

A MODEL FOR GENOMIC IMPRINTING IN THE SOCIAL BRAIN: JUVENILES

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What are imprinted genes doing in the adult brain? Genomic imprinting is when a gene's expression depends upon parent of origin. According to the prevailing view, the "kinship theory" of genomic imprinting, this effect is driven by evolutionary conflicts between genes inherited via sperm versus egg. This theory emphasizes conflicts over the allocation of maternal resources, and focuses upon genes that are expressed in the placenta and infant brain. However, there is growing evidence that imprinted genes are also expressed in the juvenile and adult brain, after cessation of parental care. These genes have recently been suggested to underpin neurological disorders of the social brain such as psychosis and autism. Here we advance the kinship theory by developing an evolutionary model of genomic imprinting for social behavior beyond the nuclear family. We consider the role of demography and mating system, emphasizing the importance of sex differences in dispersal and variance in reproductive success. We predict that, in hominids and birds, altruism will be promoted by paternally inherited genes and egoism will be promoted by maternally inherited genes. In nonhominid mammals we predict more diversity, with some mammals showing the same pattern and other showing the reverse. We discuss the implications for the evolution of psychotic and autistic spectrum disorders in human populations with different social structures.

KEY WORDS: Altruism, autism, autosomal genes, kin selection, psychosis, selfishness, sex-biased dispersal, sex-specific reproductive success, viscosity.

Genomic imprinting (GI) is the asymmetric expression of genes with different parental origin (Reik and Walter 2001). In particular, the term is used to refer to genes that are expressed either only when maternally inherited (maternally expressed) or else only when paternally inherited (paternally expressed). Why would a gene become functionally haploid, and forego the advantages of diploidy? The prevailing explanation—the kinship theory—argues that genes with different parental origin can come into conflict over their combined level of expression (Haig 2002; Tycko and Morison 2002; Wilkins and Haig 2003; Burt and Trivers 2006; Moore and Mills 2008). This theory applies to genes whose phenotype affects the kin of their carrier, and where the carrier is differentially related to the target kin via its maternally inherited and paternally inherited genes (Haig 2002; Wilkins and Haig

2003). The outcome of this conflict is self-imposed silencing of one of the conflicting genes (Haig 2002).

Imprinted genes were first identified as mediators of embryonic and placental growth (Moore and Haig 1991; Constancia et al. 2004). Thus, the kinship theory was originally formulated in terms of conflicts over the transfer of a mother's resources to her fetus (Moore and Haig 1991). In this context, kin selection models identify maternal promiscuity as the driver of GI (Haig 1996): paternally inherited genes in the fetus are favored to extract more maternal resources than are maternally inherited genes, because the cost of increased resource extraction is incurred by maternal siblings who may not be paternal siblings (Haig 1996). Later, imprinted genes were linked to appetite and behavior of infants (Moore and Haig 1991; Constancia et al. 2004). These

findings have led the kinship theory to be applied to postnatal investment of resources by the mother (Haig 2002) and, more recently, to investments made by both parents (Úbeda 2008). Again, parental promiscuity is identified as the driver of GI, although the transfer of resources is not mediated by chemical interactions of genes expressed in the fetus and placenta, but rather by behavioral interactions of genes expressed in the infant brain (Trivers 1974).

There is growing evidence that imprinted genes are also active in the brains of juveniles and adults, after the cessation of parental care (Goos and Silverman 2001; Davies et al. 2005; Plagge et al. 2005; Davies et al. 2007). This activity is not explained by the kinship theory in its original formulation, but there is potential for the theory to be extended to encompass social interactions beyond those of parent and offspring (Trivers and Burt 1999; Haig 2000). In particular, differential relatedness between neighbors with respect to maternal versus paternal genes could arise as a consequence of sex differences in dispersal rate and variance in reproductive success (Haig 2000; Isles et al. 2006). This differential relatedness might lead to GI for genes that mediate social interactions between neighbors (Haig 2000). However, no formal analysis of this idea has been undertaken. Such an analysis is necessary not only to explain GI in the juvenile and adult (post-infant) brains, but also to understand recent research linking GI to important neurological disorders within the autistic and psychotic spectra (Badcock and Crespi 2006, 2008; Crespi 2008; Crespi and Badcock 2008).

We develop an evolutionary demographic model (Grafen 1985; Taylor and Frank 1996; Rousset 2004; Wild and West 2009; Gardner 2010) that extends the kinship theory of GI to social interactions in a viscous population. Our results provide a novel and unique insight into the role of imprinted genes in the social brain, and allow us to predict how mutations and epimutations of imprinted genes will affect the balance between altruism and egoism in the social brain. The model makes clear predictions relating ancestral human demography to imprinting patterns and contemporary neurological disorders, demonstrating that, if confirmed, the link between GI and autistic-spectrum and psychotic-spectrum disorders, the social structure in which individuals evolved can affect clinical phenotype and the severity of these neurological disorders.

Results

We consider an infinite population structured into neighborhoods. At the point of census in each generation, we consider that each neighborhood contains a large number of diploid juveniles that engage in social interactions. These interactions mediate the survival of juveniles to adulthood. Adults either disperse with probability d_m and d_f for males and females respectively, or else remain in their natal neighborhood with probability $l_m = 1 - d_m$ and

$l_f = 1 - d_f$. After dispersing, adults mate at random within their neighborhood, and the next generation of juveniles is produced. We assume an even sex ratio among these offspring. We allow for a wide range of mating systems, by denoting the probability that two juveniles, randomly chosen from the same neighborhood, share the same mother by α (probability of maternal sibship), and the probability that they share the same father by β (probability of paternal sibship). Thus, α and β describe the inequity in reproductive success among females and males, respectively, and reflect variance in survival to reproductive maturity and variance in mating success. For example, monogamy with N equally fecund couples per neighborhood is represented by $\alpha = \beta = 1/N$, whereas a harem of N equally fecund females mated by a single male is represented by $\alpha = 1/N$ and $\beta = 1$ (see the Appendix for a detailed formulation of the model).

We use this sociodemographic model to determine the extent to which maternally derived and paternally derived autosomal genes underlying altruistic behavior value the survival of social partners relative to the survival of their own carrier. We term this the potential for altruism (Gardner 2010), and find that it is given by

$$A_{\bar{M}} = \frac{(1-a)r_{\bar{M}}}{1-ar_{\bar{M}}} \quad (1a)$$

$$A_{\bar{P}} = \frac{(1-a)r_{\bar{P}}}{1-ar_{\bar{P}}}, \quad (1b)$$

where

$$a = \frac{1}{2}(l_f^2 + l_m^2) \quad (2)$$

is the intensity of local competition (Frank 1998; Gardner 2010) and

$$r_{\bar{M}} = \frac{1}{2} \frac{\alpha + \frac{1}{4}[\beta(1-\alpha)l_f^2 + (\beta-\alpha)l_f l_m - \alpha(1-\beta)l_m^2]}{1 - \frac{1}{4}[(1-\alpha)l_f^2 + 2l_f l_m + (1-\beta)l_m^2]} \quad (3a)$$

$$r_{\bar{P}} = \frac{1}{2} \frac{\beta + \frac{1}{4}[\alpha(1-\beta)l_m^2 + (\alpha-\beta)l_f l_m - \beta(1-\alpha)l_f^2]}{1 - \frac{1}{4}[(1-\alpha)l_f^2 + 2l_f l_m + (1-\beta)l_m^2]} \quad (3b)$$

are the coefficient of relatedness between a focal juvenile and another juvenile in the same patch via the maternally inherited and the paternally inherited genes (see Appendix for derivation). Notice that this model accounts for the effects of competition between related individuals. Despite competition between related individuals altruism can evolve except in the extreme case in which $a = 1$ when there is no migration (see Fig. 1A) (see Gardner 2010 for the impact of sex-biased dispersal on social evolution in viscous populations in the absence of GI). Intensity of competition and relatedness have opposite effects on the potential for altruism: the greater the intensity of competition the lower the potential for

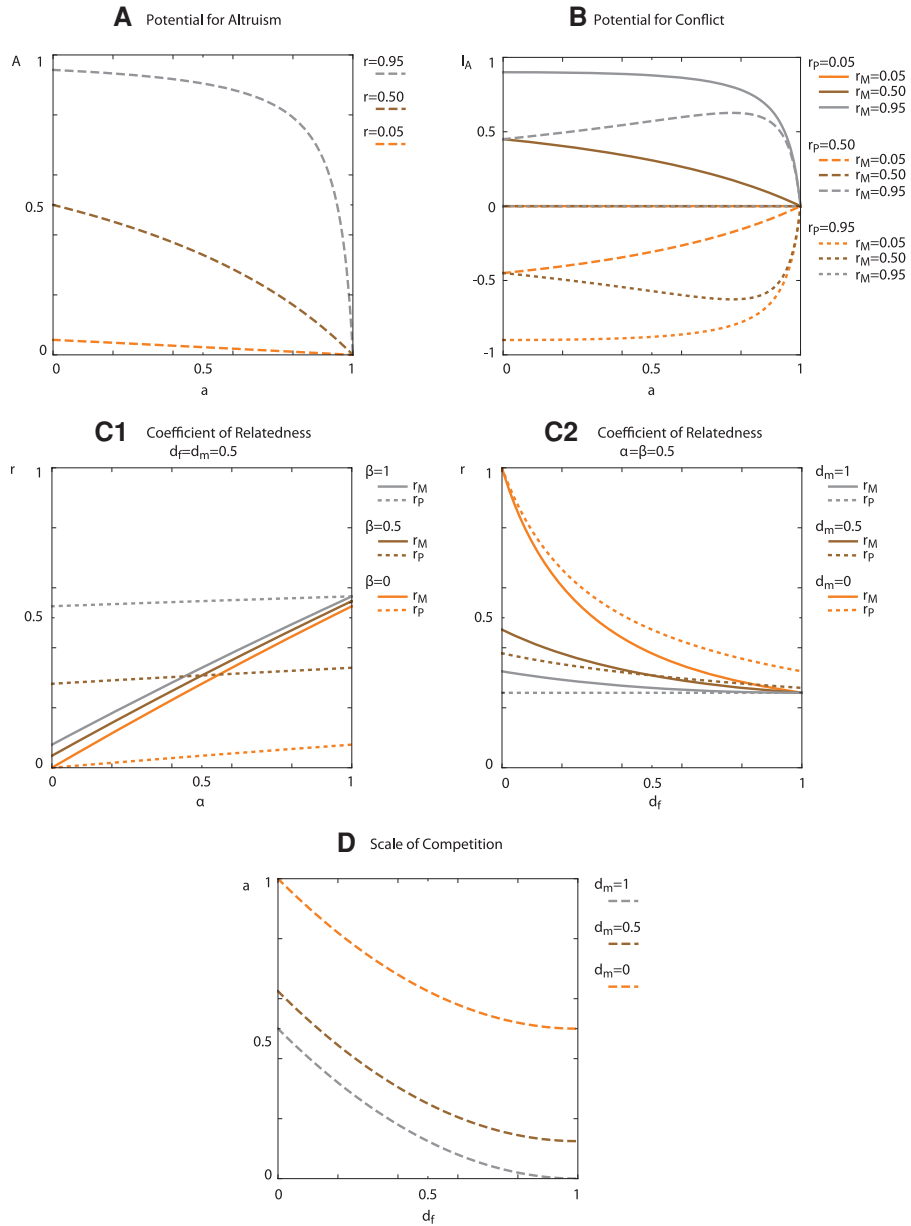


Figure 1. Potential for altruism and potential for intragenomic conflict regarding altruism. (A) Potential for altruism A_A (y -axis) as a function of the scale of competition a (x -axis) and the coefficient of relatedness r_x . The greater the competition between patchmates the lesser the potential for altruism, the greater the relatedness between juveniles the greater the potential for altruism. (B) Potential for intragenomic conflict I_A (y -axis) as a function as a function of the scale of competition a (x -axis) and the coefficient of relatedness via the maternally inherited r_M and paternally inherited r_P genes. The greater the competition between patchmates the lesser the conflict, the greater the difference in relatedness via the maternally inherited and via the paternally inherited genes the greater the conflict. Relatedness and scale of competition are determined by migration and the probability of sibship. (C1) Coefficient of relatedness r (y -axis) as a function of the probability of sibship through females α (x -axis) when there is no sex bias in dispersal $d_f = d_m = 0.5$. The greater the probability of sibship the greater the coefficient of relatedness both via the maternally inherited (continuous line) and the paternally inherited (dotted line) genes. (C2) Coefficient of relatedness r (y -axis) as a function of female dispersal d_f (x -axis) when there is no sex-difference in the probability of sibship $\alpha = \beta = 0.5$. The greater the dispersal the lower the coefficient of relatedness both via the maternally inherited and the paternally inherited genes. (D) Scale of competition a (y -axis) as a function of female dispersal d_f (x -axis). The greater the dispersal the lower the competition between related individuals. The scale of competition is the same for both gene copies with independence of their parental origin.

altruism and the greater the relatedness the greater the potential for altruism (see Fig. 1B). If altruism incurs a survival cost c to the altruist and confers a survival benefit b to the recipient, then maternally inherited genes are selected to promote altruism when $\frac{c}{b} < A_{\bar{M}}$, and paternally inherited genes are selected to promote altruism when $\frac{c}{b} < A_{\bar{P}}$.

When maternally derived and paternally derived genes differ in their valuation of the survival of social partners relative to the survival of their own carrier, there is intragenomic conflict between genes of different parental origin. We define the potential for intragenomic conflict regarding altruism I_A as the difference between the potential for altruism of the maternally inherited and the paternally inherited genes, $I_A = A_{\bar{M}} - A_{\bar{P}}$, and is given by

$$I_A = \frac{(1-a)(r_{\bar{M}} - r_{\bar{P}})}{(1-ar_{\bar{M}})(1-ar_{\bar{P}})}. \quad (4)$$

The intensity of competition a modifies the extent of the conflict making the potential for conflict bigger or smaller but does not affect its sign (see Fig. 1B). The difference in relatedness via the maternally inherited and the paternally inherited genes $r_{\bar{M}} - r_{\bar{P}}$ not only modifies the extent of the conflict but also determines the sign of the potential for conflict (see Fig. 1B). Notice that I_A ranges between $-1 < I_A < 1$.

Intragenomic conflict manifests as genes being selected for different level of combined expression (we denote the evolutionarily stable level of combined expression for maternally inherited and paternally inherited genes $g_{\bar{M}}^*$ and $g_{\bar{P}}^*$, respectively). If the locus underlies an altruistic trait, then the gene with greater potential for altruism is selected for a higher level of combined expression ($g_{\bar{M}}^* > g_{\bar{P}}^*$ if $I_A > 0$, and $g_{\bar{M}}^* < g_{\bar{P}}^*$ if $I_A < 0$). In particular, owing to the loudest voice prevails principle (Haig 1996), the gene selected for higher expression will ultimately be expressed at its optimal level whereas the other gene will be silenced ($\{g_{\bar{M}}^*, 0\}$ if $I_A > 0$ and $\{0, g_{\bar{P}}^*\}$ if $I_A < 0$, where the first position in the ordered pair $\{g_{\bar{M}}^*, g_{\bar{P}}^*\}$ corresponds to the expression of the maternally inherited gene and the second to the expression of the paternally inherited gene). In the event of no difference in potential for altruism, we predict that GI does not evolve ($g_{\bar{M}}^* = g_{\bar{P}}^*$ if $I_A = 0$).

The results of our model are presented in Figure 2. There is no conflict between genes with different parental origin, and hence GI is not expected to evolve, when there is sex bias neither in dispersal ($d_f = d_m$) nor in variance in reproductive success ($\alpha = \beta$) (Fig. 2). Assuming no sex-bias in variance in reproductive success ($\alpha = \beta$), the potential for altruism is greater for the gene derived from the least-dispersing sex, so that a gene underlying altruism will evolve to be maternally expressed if dispersal is male biased ($\{g_{\bar{M}}^*, 0\}$ if $d_f < d_m$) and paternally expressed if dispersal is female biased ($\{0, g_{\bar{P}}^*\}$ if $d_f > d_m$) (Figs. 2 and 3). Assuming no sex bias in dispersal ($d_f = d_m$), the potential for altruism is greater for the gene derived from the sex with the greatest variance

in reproductive success, so that a gene underlying altruism will evolve to be maternally expressed if females have greater variance ($\{g_{\bar{M}}^*, 0\}$ if $\alpha > \beta$) and paternally expressed if males have greater variance ($\{0, g_{\bar{P}}^*\}$ if $\alpha < \beta$) (Figs. 2 and 3).

The model predictions are more complex when there is sex-bias in both dispersal ($d_f \neq d_m$) and variance in reproductive success ($\alpha \neq \beta$). We proceed on the assumption that males have greater variance in reproductive success ($\alpha < \beta$), as this is most likely to occur in nature (Clutton-Brock 2007) (the reverse of the following results hold true if females have greater variance in reproductive success, $\alpha > \beta$). When the probability of sibship is relatively high, a gene for altruistic behavior is expected to be paternally expressed ($\{0, g_{\bar{P}}^*\}$). When the probability of sibship is relatively low, a gene for altruistic behavior evolves to be paternally expressed ($\{0, g_{\bar{P}}^*\}$) if dispersal is female biased ($d_f > d_m$), but may evolve to be either maternally or paternally expressed ($\{g_{\bar{M}}^*, 0\}$, $\{0, g_{\bar{P}}^*\}$) if dispersal is male biased ($d_f < d_m$) (Fig. 2). We focus on the more realistic case of relatively low variance in reproductive success. In this case, low bias in dispersal and high dispersal rates ($d_m \approx d_f \approx 1$) favor paternally expressed genes, but high bias in dispersal and intermediate dispersal rates ($d_m \approx 1/2$; $d_f \approx 1$) favor maternally expressed genes (Fig. 2). Consider $\alpha = 0.05$ and $\sigma_m = 0.10$ as an example: when females are philopatric ($d_f \approx 0$) and half of the males disperse ($d_m \approx 1/2$), natural selection favors maternal expression of a gene underlying altruism ($\{g_{\bar{M}}^*, 0\}$), but when all males disperse ($d_m = 1$) this gene is expected to be paternally expressed ($\{0, g_{\bar{P}}^*\}$) (Fig. 2).

The above analysis can be repeated for genes underlying egoistic behavior. We derive a potential for egoism, defined as the relative value that a maternally inherited or paternally inherited gene places on the survival of its carrier relative to the survival of a social partners ($E_M = 1/A_M$ and $E_P = 1/A_P$) (see Appendix for derivation). We find that the results derived for GI in relation to altruistic behavior apply equally to genes for egoistic behavior, but are reversed (Fig. 3).

Discussion

Our model provides a general explanation for the evolution of GI in the postinfant brain, as the identified necessary requirements—sex-bias in dispersal and/or variance in reproductive success—are the norm in the natural world. Intuitively, lower dispersal in one sex translates into higher relatedness among social partners with respect to genes inherited from that sex. Broadly speaking, natural selection favors the expression of genes encoding altruism that are derived from the more-philopatric sex, and it favors the expression of genes encoding egoism that are derived from the more-dispersing sex (Fig. 3). This is analogous to how cooperation (including traits such as helping behaviors and female-biased sex allocation) is more strongly favored among individuals of

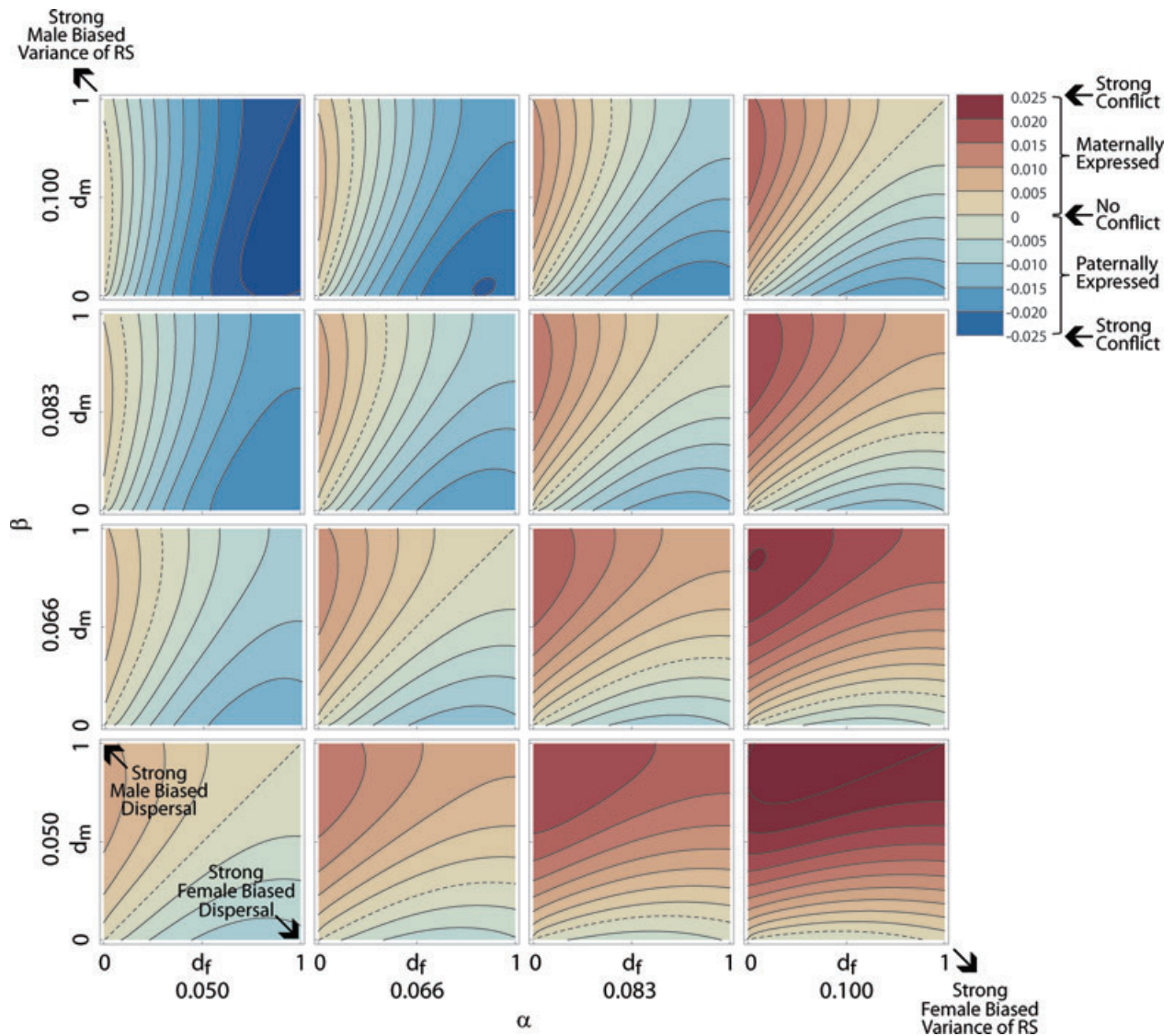


Figure 2. Potential for intragenomic conflict regarding altruism (detail; see Fig. 6 for full range). Grid using four values of the probability of sibship through female α and four values of the probability of sibship through males β covering the range [0.05, 0.10] corresponding to the case in which the probability of sibship is relatively low. Given a pair of values (α, β) , the contour lines in each figure represent the potential for conflict between genes with different parental origin regarding altruism (I_A) for values of dispersal in females and males. We use a continuous line for contours in which there is conflict ($I_A \neq 0$), and a dashed line for contours in which there is no conflict ($I_A = 0$). We use red color to represent regions in which imprinted genes will be maternally expressed ($I_A > 0$) and blue to represent regions in which imprinted genes will be paternally expressed ($I_A < 0$). The secondary diagonal corresponds to cases in which there is no sex-difference in sibship probability ($\alpha = \beta$).

the more philopatric sex (Johnstone and Cant 2008), and among nondispersing individuals in general (Taylor and Crespi 1994; El Mouden and Gardner 2008)—although with the phenotypic expression of philopatric genes occurring in the next generation. Also, higher variance in reproductive success among individuals of one sex translates into higher relatedness among social partners with respect to genes inherited from that sex. All else being equal, natural selection favors the expression of genes encoding altruism that are derived from the sex with higher variance in reproductive

success, and it favors the expression of genes encoding egoism that are derived from the sex with lower variance in reproductive success (Fig. 3).

These results suggest that GI can evolve in relation to genes encoding altruistic and egoistic behaviors exhibited by postinfants, in any species in which the opportunity for social interactions exists. There is evidence that GI of genes expressed in the embryo mediating the transfer of maternal resources is limited to plants and mammals and does not occur in birds





		Altruism	Egoism	
Dispersal	Female Bias	PI Expressed MI Silenced	MI Expressed PI Silenced	Female-biased Dispersal or Male-biased Variance in RS p —  — PI Altruism m —  — MI Egoism
	Male Bias	MI Expressed PI Silenced	PI Expressed MI Silenced	
Variance Reproductive Success	Female Bias	MI Expressed PI Silenced	PI Expressed MI Silenced	Male-biased Dispersal or Female-biased Variance in RS p —  — MI Altruism m —  — PI Egoism
	Male Bias	PI Expressed MI Silenced	MI Expressed PI Silenced	

Figure 3. Predicted pattern of expression. Pattern of expression that is expected to evolve in genes underpinning altruistic and egoistic behaviors when there is sex-biased dispersal or sex-differences in the variance in reproductive success. m and p correspond to paternally inherited and maternally inherited strands. Solid and dotted rectangles correspond to expressed and silenced genes. A and E stand for genes responsible altruistic and egoistic behaviors, respectively.

(Moore 2001). This makes sense owing to lack of an arena in which the maternal-embryonic conflict may be enacted, as yolk allocation is determined before fertilization. In contrast, it has been argued that GI of genes expressed in the infant mediating the allocation of parental care (such as those encoding begging behavior), should be found in any species providing postnatal care, including birds (Trivers and Burt 1999). We make this prediction extensive to genes encoding social behavior.

Our model predicts that genes underpinning altruistic behaviors will be paternally expressed among birds and hominids, whereas some diversity of expression is expected among nonhominid mammals (Fig. 4). Conversely, our model predicts that genes underpinning egoistic behaviors will be maternally expressed among birds and hominids, and again some diversity of expression is expected among nonhominid mammals (Fig. 4). The direction of the imprint is determined by which sex shows greater dispersal and/or variance in reproductive success. Empirical data for these quantities are best documented in birds and mammals. Variance in reproductive success is generally greater among males than females in both mammals and birds, although the difference is typically more pronounced in the former (Clutton-Brock 2007). Sex-biased dispersal is also the norm, with birds generally exhibiting female-biased dispersal (Greenwood 1980) and mammals generally exhibiting male-biased dispersal (Greenwood 1980)—with the notable exception of hominids, which typically exhibit female-biased dispersal (Greenwood 1980; Lawson Handley and Perrin 2007) (Fig. 5).

The direction of sex bias in the dispersal of ancestral humans is somewhat controversial. However, the conventional view is that ancestral humans exhibited female-biased dispersal (patrilocality) (Foley 1995), and this is supported by three lines of evidence: (1) the closest relatives of humans—gorilla, chimpanzee and bonobo—exhibit female-biased dispersal (Lawson Handley and Perrin 2007); (2) population genetic data suggest migration

has been eight times greater among ancestral human females than males (Seielstad et al. 1998) (although Wilder et al. (2004) fail to find evidence of greater female dispersal); and (3) anthropological data from modern hunter-gatherer groups indicate that women migrate more often than men (Ember 1975) (although Marlowe (Marlowe 2004) argues that modern hunter-gatherer groups do exhibit a diversity of dispersal patterns).

Assuming patrilocality is the human ancestral condition, our model can be used to explain aspects of the clinical phenotypes associated with mutations of imprinted genes expressed in the postinfant human brain. Imprinted genes are often found in clusters of paternally expressed and maternally expressed genes (Reik and Walter 2001). Our model predicts that paternally expressed genes underlie altruistic behaviors and maternally expressed genes underlie egoistic behaviors. The balance between altruistic and egoistic influences results in normal individuals showing no disorder (normal brain) (Fig. 4A). Mutations tilting the balance toward paternally expressed genes—deletion of maternally inherited genes, loss of imprint of maternally inherited genes, or paternal disomy—result in pathologies related to an excess of altruistic behavior (hyper-altruistic brain) (Fig. 4B(2)). Conversely mutations tilting the balance toward maternally expressed genes—deletion of paternally inherited genes, loss of imprint of paternally inherited genes, or maternal disomy—result in pathologies related to an excess of egoistic behavior (hyper-egoistic brain).

Crucially, the behavioral phenotype associated with hyper-altruistic or hyper-egoist brains need not (and generally will not) be functionally altruistic or egoistic, respectively. These disorders represent major disruptions at the level of the proximate mechanisms underlying social behavior, and are not well-honed adaptations operating for the good of either maternal or paternal gene. Serve as an example altruistic genes encoding reduced aggression or enhanced emotional empathy—defined as an affective

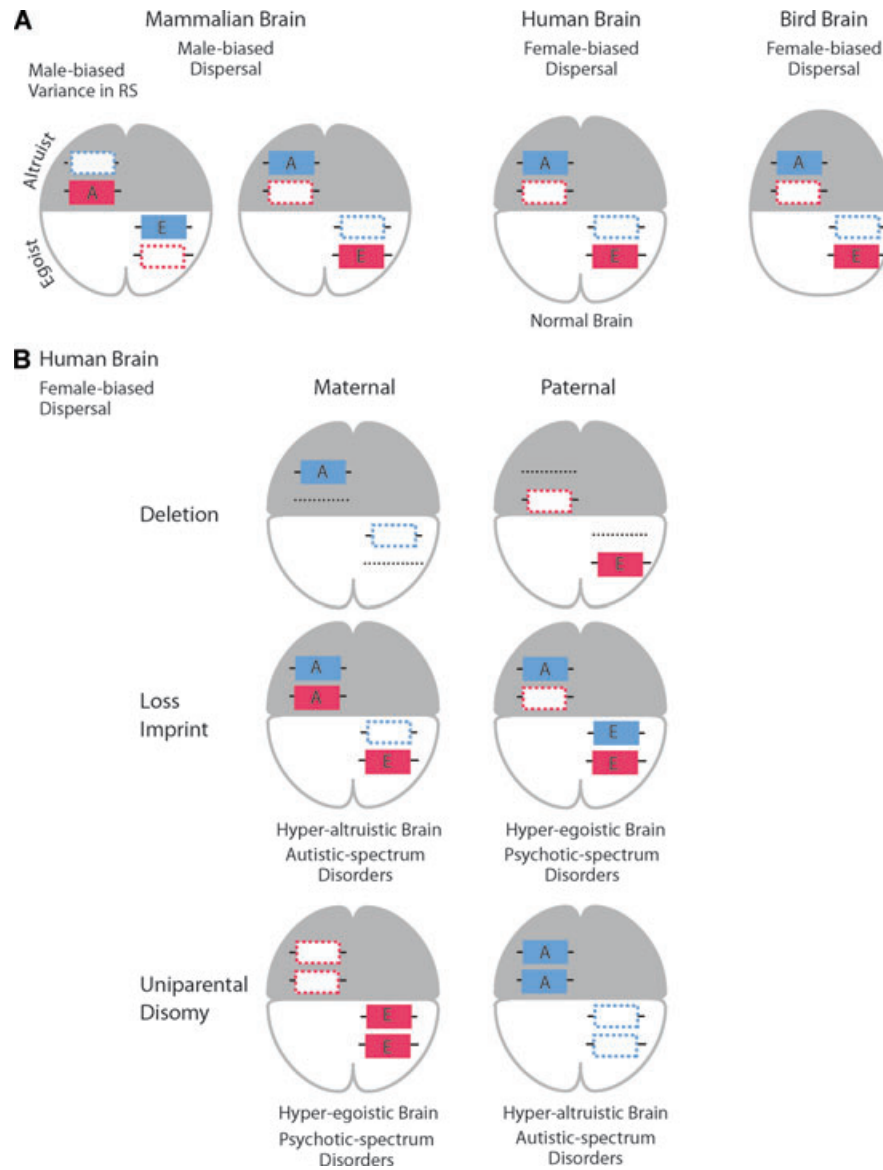

























Figure 4. Expression of imprinted genes in bird, mammalian and human brains and effects of possible mutations. (A) Expression of imprinted genes in the altruist (gray) and egoist (white) regions of the bird, mammalian, and human brains. (B). Possible mutations in human imprinted genes: (1) deletion, (2) loss of imprint, (3) uniparental disomy. Predicted clinical phenotype of associated mental disorders: hyper-egoistic brain versus hyper-altruistic brain.


state caused by sharing the emotions of another person (Hein and Singer 2008). These genes are altruistic because their expression results in the focal individual losing valuable resources to its social partner via competition or donation, respectively. Individuals suffering from a hyper-altruistic brain would be less aggressive and experience more affective empathy (less cognitive empathy) than normal individuals. On the contrary, egoistic genes encoding enhanced aggression and “Machiavellianism”—defined as a social conduct that involves manipulating others for personal gain (Hein and Singer 2008). These genes are egoistic because their expression results in the focal individual obtaining resources from its social partner via competition or manipulation, respectively.

Individuals suffering from a hyper-egoistic brain would be more aggressive and Machiavellian than normal individuals.

Recently, it has been suggested that psychotic-spectrum and autistic-spectrum disorders are two extremes of a continuum of neurological disorders (Crespi and Badcock 2008). Mutations resulting in a greater influence of maternally expressed genes give rise to psychotic-spectrum disorders, whereas mutations resulting in a greater influence of paternally expressed genes give rise to autistic-spectrum disorders (Crespi and Badcock 2008). Our model indicates that psychotic-spectrum disorders can be explained by a hyper-egoistic brain. This would manifest, for example, as juveniles being more aggressive than normal

Mammals Male-biased Dispersal		Birds Female-biased Dispersal	
Exceptions		Exceptions	
Species Name	Common Name	Species Name	Common Name
 <i>Vombatus ursinus</i>	Common Wombat	 <i>Anser caerulescens</i>	Lesser Snow Goose
 <i>Hapalemur griseus</i>	Alaotrangente Lemur	 <i>Clangula hyemalis</i>	Long Tailed Duck
 <i>Lagothrix lagotricha</i>	Woolly Monkey	 <i>Anas acuta</i>	Pintail
 <i>Ateles sp.</i>	Spider Monkey		
 <i>Brachyteles arachnoides</i>	Muriqui		
 <i>Alouatta seniculus</i>	Red Howler Monkey		
 <i>Alouatta palliata</i>	Mantled Howler Monkey		
 <i>Papio hamadryas</i>	Hamadryas Baboon		
 <i>Colobus badius rufomitratu</i>	Red Colobus	Humans Female-biased Dispersal	
 <i>Presbytis thomasi</i>	Thomas Langurs	Exceptions in Hunter-gatherers	
 <i>Gorilla gorilla</i>	Gorilla	Name Society	
 <i>Pan troglodytes</i>	Chimpanzee	EE	Vedda
 <i>Pan paniscus</i>	Bonobo	SA	Warrau
 <i>Peromyscus californicus</i>	California Mouse	SA	Siriono
 <i>Erethizon dorsatum</i>	North American Porcupine	SA	Shavante
 <i>Saccopteryx bilineata</i>	White-lined Bat		
 <i>Potos flavus</i>	Kinkajou		
 <i>Lycaon pictus</i>	African Wild Dog		
 <i>Equus caballus</i>	Horse		
 <i>Crocidura russula</i>	Greater White-toothed Shrew		

Orders

-  Marsupialia
-  Primates
-  Rodentia
-  Chiroptera
-  Carnivora
-  Perissodactyla
-  Insectivora
-  Anatidae

Regions

- EE East Eurasia
- SA South America

Figure 5. Dispersal pattern in birds and mammals. The general pattern is indicated in the heading of each column and is followed by a list of exceptions. Data proceed from Greenwood (1980); Foley (1995); Marlowe (2004); Lawson Handley and Perrin (2007).

(associated with bullying social partners, as a means of resource acquisition among ancestral humans (Hawley 2003)), or juveniles developing more cognitive empathy—ability to understand intentions and desires of another person—and less emotional empathy,

which results in Machiavellian Intelligence (associated with the capacity to understand other people’s desires but experiencing no pleasure from satisfying those desires; this allows Machiavellians to manipulate their social partners, as a means of

resource acquisition among ancestral humans (Hawley 2003)). Interestingly high cognitive empathy and extreme Machiavellianism are prominent features of psychotic spectrum disorders (McHoskey 2001). In contrast, our model indicates that autistic-spectrum disorders can be explained by a hyper-altruistic brain. This would manifest, for example, as juveniles being less aggressive than normal (associated with lack of assertion in front of social partners when competing for resources among ancestral humans), or juveniles developing less-cognitive empathy and more emotional empathy (associated with individuals that are easy to manipulate by social partners and derive pleasure from helping social partners when their social partner intention is obvious, among ancestral humans). Lack of cognitive empathy and high emotional empathy are prominent features of autistic spectrum disorders (Smith 2009). This lack of cognitive empathy explains the apparently self-centric perspective of autists (Smith 2009). Although lack of cognitive empathy has often been confused with lack of emotional empathy, autistic children do show a supranormal response to blunt emotional cues (Smith 2009).

Our analysis indicates that the direction of the imprint for a gene expressed in the postinfant brain will be the reverse of when this gene encodes the same character in the placenta or the preinfant brain. In a familial context (placenta and infant brain), paternally expressed genes are selected to extract more maternal resources and maternally expressed genes are selected to extract fewer maternal resources (Haig 2002), whereas in a social context (postinfant brain), paternally expressed genes are selected to provide more help to social partners and maternally expressed genes are selected to provide less help to social partners. In many cases, the same genes expressed in the preinfant and postinfant brains will encode very different characters. However, when these genes affect the same character, we predict a reversal in the direction of the imprint. Children suffering from Prader–Willi syndrome show a biphasic clinical phenotype, with reduced weight caused by poor sucking before weaning and obesity caused by insatiable appetite afterwards. This reversion has attracted the attention of evolutionary biologists, and two different models (Haig and Wharton 2003; Úbeda 2008) have attempted to explain it. Our model provides an alternative explanation. Prader–Willi syndrome is caused by deletion of the paternally inherited copy of the PWS/AS cluster of imprinted genes. A gene that promotes suckling is predicted to be paternally expressed (Haig 2002), and the expected clinical phenotype of Prader–Willi children before weaning is reduced suckling and under-weight. A gene involved in foraging in the native patch is predicted to be maternally expressed, and the expected clinical phenotype of Prader–Willi children after weaning is increased competition for resources and over-weight. Note that each gene encodes different features (suckling and foraging) that impact upon the same character—weight—in opposite directions. Interestingly Prader–Willi syndrome is considered a psychotic

spectrum disorder, and its postweaning clinical phenotype is consistent with that of a hyper-egoistic brain.

The kinship theory has been extremely successful in making testable predictions for the role of imprinted genes in different organs (Constancia et al. 2004). Our model allows us to make predictions about the spatial distribution of imprinted genes in the human brain. Within the brains of mouse chimeras containing a mixture of normal cells and cells containing two maternal genomes, the latter cell type becomes concentrated in the frontal neocortex (Keverne et al. 1996). In contrast, within the brains of mouse chimeras containing a mixture of normal cells and cells containing two paternal genomes, the latter cell type becomes concentrated in the hypothalamic and septal regions of the brain (Keverne et al. 1996). Data on sex-biased dispersal in mice are surprisingly sparse (Greenwood 1980) but, upon the assumption of male-biased dispersal that is usual for nonprimate mammals, we might infer that the frontal neocortex specializes in governing altruistic behavior, whereas the hypothalamic and septal regions specialize in governing egoistic behavior. Thus, we would predict the reverse localization of double-maternal and double-paternal cells in the brains of human chimeras. Unfortunately, there is little information on the role of these brain regions in mediating human social behavior. More generally, our model illustrates the importance of reliable ecological and demographic data (including in relation to historical populations), for humans and nonhuman study organisms, if we are to fully understand even basic neurobiology, and to tackle human brain disorders in an informed way.

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Appendix

ALTRUISM

Expected fitness

We denote an individual's probability of survival to adulthood by $S(x, y)$, where x is the level of helping invested by the individual into its social partners, and y is the average level of helping invested by the individual's social partners. We assume that S is a monotonically decreasing function of its first argument and a monotonically increasing function of its second argument. We assume that the average investment into helping over all the

individuals in the population is z . Moreover, following Taylor and Frank (1996), we assume that there is vanishingly little variation around this population average, allowing us to employ differential calculus methodology.

As we are considering evolution in a class-structured population (i.e., individuals may be female or male), we must treat fitness for individuals of each class separately, and then combine these using class reproductive value weightings (Fisher 1930; Price and Smith 1972; Taylor 1990, 1996; Taylor and Frank 1996; Grafen 2006; details below). We begin by calculating the expected fitness of a focal juvenile female as a function of the helping strategy of herself and her social partners (Gardner 2010). If the focal female survives to adulthood, then she may either disperse or remain in her natal patch. The expected number of offspring she produces is assumed to be inversely proportional to the number of females competing within the patch upon which she finds herself after the dispersal phase. Hence, her expected fitness is given by

$$w_f = S(x, y)k \left[\frac{l_f}{l_f S(y, y) + (1 - l_f)S(z, z)} + \frac{1 - l_f}{S(z, z)} \right], \quad (A1)$$

where k is a constant of proportionality. Notice that the average fitness of all females in the population is $\bar{w}_f = k$. Hence the expected fitness of the focal female, relative to the average fitness for her class, is

$$W_f = S(x, y) \left[\frac{l_f}{l_f S(y, y) + (1 - l_f)S(z, z)} + \frac{1 - l_f}{S(z, z)} \right]. \quad (A2)$$

We now calculate the expected fitness of a focal juvenile male (Gardner 2010). If the focal male survives to adulthood, then he may either disperse or remain in his natal patch. If the focal male does not disperse, his expected mating success is given by the ratio of adult females to adult males within his natal patch, which is

$$q = \frac{l_f S(y, y) + (1 - l_f)S(z, z)}{l_m S(y, y) + (1 - l_m)S(z, z)}. \quad (A3)$$

The expected number of offspring produced by his mates will be inversely proportional to the number of females in his natal patch. If the focal male does disperse, his expected mating success is given by the ratio of adult females to adult males in the entire population, which is 1. The expected number of offspring produced by his mates will be inversely proportional to the number of females in his nonnatal patch. Hence, his expected fitness is given by:

$$w_m = S(x, y)k \left[q \frac{l_m}{l_f S(y, y) + (1 - l_f)S(z, z)} + \frac{1 - l_m}{S(z, z)} \right]. \quad (A4)$$

The average fitness for all males in the population is $\bar{w}_m = k$. Hence the expected fitness of the focal male, relative to the average fitness for his class, is

$$W_m = S(x, y) \left[\frac{l_m}{l_m S(y, y) + (1 - l_m)S(z, z)} + \frac{1 - l_m}{S(z, z)} \right]. \quad (A5)$$

In the context of a class-structured population, the contribution of each class to a gene's expected fitness is weighted by that class' reproductive value (Fisher 1930; Price and Smith 1972; Taylor 1990; Grafen 2006). Hence, the expected fitness of a gene is given by

$$W = \frac{1}{2}W_f + \frac{1}{2}W_m \quad (A6)$$

where the $1/2$ factors correspond to the class reproductive value for males and females (Fisher 1930; Price 1970; Taylor 1996).

Hamilton's Rule

Let χ denote the maternal ($\chi = \bar{M}$) or paternal ($\chi = \bar{P}$) origin of a gene copy. The χ -inherited copy of a gene for helping is favored by natural selection when Hamilton's rule is satisfied (Hamilton 1963, 1964, 1970; Taylor 1996; Taylor and Frank 1996; Wild and West 2009):

$$-C + r_\chi B > 0 \quad (A7)$$

where $-C = \partial W/\partial x|_z$ is the personal fitness cost of helping experienced by the actor, $B = \partial W/\partial y|_z$ is the personal fitness benefit of helping experienced by the recipient (partial derivatives are evaluated at $x = y = z$), and r_χ is the kin-selection coefficient of relatedness between the χ -inherited gene copy of a focal individual and its social partners.

Cost and benefit

From equation (6), we can derive an expression for the fitness cost and benefit experienced by a helping individual.

The fitness cost $-C = \partial W/\partial x|_z$ is

$$-C = \frac{\partial S/\partial x|_z}{S(z, z)}. \quad (A8)$$

Let $\partial S/\partial x|_z = -c$ and $\partial S/\partial y|_z = b$ be the survival cost and benefit experienced by a helping individual then

$$-C = -\frac{c}{S(z, z)}. \quad (A9)$$

The fitness benefit $B = \partial W/\partial y|_z$ is

$$B = \frac{1}{S(z, z)} \left[b - \frac{1}{2}(l_f^2 + l_m^2)(b - c) \right]. \quad (A10)$$

Let

$$a = \frac{1}{2}(l_f^2 + l_m^2) \quad (A11)$$

be the scale of competition between neighboring siblings (Frank 1998) (notice that $0 < a < 1$). Thus

$$B = \frac{b - a(b - c)}{S(z, z)}. \quad (\text{A12})$$

Because C and B are positive, investment into promoting the survival of patchmates is formally altruistic (Hamilton 1964; West et al. 2007).

Coefficient of relatedness

The coefficient of consanguinity of the focal individual to itself, p_S , is the same via its maternally inherited (MI) and paternally inherited (PI) genes, namely:

$$p_S = \frac{1}{2} + \frac{1}{2}\varphi, \quad (\text{A13})$$

where φ is the coefficient of inbreeding or consanguinity between mating partners

$$\varphi = l_f l_m p_X. \quad (\text{A14})$$

The coefficient of consanguinity between the focal individual and its neighbors via the MI gene is

$$p_{X\bar{M}} = \frac{1}{2}[\alpha p_S + (1 - \alpha)l_f^2 p_X] + \frac{1}{2}\varphi, \quad (\text{A15})$$

and via the PI gene is

$$p_{X\bar{P}} = \frac{1}{2}[\beta p_S + (1 - \beta)l_m^2 p_X] + \frac{1}{2}\varphi, \quad (\text{A16})$$

where α and β are the probability of maternal and paternal sib-ship for same-patch juveniles, respectively.

The coefficient of consanguinity of the focal individual to its neighbors is

$$p_X = \frac{1}{2}(p_{X\bar{M}} + p_{X\bar{P}}). \quad (\text{A17})$$

Substituting (A13)–(A16) in (A17) yields an expression of p_X as a function of itself that can be solved for p_X

$$p_X = \frac{\alpha + \beta}{8 - 2(1 - \alpha)l_f^2 - (4 + \alpha + \beta)l_f l_m - 2(1 - \beta)l_m^2}. \quad (\text{A18})$$

Substituting (A14) and (A18) in (A13), we get an explicit expression for p_S

$$p_S = \frac{4 - (1 - \alpha)l_f^2 - (1 - \beta)l_m^2 - 2l_f l_m}{8 - 2(1 - \alpha)l_f^2 - (4 + \alpha + \beta)l_f l_m - 2(1 - \beta)l_m^2}. \quad (\text{A19})$$

Substituting (A13), (A14), and (A18) in (A15) we get an explicit expression for $p_{X\bar{M}}$

$$p_{X\bar{M}} = \frac{1}{2} \frac{4\alpha + [\beta(1 - \alpha)l_f^2 + (\beta - \alpha)l_f l_m - (1 - \beta)\alpha l_m^2]}{8 - 2(1 - \alpha)l_f^2 - (4 + \alpha + \beta)l_f l_m - 2(1 - \beta)l_m^2}. \quad (\text{A20})$$

Substituting (A13), (A14), and (A18) in (A16) we get an explicit expression for $p_{X\bar{P}}$

$$p_{X\bar{P}} = \frac{1}{2} \frac{4\beta + [\alpha(1 - \beta)l_m^2 + (\alpha - \beta)l_f l_m - \beta(1 - \alpha)l_f^2]}{8 - 2(1 - \alpha)l_f^2 - (4 + \alpha + \beta)l_f l_m - 2(1 - \beta)l_m^2}. \quad (\text{A21})$$

The relatedness of a focal juvenile to another juvenile in the same patch via its MI gene results from dividing $p_{X\bar{M}}$ and p_S (Grafen 1985; Gardner et al. 2007)

$$r_{\bar{M}} = \frac{1}{2} \frac{\alpha + \frac{1}{4}[\beta(1 - \alpha)l_f^2 + (\beta - \alpha)l_f l_m - \alpha(1 - \beta)l_m^2]}{1 - \frac{1}{4}[(1 - \alpha)l_f^2 + 2l_f l_m + (1 - \beta)l_m^2]} \quad (\text{A22})$$

and the relatedness of a focal juvenile to another juvenile in the same patch via its PI gene results from dividing $p_{X\bar{P}}$ and p_S

$$r_{\bar{P}} = \frac{1}{2} \frac{\beta + \frac{1}{4}[\alpha(1 - \beta)l_m^2 + (\alpha - \beta)l_f l_m - \beta(1 - \alpha)l_f^2]}{1 - \frac{1}{4}[(1 - \alpha)l_f^2 + 2l_f l_m + (1 - \beta)l_m^2]} \quad (\text{A23})$$

See Figure 1 for the graphical representation of the coefficients of relatedness and the scale of competition as functions of l_m , l_f , α , and β .

Condition for increase

The net effect of dispersal on the evolution of altruism will be given by substituting the expression for fitness cost (A9) and fitness benefit (A12) in Hamilton's rule (A7)

$$\frac{c}{b} < \frac{(1 - a)r_\chi}{1 - ar_\chi}. \quad (\text{A24})$$

The right-hand side of the above inequality measures the value of neighbors' survival relative to an individual's personal survival, from the perspective of the χ -inherited gene, and summarizes the extent to which natural selection favors altruism encoded by this gene. We will refer to this term as the potential for altruism of the χ -inherited gene, A_χ . The relation between natural selection and the analogy of the gene as a decision maker is discussed more fully by Dawkins (1976).

The potential for altruism of the maternally and paternally inherited genes are

$$A_{\bar{M}} = \frac{(1 - a)r_{\bar{M}}}{1 - ar_{\bar{M}}} \quad (\text{A26})$$

$$A_{\bar{P}} = \frac{(1 - a)r_{\bar{P}}}{1 - ar_{\bar{P}}}, \quad (\text{A27})$$

where a , $r_{\bar{M}}$ and $r_{\bar{P}}$ are defined as functions of l_m , l_f , α , and β in (A11), (A22), and (A23), respectively.

The difference between the potential for altruism of the maternally inherited and the paternally inherited genes corresponds

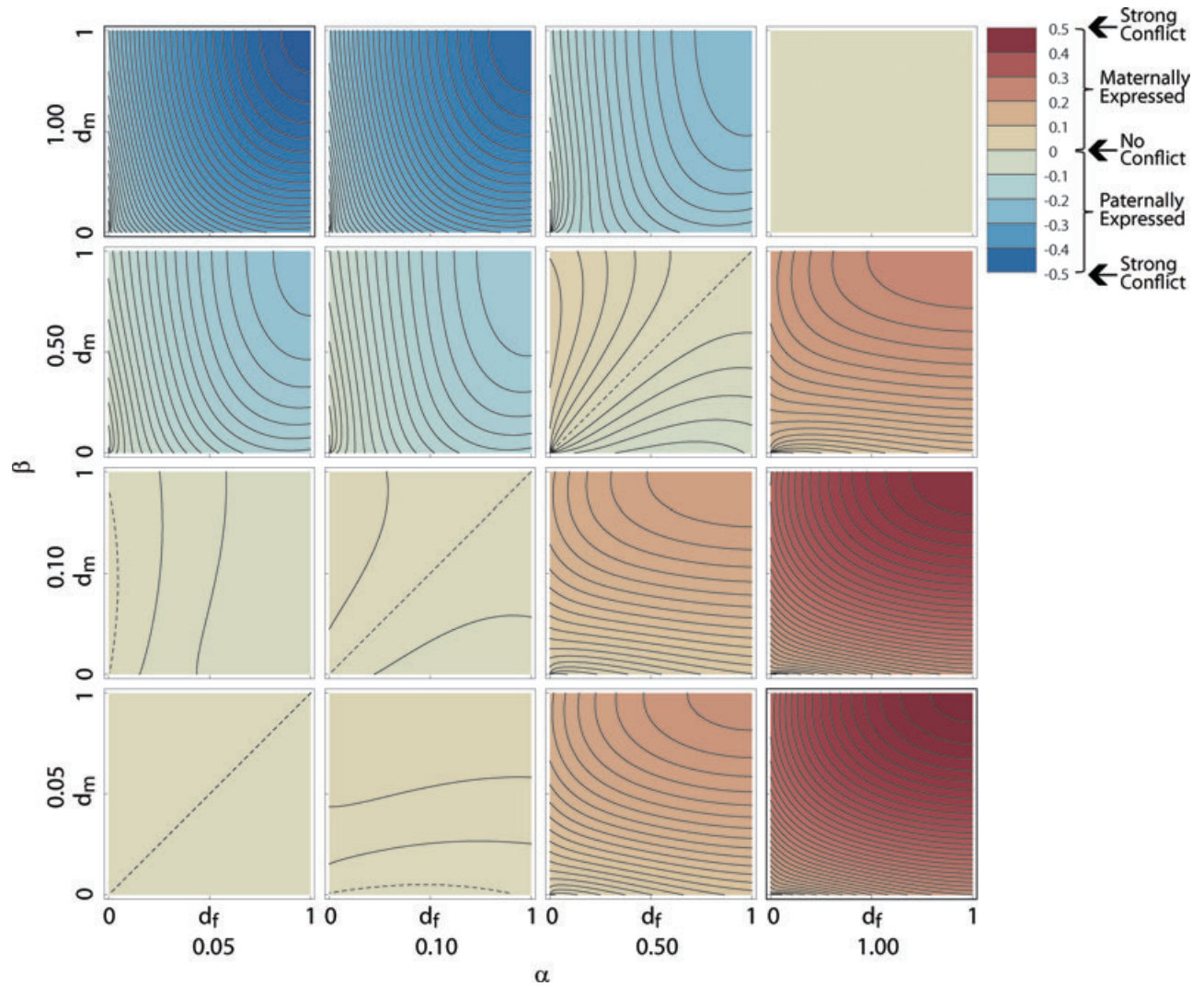


Figure 6. Potential for intragenomic conflict regarding altruism (full range). Grid using four values of the probability of sibship through female α and four values of the probability of sibship through males β covering the range [0.05, 1]. Given a pair of values (α, β) , the contour lines in each figure represent the potential for conflict between genes with different parental origin regarding altruism (I_A) for values of dispersal in females and males.

to the potential for intragenomic conflict between genes with different parental origin regarding altruism

$$I = \frac{(1-a)(r_M - r_P)}{(1-ar_M)(1-ar_P)}, \quad (\text{A26})$$

where a , r_M and r_P are defined as functions of l_m , l_f , α , and β in (A11), (A22), and (A23), respectively. See Figure 1 for the graphical representation of the potential for intragenomic conflict regarding altruism as functions of a and r . See Figures 2 and 6 for the graphical representation of the potential for intragenomic conflict regarding altruism as a function of l_m , l_f , α , and β .

Evolutionarily Stable Strategy

In the previous section, we have established whether a small increase in helping juveniles in the same neighborhood will be

favored by natural selection or not. In this section, we will determine the level of helping such that no deviation from this level will be favored by natural selection, that is the ESS level of helping determined by the maternally and paternally inherited copies of gene g_χ .

Consider a gene for altruistic behavior. Greater expression of this gene results in a survivorship cost for its carrier and a survivorship benefit to neighboring juveniles. The increments in cost and benefit experienced by χ -inherited mutant gene \hat{g}_χ when wild-type gene is g_χ are $S(\hat{x}, y) - S(x, y) = (\partial S/\partial x)(\hat{x} - x) = c$ and $S(x, \hat{y}) - S(x, y) = (\partial S/\partial y)(\hat{y} - y) = b$, respectively.

From invasion condition (A24) we find that a small increment in helping ($\hat{g}_\chi > g_\chi$) is favored when $c < A_\chi b$, a small decrease in helping ($\hat{g}_\chi < g_\chi$) is favored when $c > A_\chi b$, and a resident gene g_χ^* that cannot be invaded by any mutant gene \hat{g}_χ must satisfy

$$\left. \frac{c}{b} \right|_{g_x^*} = A_x. \quad (\text{A28})$$

EGOISM

The previous analysis applies to genes for altruism but similar conditions can be derived for genes for egoism.

Let us redefine fitness cost and benefit as $-C = \partial W/\partial y|_z$, $B = \partial W/\partial x|_z$, and survival cost and benefit as $-c = \partial S/\partial y|_z$, $b =$

$\partial S/\partial x|_z$, respectively. Because C and B are positive, investment into promoting one's survival is formally selfish (Hamilton 1964; West et al. 2007). These changes result in a new term E that we refer to as the potential for egoism. The potential for egoism E corresponds to the inverse of the potential for altruism, that is $E = 1/A$. Keeping these in mind, we can derive the same results as above but now all maxima for the potential for altruism are minima for the potential for egoism.