau g_M + (1-\tau) g_P$ is the breeding value of the focal individual, g_M is the genic value of the individual's maternal-origin gene, g_P is the genic value of the individual's paternal-origin gene, and τ modulates the relative control these two parties have over the phenotype, $G' = \tau g'_M + (1-\tau) g'_P$ is the average breeding value of social partners, g'_M is the average genic value of a social partner's maternal-origin gene, g'_P is the average genic value of a social partner's maternal-origin gene, and $dx/dG = dy/dG' = \gamma$ describes the mapping of genotype to phenotype. The consanguinity of a gene chosen at random from the population to the individual she is in is $dG/dg = p$, and the consanguinity this gene to her social partners is $dG/dg = p'$. Evaluating all the derivatives at the population average, $x = y = z$, then we may rewrite the condition for increase $dW/dg > 0$ in the form of Hamilton's rule:

$$-C(z) + B(z) r > 0 \quad (2)$$

Where $C(z) = -\partial W/\partial x$ is the marginal direct fitness cost of increased investment into language, $B(z) = -\partial W/\partial y$ is the marginal indirect benefit of increased investment into language, and $r = p'/p$ is the kin selection coefficient of relatedness [2].

To investigate the agendas of a maternal-origin gene, paternal-origin gene, and the individual as whole, we alter the weighting τ of the maternal-origin and paternal-origin contributions to the breeding value [3]. If we assign full control to the maternal-origin gene then $\tau = 1$, if we assign control to the paternal-origin gene then $\tau = 0$, and for the

32 individual where control is shared equally then $\tau = 1/2$. We can calculate the
33 consanguinities to self and social partners in these three cases. The consanguinity to self p
34 is:

$$\frac{\mathrm{d}G}{\mathrm{d}g} = \tau\frac{\mathrm{d}g_M}{\mathrm{d}g} + (1-\tau)\frac{\mathrm{d}g_P}{\mathrm{d}g} =$$
35 $$\tau\left(\frac{1}{2}+\frac{1}{2}\frac{\mathrm{d}g_M}{\mathrm{d}g_P}\right) + (1-\tau)\left(\frac{1}{2}+\frac{1}{2}\frac{\mathrm{d}g_P}{\mathrm{d}g_M}\right) =$$
$$\tau\left(\frac{1}{2}+\frac{1}{2}\varphi\right) + (1-\tau)\left(\frac{1}{2}+\frac{1}{2}\varphi\right) = \frac{1+\varphi}{2} = p_s \quad (3)$$

36 And the consanguinity to social partners p' is:

$$\frac{\mathrm{d}G'}{\mathrm{d}g} = \tau\frac{\mathrm{d}g'_M}{\mathrm{d}g} + (1-\tau)\frac{\mathrm{d}g'_P}{\mathrm{d}g} =$$
37 $$\tau\frac{\mathrm{d}g'_M}{\mathrm{d}g_M}\left(\frac{1}{2}+\frac{1}{2}\varphi\right) + (1-\tau)\frac{\mathrm{d}g'_P}{\mathrm{d}g_P}\left(\frac{1}{2}+\frac{1}{2}\varphi\right) = p_X \quad (4)$$

38 Where φ is the inbreeding coefficient. The value of τ has no impact on the consanguinity to
39 self, such that $p_s = p_M = p_P = p_I$. Conversely, the consanguinities to social partners is
40 dependent on the value of τ. When we assign control of the phenotype to the maternal-
41 origin genes:

42 $$\left.\frac{\mathrm{d}G'}{\mathrm{d}g}\right|_{\tau=1} = \frac{\mathrm{d}g'_M}{\mathrm{d}g_M}\left(\frac{1}{2}+\frac{1}{2}\varphi\right) = p'_M \quad (5)$$

43 When we assign control of the phenotype to the paternal-origin genes:

44 $$\left.\frac{\mathrm{d}G'}{\mathrm{d}g}\right|_{\tau=0} = \frac{\mathrm{d}g'_P}{\mathrm{d}g_P}\left(\frac{1}{2}+\frac{1}{2}\varphi\right) = p'_P \quad (6)$$

45 And when we assign joint control of the phenotype:

46 $$\left.\frac{\mathrm{d}G'}{\mathrm{d}g}\right|_{\tau=1/2} = \tau p_M + (1-\tau)p_P = p'_I \quad (7)$$

47 Thus the relatedness coefficients are: $r_M = p'_M/p_s$ for a maternal-origin controlled locus,
48 $r_P = p'_P/p_s$ for a paternal-origin gene controlled locus, and $r_I = p'_I/p_s$ for a locus under
49 joint control. The relatedness for the individual is the average of the relatedness for a
50 maternal-origin and paternal-origin controlled locus:

51 $$r_I = \frac{p'_I}{p_s} = \frac{\tau p_M + (1-\tau)p_P}{p_s} = \tau r_M + (1-\tau)r_P = \frac{r_M + r_P}{2} \quad (8)$$

52 We can rewrite the LHS of equation 2, where ρ can represent any relatedness coefficient,
53 including the three previously defined (r_M, r_P, r_I):

$$-C(z) + B(z)\rho \quad (9)$$

We also define a function $J(z^*, \rho) = -C(z^*) + B(z^*)\rho$, where the value z^* is a convergent stable equilibrium [4, 5]. We can consider this to be a local inclusive fitness optimum. To be a convergent stable point, it must satisfy the condition $J(z^*, \rho) = 0$ [5], and hence we can write:

$$\frac{\mathrm{d}J}{\mathrm{d}\rho} = \frac{\partial J}{\partial \rho} + \frac{\partial J}{\partial z^*} \times \frac{\mathrm{d}z^*}{\mathrm{d}\rho} = 0 \quad (10)$$

Which we can rearrange to:

$$\frac{\mathrm{d}z^*}{\mathrm{d}\rho} = -\frac{\partial J/\partial \rho}{\partial J/\partial z^*} \quad (11)$$

It must also satisfy the condition $\partial J/\partial z^* < 0$, hence:

$$S\left(\frac{\mathrm{d}z^*}{\mathrm{d}\rho}\right) = S\left(\frac{\partial J}{\partial \rho}\right) = S\big(B(z)\big) \quad (12)$$

Furthermore, $J(z^*, \rho) = -C(z^*) + B(z^*)\rho = 0$. Provided we assume that the relatedness coefficient is positive ($\rho > 0$), then:

$$S\big(B(z)\big) = S\big(C(z^*)\big) \quad (13)$$

We can interpret this as meaning that at a convergent stable equilibrium, the signs of the marginal effect on self and on social partners must oppose one another. According to Hamilton's classification of social behaviours [6], this means that the optimal language investment must be either: selfish, in which case the gene copy with lower relatedness will favour a higher level of the trait; or altruistic, in which case the gene copy with the higher relatedness will favour a higher level of the trait [7, 8].

Full set of predictions from the kinship theory

Higher relatedness?	Trait type?	Gene type?	Prediction from kinship theory
Matrilines	Altruistic	Promoter	Maternal expression
Matrilines	Altruistic	Inhibitor	Paternal expression
Matrilines	Selfish	Promoter	Paternal expression
Matrilines	Selfish	Inhibitor	Maternal expression
Patrilines	Altruistic	Promoter	Paternal expression
Patrilines	Altruistic	Inhibitor	Maternal expression
Patrilines	Selfish	Promoter	Maternal expression

Patrilines	Selfish	Inhibitor	Paternal expression

Illustrative models of language function

To illustrate how this general analysis applies to specific ideas about language function, we have constructed three variations of a Haldane's tribe splitting model [9]. In this model, the population exists as an infinite series of patches [10] in which adults give rise to a large number of offspring. Those offspring then develop into adults, with their probability of survival to adulthood modulated by their, and their social partners, investment into language. The tribe in each patch then splits into smaller tribes, or buds. These buds may disperse, before competing against other buds for breeding spots, individuals may then migrate before reproducing [11]. An illustration of this life cycle can be seen in Supplementary Figure 1.

The probability of a juvenile surviving to adulthood $S(x, y)$ is a function of the language investment of the focal individual x, and their social partners y. In the three models, this is the aspect which changes, with the survival modulated differently to represent a scenario where language is a tool of: cooperation, coordination, or competition. To analyse these, we follow Taylor and Frank and assume vanishingly small variation around the population average, enabling us to use their calculus based method [1].

The fitness of a female in this population is determined by: her probability of survival to adulthood $S(x, y)$, the average fecundity k, and her probability of finding a breeding spot $F_f(x, y, z)$. For simplicity, in this model we have assumed that the size of a bud is equal to the number of available breeding spots in a patch, hence competition for breeding spots is between buds. As individual females compete for breeding spots in buds, then the probability of finding a breeding spot is inversely proportional to the number of other buds that the focal bud is competing against. The probability of a bud remaining in its natal patch is $1 - d_b$, and of dispersing being d_b. The average fitness of a female in the population is $\overline{w}_f = k$, so we can write the relative fitness for a female in the population as [11, 12]:

$$W_f = S(x, y)\left[\frac{1 - d_b}{(1 - d_b)S(y, y) + d_b S(z, z)} + \frac{d_b}{S(z, z)}\right] \quad (14)$$

The fitness of a male in the population is determined by: his probability of survival to adulthood $S(x,y)$, the average fecundity of females k, and the proportion of those breeding females that our focal individual mated with $F_m(x,y,z)$. The total proportion of those offspring which are his F_m is the product of his mating success q, which is given by the proportion of the adult males to adult females in a patch, and the probability that a given female finds a breeding spot. The mating success, in both the natal and in the average patch, is simply the sex ratio of the patch, as buds do not form or disperse in a manner dependent on sex. We assume for mechanical purposes that there is at least one male and one female in each bud. This means that mating success of our focal male q_n is equal to that of the average male $\bar{q}$, $q_n = \bar{q}$. The average fitness for all males in the population is $\overline{w}_m = k$. Hence the relative fitness for a male is:

$$W_m = S(x,y)\left[\frac{(1-d_b)}{(1-d_b)S(y,y)+d_bS(z,z)}+\frac{d_b}{S(z,z)}\right] \quad (15)$$

In a class structured population, the contribution of each class to a gene's expected fitness is given by that class' reproductive value [13]. Here, the class reproductive value for males and females are both ½ , hence[1]:

$$W = c_fW_f + c_mW_m = \frac{1}{2}W_f + \frac{1}{2}W_m \quad (16)$$

Inserting equations 14 and 15 into this and simplifying gives:

$$W = S(x,y)\left[\frac{(1-d_b)}{(1-d_b)S(y,y)+d_bS(z,z)}+\frac{d_b}{S(z,z)}\right] \quad (17)$$

If we let:

$$\left.\frac{\partial S}{\partial x}\right|_{x=y=z} = -c \quad (18)$$

And:

$$\left.\frac{\partial S}{\partial y}\right|_{x=y=z} = b \quad (19)$$

From this we can get the partial derivative with respect to individual language investment:

$$\left.\frac{\partial W}{\partial x}\right|_{x=y=z} = -\frac{c}{S(z,z)} \quad (20)$$

And the partial derivative with respect to neighbours' language investment [14]:

$$\left.\frac{\partial W}{\partial y}\right|_{x=y=z} = \frac{b-a(b-c)}{S(z,z)} \quad (21)$$

Where $a = (1 - d_b)^2$ is the 'scale of competition' [14–16].The condition the trait to increase is:

$$\left.\frac{\partial W}{\partial x}\right|_{x=y=z} + r\left.\frac{\partial W}{\partial y}\right|_{x=y=z} > 0 \tag{22}$$

Which is equivalent to [14]:

$$\frac{-c + r\big(b - a(b-c)\big)}{S(z,z)} > 0 \tag{23}$$

Cooperation:

Language has been suggested to be a tool to share information about the world to others. These hypotheses also indicate that the information is valuable and improves an individual's fitness. To represent this in our model, an individual pays a cost to invest into language $(1 - x)$, they then gain a multiplicative benefit from the investment into language from others y. Hence, we represent a juvenile's survival to adulthood as:

$$S(x, y) = y(1 - x) \tag{24}$$

Giving the condition for increase:

$$\frac{-z + r\big((1-z) - a(1-2z)\big)}{z(1-z)} > 0 \tag{25}$$

To determine the boundary conditions, let:

$$f(z) = \frac{-z + r\big((1-z) - a(1-2z)\big)}{z(1-z)} \tag{26}$$

At the lower boundary:

$$\lim_{z \to 0^+} f(z) = +\infty \tag{27}$$

Hence the trait will increase. And as we approach the upper boundary:

$$\lim_{z \to 1^-} f(z) = -\infty \tag{28}$$

Hence the trait will decrease. Any intermediate optima will be reached at $f(z) = 0$, which gives us:

$$z^* = \frac{r(1-a)}{1 + r(1-a)} \tag{29}$$

This point is convergent stable as $f'(z^*) < 0$.

Coordination:

Language has also been suggested to be a tool which enables individuals to coordinate actions, such as hunting or scavenging. In this case individuals pay a cost to invest into language $(1-x)$, but get a multiplicative benefit xy from both their investment and the average investment of social partners. Hence, we can write the survival function as:

$$S(x,y) = xy(1-x) \tag{30}$$

Giving the condition for increase as:

$$\frac{z-2z^2+r\left(z-z^2-a(2z-3z^2)\right)}{z^2(1-z)} > 0 \tag{31}$$

Again, we define a function to determine the boundary conditions.

$$f(z) = \frac{z-2z^2+r\left(z-z^2-a(2z-3z^2)\right)}{z^2(1-z)} \tag{32}$$

At the lower boundary:

$$\lim_{z\to 0^+} f(z) = +\infty \tag{33}$$

Hence the trait will increase. And as we approach the upper boundary:

$$\lim_{z\to 1^-} f(z) = -\infty \tag{34}$$

Hence the trait will decrease. Any intermediate optima will be reached at $f(z) = 0$:

$$z^* = \frac{1+r(1-2a)}{2+r(1-3a)} \tag{35}$$

This point is convergent stable as $f'(z^*) < 0$.

Competition

Language has also been suggested to serve as a tool with which individuals can compete against others in their group for status, allegiances, or mates. To represent this, an individual pays a cost to invest into language $(1-x)$, they then gain a multiplicative benefit of their investment relative to the average investment of social partners x/y. This gives us the survival function:

$$S(x,y) = \frac{x}{y}(1-x) \tag{36}$$

And thus, the condition for increase is:

$$\left[\frac{1-2z}{z} + r\left(\frac{z(1-z)}{z^2} - a\left(\frac{z(1-z)}{z^2} + \frac{1-2z}{z}\right)\right)\right] \times (1-z) > 0 \tag{37}$$

To determine the boundary conditions, we define a function:

$$f(z) = \left[\frac{1-2z}{z} + r\left(\frac{z(1-z)}{z^2} - a\left(\frac{z(1-z)}{z^2} + \frac{1-2z}{z}\right)\right)\right] \times (1-z) \quad (38)$$

At the lower boundary:

$$\lim_{z\to 0^+} f(z) = +\infty \quad (39)$$

Hence the trait will increase. And as we approach the upper boundary:

$$\lim_{z\to 1^-} f(z) = -\infty \quad (40)$$

Hence the trait will decrease. Any intermediate optima will be reached at $f(z) = 0$.

$$z^* = \frac{1-r}{2-r(a+1)} \quad (41)$$

This point is convergent stable as $f'(z^*) < 0$.

Relatedness

To calculate consanguinity and relatedness, we assume that the consanguinity coefficients have reached their quasi-equilibrium values which is a reasonable assumption if selection is weak [17]. The consanguinity of an individual to herself is defined by drawing two genes at random from the same locus with replacement and is equal to the probability that these two genes are identical by descent [2]. For a diploid individual in a sexual population, the probability of the same gene being drawn twice (either the maternal or paternal copy) is 1/2. In this scenario, they are identical by descent (IBD) with probability 1. With probability 1/2, the maternal origin and the paternal origin gene are both drawn, in this case the genes are IBD with probability φ, where φ is the consanguinity of mating partners (inbreeding coefficient). Hence consanguinity to self is:

$$p_S = \frac{1}{2} + \frac{1}{2}\varphi \quad (42)$$

The inbreeding coefficient is:

$$\varphi = \left(1-d_f\right)(1-d_m)p_X \quad (43)$$

The coefficient of consanguinity to another individual in the same patch via the maternal-origin gene is:

$$p'_M = \frac{1}{2}\left[\alpha p_S + (1-\alpha)(1-d_f)^2 p_X\right] + \frac{1}{2}\varphi \quad (44)$$

And via a paternal-origin gene it is:

$$p'_P = \frac{1}{2}[\beta p_S + (1-\beta)(1-d_m)^2 p_X] + \frac{1}{2}\varphi \quad (45)$$

Where α is the probability of two individuals in the same patch having the same mother, and β is the probability of two individuals in the same patch having the same father. The consanguinity of a focal individual to their neighbours can then be given by:

$$p_X = \frac{1}{2}(p'_M + p'_P) \tag{46}$$

We can put into equations 44 and 45 into this, and then rearrange to give:

$$p_X = \frac{\alpha + \beta}{8 - 2(1-\alpha)(1-d_f)^2 - (4+\alpha+\beta)(1-d_f)(1-d_m) - 2(1-\beta)(1-d_m)^2} \tag{47}$$

Using this we can then get an equation for the consanguinity to self:

$$p_s = \frac{4 - (1-\alpha)(1-d_f)^2 - (1-\beta)(1-d_m)^2 - 2(1-d_f)(1-d_m)}{8 - 2(1-\alpha)(1-d_f)^2 - (4+\alpha+\beta)(1-d_f)(1-d_m) - 2(1-\beta)(1-d_m)^2} \tag{48}$$

Putting these two equations back into equations 44 and 45 we get:

$$p'_M = \frac{1}{2}\frac{4\alpha + [\beta(1-\alpha)(1-d_f)^2 + (\beta-\alpha)(1-d_f)(1-d_m) - (1-\beta)\alpha(1-d_m)^2]}{8 - 2(1-\alpha)(1-d_f)^2 - (4+\alpha+\beta)(1-d_f)(1-d_m) - 2(1-\beta)(1-d_m)^2} \tag{49}$$

$$p'_P = \frac{1}{2}\frac{4\beta + \left[\alpha(1-\beta){l_m}^2 + (\alpha-\beta)l_f l_m - \beta(1-\alpha){l_f}^2\right]}{8 - 2(1-\alpha)(1-d_f)^2 - (4+\alpha+\beta)(1-d_f)(1-d_m) - 2(1-\beta)(1-d_m)^2} \tag{50}$$

Giving us the consanguinities through a maternal-origin and paternal-origin gene in terms of our demographic parameters. Relatedness can be calculated by dividing the consanguinity to a social partner by the consanguinity to self.

$$r_X = \frac{p_X}{p_s} \tag{51}$$

And thus, the relatedness through a maternal-origin gene is:

$$r_{XM} = \frac{p'_M}{p_s} = \frac{1}{2}\frac{\alpha + \frac{1}{4}[\beta(1-\alpha)(1-d_f)^2 + (\beta-\alpha)(1-d_f)(1-d_m) - \beta(1-\alpha)(1-d_m)^2]}{1 - \frac{1}{4}[(1-\alpha)(1-d_f)^2 + 2(1-d_f)(1-d_m) + (1-\beta)(1-d_m)^2]} \tag{52}$$

And through a paternal-origin gene is:

$$r_{XP} = \frac{p'_P}{p_s} = \frac{1}{2}\frac{\beta + \frac{1}{4}[\alpha(1-\beta)(1-d_m)^2 + (\alpha-\beta)(1-d_f)(1-d_m) - \beta(1-\alpha)(1-d_f)^2]}{1 - \frac{1}{4}[(1-\alpha)(1-d_f)^2 + 2(1-d_f)(1-d_m) + (1-\beta)(1-d_m)^2]} \tag{53}$$

The imprinting status of current genes associated with language phenotypes

We took the set of genes that had been highlighted in previous reviews as being identified with language phenotypes [18, 19], we then compared these against current databases of imprinted or putatively imprinted genes in humans [20–22]. These were then identified as either being imprinted/putatively imprinted (Y), or not known/not imprinted (N). Of the set we used, none were currently known to be imprinted.

Gene name	Chromosome location	Uniprot protein name	Ensembl ID	Otago	Gene-imprint	Meta-imprint
ABCC13	21q11.2	Putative ATP-binding cassette sub-family C member 13	ENSG00000243064	N	N	N
AP4E1	15q21.2	Adaptor related protein complex 4 subunit e 1	ENSG00000081014	N	N	N
ATP2C2	16q24.1	Calcium-transporting ATPase type 2C member 2	ENSG00000064270	N	N	N
AUTS2	7q11.22	Autism susceptibility gene 2 protein	ENSG00000158321	N	N	N
BCL11A	2p16.1	B-cell lymphoma/leukemia 11A	ENSG00000119866	N	N	N
BCL11A (CTIP1)	2p16.1	B-cell lymphoma/leukemia 11A	ENSG00000119866	N	N	N
CAMK4	5q22.1	Calcium/calmodulin-dependent protein kinase type IV	ENSG00000152495	N	N	N
CAND1	12q15	Cullin-associated NEDD8-dissociated protein 1	ENSG00000111530	N	N	N
CCDC136/ FLNC	7q32.1	Coiled-coil domain-containing protein 136	ENSG00000128596	N	N	N
CMIP	16q23.2	C-Maf-inducing protein	ENSG00000153815	N	N	N
CNTNAP2 (CASPR2)	7q35	Contactin-associated protein-like 2	ENSG00000174469	N	N	N
COL4A2	13q34	Collagen alpha-2(IV) chain	ENSG00000134871	N	N	N
CTNND2	5p15.2	Catenin delta-2	ENSG00000169862	N	N	N
DAPK3	19p13.3	Death-associated protein kinase 3	ENSG00000167657	N	N	N
DCDC2	6p22.3	Doublecortin domain-containing protein 2	ENSG00000146038	N	N	N
DNAAF4 (DYX1C1)	15q21.3	Dynein assembly factor 4, axonemal	ENSG00000256061	N	N	N

DOCK4	7q31.1	Dedicator of cytokinesis protein 4	ENSG00000128512	N	N	N
ERC1 (ELKS)	12p13.33	ELKS/Rab6-interacting/CAST family member 1	ENSG00000082805	N	N	N
FGF18	5q35.1	Fibroblast growth factor 18	ENSG00000156427	N	N	N
FOXP1	3p13	Forkhead box protein P1	ENSG00000114861	N	N	N
FOXP2	7q31.1	Forkhead box protein P2	ENSG00000128573	N	N	N
GCFC2 (C2ORF3)	2p12	GC-rich sequence DNA-binding factor 2	ENSG00000005436	N	N	N
GNPTAB	12q23.2	N-acetylglucosamine-1-phosphotransferase subunits alpha/beta	ENSG00000111670	N	N	N
GNPTG	16p13.3	N-acetylglucosamine-1-phosphotransferase subunit gamma	ENSG00000090581	N	N	N
GRIN2A (NR2A)	16p13.2	Glutamate receptor ionotropic, NMDA 2A	ENSG00000183454	N	N	N
IMMP2L	7q31.1	Mitochondrial inner membrane protease subunit 2	ENSG00000184903	N	N	N
INSC	11p15.2	Protein inscuteable homolog	ENSG00000188487	N	N	N
KIAA0319	6p22.3	Dyslexia-associated protein KIAA0319	ENSG00000137261	N	N	N
MRPL19	2p12	39S ribosomal protein L19, mitochondrial	ENSG00000115364	N	N	N
NAGPA	16p13.3	N-acetylglucosamine-1-phosphodiester alpha-N-acetylglucosaminidase	ENSG00000103174	N	N	N
NDST4	4q26	Bifunctional heparan sulfate N-deacetylase/N-sulfotransferase 4	ENSG00000138653	N	N	N
NFXL1	4p12	NF-X1-type zinc finger protein NFXL1	ENSG00000170448	N	N	N
NOP9	14q12	Nucleolar protein 9	ENSG00000196943	N	N	N
PCDH11X	Xq21.31	Protocadherin-11 X-linked	ENSG00000102290	N	N	N
PCDH11Y	Yp11.2	Protocadherin-11 Y-linked	ENSG00000099715	N	N	N
PLCL1 (PRIP)	2q33.1	Inactive phospholipase C-like protein 1	ENSG00000115896	N	N	N
RBFOX2	22q12.3	RNA binding protein fox-1 homolog 2	ENSG00000100320	N	N	N
ROBO1	3p12.3	Roundabout homolog 1	ENSG00000169855	N	N	N

ROBO2	3p12.3	Roundabout homolog 2	ENSG00000185008	N	N	N
SCN11A	3p22.2	Sodium channel protein type 11 subunit alpha	ENSG00000168356	N	N	N
SETBP1	18q12.3	SET-binding protein	ENSG00000152217	N	N	N
SRPX2	Xq22.1	Sushi repeat-containing protein SRPX2	ENSG00000102359	N	N	N
TBR1	2q24.2	T-box brain protein 1	ENSG00000136535	N	N	N
TM4SF20	2q36.3	Transmembrane 4 L6 family member 20	ENSG00000168955	N	N	N
ZNF277	7q31.1	Zinc finger protein 277	ENSG00000198839	N	N	N
ZNF385D	3p24.3	Zinc finger protein 385D	ENSG00000151789	N	N	N

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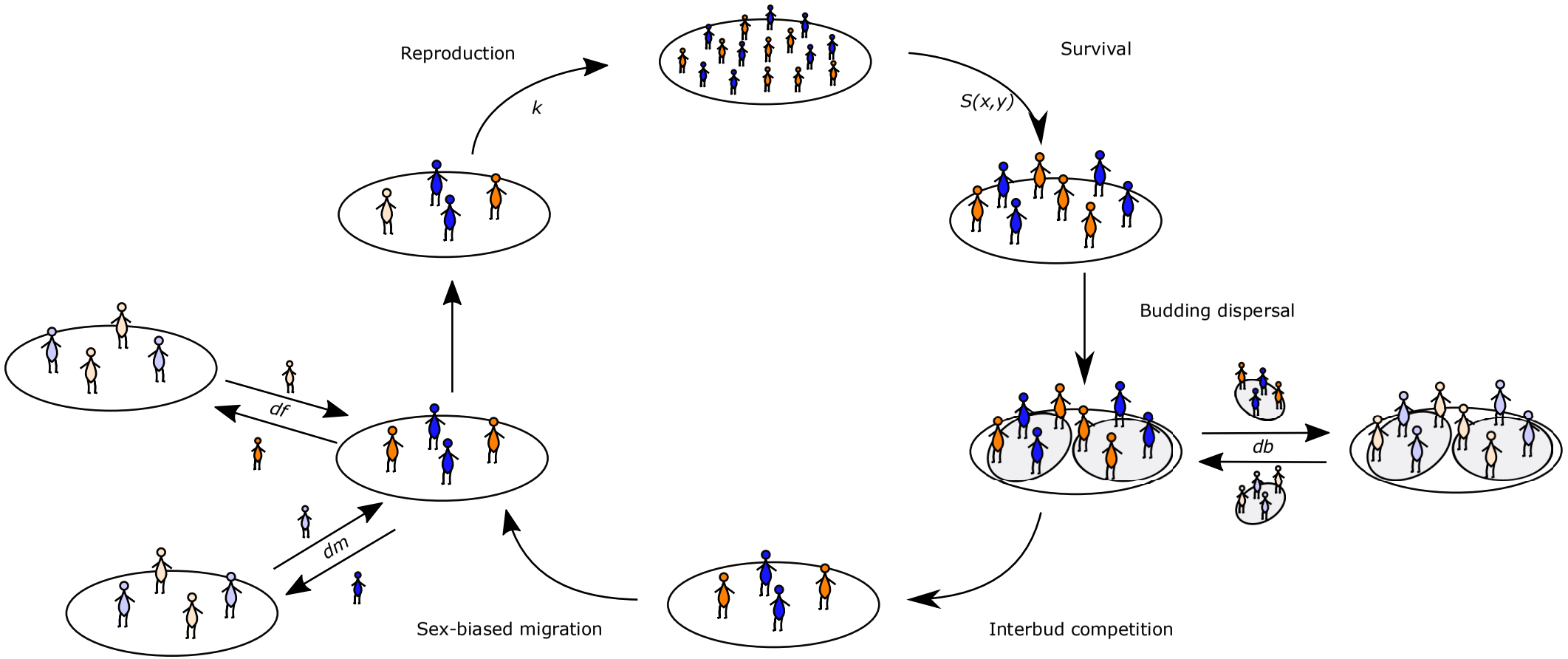
Legends for supplementary figures

Supplementary Figure 1. The life cycle of the population in the three models of language function. A large number of individuals are born, and language investment modulates their survival to adulthood. Patches then subdivide into buds, and either disperse or remain in their patch. Buds then compete for the breeding spots within the patch. After breeding spots have been attained, individuals can migrate with sex-specific rates.

Supplementary Figure 2. The gene expression patterns, and phenotypic consequences, of four types of mutations (gene deletions, gene duplications, epimutations, and uniparental disomies) to imprinted genes. This is done for a scenario where language is selfish (a), or altruistic (b). A hyperlinguistic scenario is where those pathways which marginally increase

295 language investment are overallocated, and a hypolinguistic scenario is where such
296 pathways are underallocated. In both cases the paternal-origin genes are assumed to be
297 more related to social partners. Maternal-origin genes are coloured orange, and paternal-
298 origin genes are blue.


Reproduction
k
Survival
S(x,y)
Budding dispersal
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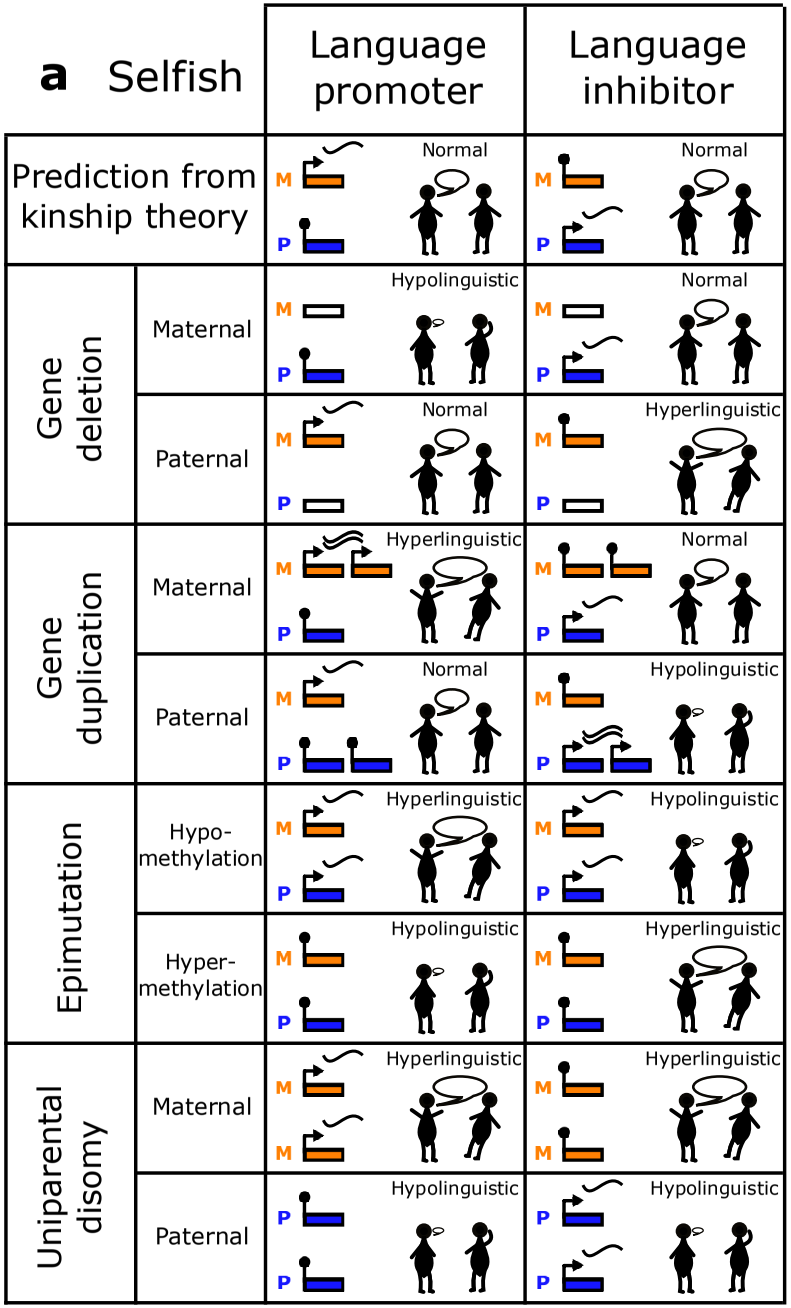
df
dm
Sex-biased migration
Interbud competition


a Selfish
Language promoter
Language inhibitor
Prediction from kinship theory
Normal
Normal
Gene deletion
Maternal
Hypolinguistic
Normal
Paternal
Normal
Hyperlinguistic
Gene duplication
Maternal
Hyperlinguistic
Normal
Paternal
Normal
Hypolinguistic
Epimutation
Hypo-methylation
Hyperlinguistic
Hypolinguistic
Hyper-methylation
Hypolinguistic
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Uniparental disomy
Maternal
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Hyperlinguistic
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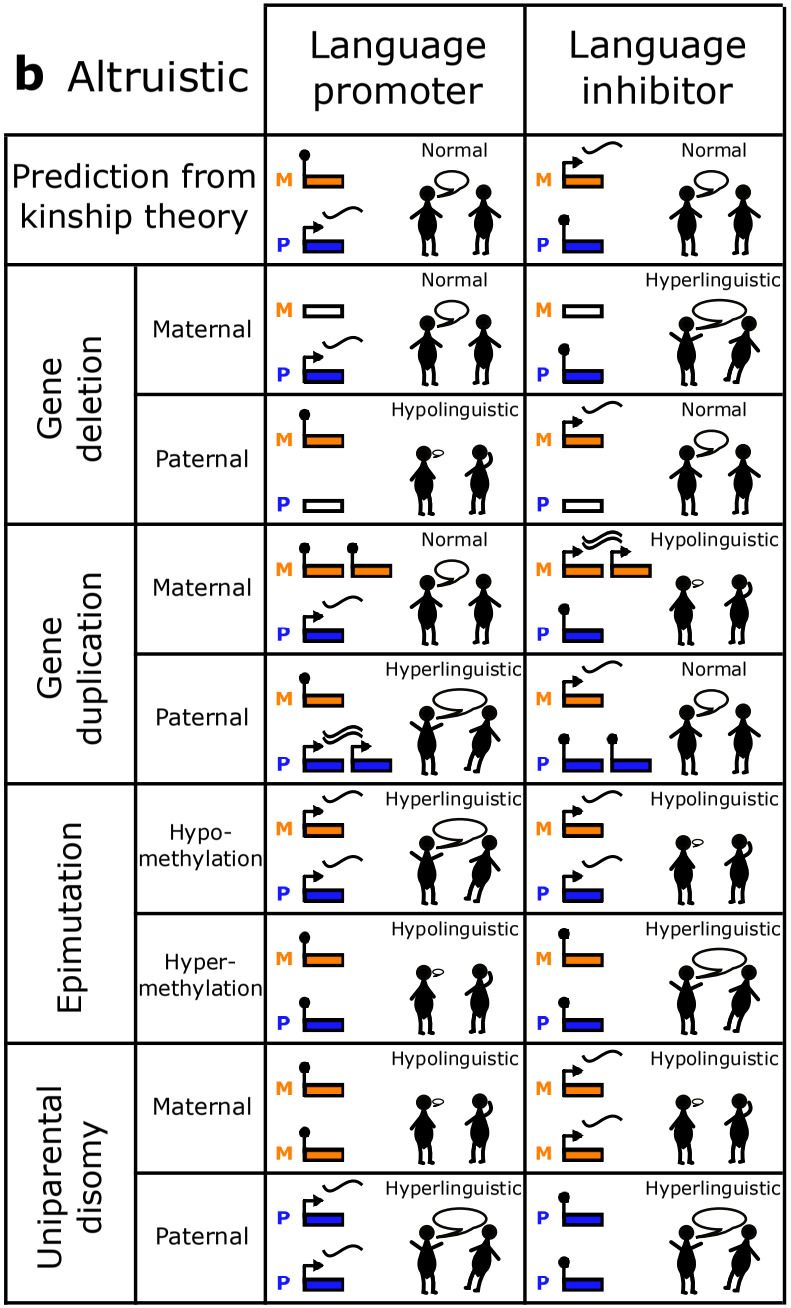
b Altruistic
Language promoter
Language inhibitor
Prediction from kinship theory
Normal
Normal
Gene deletion
Maternal
Normal
Hyperlinguistic
Paternal
Hypolinguistic
Normal
Gene duplication
Maternal
Normal
Hypolinguistic
Paternal
Hyperlinguistic
Normal
Epimutation
Hypo-methylation
Hyperlinguistic
Hypolinguistic
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Hypolinguistic
Hyperlinguistic
Uniparental disomy
Maternal
Hypolinguistic
Hypolinguistic
Paternal
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