The Parental Antagonism Theory of Language Evolution: Preliminary Evidence for the Proposal

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Abstract   Language—as with most communication systems—likely evolved by means of natural selection. Accounts for the genetical selection of language can usually be divided into two scenarios, either of which used in isolation of the other appear insufficient to explain the phenomena: (1) there are group benefits from communicating, and (2) there are individual benefits from being a better communicator. In contrast, it is hypothesized that language phenotypes emerged during a coevolutionary struggle between parental genomes via genomic imprinting, which is differential gene expression depending on parental origin of the genetic element. It is hypothesized that relatedness asymmetries differentially selected for patrigene-caused language phenotypes to extract resources from mother (early in development) and matrigene-caused language phenotypes to influence degree of cooperativeness among asymmetric kin (later in development). This paper reports that imprinted genes have a high frequency of involvement in language phenotypes (~36%), considering their presumed rarity in the human genome (~2%). For example, two well-studied genes associated with language impairments (*FOXP2* and *UBE3A*) exhibit parent-of-origin effects. Specifically, *FOXP2* is putatively paternally expressed, whereas *UBE3A* is a maternally expressed imprinted gene. It is also hypothesized that the more unique and cooperative aspects of human language emerged to the benefit of matrilineal inclusive fitness. Consistent with this perspective, it is reported here that the X-chromosome has higher involvement in loci that have associations with language than would be expected by chance. It is also reported, for the first time, that human and chimpanzee maternally expressed overlapping imprinted genes exhibit greater evolutionary divergence (in terms of the degree of overlapping transcripts) than paternally expressed overlapping imprinted genes. Finally,

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an analysis of global language patterns reveals that paternally but not maternally silenced *Alu* elements are positively correlated with language diversity. Furthermore, there is a much higher than expected frequency of *Alu* elements inserted into the protein-coding machinery of imprinted and X-chromosomal language loci compared with nonimprinted language loci. Taken together these findings provide some support for parental antagonism theory. Unlike previous theories for language evolution, parental antagonism theory generates testable predictions at the proximate (e.g., neurocognitive areas important for social transmission and language capacities), ontogenetic (e.g., the function of language at different points of development), ultimate (e.g., inclusive fitness), and phylogenetic levels (e.g., the spread of maternally derived brain components in mammals, particularly in the hominin lineage), thus making human capacities for culture more tractable than previously thought.

Evolutionary analyses of a neurocognitive phenotype such as language are principally concerned with adaptive mechanisms given a species' natural history, life history, and ecology. Linguistic information-processing mechanisms can evolve by means of natural selection provided there were (1) alternative linguistic information-processing mechanisms in ancestral populations and that (2) differences in linguistic information-processing mechanisms were heritable. Specifically variation in the underlying mechanisms was based on genetic variation, and (3) some underlying linguistic information-processing mechanisms conferred an inclusive fitness advantage, whereas others did not (Pinker and Jackendoff 2005). Inclusive fitness is defined as the reproductive success of the individual adjusted for the coefficient of relatedness plus the reproductive success of all close kin multiplied by the coefficient of relatedness (e.g., you have one child and your sister has two additional children, due to your assistance; your inclusive fitness would be 1.00 gene copies transmitted on average or $1 \times 0.50 + 2 \times 0.25$). Inclusive fitness allows you to calculate roughly how many gene copies are transmitted to the next generation due to social actions (Hamilton 1964). However, it is important to appreciate that the above preconditions for the natural selection of language can be split by the parental origin of language loci. In species with sex-biased dispersal, sex-chromosomes and relatedness asymmetries, conflicts of interests between matrilineal and patrilineal inclusive fitness emerge and are expected to cause conflicts in the development and activation of neurocognitive machinery mediating social behavior (Haig 2000) and vocal communication. Such divisions are necessary because each parental genome does not "agree" over adaptive choices at the individual level as these "choices" have differential effects on asymmetric kin (i.e., relatives related to decision makers on one parental side but not the other). This hypothesis is called parental antagonism theory (Haig 1997, 2002) and may appear speculative; however, data are accumulating that mammalian neurobiology is influenced strongly by imprinted genes, considering their rarity in the genome. For example, two recent papers studying mice (Gregg et al. 2010a, 2010b), show that 1,300 loci exhibit

parent-of-origin effects on neural development and that 347 autosomal loci exhibit sex-specific imprinting biases.

The driving premise behind this paper is that the signal-receptor neuro-cognitive systems mediating human language are susceptible to super-stimulation (Ryan 1990), and socially transmitted linguistic information provides ample opportunities for sensory exploitation and counteradaptations to filter exploitation attempts (Rice and Holland 1997). Language, like other maternal epigenetic phenomenon (Meaney 2001), is hypothesized to influence parental investment conflicts within and between offspring. This approach contrasts sharply with the idea that language is solely about cooperative communication, which is currently the dominant, albeit theoretically naïve view, held by some language theorists (for review of different language theories, see Szamado and Szathmary 2006).

Taking a parental antagonism perspective on language evolution raises the possibility that language phenotypes are not necessarily adaptive for the same reason across ontogeny. Further, language and its associated phenotypes in part may be byproducts of an antagonistic tug-of-war between imprinted genes (within the individual) designed to optimize divergent patrilineal and matrilineal inclusive fitness interests. The psychological mechanisms determining the transmission and reception of social information are predicted to be sensitive to these asymmetries in genetic relatedness (Brown 2001, 2008), a key driver of divergent inclusive fitness for parental genomes. In the following sections parental antagonism theory is elucidated with regards to social behavior and language development.

Parental Antagonism Theory

For most of the genome whether a given gene was transmitted matrilineally or partrilineally does not matter for phenotypic development. However, for a small group of genes parent of origin matters, a phenomenon referred to as genomic imprinting (Murphy and Jirtle 2003). Genomic imprinting is the inactivation of a particular allele, depending on parent of origin. The parental antagonism hypothesis for genomic imprinting (Haig 2002, 2003, 2006a, 2006b, 2006c) hypothesizes that asymmetries in relatedness (e.g., due to multiple paternity and/or sex-biased dispersal) (see Figure 1) favors the differential expression of maternal and paternal alleles so that (1) paternal alleles increase the cost to the offspring's mother (at some benefit to themselves), and (2) the maternal alleles reduce these costs (Haig 2002).

Imprinting is expected to evolve at loci when the gene expression levels that maximize matrilineal inclusive fitness differ from the gene expression levels that maximize patrilineal inclusive fitness (Haig 1999a; Haig and Wharton 2003). Parental antagonism theory predicts that paternal genes within children (called patrigenes) will lead to behaviors that *increase* a mother's costs of child rearing (provided it benefits patrigenes to do so), and conversely, maternal genes within the child (called matrigenes) will be selected to *reduce* these costs. Thus this
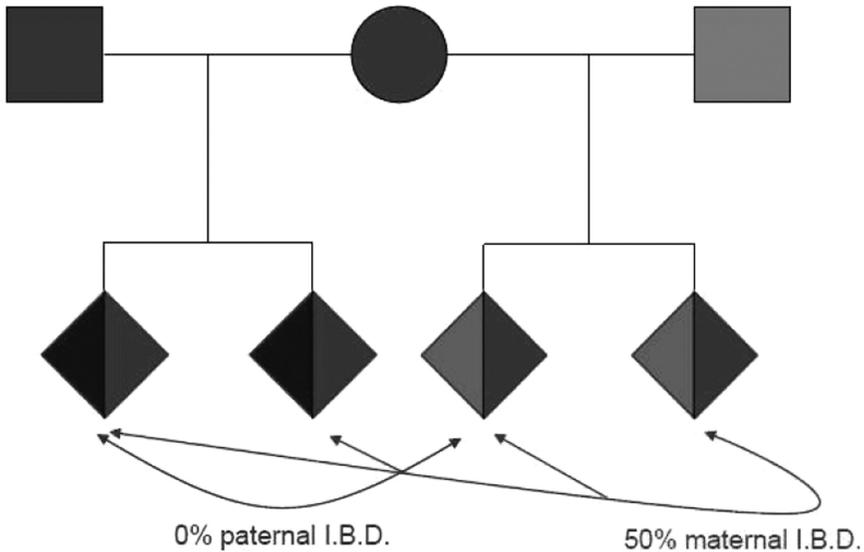
Figure 1. Asymmetric relations using standard pedigree symbols. If mother mates with more than one male, maternally and paternally derived alleles in her offspring will show asymmetric patterns of relatedness within the family group. For autosomal genes maternal alleles in half-sibs will be related to each other by descent with a probability of 50% ($r = 0.50$), but paternal alleles will not be shared. This can be seen by the arrows in the diagram. Due to multiple paternity (and sex-biased dispersal) during mammalian evolutionary history, the average relatedness of paternal alleles between siblings would have been less than 0.50. This asymmetry is the basis of parental antagonism theory. IBD, identical-by-descent. Courtesy of Dr Ben Dickins.

intrapersonal conflict (i.e., within children) regards securing the optimal level of parental investment from mother (Trivers 1972). Parental genomes disagree over the optimal level of maternal parental investment. Parental investment was defined by Trivers (1972) as care delivered by a parent to offspring that increases the likelihood that the offspring survives at a cost to the parent's capacity to care for other offspring. Examples of parental investment include but are not limited to gamete size, lactation, feeding, protection, and teaching.

Early in the development of parental antagonism theory, imprinting researchers focused primarily on prenatal growth (Haig 1999b) and neonatal investment (Haig and Wharton 2003). Recent theoretical work by Haig (2010) makes a persuasive argument that genomic imprinting and parental antagonisms have shaped human life history evolution. Beyond the prenatal period there are additional opportunities for intragenomic conflict. For example, patrigenes in infants influence night waking, increasing breastfeeding costs for cosleeping mothers (McNamara 2004). Corroborating this conjecture are recent reviews of REM sleep patterns and the neural areas mediating milk let-down in humans (McNamara 2004; McNamara et al. 2002; Messinger et al. 2002). McNamara and

colleagues have hypothesized that infant REM facilitates positive attachment to mother (to gain resources from her) and may be regulated via paternal genes.

There are other reasons to expect that intragenomic conflicts persist after birth and shape neurocognitive mechanisms and decision making. In experiments with chimeric mice, maternal genes are overexpressed in cells found in the neocortex (important for flexible decision making) and paternal genes are overexpressed in the cells of the hypothalamus, involved in homeostasis, emotion, hunger, and sex (Allen et al. 1995; Keverne et al. 1996a, 1996b). Theoretically nonfeeding costs to mother are important (Brown and Consedine 2004; Haig and Wharton 2003; McNamara 2004). Ethologists and psychologists have long been aware that mammalian mothers provide more than nourishment; they also provide social learning opportunities and bonds that are crucial for child development (Ainsworth 1979; Altmann 1980; Brown 2001; Bowlby 1969; Harlow 1958; Hinde 1976). One strategy for securing investment from mother lies in the process of forming emotional bonds and eliciting maternal investment through nonverbal signals. Brown and Consedine (2004) suggested that the emotion signals produced by infants (and young children more generally) are best understood within an intragenomic conflict framework. Specifically paternal genes may have designed emotion signals in children to increase the costs on matrilineal inclusive fitness. Lack of care-eliciting signals of need from children would suggest an overexpression of maternal genes within the child, designed to reduce maternal costs. Recent theoretical articles, i.e., reviews and empirical papers based on a parental antagonism approach, are yielding novel insights on neurocognitive development (Badcock and Crespi 2006; Chamberlain and Lalande 2010; Crespi 2007, 2008; Curley and Mashoodh 2010; Oliver et al. 2007; Úbeda and Gardner 2010, 2011).

Does the acquisition of language influence level of maternal investment? Crespi (2007) hypothesized that language is used to extract resources from mother. The parent-of-origin effects of *FOXP2* were cited as an example. Specifically, individuals with underexpression of a paternal copy of *FOXP2* have more severe speech and language disorder (Feuk et al. 2006). It is important to note that there is only indirect correlational evidence of imprinting in *FOXP2* (i.e., parent-of-origin effects). Therefore, it could be that *FOXP2* is seemingly "imprinted" due to regulation by other imprinted genes (Crespi, personal communication, June 2010). One of the reasons that the evidence remains indirect is due to the fact that there were no single-nucleotide polymorphisms within the transcript to allow for the identification of transcription from the different parental alleles (Haig, personal communication).

In children who have Angelman syndrome we see a different pattern in that the absence of maternal *UBE3A* disrupts language production (Clayton-Smith and Laan 2003). Children with Angelman syndrome have an overexpression of paternal genes and impaired language abilities. One way to reconcile these findings is that language learning can be broken down into two antagonistic functional components across ontogeny: (1) information transmission and

reception beneficial to patrigenes and (2) information transmission and reception beneficial to matrigenes. The evolutionary dynamics of this contest are depicted in Figure 2.

Feuk et al. (2006) found that the absence of paternal *FOXP2* gene among individuals with Silver-Russell syndrome (SRS) causes developmental verbal dyspraxia in the expressive but not receptive domains. Specifically all subjects suffered from impairments in expressive language, but receptive skill ranged
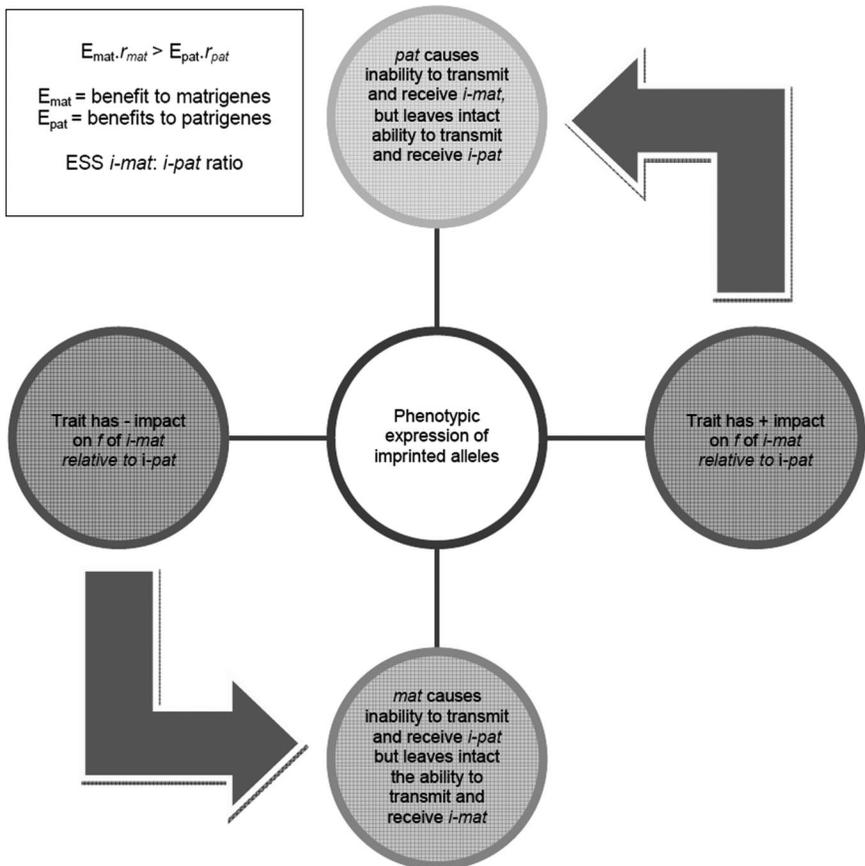

Figure 2. Parental antagonism theory of language and hypothesized effects. The diagram depicts a response model of parental antagonistic effects when a phenotypic variant emerges that favors information from one parental genome over another. Specifically imagine a trait (e.g., helping mother forage for food) has a positive (+) effect on the production of *i-mat* ("I can help you get food") and a negative (−) effect on the production of *i-pat* ("Feed me or too tired to help"). Dynamics are inset that could be used to model the evolutionarily stable solution for the frequency of matrilineally and patrilineally beneficial information. *pat,* paternal gene action on child development; *mat,* maternal gene action on child development; *i-mat,* maternally beneficial information; *i-pat,* paternally beneficial information; *f,* frequency; ESS, Evolutionarily Stable Strategy.

from mild to average (Feuk et al. 2006). SRS is a maternally expressed disorder. The fact that verbal IQ is within the normal range among matrigene expressed individuals with SRS is further evidence for my hypothesis that conflicts between matrigenes and patrigenes are important for human language development.

Neonatal vocalizations are an important example of how matrigenes and patrigenes could both be involved in language development but in different ways. During child development some of the earliest vocalizations are cues to satisfaction/satiation (cooing) and signals of need (crying). It is predicted that patrigenes will increase signals of need (crying to secure more resources at a cost to mother), whereas matrigenes will be selected to increase satisfaction or satiation cues (cooing to reduce maternal investment costs). The reason it is more beneficial to patrigenes to increase crying and other verbal signals of need for self is that current or future siblings may not be related paternally but will more often than not be closely related maternally.

When children age they develop more sophisticated forms of language, and the same evolutionary logic is expected to apply. Upon weaning, feeding conflicts change; for example, foraging by children can reduce the costs on their mother (Haig 2010; Haig and Wharton 2003). Thus parental antagonisms are predicted to track these changes in how children get nourished. Specifically patrigenes are expected to exert their costs on mother early in development (e.g., to secure more than necessary parental investment from mother) and gradually weaken as socially learned norms of the collective good (mediated by matrigenes) develop (Brown 2001). Theoretical justification for this comes from the X-linked inhibitory bias hypothesis (Haig 2006b). The X-linked inhibitory bias hypothesis is based on the fact that the X-chromosome spends half its time in males and *two-thirds* of its time in females; thus, according to the X-linked inhibitory bias hypothesis (Haig 2006b), it is expected that genes on the X-chromosome will favor matrilineal inclusive fitness (reducing mother's parental investment burden) relative to patrilineal inclusive fitness. Considering that there is a preponderance of loci involved in the development of higher cognitive functions including language on the X-chromosome (for review see Zechnera et al. 2001), this seems to provide some support for the hypothesis that matrilineal inclusive fitness may drive (in part) social learning and associated language abilities. Other sources of justification for the idea that the more cooperative uses of language are in part shaped by matrilineal inclusive fitness is the preponderance of matrigenes in neural areas involved in higher cognitive functions (Allen et al. 1995; Keverne et al. 1996a, 1996b). Finally, in the case of nonshivering thermogenesis, there is now empirical evidence that matrigenes are involved with collective good and patrigenes are more selfish (Bittel et al. 2007; Cannon and Nedergarrd 2004; da Rocha et al. 2007; Haig 2008). Specifically, heat generated by huddling littermates is a collective good (Haig 2008), and the empirical evidence suggests that there are two paternally expressed loci for reducing heat and one maternal loci involved with increasing heat to share with huddling littermates.

Patrigenes would benefit from exploiting maternal relatives (depending on opportunity costs, such as the costs of social sanctions for exploiting perceived kin). Subtle exploitation may be less costly and paternal gene expression causing young children to make larger or more frequent requests for assistance may be favored, whereas maternal inclusive fitness will be maximized when children downplay their needs (e.g., reducing demands and learning to help mother and siblings forage).

To reiterate, the role of differential parental gene expression is expected to change during development. Specifically it is predicted that early in child development patrigenes exert effects on nonverbal and verbal signals of need to mother. However later in child development—when cooperation with siblings and collateral kin becomes increasingly important—language skills favoring matrilineal inclusive fitness are predicted to increase (e.g., being receptive to maternally beneficial social rules and norms such as sharing among siblings or offsetting mother's parental investment via cooperative foraging) (Haig 2010).

Consistent with the model presented in Figure 2, language reception can also be altered by parent-of-origin gene expression in Turner's syndrome (Hamelin et al. 2006). Turner's syndrome is a developmental disorder whereby the individual is missing an X-chromosome. Because human females are homogametic for their sex chromosomes (i.e., XX), females affected by the condition will have either a paternal X-chromosome or maternal X-chromosome remaining. Those individuals with an overexpression of paternal genes are more likely to have neural-based hearing impairments than individuals with an overexpression of maternal genes. That is, Hamelin et al. (2006) found that Turner's syndrome subjects with an X-chromosome of maternal origin were less likely to have sensorineural hearing loss compared with those with an X of paternal origin. In some cases of sensorineural hearing impairment sounds can be heard at normal thresholds, but there is a selective impairment of speech perception. It appears that the rapid tonal fluctuations of speech are not perceived as well among individuals with sensorineural hearing impairment (Lorenzi et al. 2006). These speech sounds share a similarity to the exaggerated intonation contours of infant-directed speech (Fernald and Kuhl 1987). Parental antagonism theory suggests that inability to process maternally transmitted infant-directed speech information may be a strategy caused by patrigenes to filter out information benefiting mothers or more generally matrilineal inclusive fitness.

Relatedness asymmetries are expected to cause parental antagonism over behavior in social transmission domains. For example, let's consider an example of increased inclusive fitness for the adopting socially transmitted religious celibacy. In this selective scenario the celibate's family (e.g., sibs) and collateral kin (e.g., cousins) could receive social benefits (trust, mating opportunities) from others by having a celibate sibling. However if there is multiple paternity within the family, matrilineal coefficients of relatedness will be higher (all sibs share the same mother) compared with patrilineal coefficients (i.e., $r^{pat} = 0$) because sibs were sired by different fathers. An intragenomic conflict approach predicts a

divided mind in this situation even though religious celibacy may be socially desirable in some societies (Brown 2001). Specifically it is predicted that the paternal genome within self will reject the celibacy norm, whereas the maternal genome will favor its acceptance. An escalation of conflicting parental gene expression within self is expected when there are relatedness asymmetries either due to sex-biased dispersal or multiple paternity. Likewise different transmission rules between sex chromosomes (X, Y) can cause conflicts over social behavior (Brown 2001a; Haig 2000; Haig 2006b; Trivers 2000). For example, the X-chromosome spends half its time in males and *two-thirds* of its time in females; thus, according to the X-linked inhibitory bias hypothesis (Haig 2006b), it is expected that genes on the X-chromosome will favor matrilineal inclusive fitness (reducing mother's parental investment burden) relative to patrilineal inclusive fitness. One way this could occur if older offspring and collateral kin help rear younger offspring, offsetting mother's direct invest-ment costs. Also siblings who share a maternal X-chromosome will be less likely to engage in resource competition and prefer more cooperative solutions to resource scarcity and limited maternal care. Presuming that language to some degree facilitates cooperative behavior, it is expected that a greater number of language loci will emerge on the X-chromosome than would be expected by chance. To test the hypothesis, the Online Mendelian Inheritance in Man (OMIM) database (www.ncbi.nlm.nih.gov/omim) was consulted for allelic variants that have effects on language (i.e., where the word "language" was found in the full-text search of genes with and without allelic variants). It is important to note that so-called 'language' loci uncovered in this simple search may be an under-estimate and further, it would be mistake to consider these as specific language genes as they have diverse developmental functions, only one of which is linguistic.

Since chromosomal size could bias the results, it was held constant in both analyses by dividing the frequency of language search results by the estimated number of genes on that chromosome. If language alleles were randomly distributed to each chromosome, then given its size, we would expect the likelihood of such an allele to occur on the X chromosome to be approximately seven percent. As seen in Table 1, the observed frequency of so-called language loci on the X-chromosome is 27%, which is much higher than would be expected by chance (binomial test, $p < 0.001$). This finding is consistent with more than one hypothesis, namely (1) X-linked inhibitory bias hypothesis (Haig 2006b), whereby relatedness asymmetries could favor matrilineal cooperative exchanges via language, and (2) language facilitating the male exploitation of females' sensory systems, causing sexually antagonistic language genes to accumulate on the X-chromosome (Rice and Holland 1997). In heterogametic mammals, such as humans, both sexes inherit a maternally derived X-chromosome, but only females have paternally derived X-chromosomes. It has been suggested that the X-chromosome is where sexually antagonistic genetic variation will accumulate (Rice 1984). Gibson et al. (2002) hypothesized that X-linkage may increase

Table 1. Frequency (*f*) of "language" allelic variants; variant, phenotypic effects; and whether or not the variant is subject to parent-of-origin effects

Chromosome	*f* "language" OMIM results[a]	*Language locus*	*Imprinting status*
3	8 (0.05)	SLC9A9	No evidence of imprinting
6	9 (0.05)	SYNGAP1	No evidence of imprinting
7	17 (0.10)	FOXP2; CNTNAP2; SGCE; GTF2I	Possible paternal expression (Feuk et al. 2006); possible paternal expression (Arking et al. 2008); paternal expression (Asmus et al. 2002; Grabowski et al. 2003); maternal expression (Collette et al. 2009; Perez-Jurado et al. 1996)
10	9 (0.05)	OAT	No evidence of imprinting
14	7 (0.04)	FOXG1	Possible paternal expression
15	5 (0.03)	UBE3A	Maternal expression
17	9 (0.05)	NF1	Paternal expression
22	9 (0.05)	SHANK3	No evidence of imprinting
X	24 (0.15)	MECP2; SRPX2; OPHN1; FLNA	No evidence of imprinting, but *MECP2* deficiency affects the level of expression of imprinted gene *UBE3A* (Makedonski et al. 2005; Samaco et al. 2005)

OMIM, Online Mendelian Inheritance in Man (OMIM) database; www.ncbi.nlm.nih.gov/omim/.
a. Proportion of results given the number of genes on the chromosome in parentheses.

sexually antagonistic polymorphism. Specifically, imagine a rare recessive mutant allele with a large disadvantage for homogametic females (XX) and a small advantage for heterogametic males (XY). When averaged across the sexes, the mutation would not be favored by selection and would be less likely to accumulate on an autosome (i.e., not a sex chromosome). However, when X-linked the allele accumulates, because it is more commonly expressed in males than females, as the recessive variant is not hidden by dominance in heterogametic males (XY). Interestingly, language has been hypothesized to be caused by sexually antagonistic coevolution (Rice and Holland 1997), and thus genes costly for females (but beneficial to males) are expected to accumulate on the X-chromosome. If this is true, it is expected that we would find a higher frequency of female detrimental language loci on the X-chromosome (e.g., making females more susceptible to male sensory exploitation attempts). Therefore language genes are expected to accumulate on the X-chromosome at higher rates than expected by chance. Sexual antagonistic coevolution may be a less viable explanation than parental antagonism theory because many of the mutant forms of these genes are not particularly harmful to females relative to males. Nonetheless, both sexual and parental antagonism theories predict an abundance of language loci on the X-chromosome. The way to differentiate between the two hypotheses is to investigate the language phenotype directly. Specifically, parental antagonism hypotheses predict that language loci on the

X-chromosome facilitate cooperative behavior among matrilineal kin, whereas sexual antagonism hypotheses predict that language loci will harm females but be beneficial to males. Granted, it is possible that both theories have played a role in the evolution of language. The comparative method may be particularly invaluable with regards to disentangling the sexual and parental antagonism hypotheses regarding sex chromosomal involvement in language phenotypes. Parent-of-origin gene expression for prenatal growth factors does not occur in birds (O'Neill et al. 2000), which was expected by kinship theory because maternal allocation to offspring is completed before fertilization. However, recent work suggests that parent-of-origin effects may exist in chickens (Campos et al. 2009; Rowe et al. 2009). Interestingly, Brandvain (2010) interprets this partial evidence for the possibility that genes influencing social behavior may be imprinted even in species that do not experience conflicts of interest over prenatal investment. Nonetheless, parental antagonism may occur via haploid sex chromosomes. Because heterogamety is sex reversed in birds (i.e., females are ZW and males are ZZ) and some species have elaborate begging displays, signaling capacities, and male parental care, avian species may be an ideal group to focus future empirical studies exploring antagonism accounts of communication and social behavior. The presence of cooperative breeding, female-biased dispersal, high levels of monogamy (compared with mammals), and male parental investment may be suggestive of the existence of a Z-linked inhibitory bias similar to X-linked inhibitory bias theorized by Haig (2006b). Granted the absence of evidence that male birds do not show an inactivation of the maternally transmitted Z-chromosome could suggest that for avian species parental antagonisms are minimized due to social ecological factors such as monogamy. Nonetheless, it cannot yet be ruled out that transient inactivation of the Z-chromosome in male birds (Shoenmakers et al. 2009) has consequences on other genes mediating postnatal investment. Despite the fact that maternal prenatal investment precedes fertilization, it is still a theoretical possibility that avian postnatal conflicts could be mediated by parental genome silencing on sex chromosomes. A Z-linked inhibitory bias hypothesis would predict that enhancers of paternal investment will be preferentially located on autosomes in birds, whereas inhibitors of male demand will be preferentially located on the Z-chromosome.

Table 1 also shows the allelic variants associated with language phenotypes on the autosomes. Of interest, is the number of language loci that are subject to genomic imprinting (i.e., where the effects of a gene depend on whether it was transmitted from mother or father). Because only 1–2% of the mammalian genome is imprinted (Murphy and Jirtle 2003), we would not expect any of the 14 language genes to be imprinted unless parental antagonism had played a role in the origin of language. Interestingly 36% of language loci uncovered in the OMIM database are subject to possible parent-of-origin effects: binomial test, $p < 0.001$. In Table 1, seven imprinted loci—five patrigenes and two matrigenes—have been identified as important for language development. A

notable putatively imprinted language-associated locus is *FOXP2,* which is also in close proximity to two other genes also associated with speech production: *CNTNAP2* (see Vernes et al. 2008 for its relation to specific language impairment) *and SGCE. SGCE*, a conserved gene, is important for placental growth in mammals in association with paternally expressed gene 10 (*PEG10*). It should be pointed out that the so-called "language" loci in Table 1 may reflect genes affecting global mental retardation, which in turn causes language impairment as a secondary consequence. Also the database search conducted was limited by the word "language" appearing in the report of phenotypic character-istics of the allelic variant. Thus, this analysis should be taken with caution with regards to imprinted genes involvement in language.

One of the important questions for the study of language evolution is the degree to which there are language-specific loci that have had different evolutionary pathways among closely hominid species. In the following section this hypothesis is explored in greater detail in light of the evidence that imprinted genes may be important for language development.

Chimpanzee-Human Divergence in Overlapping Imprinted Genes

Overlapping genes are genes whose transcription regions are shared (Kim et al. 2009). There is now a growing body of evidence that overlapping genes may regulate key gene expression mechanisms in genomic imprinting (Runte et al. 2001). Specifically imprinting related *SNURF-SNRPN* and *UBE3A* overlap-ping genes are associated with Prader–Willi (PWS) and Angelman syndromes (Runte et al. 2001). If imprinted genes played a special role in the evolution of human language, one may expect to find that there are significant differences between chimpanzees' and humans' overlapping imprinted loci.

For investigating of evolutionary relationships of overlapping genes, Kim et al. (2009) designed the Evolution Visualizer for Overlapping Genes (EvoG), a web-based database (http://neobio.cs.pusan.ac.kr/evog/) that contains overlap-ping genes common across human, chimpanzee, cow, mouse, chicken, rat, zebrafish, *Drosophila melanogaster*, and *Xenopus tropicalis*. The EvoG database includes the following numbers of overlapping genes: 10,120 human, 10,026 chimpanzee, 853 orangutan, 213 rhesus, 3595 cow, 99 horse, 1 platypus, 213 dog, 25 cat, 1451 chicken, 3 zebrafinch, 115 opossum, 8667 mouse, 6892 rat, 535 *X. tropicalis*, 50 medaka, and 1805 zebrafish.

In the following analyses I compare overlapping imprinted genes across species contained in the EvoG database. It is predicted that there will be significantly less chimpanzee-human similarity in imprinted overlapping genes on language rich chromosomes compared with other chromosomes. There are nine relevant imprinted genes on chromosome 7 with 36 similarity indices between chimpanzee and human. EvoG calculates similarity in overlapping genes by the number of overlapping genes between each gene pair (Kim et al.

2009). Do human chromosomes with language adaptations exhibit less similarity in their overlapping imprinted genes compared with chimpanzees? Seemingly not, as chromosome 11 has significantly lower (both $ps < 0.01$) similarity to chimpanzees (mean $= 0.89$, SD $= 0.14$) than chromosomes 7 (mean $= 0.99$, SD $= 0.01$) and 15 (mean $= 0.97$, SD $= 0.03$): $F (2,157) = 23.22$, $p < 0.001$. There was no statistically significant difference between chimpanzee-human similarity indices for chromosomes 7 and 15 ($p = 0.75$). Chimpanzee and human similarity of overlapping imprinted genes indicate that these particular overlapping imprinted genes are more evolutionarily conserved on chromosomes 7 and 15 compared with 11.

Nonetheless, the human-chimp divergence between overlapping imprinted genes on chromosome 11 is notable, because there appears to have been recent natural selection (Voight et al. 2006), and chromosome 11 contains genes associated with schizophrenia (Klar 2004), a disorder with links to neural areas involved in language (for review see Mitchell and Crow 2005). Interestingly, on chromosome 11 there has been greater chimpanzee-human evolutionary divergence in the maternally expressed overlapping gene region *H19, OSBPL5* (similarity index $= 0.5392$) compared with all other overlapping imprinted gene pairs (mean similarity index $= 0.9471$): $t(174) = 57.86$, $p < 0.001$. *H19* is also noteworthy, because it is a growth suppressor gene, consistent with parental antagonism theory. It is an open empirical question whether or not there has been recent selection on this overlapping genetic region.

Evolutionary conservation (i.e., similarity between chimpanzees and humans in overlapping imprinted genes) could either be due to constraint or selection maintaining gene proximity. Interestingly, there is greater variation in chimp-human similarity indices for maternal gene-pairs compared paternal expression gene pairs (Levene's test, $p < 0.001$). As seen in Figure 3, maternal gene pairs are more evolutionarily distant between humans and chimpanzees (mean $= 0.91$; SD $= 0.15$) compared with paternal gene pairs (mean $= 0.97$; SD $= 0.03$): $t(100) = 3.41$, $p < 0.05$ (see Figure 3). This indicates that there has been greater divergence between humans' and chimpanzees' overlapping maternal gene pairs compared with paternal gene pairs. Considering that parental antagonism theory has previously suggested that social cognition and learning are mediated by maternal gene expression (Brown 2001), then the finding of greater evolutionary divergence between chimps and humans in overlapping matrigenes compared with overlapping patrigenes is notable. This finding may also be consistent with comparative psychological assumptions that humans are unique with regards to social cognitive adaptations relative to chimpanzees (Tomasello 2009). Based on kinship theory, Smit (2009; 2010) has theorized that imprinted genes are important for human collaborative action and communication. The parental antagonism model presented here is in agreement with Smit (2009) in that the more cooperative functions of human communication (e.g., shared intentionality, pointing, etc.) may be mediated in part by maternal gene expression within offspring. Smit (personal communication, 5 January 2011)
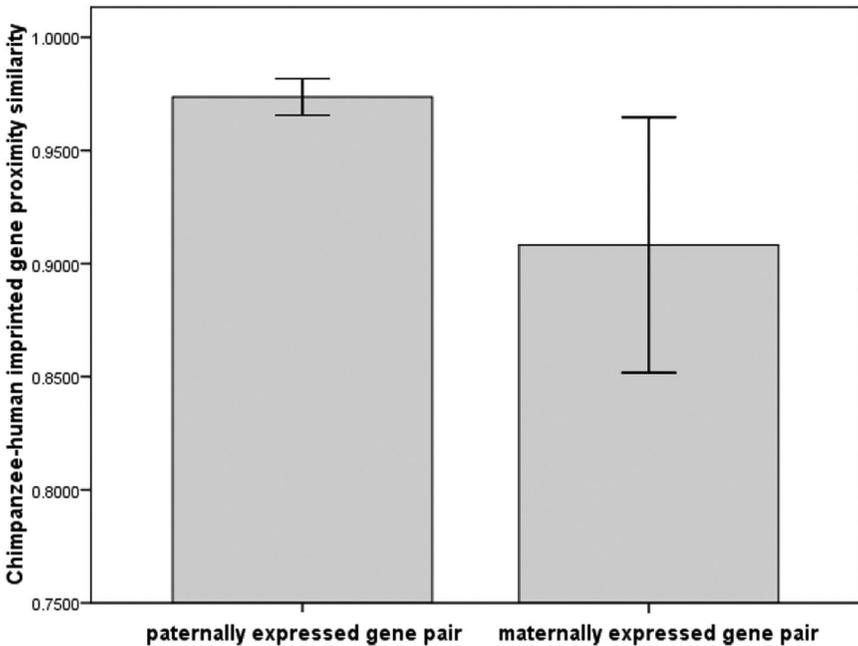
Figure 3. Maternal versus paternal overlapping gene pair similarity between chimpanzee and human (error bars, 95% confidence intervals). Data source: http://neobio.cs.pusan.ac.kr/evog/.

rightly points out that the parental antagonism model of language is consistent with Tomasello's (2008) cognitive developmental perspective of human communication. Specifically, Tomasello (2008) hypothesized that as children age they become less reliant on demand action, but rather begin to offer assistance to others due to the development of psychological mechanisms mediating understanding and shared intentions. The major difference between Tomasello (2008) and the work presented here, is that I hypothesize specific antagonistic genetic processes underlying these cognitive developmental trends, whereas Tomasello (2008) may argue that these processes are only beneficial at the individual organism level of selection.

General Predictions Derived from Parental Antagonism Theory

Any good theory for understanding the evolution of language must generate predictions that are distinct from alternative adaptationist hypotheses. The ideal adaptationist account of language, will be a proposal where the exceptions prove the rule. For example the theory of parental investment in relation to sexual selection does splendidly when it predicts generally that the sex investing most in offspring will be a more selective mate chooser; and the least investing sex will invest more in sexually selected display traits (Trivers 1972).

In sex-role-reversed species where males invest more in offspring care, males indeed are choosier, whereas females invest more in secondary sexual display traits (Trivers 1972).

Parental antagonism theory of language may prove useful in explaining the exceptions to the observed patterns. For example, when there are relatedness asymmetries, patrigenes are predicted to be selected to extract more resources from mother, and offspring matrigenes will be selected to minimize extractions (this effect will be diminished when relatedness asymmetries among kin are reduced) (see Brown 2001). Specifically, recent modeling work (Úbeda and Gardner 2010, 2011) make this case particularly clear that social ecological factors such as sex-biased dispersal and sex-specific variance in reproductive success will have major consequences for the evolution of parent-of-origin gene expression. In most species with parental investment, sex-biased dispersal, multiple paternity, and sex chromosomes conditions will be ripe for parental antagonism; but when these factors are limited, parental antagonistic effects are predicted to be reduced. Species with variable mating systems and sex-biased dispersal patterns are ideal to test whether degree of parental antagonism is minimized, dependent on within-species variation in relatedness asymmetries. If mating systems and dispersal patterns exhibit wide intraspecific variation (as they seem to in humans), patrigene and matrigene expression may change expression patterns, depending on these social ecological factors. It is hypothesized that within-species variation in relatedness asymmetries caused a coevolutionary arms race between parentally derived language loci in species capable of socially transmissible dispersal and mating patterns.

Three general predictions regarding language systems derived from parental antagonism theory are presented. However, these general patterns are expected to be altered accordingly when relatedness asymmetries among collateral kin vary across societies.

General prediction 1.   Some of the first verbal utterances used by children are requests and demands called "manding" (as in demanding). Mands are controlled by deprivation and aversion states. The function of a mand is to request or to obtain what is wanted (e.g., "cookie *or* biscuit!"). Mand use early in child development is hypothesized to be caused by increased patrigene expression relative to matrigene expression. Throughout mammalian evolutionary history a mother would have accrued the costs of mands; thus, maternal genes within the child (matrigenes) would be selected to decrease the number of mands toward the child's actual caloric need. Children with paternally overexpressed Angelman syndrome appear to confirm this expectation, because they primarily use manding to communicate compared with other children (Didden et al. 2004). Consistent with the parental antagonism approach, new evidence in newborn mice investigating the communication effects of *Ube3a*/*Gabrb3* deletions, shows that m−/p+ pups emitted significantly more ultrasonic vocalizations than m+/p+ littermates in mother's bedding (Jiang et al. 2010). Neonatal ultrasonic

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vocalizations in mice are associated with securing maternal investment (Portfors 2006). This finding indicates that patrigenes – as predicted – cause signals of need in mice. Future work in humans is warranted to determine if mands perform a similar function and are mediated by *UBE3A/GABRB3*.

General prediction 2.   Cognitive mechanisms for being receptive and transmitting utterances fostering kin-based cooperativeness is hypothesized to be caused by increased matrigene expression—relative to patrigene expression— during child development. Recent work suggests that one of the genes associated with the development of William's syndrome confirm the hypothesis that there is matrigene overexpression relative to patrigene (Collette et al. 2009). This is notable due to type of hypersociality exhibited among many children with William's syndrome. Specifically children with William's syndrome have enhanced expressive language abilities in domains of cooperativeness, empathy, and sympathy (Jones et al. 2000). This may appear to contradict Crespi's (2007) hypothesis that language use among young children is mostly about getting parental resources (mands serve this function, but other more cooperative aspects of child language use do not) (see Jones et al. 2000). Crespi's (2007) hypothesis aligns with the parental antagonism theory of language presented here, which expects that resource extraction utterances (but not later developing socially cooperative features of language) are products of increased patrigene expression during early child development.

There is further evidence in support for the hypothesis that the matrigene-mediated cooperative use of language emerges later in development. On the false belief question used to assess theory of mind abilities (i.e., the ability to understand others who have beliefs different from one's own; this ability is believed to be an important feature of social behavior in humans), more children with William's syndrome and PWS passed the false-belief task compared with children with nonspecific mental retardation (Sullivan and Tager-Flusberg 1999). Considering that both William's syndrome and PWS are due (in part) to an overexpression of matrigenes, this finding is important corollary evidence for a parental antagonism theory of language.

General prediction 3.   Intrapersonal reciprocity (Haig 2003) between patrigenes and matrigenes at language loci will lead to improved language competency. The rationale here is that conflicts within are not an adaptive division of labor, but rather a source of inefficiency at the individual level. A pathway to intrapersonal reciprocity may be determined by level of maternal investment during early child development. Specifically, patrigenes are predicted to "reciprocate" increased maternal investments by lowering demands from mother (Brown 2004, 2008). There is evidence that maternal investment alters offspring gene expression via epigenetic mechanisms, including DNA methylation (Francis et al. 1999; Meaney 2001; Weaver et al. 2006). This work was largely done in rats, but there is recent evidence that a similar process

may operate in humans (McGowan, Sasaki, Huang et al. 2008). Specifically, McGowan et al. (2008) found evidence for hypermethylation in a rRNA promoter region of the hippocampus of suicide subjects (compared with a control group of postmortem subjects). These suicide victims had a history of child neglect and abuse. More germane to the parental antagonism theory of language evolution, Keller et al. (2010) found increased *BDNF* methylation in Wernicke's language area of brain among 44 suicide completers (compared with 33 nonsuicide control subjects). Increased methylation corresponded to lower *BDNF* messenger RNA levels. It is important to note that it has previously been found in a sample of schizophrenic subjects that *BDNF* could be subject to parent-of-origin effects, being more likely to be transmitted maternally (Muglia et al. 2003). Regardless, *BDNF* is located between two imprinted genes, one paternally and the other maternally expressed in humans: *OBPH1* and *WT1*.

Increased stress (a signal of need) response in offspring raised by low investing mothers could be a part of patrigene strategy to increase maternal investment (i.e., causing offspring to produce more signals of need to mother or matrilineal kin), which in turn escalates intrapersonal conflicts. Intrapersonal reciprocity could subsequently be favored by natural selection (or an epiphenomenon of an equilibria between patrigenes and matrigenes). Both of these possibilities are beyond the scope of this paper but deserve theoretical consideration.

Does Antagonism Theory Tell Us Anything about Human Uniqueness?

Languages use arbitrary conventions to signal meaning and most theorists perceive this to be a peculiar feature of an animal signaling system (Szamado and Szathmary 2006). This arbitrariness could be a clue to its origins. Signals restricted to families of relatives, such as shibboleths (i.e., signals only decodable by those who are part of the group), probably are a key characteristic of early hominin language evolution. Indeed, the kin basis for the origin of language is critical for parental antagonism theory, which deals with conflicts of interest between all collateral kin social relations (Haig 2000). Local accents and dialects are seemingly arbitrary, but within the family groups in which they emerge, these signals are easily decodable (i.e., not perceived as irregular or a deviation from the norm). According to the parental antagonism proposal presented here, these local accents and utterances act as shibboleths of kin membership and would be associated with relatedness asymmetries and subsequently parental antagonism within the child. For example, the degree to which diverse accents are tolerated may be negatively associated with underlying intragenomic conflicts due to asymmetries in relatedness.

How a standard reliable signaling system found in most organisms made a transition to a seemingly arbitrary extensive communication system provides a challenge for parental antagonism theory in its current formulation. However, maternal effect genes are considered to be primarily driving cooperation among

individuals not related on the paternal side. One of the byproducts of cognitive mechanisms designed to suppress paternal nepotism is that organisms will trust and cooperate with nonkin via proximate mechanisms for hypersociability. Thus, the parental antagonism theory of language evolution posits a parsimonious phylogenetic account of animal begging systems toward cooperative use of language via socially transmitted norms of conduct. That is, early in evolution of kin-based communication systems antagonisms are primarily driven by sexually

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antagonistic coevolutionary arms races (Rice and Holland 1997); however, once parent-of-origin gene expression emerged during evolution, kin-based communication systems began to evolve by parental antagonism (Haig 2000). Specifically, the ancestral state of proto-language use would primarily function for extracting resources from mother via vocal utterances: a taxonomically widespread phenomenon among species with parental investment. However, it is important to note that the selection for extraction of maternal resources among altricial offspring may be particularly strong in humans. Recent work (Wawrzik et al. 2010) suggests that the imprinted patrigene *C15orf2* was positively selected in humans and is important for fetal brain growth (e.g., hypothalamus). It remains to be seen whether or not this gene functions to produce signals of need during child development. Matrigenes are predicted to have been selected to switch off

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such patrigene-mediated demands as weaning approaches. Thus, as individuals age, outside the window of being extremely altricial, matrigene expression is expected to more vigorously suppress signals of need from dependent offspring.

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Recently, Williams et al. (2010) studied language-associated matrigene (i.e., *UBE3A*) expression levels in postmortem brain samples from 28 individuals (with no history of neurological or mental health problems), ranging from 20 days to 79 years of age. It was found that *UBE3A* expression in the cortex begins to drop after puberty and declines substantially after thirty years of age in the cross-sectional sample (Williams et al. 2010). For example, *UBE3A* expression in the primary visual cortex was relatively constant in children, teenagers, and young adults. However after the young adult years (older than 30 years) there was reduced *UBE3A* expression in the neocortex. *UBE3A* expression in adults more than 50 years fell to about 50% of the younger adult levels (Williams et al. 2010). At this point, it is pure speculation whether or not these expression levels in the neocortex are associated with language and social learning in humans. Consistent with the proposition that matrigenes are important for social learning in humans, there is evidence that *UBE3A* (a matrigene) is required for experience-dependent neocortical plasticity in mice (Yashiro et al. 2009; Greer et al. 2010). Finally, these findings may also be consistent with the proposal of

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Keverne et al. (1996a) that the general primate pattern of brain evolution fits with an increasing expansion of maternally derived brain components fostering flexible decision making (e.g., social learning mediated by refined language abilities) over endocrine-based demands mediated by paternally derived brain components.

Parental Antagonism and Gene-Culture Coevolution

If parental antagonism theory proves to be useful for elucidating culture, as previously hypothesized (Brown 2001), then parent-of-origin effect genes may predict the diversity of languages and/or their spread globally. Indeed, any good theory of language should be able to predict language form and spread. If fissioning of parental language demes is caused (in part) by conflicts within families—a reasonable source of human dispersal, then paternally and maternally biased gene expression patterns could be positively correlated with present-day language diversity patterns. The mechanism here may be similar to "cultural hitchhiking" previously hypothesized by Whitehead (1998) to explain why socially transmitted dialects and mtDNA diversity are correlated across cetacean species. Essentially, in some species of whales, males are the dispersing sex, which increases mtDNA relatedness among pod members (i.e., reduced mtDNA diversity), causing a positive association between dialect and mtDNA diversity. Whitehead (1998) suggests that this is an example of how socially transmitted information can affect genetic diversity. Therefore, it seems reasonable to posit that if parental antagonisms have played a role in language evolution, parentally derived genetic elements may be associated with language diversity via a cultural hitchhiking or similar process.

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One way to test this hypothesis is to have information on a genetic element that exhibits parent-of-origin effects, varying in frequency across human populations with differing levels of language diversity. An example of "markers" that vary across human populations are *Alu* elements (Kass et al. 2007; Rowold and Herrera 2000). *Alu* elements are a family of retrotransposons, and more than a million copies reside in the human genome (Lander et al. 2001). Alu elements are a primate-specific family of DNA elements (often referred to as molecular fossils) that were integrated early during primate evolution (Shen et al. 1991). Interestingly, there are approximately 5,000–7,000 *Alu* insertions unique to humans (i.e., arose after the split between humans and chimpanzees) (see Mikkelsen et al. 2005; Roy-Engel et al. 2001).

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Imprinted genes and *Alu* insertion sites share several characteristics (Waterland and Jirtle 2004). One shared characteristic is that imprinted genes and *Alu* elements are transcriptionally regulated by CpG methylation (Reik and Walter 2001). Another characteristic shared by imprinted genes and *Alu* elements are their tendency to be clustered within the genome. The clustering of imprinted genes is in some cases necessary to facilitate long-range coordinate regulation between pairs of genes within an imprinted domain (Arney 2003). Similarly, transposons tend to cluster within the genome, being enriched in areas where they cause the least genetic and epigenetic harm to the host organism (Smit 1999).

Alu elements have been used to track human historical patterns (Watkins et al. 2003), and databases now exist to measure insertion frequencies across cultures. Also of interest from parental antagonism theory, Sandovici et al. (2005) investigated parent-of-origin effects among 19 *Alu* elements in a sample

of 48 three-generation families. It was found that the following *Alu* polymorphisms were more strongly methyated paternally relative to maternally: Ya5NBC345, Yb8NBC437, Yb8NBC146, Yb8NBC479, Yb8NBC80, and Ya5NBC221 (GenBank accession numbers: AC008249, AL390755, AC009028, AL121919, AC006249, AC004019). However, Sandovici et al. (2005) found only one *Alu* element that exhibited significantly stronger maternal relative to paternal methylation: Ya5NBC171 (GenBank accession number: AL035688). Control *Alu* elements (i.e., ones that did not exhibit significant parental bias in DNA methylation patterns) were Yb8NBC405, Yb8NBC93, TPA25, Yb9NBC50, Yc1NBC50, Yb8NBC65, Yc1NBC63, Yc1NBC2, Yb8NBC412, APO, Yb9NBC30, and Ya5NBC102 (GenBank accession numbers: AC024057, AL035461, K03021, AL109865, AC010382, AL031228, AL121964, AC006195, AC018634, X53550, AC003003, X62855).

To test this possibility that parentally biased *Alu* element insertions frequencies are positively related to language diversity, three data sets were combined from previously published papers and www.ethnologue.com: (1) language diversity index (www.ethnologue.com) and regional location to reduce Galton's problem of autocorrelation or nonindependent observations (see Fincher and Thornhill 2008); (2) *Alu* elements insertion frequencies across human populations (Watkins et al. 2003); and (3) the recent evidence of parent-of-origin effects among 19 *Alu* elements(Sandovici et al. 2005). Control *Alu* elements Yc1NBC50 and Yb9NBC30 were not included in the following analyses because insertion frequencies were not available by culture from Watkins et al. (2003).

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Language diversity was calculated using Greenberg's index values from www.ethnologue.com. Greenberg's language diversity index is the probability that two randomly selected people in a country would have different first languages (Lieberson 1981). A value of 1 indicates that no two individuals have the same first language, whereas lower values (e.g., 0) indicate little to no diversity (i.e., most people have the same first language). The derivation of the language diversity index is based on the number of individuals using the language as a proportion of the total population (www.ethnologue.com). To determine the language diversity for a culture that resides across multiple countries the mean index was used in analyses. For example, if the genetic sample was taken from individuals who were members of the San culture, then the mean language diversity index would be the mean language diversity across South Africa, Zimbabwe, Lesotho, Mozambique, Swaziland, Botswana, Namibia, and Angola, where the San primarily reside.

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There are clear cultural group differences in *Alu* insertion frequencies (Table 2). Of note were the statistically significant differences in paternally silenced *Alu* insertions, whereby Indian samples have a significantly higher frequency of paternally silenced *Alu* insertions than African and European samples. Finally, Europeans have a higher frequency of paternally silenced *Alu* insertions than African samples (all $ps < 0.04$). With regards to maternally

silenced *Alu* insertions only one significant difference was found: Indian samples had a higher frequency than European samples ($p < 0.05$).

Because there were also group differences in language diversity, it was decided that larger cultural groups (i.e., African, Asian, European, and Indian) would be included as a covariate in subsequent analyses to help reduce to some extent the effects of shared geographic region. Controlling for larger cultural group, it was found that the frequency of paternally silenced, but not maternally silenced *Alu* element insertions (relative to *Alu* insertions without a parental bias) was positively associated with language diversity across cultures: partial r^{pat} (28) = 0.54, $p < 0.01$; partial r^{mat} (28) = 0.35, $p = 0.058$. The paternally silenced *Alu* elements that exhibit the strongest correlations with language diversity are Yb8NBC437 (chromosome 13) and Yb8NBC479 (chromosome 20).

These findings suggest that as language diversity increases so does the number of paternally silenced *Alu* insertions. Future work should investigate whether or not there are more *Alu* insertions exhibiting parent-of-origin effects and if indeed their insertion frequencies are associated with language diversity

Table 2. *Alu* insertion frequency by parent-of-origin (or lack thereof) and language diversity indices across four major cultural groups[a]

	Group	Mean	SD
Paternally silenced *Alu* insertion frequency	African	0.628	0.066
	Asian	0.683	0.057
	European	0.701	0.020
	Indian	0.757	0.040
	Total	0.697	0.074
Games-Howell post hoc tests	African < European, Indian; Indian > African, European		
Maternally silenced *Alu* insertion frequency	African	0.212	0.134
	Asian	0.243	0.138
	European	0.089	0.077
	Indian	0.253	0.106
	Total	0.217	0.124
Games-Howell post hoc tests	Indian > European		
No parental bias *Alu* insertion frequency	African	0.409	0.032
	Asian	0.507	0.019
	European	0.494	0.023
	Indian	0.412	0.033
	Total	0.437	0.051
Games-Howell post hoc tests	African < Asian, European; Indian < Asian, European		
Language diversity index	African	0.864	0.039
	Asian	0.339	0.287
	European	0.153	0.084
	Indian	0.940	0.000
	Total	0.717	0.325
Games-Howell post hoc tests	African, Indian > European; Indian > Asian		

a. Total sample size = 31; sample sizes for each cultural grouping: African = 10; Asian = 5; European = 4; Indian = 12.

patterns. The majority of transcriptionally active *Alu* elements are transmitted paternally (Jurka et al. 2002). The fact that paternally silenced insertions covary with language diversity is tantalizing from a parental antagonism perspective. Indeed the current findings point to the interesting possibility that *Alu* paternal silencing functions to reduce intragenomic conflicts in cultural groups that may have higher than average relatedness asymmetries, a possible positive correlate of living in areas with more outgroup languages. For example, maintaining cooperative networks among asymmetric collateral kin may involve silencing potentially disruptive paternally derived genetic elements. Future work may wish to test whether *Alu* paternal silencing correlates with socially transmitted signals of relatedness asymmetries and correlates positively with linguistically mediated cooperation among kin, as would be expected by parental antagonism theory. Regardless of the causes of these associations, it is noteworthy that linguistic diversity (or one of its associated correlates) may well affect epigenetic gene silencing in a pattern possibly consistent with parental antagonism theory. Recently, Van Cleve et al. (2010) mathematically modeled that when selection is stronger on one sex, there would be an increased benefit to gene expression of an allele transmitted by the parent of the sex under weaker selection, regardless of sex-biased dispersal. Van Cleve et al. (2010) suggest that parental antagonisms may be driven by factors other than sex-biased dispersal and multiple paternity (recall these are considered to be the primary forces that drive intragenomic conflicts). Thus, based on the Van Cleve et al. (2010) model, future work should look at how *Alu* element silencing is affected by population demography, life history and factors associated with kinship.

Because *Alu* elements have often been considered "junk DNA" or molecular parasites (i.e., genes that play no functional or regulatory roles) (see Muotri et al. 2007), making them ideal for testing the cultural hitchhiking hypothesis proposed by Whitehead (1998). However, recent studies suggest that *Alu* elements regulate gene expression in humans (Hasler and Strub 2006). Despite *Alu* elements being genetically functionless, they may affect gene structures, protein sequences, splicing motifs, and expression patterns (Dagan et al. 2004). The TranspoGene database, formally known as AluGene (http://transpogene.τ.ac.il/) provides a complete *Alu* map of the human genome, whereby each *Alu* element is annotated with respect to coding region and exon/intron location.

To investigate whether *Alu* elements are particularly important for imprinted language loci expression, each language loci from Table 1 (imprinted and not imprinted) was entered into the TranspoGene database to determine the frequency of *Alu* elements that have inserted themselves into the protein-coding areas of the loci in question. As predicted, there was significantly higher than expected frequency of *Alu* elements inserted in the protein coding machinery of imprinted language loci (n = 472) versus the number of *Alu* elements inserted into nonimprinted autosomal language loci (124): $\chi^2 (1) = 203.20$, $p < 0.001$. A similar pattern was revealed for the X-chromosomal language loci. Specifically, there was significantly higher than expected frequency of *Alu* elements inserted

into the protein coding machinery of X-chromosomal language loci (281) versus the number of *Alu* elements inserted into nonimprinted autosomal language loci (95): $\chi^2(1) = 60.86$, $p < 0.001$. Future research will need to investigate how *Alu* elements contribute to imprinted gene expression and X-chromosomal loci.

In the following sections of this paper previous theories of language evolution will be presented, and there will be a discussion of how each may (or may not) relate to parental antagonism theory. A demarcation between individual-benefit and group-benefit accounts will be made.

Previous Theories of Language Evolution

An evolutionary explanation for language need not (and probably should not) propose the same adaptive function across ontogeny as fitness contingencies change. Proposing a unitary function for language irrespective of an individual's life history is a major weakness to most theories of language evolution (for review see Szamado and Szathmary 2006). In light of this criticism, parental antagonism theory may be more important for understanding the development of language abilities, as opposed to the varied uses or elaborations of adult language. Earlier in the paper it was claimed that the parental antagonism theory of language evolution is preferable to individual-benefit and group-benefit approaches. The reason this is the case is that parental antagonism hypotheses that language serves both manipulative and cooperative functions, a point that is rarely considered by most theories of language evolution.

Below I present some of the previous hypotheses for the evolution of language and reinterpret them in light of parental antagonism theory. In general, previous theories of language evolution can be categorized as individual- or group-level benefit explanations. In each case, testable hypotheses will be provided to distinguish between the alternative accounts.

Individual-Benefit Hypotheses

The mental toolkit hypothesis.   This hypothesis argues that language evolved primarily to facilitate cognition and was later co-opted for the purpose of communication (Burling 1993). This hypothesis suggests that internal mental life is a way to organize future behavior. According to this hypothesis our internal mental life should be unified; however, this does not seem to be the case (e.g., a moderate level of schizotypy is typical part of human cognitive function) (Nettle and Clegg 2006). It is important to note that schizotypal aspects of human cognition are primarily language-based and figure prominently in some functional accounts of language (e.g., Crow 2000; Mitchell and Crow 2005).

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The mental toolkit hypothesis seems to ignore internal neurocognitive conflicts. It is important to note, however, that internal neurocognitive conflict could be an individual level adaptation, a malfunction of a complex neural system or a manifestation of internal genetic conflicts (Haig 2006). This hypothesis is difficult to test, but has been elucidated in the study of REM sleep

states. Specifically REM sleep states are largely regulated by patrigenes (McNamara 2004). Perhaps intragenomic conflicts occur during sleeping and waking mental states. If McNamara (2004) is right, then rather than providing us with an adaptive mental toolkit for better decisions, imprinted language loci could drive cognition in opposite directions, depending on the relatedness asymmetries a child experiences during development. The most obvious distinction between the mental toolkit and parental antagonism theories is that the latter does not presume that internal mental conflicts improve cognition at the individual level.

The mating contract hypothesis.   It has been hypothesized that increasing hominin group size and a need for male provisioning selected for linguistically based social contracts between the sexes (Deacon 1997). This hypothesis could be reframed in terms of sexually antagonistic coevolution, whereby good genes for one sex may not be beneficial for the other (Rice and Holland 1997). Sexual and parental antagonism theories are not necessarily mutually exclusive, especially in the case of sex-chromosome imprinting (Gregg et al. 2010b). Regardless, the parental antagonism hypothesis proposes that these two capacities are separable insofar that the language and social cognitive phenotype benefits both matrigenes and patrigenes differentially. Specifically, a child's theory of mind may be used to manipulate mother's parental investment to the benefit of patrigenes, but it could also be used by matrigenes to increase male provisioning. Parental antagonism theory does specify (unlike the mating contract hypothesis) that matrigenes (e.g., on the X-chromosome) will mediate the cooperative use of language and that this will develop first within the family as opposed to mating contexts.

The sexual selection hypothesis.   This hypothesis argues that language elaborate sexually selected ornament, which enabled females to assess the quality of a male (Miller 2001). Investment in sexual display traits appears to be less subject to parental antagonism because there should be few parental disagreements over mate selection criteria. However, when mating with a particular

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individual costs matrilineal kin more than patrilineal kin, intrapersonal conflicts are expected to occur. For example, socially transmitted norms discouraging incestuous relations among close kin is an example of where parental antagonism theory could apply (Brown 2001). The costs of inbreeding to the matriline are higher than the patriline, because paternal relatedness between sibs will be lower on average compared with maternal relatedness. Interestingly, urinary odor preferences are transmitted via maternal genes in mice, consistent with the hypothesis that maternal genes are critical for the development of olfactory mechanisms to avoid inbreeding (Isles et al. 2001). In the case of language, the ease to which we socially acquire verbally transmitted norms to avoid mating with close kin is expected to be influenced by the actions of matrigenes within the child (Brown 2001).

If aspects of language are sexually selected, then sex-specific fitness costs and benefits could emerge. It has been suggested that genomic imprinting mechanisms could have facilitated sex-specific trait expression and ameliorate the costs of sexually antagonistic genes where good genes in one sex are bad for the other (Day and Bonduriansky 2004). Another source of parental antagonism may occur due to sex-specific trait expression. For example, if "sexy sons" damage female-specific fitness (e.g., due to decreased parental investment in offspring), then matrigenes in sons could express themselves in such a way as to minimize such costs to the matriline. Essentially, verbally skilled social manipulators may decrease the fitness of female-specific genes. Thus, the maternal X-chromosomes in male offspring are predicted to reduce these costs on the matriline.

Motherese. According to the motherese hypothesis, language evolved because mothers could not simultaneously carry offspring and forage efficiently, so to calm their infants, ancestral mothers used vocal communication (Falk 2004). This hypothesis is most obviously linked to parental antagonism theory, in that it involves parental investment and mother-infant bonding. To distinguish between the two theories, evidence for genetic disagreement within the child is needed. Specifically, patrigene expression is predicted to interfere with a child's ability to respond to motherese, whereas matrigenes will encourage the child to be susceptible to the calming effects of language.

Group Benefit Hypotheses

The gossip hypothesis. Tightly committed female groups have strong incentives to share information about one another (i.e., gossiping), and this force selected for human language (Power 1998). According to parental antagonism theory, female groups may be tightly related provided there was a history of male-biased dispersal (also see Úbeda and Garner 2010 for a discussion of how sex-specific variance in reproductive success can cause a similar pattern). When there is male-biased dispersal, offspring will be more closely related to collateral kin matrilineally than patrilineally (e.g., $r^{pat} = 0$). This creates a strong relatedness asymmetry and parental antagonistic effects may escalate. Thus, language abilities for signaling and receiving gossip designed to increase social cohesion will be caused by the effects of matrigenes. However, signaling and receiving negative gossip (i.e., spreading false information regarding matrilineal collateral kin) would be selected for by patrigenes because they are no more related to others in the group than strangers. The reason that parental antagonism is a more powerful account for gossip is that it explains positive and negative gossip and makes differential predictions regarding the content of gossip mediated by differential parental genome expression. The group bonding through ritual hypothesis (Knight 1998) is similar to Power's (1998) hypothesis and posits that language evolved in a context of between group female rituals, whereby female participants perform a "strike action" against low parental investing males. Once again, provided matrilineal coefficients of

relatedness among kin are higher relative to patrilineal coefficients of relatedness, cooperative norms will spread via matrigenes. In contrast to Knight's (1998) hypothesis increased female cooperation via language is predicted to be associated with sex-specific imprinting on the X-chromosome. Recent work in mice by Gregg et al. (2010b), shows that sex-specific imprinted genes are mostly found in females and have influence on hypothalamic function, a neural area associated thermoregulation, mating, and maternal behavior.

The grooming hypothesis.   According to the grooming hypothesis, language evolved as a substitution for physical grooming found in many nonhuman primate lineages (Dunbar 1998). The selection pressure for verbal or symbolic grooming was the increasing size of the early hominin groups, making physical grooming costly in terms of time and effort. Much like the gossip hypothesis, the grooming hypothesis posits that verbal communication makes for more cohesive groups. However, parental antagonism theory alternatively proposes that group social cohesion is the consequence of matrilineal inclusive fitness maximization mediated by matrigenes when there are relatedness asymmetries. That is, cooperative behavior between collateral kin (i.e., positive information sharing) will be selected matrilineally when there are relatedness asymmetries (i.e., when maternal coefficients are higher than paternal). Specifically, learning to vocalize prosocial utterances and adopt norms to be cooperative with maternal relatives are consequences of matrigene expression within children. Disruptions of these processes will be selected for at the patrigene level.

The hunting hypothesis.   Washburn and Lancaster (1968) and Hewes (1973) hypothesized that language evolved to coordinate hunting activities. For example, gestural symbols could coordinate cooperative hunting efficiency without alerting prey. Parental antagonisms over cooperative hunting ventures have not been considered previously. A division of labor can make for more efficient hunting parties; however, there is a potential for free-riding because tracking others' efforts may be difficult. High degrees of matrilineal relatedness among hunters would favor increased cooperative gestures during hunts, whereas low patrilineal relatedness would favor free-riding on the hunting efforts of others (i.e., when the kill is shared equally with paternal nonrelatives).

Also worth considering are offspring food preferences and how these could exert pressures on parental foraging patterns. It has been suggested that matrigenes would be selected to minimize maternal foraging costs by causing nonchoosy eating habits among offspring (Haig and Wharton 2003). Further, maternal gene expression in offspring could favor fewer verbal demands for maternally acquired foods. Hinton et al. (2006) in a positron emission tomography study of individuals with PWS, a condition where there is an overexpression of matrigenes, it was found that the areas of the brain associated with food reward (i.e., amygdala and orbitofrontal cortex) did not show an increased response to high incentive foods (e.g., high-caloric sugar-based foods). If hunting and

gathering was a sexual division of labor for most of hominin evolutionary history, then it would be expected that maternal genes would favor a decreased preference for foods foraged by mother and an accelerated demand for foods acquired by father or maternally unrelated kin (e.g., meat). Thus the actions of matrigenes could place higher demands on males to cooperatively hunt.

Conclusions

It has been reported that imprinted genes are more likely than expected to be involved in language phenotypes. Genes with language effects also appeared more often on the X-chromosome given its size. Initially it was predicted that human language-rich chromosomes such as 7 and 15 would exhibit less similarity to chimpanzees in overlapping genes (compared with other chromosomes); however, this was not the case. Indeed it was chromosome 11 that exhibited the biggest difference between chimps' and humans' overlapping imprinted genes. This was considered notable in retrospect because schizophrenia, a disease with language impairments (Mitchell and Crow 2005), is associated with chromosome 11 imprinting (Abel 2004). It has also been reported that maternally expressed overlapping genes show less similarity to chimpanzees compared with paternally expressed overlapping genes. Finally, it was found that the frequency of paternally silenced *Alu* insertions (but not maternally silenced *Alu* insertions) were positively associated with language diversity in a sample of 30 cultures. Furthermore, a higher than expected frequency of *Alu* elements have inserted themselves into the protein-coding machinery of imprinted and X-chromosomal loci. These novel empirical findings provide preliminary evidence in support of the parental antagonism theory of language.

This paper proposed that it is incorrect to assume that human language must have evolved as a system of harmonious cooperation. When cooperation is observed, it must be explained rather than assumed. In contrast, the parental antagonism theory of language predicts cooperation *and* conflict over information transmission (whether sent or received), depending on the asymmetries in the coefficients of relatedness between signalers and receivers. Language can be manipulative, and organisms are expected to transmit information to shift the receiver from its fitness optimum; and receivers will be selected to avoid these exploitation attempts. In sum, the parental antagonism theory of language evolution hypothesizes that early in ontogeny signals of need will be mediated by patrigene expression to the benefit (but at a cost to matrilineal) of patrilineal inclusive fitness, whereas signals of satiation of needs will be mediated by matrigene expression within the child in order to counteract the costly effects of patrigenes. As children age, it is predicted that matrigene expression will favor the transmission and reception of socially transmitted linguistic information favoring kin-based cooperation, whereas patrigenes will attempt to counteract these maternal effects provided that patrilineal relatedness is low (relative to matrilineal relatedness). These predictions are specific in terms of social ecological forces and expression patterns of language-associated loci, making the

paternal antagonism theory of language evolution amiable to further empirical scrutiny.

In conclusion, Haig (2010) based on parental antagonism theory has called for longitudinal work on childhood feeding adaptations. Likewise, to test the parental antagonism theory of language presented here, longitudinal studies of language development are needed to investigate how language relates to parental investment and cooperation among asymmetric kin across the life course and across cultures. Another avenue of future empirical exploration of parental antagonism theory is to employ a sociogenomics perspective (Robinson 1999) on imprinted language loci across the life course and in different social ecologies. Regardless of the empirical success of a parental antagonism view of language development it is reasonable to believe that clinical value (Haig 2010) could be gained from long-term investigations of imprinted diseases that have language disorder sequelae.

Literature Cited

Abel, K. M. 2004. Foetal origins of schizophrenia: Testable hypotheses of genetic and environmental influences. *Br. J. Psychiatry* 184:383–385.

Ainsworth, M. D. S. 1979. Attachment as related to mother—infant interaction. *Adv. Study Behav*. 9:2–52.

Allen, N. D., K. Logan, G. Lally et al. 1995. Distribution of parthenogenetic cells in the mouse brain and their influence on brain development and behavior. *Proc. Natl. Acad. Sci. USA.* 92:10782–10786.

Altmann, J. 1980. *Baboon Mothers and Infants.* Cambridge, MA: Harvard University Press.

Arking, D. E., D. J. Cutler, C. W. Brune et al. 2008. A common genetic variant in the neurexin superfamily member *CNTNAP2* increases familial risk of autism. *Am. J. Hum. Genet.* 82:160–164.

Arney, K. L. 2003. H19 *and Igf2*-enhancing the confusion? *Trends Genet.* 19:17–23.

Asmus, F., A. Zimprich, S. Tezenas du Montcel et al. 2002. Myoclonus dystonia syndrome: Epsilon-sarcoglycan mutations and phenotype. *Ann. Neurol.* 52:489–492.

Badcock, C. and B. J. Crespi. 2006. Imbalanced genomic imprinting in brain development: An evolutionary basis for the aetiology of autism. *J. Evol. Biol.* 19:1007–1032.

Bittel, D. C., N. Kibiryeva, S. G. McNulty et al. 2007. Whole genome microarray analysis of gene expression in an imprinting center deletion mouse model of Prader-Willi syndrome. *Am. J. Med. Genet.* 143A:422–429.

Bowlby, J. 1969. Attachment and Loss, Vol. 1: *Attachment.* New York, NY: Basic Books.

Brandvain, Y. 2010. Matrisibs, patrisibs, and the evolution of imprinting on autosomes and sex chromosomes. *Am. Nat.* 176:511–521.

Brown, W. M. 2001. Genomic imprinting and the cognitive architecture mediating human culture. *J. Cogn. Culture* 1:251–258.

Brown, W. M. 2004. Evolved cognitive architecture mediating fear: A genomic conflict approach. In: *The Psychology of Fear,* P. L. Gower, ed. New York, NY: Nova Science Publishers, 171–182.

Brown, W. M. 2008. Sociogenomics for the cognitive adaptationist. In *Foundations of Evolutionary Psychology,* C. Crawford and D. Krebs, eds. Hillsdale, NJ: Psychology Press/Lawrence Erlbaum, 171–182.

Brown, W. M., and N. S. Consedine. 2004. Just how happy is the happy puppet? An emotion signalling and kinship theory perspective on the behavioral phenotype of children with Angelman syndrome. *Med. Hypotheses* 63:377–385.

Burling, R. 1993. Primate calls, human language, and nonverbal communication. *Curr. Anthropol.* 34:25–53.

Campos, R. L. R., K. Nones, M. C. Ledur et al. 2009. Quantitative trait loci associated with fatness in a broiler-layer cross. *Anim. Genet.* 40:729–736.

Cannon, B., and J. Nedergaard. 2004. Brown adipose tissue: Function and physiological significance. *Phys. Rev.* 84:277–359.

Chamberlain, S. J., and M. Lalande. 2010. Neurodevelopmental disorders involving genomic imprinting at human chromosome 15q11–q13. *Neurobiol. Dis.* 39:13–20.

Clayton-Smith, J., and L. Laan. 2003. Angelman syndrome: A review of the clinical and genetic aspects. *J. Med. Genet.* 40:87–95.

Collette, J. C., C. Xiao-Ning, D. L. Mills et al. 2009. William's syndrome: Gene expression is related to parental origin and regional coordinate control. *J. Hum. Genet.* 54:193–198.

Crespi, B. J. 2007. Sly *FOXP2*: Genomic conflict in the evolution of language. *Trends Ecol. Evol.* 22 (4):174–175.

Crespi, B. J. 2008. Language unbound: Genomic conflict and psychosis in the origin of modern humans. In *Sociobiology of Communication: An Interdisciplinary Perspective*, P. d'Ettorre and D. Hughes, eds. Oxford, U.K.: Oxford University Press: 225–249.

Curley, J. P., and R. Mashoodh. 2010. Parent-of-origin and trans-generational germline influences on behavioral development: The interacting roles of mothers, fathers, and grandparents. *Dev. Psychobiol.* 52:312–330.

Crow, T. J. 2000. Schizophrenia as the price that *Homo sapiens* pays for language: A resolution of the central paradox in the origin of the species. *Brain Research Reviews* 31:118–129.

da Rocha, S. T., M. Tevendale, E. Knowles et al. 2007. Restricted co-expression of Dlk1 and the reciprocally imprinted non-coding RNA, Gtl2: Implications for cis-acting control. *Dev. Biol.* 306:810–823.

Dagan, T., R. Sorek, E. Sharon et al. 2004. *Alu*Gene: A database of *Alu* elements incorporated within protein-coding genes. *Nucleic Acids Res.* 32:D489–D492.

Day, T., and R. Bonduriansky. 2004. Intralocus sexual conflict can drive the evolution of genomic imprinting. *Genetics* 167:1537–1546.

Deacon, T. 1997. *The Symbolic Species.* New York, NY: Penguin Books.

Didden, R., H. Korzilius, P. Duker et al. 2004. Communicative functioning in individuals with Angelman syndrome: A comparative study. *Disabl. Rehabil.* 26:1263–1267.

de Bono, M., and C. I. Bargmann 1998. Natural variation in a neuropeptide Y receptor homolog modifies social behavior and food response in *C. elegans. Cell* 94:679–689.

Dunbar, R. 1998. Theory of mind and the evolution of language. In *Approaches to the Evolution of Language,* J. R. Hurford, M. Studdert-Kennedy, and C. Knight, eds. Cambridge, U.K.: Cambridge University Press, 92–110.

Falk, D. 2004. Prelinguistic evolution in early hominins: Whence motherese? *Behav. Brain Sci.* 27:491–503.

Fernald, A., and P. Kuhl. 1987. Acoustic determinants of infant preference for motherese speech. *Infant Behav. Dev.* 10:279–293.

Feuk, L., A. Kalervo, M. Lipsanen-Nyman et al. 2006. Absence of a paternally inherited FOXP2 gene in developmental verbal dyspraxia. *Am. J. Hum. Genet.* 79:965–972.

Fincher, C. L., and R. Thornhill. 2008. A parasite-driven wedge: Infectious diseases may explain language and other biodiversity. *Oikos* 117:1289–1297.

AQ: JJ

AQ: KK

AQ: LL

Francis, D., J. Diorio, D. Liu et al. 1999. Nongenetic transmission across generations of maternal behavior and stress responses in the rat. *Science* 286:1155–1158.

Gibson, J. R., A. K. Chippindale, and W. R. Rice. 2002. The X chromosome is a hot spot for sexually antagonistic fitness variation. *Proc. Roy. Soc. Lond. Ser. B Biol. Sci.* 269:499–505.

Grabowski, M., A. Zimprich, B. Lorenz-Depiereux et al. 2003. The epsilon-sarcoglycan gene (*SGCE*), mutated in myoclonus-dystonia syndrome, is maternally imprinted. *Eur. J. Hum. Genet.* 11:138–144.

Greer, P. L., R. Hanayama, B. L. Bloodgood, A. R. Mardinly, D. M. Lipton, S. W. Flavell, T. K. Kim, E. C. Griffith, Z. Waldon, R. Maehr, H. L. Ploegh, S. Chowdbury, P. F. Worley, J. Steen, and M. E. Greenberg. 2010. The Angelman Syndrome protein Ube3a regulates synapse development by ubiquitinating arc. *Cell* 140:704–716.

Gregg, C., J. Zhang, B. Weissbourd et al. 2010a. High-resolution analysis of parent-of-origin allelic expression in the mouse brain. *Science* 329:643–648.

Gregg, C., J. Zhang, J. Butler et al. 2010b. Sex-specific parent-of origin allelic expression in the mouse brain. *Science* 329:682–685.

Haesler, S., K. Wada, A. Nshdejan et al. 2004. FoxP2 expression in avian vocal learners and non learners. *J. Neurosci.* 24:3164–3175.

Haig, D. 1997. Parental antagonism, relatedness asymmetries, and genomic imprinting. *Proc. Roy. Soc. Lond. B Biol. Sci.* 264:1657–1662.

Haig, D. 1999a. Multiple paternity and genomic imprinting. *Genetics* 151:1229–1231.

Haig, D. 1999b. Genetic conflicts of pregnancy and childhood. In *Evolution in Health and Disease,* S. C. Stearns, ed. Oxford, U.K.: Oxford University Press, 7–90.

Haig, D. 2000. Genomic imprinting, sex-biased dispersal, and social behavior. *Ann. NY Acad. Sci.* 907:149–163.

Haig, D. 2002. *Genomic Imprinting and Kinship.* Piscataway, NJ: Rutgers University Press.

Haig, D. 2003. On intrapersonal reciprocity. *Evol. Hum. Behav.* 24:418–425.

Haig, D. 2006a. Intrapersonal conflict. In *Conflict,* M. K. Jones and A. Fabian, eds. Cambridge, U.K.: Cambridge University Press: 8–22.

Haig, D. 2006b. Intragenomic politics. *Cytogenet. Genome Res.* 113:68–74.

Haig, D. 2006c. Self-imposed silence: Parental antagonism and the evolution of X chromosome inactivation. *Evolution.* 60:440–447.

Haig, D. 2008. Huddling: Brown fat, genomic imprinting and the warm inner glow. *Curr. Biol.* 18:R172–R175.

Haig, D. 2010. Transfers and transitions: Parent-offspring conflict, genomic imprinting, and the evolution of human life history. *Proc. Natl. Acad. Sci. USA* 107:1731–1735.

Haig, D., and R. Wharton. 2003. Prader–Willi syndrome and the evolution of human childhood. *Am. J. Hum. Biol.* 15:320–329.

Hamelin, C. E., G. Anglin, C. A. Quigley, and C. L. Deal. 2006. Genomic imprinting in Turner syndrome: Effects on response to growth hormone and on risk of sensorineural hearing loss. *J. Clin. Endocrinol. Metab.* doi:10.1210/jc.2006-0490

Hamilton, W. D. 1964. The genetical evolution of social behaviour I and II. *J. Theoret. Biol.* 7:1–52.

Harlow, H. F. 1958. The nature of love. *Am. Psychol.* 13:673–685.

Hasler, J., and K. Strub. 2006. Alu elements as regulators of gene expression. *Nucleic Acids Res.* 34:5491–5497.

Hewes, G. 1973. Primate communication and the gestural origin of language. *Curr. Anthropol.* 14:5–25.

Hinde, R. A. 1976. On describing relationships. *J. Child Psychol. Psychiatry* 17:1–19.

Hinton, E. C., A. J. Holland, M. S. N. Gellatly et al. 2006. An investigation into food preferences and the neural basis of food-related incentive motivation in Prader–Willi syndrome. *J. Intellect. Disabil. Res.* 50:633–642.

Isles, A. R., and L. S. Wilkinson. 2000. Imprinted genes, cognition and behaviour. *Trends Cogn. Sci.* 4:309–318.

AQ: MM

Isles, A. R., M. J. Baum, D. Ma, E. B. Keverne, and N. D. Allen. 2001. Genetic imprinting: Urinary odour preferences in mice. *Nature* 409:783–784.

Jiang, Y., Y. Pan, L. Zhu, L. Landa, J. Yoo et al. 2010. Altered ultrasonic vocalization and impaired learning and memory in Angelman Syndrome mouse model with a large maternal deletion from Ube3a to Gabrb3. *PLoS ONE* 5(8):e12278. doi:10.1371/journal.pone.0012278.

Jones, W., U. Bellugi, Z. Lai et al. 2000. Hypersociability in William's syndrome. *J. Cogn. Neurosci.* 12:S30–S46.

Jurka, J., M. Krnjajic, et al. 2002. Active Alu elements are passed primarily through paternal germlines. *Theor. Popul. Biol.* 61:519–530.

Kass, D. H., N. Jamison, M. M. Mayberry et al. 2007. Identification of a unique Alu-based polymorphism and its use in human population studies. *Gene* 390:146–152.

Keller, S., M. Sarchiapone, F. Zarrilli et al. 2010. Increased BDNF promoter methylation in the Wernicke area of suicide subjects. *Arch. Gen. Psychiatry* 67:258–267.

Keverne, E. B., F. L. Martel, and C. M. Nevison. 1996a. Primate brain evolution: Genetic and functional considerations. *Proc. Roy. Soc. Biol. Sci. Part B* 263:689–696.

Keverne, E. B., R. Fundele, M. Narasimha et al. 1996b. Genomic imprinting and the differential roles of parental genomes in brain development. *Dev. Brain Res.* 92:91–100.

Kim, D., C. Cho, J. Huh et al. 2009. EVOG: A database for evolutionary analysis of overlapping genes. *Nucleic Acids Res.* 37:D698–D702.

Klar, A. J. S. 2004. A genetic mechanism implicates chromosome 11 in schizophrenia and bipolar diseases. *Genetics* 167:1833–1840.

Knight, C. 1998. Ritual/speech coevolution: A solution to the problem of deception. In *Approaches to the Evolution of Language,* J. R. Hurford, M. Studdert-Kennedy, and C. Knight, eds. Cambridge, U.K.: Cambridge University Press, 68–91.

Lander, E. S., L. M. Linton et al. 2001. Initial sequencing and analysis of the human genome. *Nature* 409:860–921.

Lieberson, S. 1981. *Language Diversity and Language Contact*. Stanford, CA: Stanford University Press.

Liu, D., J. Diorio, B. Tannenbaum et al. 1997. Maternal care, hippocampal glucocorticoid receptors, and hypothalamic-pituitary-adrenal responses to stress. *Science* 277:1659–1662.

Lorenzi, C., G. Gilbert, H. Carn, S. Garnier, and B. C. J. Moore. 2006. Speech perception problems of the hearing impaired reflect inability to use temporal fine structure. *Proc. Nat. Acad. Sci. USA* 103:18866–18869.

Makedonski, K., L. Abuhatzira, Y. Kaufman et al. 2005. MeCP2 deficiency in Rett syndrome causes epigenetic aberrations at the PWS/AS imprinting center that affects *UBE3A* expression. *Hum. Mol. Genet.* 14:1049–1058.

McGowan, P. O., A. Sasaki, T. C. T. Huang et al. 2008. Promoter-wide hypermethylation of the ribosomal RNA gene promoter in the suicide brain. *PLoS ONE* 3(5):e2085. doi:10.1371/journal.pone.0002085.

McNamara, P. 2004. Genomic imprinting and neurodevelopmental disorders of sleep. *Sleep Hypnosis* 6:82–90.

McNamara, P., J. Dowdall, and S. Auerbach. 2002. REM sleep, early experience, and the development of reproductive strategies. *Hum. Nat.* 13:405–435.

Meaney, M. J. 2001. Maternal care, gene expression, and the transmission of individual differences in stress reactivity across generations. *Annu. Rev. Neurosci.* 24:1161–1192.

Messinger, D., M. Dondi, G. C. Nelson-Goens et al. 2002. How sleeping neonates smile. *Dev. Sci.* 5:48–54.

Mikkelsen, T. S., L. D. Hillier, E. E. Eichler et al. 2005. Initial sequence of the chimpanzee genome and comparison with the human genome. *Nature* 437:69–87.

Miller, G. 2001. *The Mating Mind.* New York, NY: Anchor Books.

Mitchell, R. L. C., and T. J. Crow. 2005. Right hemisphere language functions and schizophrenia: The forgotten hemisphere? *Brain* 128:963–978.

AQ: NN

Muglia, P., A. M. Vicente, M. Verga, N. King, F. Macciardi, and J. L. Kennedy. 2003. Association between the BDNF gene and schizophrenia. *Molecular Psychiatry* 8:146–147. doi:10.1038/sj.mp.4001221.

Muotri, A. R., M. C. Marchetto, N. G. Coufal et al. 2007. The necessary junk: New functions for transposable elements. *Hum. Mol. Genet.* 16:R159–R167.

Murphy, S. K., and R. L. Jirtle. 2003. Imprinting evolution and the price of silence. *BioEssays* 25:577–588.

Nettle, D., and H. Clegg. 2006. Schizotypy, creativity and mating success in humans. *Proc. Roy. Soc. B Biol. Sci.* 273:611–615.

Oliver, C., K. Horsler, K. Berg et al. 2007. Genomic imprinting and the expression of affect in Angelman syndrome: What's in the smile? *J. Child Psychol. Psychiatry* 48(6):571–579.

O'Neill, M. J., R. S. Ingram, P. B. Vrana et al. 2000. Allelic expression of *IGF2* in marsupials and birds. *Dev. Genes Evol.* 210:18–20.

Perez-Jurado, L. A., R. Peoples, P. Kaplan et al. 1996. Molecular definition of the chromosome 7 deletion in Williams syndrome and parent-of-origin effects on growth. *Am. J. Hum. Genet.* 59:781–792.

Pinker, S., and R. Jackendoff. 2005. The faculty of language: What's special about it? *Cognition* 95:201–236.

Power, C. 1998. Old wives' tales: The gossip hypothesis and the reliability of cheap signals. In *Approaches to the Evolution of Language,* J. R. Hurford, M. Studdert-Kennedy, and C. Knight, eds., Oxford, U.K.: Cambridge University Press, 111–129.

Portfors, C. V. 2007. Types and functions of ultrasonic vocalizations in laboratory rats and mice. *J. Amer. Assoc. Lab. Anim. Sci.* 46:128–134.

Reik, W., and J. Walter. 2001. Genomic imprinting: Parental influence on the genome. *Nat. Rev. Genet.* 2:21–32.

Rice, W. R. 1984. Sex chromosomes and the evolution of sexual dimorphism. *Evolution* 38:735–742.

Rice, W. R., and B. Holland. 1997. The enemies within: Intergenomic conflict, interlocus contest evolution (ICE), and the intraspecific Red Queen. *Behav. Ecol. Sociobiol.* 41:1–10.

Robinson, G. 1999. Integrating animal behaviour and sociogenomics. *Trends Ecol. Evol.* 14:202–205.

Rowe, S. J., R. Pong-Wong, C. S. Haley et al. 2009. Detecting parent of origin and dominant QTL in a two-generation commercial poultry pedigree using variance component methodology. *Genet. Selection Evol.* 41:1–11.

Rowold, D. J. and R. J. Herrera. 2000. Alu elements and the human genome. *Genetica* 108:57–72.

Roy-Engel, A. M., M. L. Carroll, E. Vogel, R. K. Garber, S. V. Nguyen, A. H. Salem, M. A. Batzer, and P. L. Deininger. 2001. Alu insertion polymorphisms for the study of human genomic diversity. *Genetics* 159:279–290.

Runte, M., A. Huttenhofer, S. Gross, M. Kiefmann, B. Horsthemke, K. Buiting. 2001. The IC-*SNURF-SNRPN* transcript serves as a host for multiple small nucleolar RNA species and as an antisense RNA for *UBE3A*. *Hum. Mol. Genet.* 10:2687–2700.

Ryan, M. J. 1990. Sexual selection, sensory systems, and sensory exploitation. *Oxf. Surv. Evol. Biol.* 7:157–195.

Samaco, R. C., A. Hogart, and J. M. LaSalle. 2005. Epigenetic overlap in autism spectrum neurodevelopmental disorders: *MECP2* deficiency causes reduced expression of UBE3A and GABRB3. *Hum. Mol. Genet.* 14:483–492.

Sandovici, I., S. Kassovska-Bratinova, J. C. Loredo-Osti et al. 2005. Interindividual variability and parent of origin DNA methylation differences at specific human Alu elements. *Hum. Mol. Genet.* 14:2135–2143.

Schoenmakers, S., E. Wassenaar, J. W. Hoogerbrugge et al. 2009. Female meiotic sex chromosome inactivation in chicken. *PloS Genet.* 5:e1000466. doi: 10.1371/journal.pgen.

Shen, M. R., M. A. Batzer, and P. L. Deininger. 1991. Evolution of the master Alu gene(s). *J. Mol. Evol.* 33:311–320.

Smit, A. F. 1999. Interspersed repeats and other mementos of transposable elements in mammalian genomes. *Curr. Opin. Genet. Dev.* 9:657–663.

AQ: QQ

AQ: SS

AQ: UU

Smit, H. 2009. Genomic imprinting and communicative behaviour: Prader-Willi and Angelman syndrome, *Netherlands Journal of Psychology* 65:78–88.

Smit, H. 2010. A conceptual contribution to battles in the brain. *Biol. Philos.* 25:803–821.

Sullivan, K., and H. Tager-Flusberg. 1999. Second-order belief attribution in Williams syndrome: Intact or impaired? *Am. J. Ment. Retard.* 104:523–532.

Szamado, S., and E. Szathmary. 2006. Selective scenarios for the emergence of natural language. *Trends Ecol. Evol.* 21:555–561.

Tomasello, M. 2008. *Origins of Human Communication*, Cambridge, MA:MIT Press.

Tomasello, M. 2009. *Why We Cooperate.* Cambridge, MA: MIT Press.

Trivers, R. 1972. Parental investment and sexual selection. In: *Sexual Selection and the Descent of Man 1871–1971,* B. Campbell, ed. Chicago, IL: Aldine Press, 139–179.

Trivers, R. 2000. The elements of a scientific theory of self deception. *Ann. NY Acad. Sci.* 907:114–131.

Úbeda, F., and A. Gardner. 2010. A model for genomic imprinting in the social brain: Juveniles. *Evolution* 64:2587–2600.

Úbeda, F., and A. Gardner. 2011. A model for genomic imprinting in the social brain: Adults. *Evolution* 65:462–475.

Van Cleve, J., M. W. Feldman, and L. Lehmann. 2010. How demography, life history, and kinship shape the evolution of genomic imprinting. *Am. Nat.* 176:400–455.

Vernes, S. C., D. F. Newbury, B. S. Abrahams, L. Winchester, J. Nicod, M. Groszer, M. Alarcón, P. L. Oliver, K. E. Davies, D. H. Geschwind, A. P. Monaco, S. E. Fisher. 2008. A functional genetic link between distinct developmental language disorders. *N. Engl. J. Med.* 359:2337–2345.

Voight, B. F., S. Kudaravalli, X. Wen et al. 2006. A map of recent positive selection in the human genome. *PLoS Biol.* 4(3):e72.

Washburn, S., and C. Lancaster. 1968. The evolution of hunting, in *Man the Hunter.* R. B. Lee and I. Devore, eds., Chicago, IL: Aldine, 293–303.

Waterland, R. A., and R. L. Jirtle. 2004. Early nutrition, epigenetic changes at transposons and imprinted genes, and enhanced susceptibility to adult chronic diseases. *Nutrition.* 20:63–68.

Watkins, W. S., A. R. Rogers, C. T. Ostler et al. 2003. Genetic variation among world populations: Inferences from 100 Alu insertion polymorphisms. *Genome Res.* 13:1607–1618.

Wawrzik, M., U. A. Unmehopa, D. F. Swaab, J. van de Nes, K. Buiting, and B. Horsthemke. 2010 The *C15orf2* gene in the Prader -Willi syndrome region is subject to genomic imprinting and positive selection. *Neurogenetics* 11:153–161.

Weaver, I. C. G., M. J. Meaney, and M. Szyf. 2006. Maternal care effects on the hippocampal transcriptome and anxiety-mediated behaviors in the offspring that are reversible in adulthood. *Proc. Natl. Acad. Sci. USA.* 103:3480–3485.

Whitehead, H. 1998. Cultural selection and genetic diversity in matrilineal whales. *Science* 282:1708–1711.

Williams, K., D. A. Irwin, D. G. Jones, K. M. Murphy. 2010. Dramatic loss of Ube3A expression during aging of the mammalian cortex. Frontiers in Aging Neuroscience 2:1–9.

Yashiro, K., T. Riday, K. Condon et al. 2009. *Ube3A* is required for experience-dependent maturation of the neocortex. *Nat. Neurosci.* 12:777–783.

Zechnera, U., M. Wildab, H. Kehrer-Sawatzkib et al. 2001. A high density of X-linked genes for general cognitive ability: A run away process shaping human evolution? *Trends Genet.* 17:697–701.

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