

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 patrilocal 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

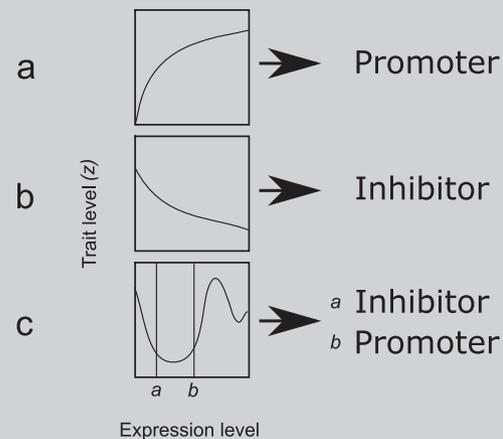
While we may talk about the different agendas of the maternal- and paternal-origin genes, these agendas are not directly visible. However, what makes this intragenomic conflict open to empirical investigation is that, according to the kinship theory of genomic imprinting,^[34] these different agendas 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 versus 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.

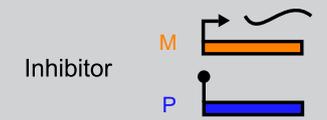
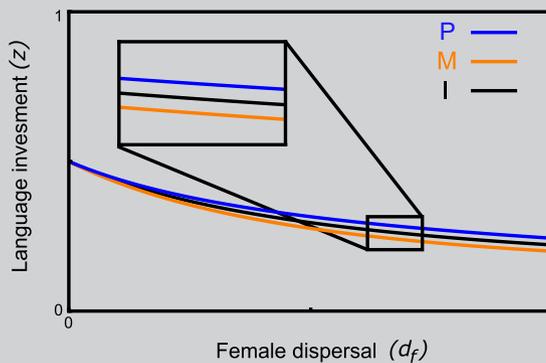
But despite the rarity of imprinting in the genome, there is evidence that some imprinted genes affect linguistic and communicative behavior.^[68,69] While none of the genes that

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 $S(x, \gamma)$ is modulated in different ways by their own investment x and the investment of social partners γ . 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).

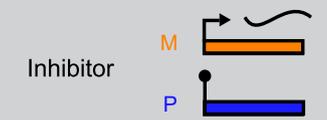
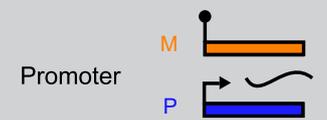
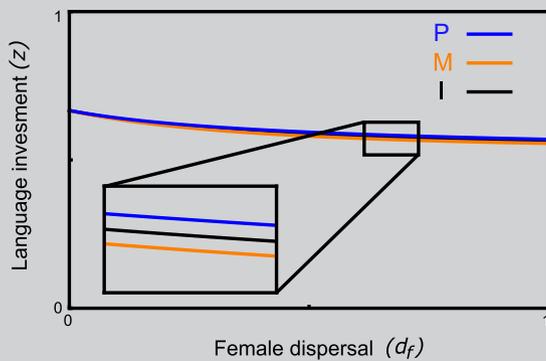
Cooperation| Language may be used to share information about the environment or about other group members or teach others new skills,

$$S(x, \gamma) = \gamma(1-x)$$



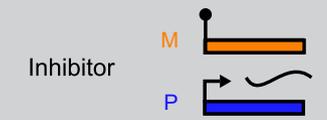
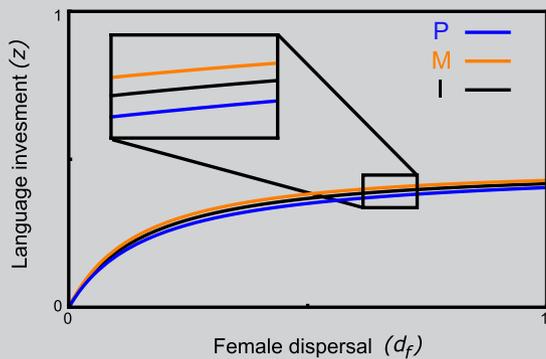
Coordination| Language may be used to coordinate actions and divide labor among group members. This may include tasks such as hunting or scavenging,

$$S(x, \gamma) = x\gamma(1-x)$$



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

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



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

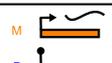
Type of social trait?	Gene type?	Predictions from the kinship theory
Altruistic	Promoter	Paternal Expression 
Altruistic	Inhibitor	Maternal Expression 
Selfish	Promoter	Maternal Expression 
Selfish	Inhibitor	Paternal Expression 

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 *LRR16A* gene, in which risk variants are maternally over-transmitted 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

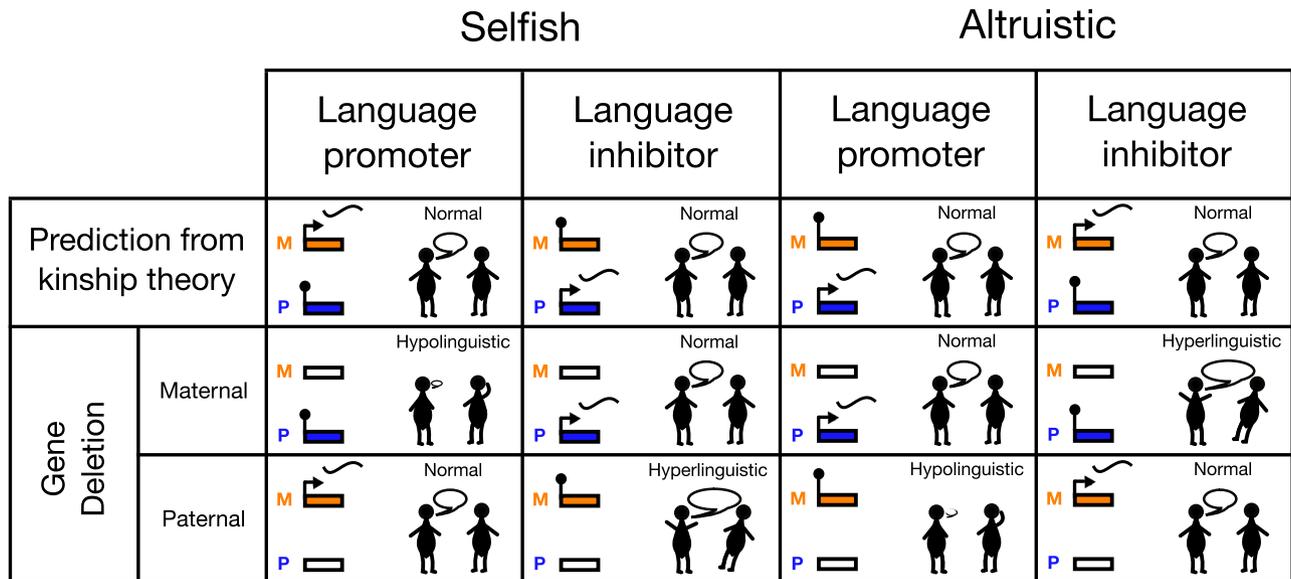


Figure 2. The gene expression patterns and phenotypic consequences of a gene deletion at an imprinted locus. This is done for a scenario where language is selfish or altruistic. A hyperlinguistic scenario is where those pathways which marginally increase language investment are overallocated, and a hypolinguistic 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 it is not possible to quantitatively compare language between species, and link this to differences in relatedness, as one may be able to 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 patriline versus matriline) 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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