doi:10.1111/j.1558-5646.2010.01115.x

A MODEL FOR GENOMIC IMPRINTING IN THE SOCIAL BRAIN: ADULTS

Francisco Úbeda[1,2] and Andy Gardner[3]

[1]*Department of Ecology and Evolutionary Biology, University of Tennessee, Knoxville, Tennessee 37996*

[2]*E-mail: fubeda@utk.edu*

[3]*Department of Zoology, University of Oxford, Oxford OX1 3PS, United Kingdom*

Received April 16, 2010
Accepted August 6, 2010

Genomic imprinting refers to genes that are silenced when inherited via sperm or via egg. The silencing of genes conditional upon their parental origin requires an evolutionary explanation. The most widely accepted theory for the evolution of genomic imprinting—the kinship theory—argues that conflict between maternally inherited and paternally inherited genes over phenotypes with asymmetric effects on matrilineal and patrilineal kin results in self-imposed silencing of one of the copies. This theory has been applied to imprinting of genes expressed in the placenta, and infant brain determining the allocation of parental resources being the source of conflict parental promiscuity. However, there is growing evidence that imprinted genes are expressed in the postinfant brain where parental promiscuity per se is no longer a source of conflict. Here, we advance the kinship theory by developing an evolutionary model of genomic imprinting in adults, driven by intragenomic conflict over allocation to parental versus communal care. We consider the role of sex differences in dispersal and variance in reproductive success as sources of conflict. We predict that, in hominids and birds, parental care will be expressed by maternally inherited genes. In nonhominid mammals, we predict more diversity, with some mammals showing the same pattern and other showing the reverse. We use the model to interpret experimental data on imprinted genes in the house mouse: specifically, paternally expressed *Peg1* and *Peg3* genes, underlying maternal care, and maternally expressed *Gnas* and paternally expressed *Gnasxl* genes, underlying communal care. We also use the model to relate ancestral demography to contemporary imprinting disorders of adults, in humans and other taxa.

KEY WORDS: **Autosomal genes, communal care, kin selection, maternal care, paternal care, 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 when maternally inherited (MI) but silenced when paternally inherited (PI, maternally expressed), or expressed when PI but silenced when MI (paternally expressed) (Reik and Walter 2001). This pattern of expression has generated great interest among biologists (Trivers and Burt 1999; Reik and Walter 2001; Haig 2002; Tycko and Morison 2002; Úbeda and Haig 2003; Wilkins and Haig 2003b; Constancia et al. 2004; Burt and Trivers 2006).

Over the years a number of theories have been proposed to explain the evolution of GI (see Wilkins and Haig 2003b for a summary). Recent work hypothesizes that GI is the outcome of genes being selected in the opposite direction in each sex (Day and Bonduriansky 2004). Genes contributed by the sex experiencing stronger selection are predicted to be expressed, whereas genes contributed by the other sex are predicted to be silenced (Day and Bonduriansky 2004). Another recent theory argues that GI results from the coevolution between genes expressed in a mother and her offspring (Wolf and Hager 2006). Silencing of the genes contributed by the father facilitates the co-adaptation of maternal and offspring traits during pregnancy (Wolf and Hager 2006).

However, the most widely accepted explanation for the evolution of GI —the kinship theory— argues that genes with different

parental origin come into conflict over their combined level of expression (Moore and Haig 1991; Trivers and Burt 1999; Tycko and Morison 2002; Úbeda 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 her MI versus her PI genes (Haig 1997). The outcome of this conflict is self-imposed silencing of one of the conflicting genes (Haig 1997; Úbeda and Haig 2003).

Imprinted genes were first identified as mediators of embryonic and infant growth (Moore and Haig 1991; Constancia et al. 2004). Thus, the kinship theory was formulated in terms of conflict between genes expressed in an offspring, and promiscuity was identified as the source of conflict (Moore and Haig 1991; Haig 1997; Úbeda 2008). In particular, genes expressed in an offspring that determine the allocation of parental resources to herself, as opposed to future siblings, are expected to be imprinted when her parents change reproductive partners over their lifetime (Haig 1997; Úbeda 2008). This is because future maternal siblings need not be paternal siblings and future paternal siblings need not be maternal siblings, so future siblings are valued differently by an individual's MI and PI genes (Haig 1997; Úbeda 2008).

There is growing evidence that imprinted genes are also active in the brain of juveniles and adults (postinfants) (Goos and Silverman 2001; Davies et al. 2005, 2007; Plagge et al. 2005). This activity is not readily explained by the formulation of the kinship theory in a familial context, but might be explained by its application in a social context (Trivers and Burt 1999; Haig 2000, 2010; Úbeda and Gardner 2010). A first step toward formulating a kinship theory for the social brain has been to develop a general model for the expression of genes in the brain of juveniles that engage in social interactions with neighboring juveniles (Úbeda and Gardner 2010). This work has shown that genes underlying altruistic and egoistic interactions among juveniles are expected to be imprinted when migration and/or variance in reproductive success (RS) are sex biased (Úbeda and Gardner 2010).

Here, we go a step further, and elaborate a general evolutionary demographic model (Grafen 1985; Taylor 1996; Rousset 2004; Wild and West 2009) for the expression of genes in the brains of adults that engage in social interactions with their own offspring and with neighboring juveniles. We assume that parents have a limited amount of resources that can be allocated either to parental care or to communal care. The allocation of resources to either type of care reduces the resources available for the other type of care. Therefore this model applies to societies in which communal care is common practice (see Discussion for communal care in the animal kingdom).

This model provides insights into the role of imprinted genes in the adult social brain. Our model allows us to relate ancestral demography to contemporary imprinting patterns, and we make clear predictions as to the extent of the conflict (if any), the direction of the imprint, and the effects of mutations and epimutations. We relate the predictions of our model to empirical data on imprinting of mouse genes *Peg1* and *Peg3* underpinning pup retrieval and nest building (forms of maternal care) (Kaneko-Ishino et al. 1995; Lefebvre et al. 1998; Li et al. 1999), and *Gnas* and *Gnasxl* underlying thermoregulation (a form of communal care) (Frontera et al. 2008; Haig 2008).

Model and Results

We consider an infinite population structured into neighborhoods of diploid individuals. Adults engage in social interactions that mediate the survival of juveniles. For simplicity, we focus upon maternal care and female communal care in the main text, but we also present analogous results for paternal care and male communal care in the Appendix. In particular, we consider that mothers can either provide care to their own offspring only (maternal care), or provide care that benefits all juveniles in the neighborhood equally (female communal care) (König 1997). We assume that increasing investment into maternal care leads to a reduced investment into female communal care, and vice versa. We say that a gene underlies maternal care when greater expression of that gene in an adult female results in a greater investment into her own offspring, and we say that a gene underlies female communal care when greater expression of that gene in an adult female results in a greater investment into all local juveniles.

Upon reaching adulthood, individuals disperse with probability d_m for males and d_f for females, respectively, or else they remain in their natal neighborhood with probability $l_m = 1 - d_m$ and $l_f = 1 - d_f$, respectively. After the dispersal phase, 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). The parameters α and β capture the inequity in RS among females and males, respectively (Úbeda and Gardner 2010), and reflect variance in survival as well as variance in mating success among those surviving to adulthood. Note that, owing the assumption of an even sex ratio, $\alpha = \beta$ implies that the variance in RS among females is the same as that among males. Henceforth, we refer to "probability of sibship" when discussing the mathematical model parameter, and we refer to "variance in RS" when discussing the biological phenomenon that this parameter captures.

We use this model to determine the extent to which MI versus PI autosomal genes, expressed in adult females and underlying

communal care, value the survival of neighboring juveniles relative to the survival of their own carrier's offspring: the "potential for female communal care" (see Appendix). When the gene considered is either MI or PI, the potentials are given by

$$Q_{\bar{M}} = \frac{(1-a)r_{\bar{M}}}{1-ar_{\bar{M}}} \tag{1a}$$

$$Q_{\bar{P}} = \frac{(1-a)r_{\bar{P}}}{1-ar_{\bar{P}}}. \tag{1b}$$

Variables r are the coefficients of relatedness between an adult female and a juvenile in the same patch, via the female's MI or PI genes according to the subscript, and are defined as

$$r_{\bar{M}} = \frac{\alpha(1+h_\alpha) + \left(\alpha l_f l_m + 2h_\alpha^2\right)p_X}{2+\alpha l_f l_m + 2l_f l_m(1+h_\alpha)p_X} \tag{2a}$$

$$r_{\bar{P}} = \frac{\alpha+\beta h_\alpha + (\alpha l_f l_m + 2h_\alpha h_\beta)p_X}{2+\beta l_f l_m + 2l_f l_m(1+h_\beta)p_X}, \tag{2b}$$

where p_X is the consanguinity between two juvenile patch-mates, defined as

$$p_X = \frac{\frac{1}{2}(\alpha+\beta)}{4-\frac{1}{2}(h_\alpha+h_\beta)-\frac{1}{4}(4+\alpha+\beta)l_f l_m} \tag{3}$$

and $h_\alpha = (1+\frac{1}{2}\alpha)l_f l_m + (1-\alpha)l_f^2$ and $h_\beta = (1+\frac{1}{2}\beta)l_f l_m + (1-\beta)l_m^2$.

Variable a is the intensity of local competition between neighboring juveniles (Frank 1998), and is defined as

$$a = \frac{1}{2}\left(l_f^2 + l_m^2\right). \tag{4}$$

This quantity accounts for the effects of kin competition (Queller 1992; Taylor 1992; West et al. 2002), a phenomenon that has received much interest in recent years owing to its negative impact upon the evolution of social traits in viscous populations. Although the effects of kin competition exactly cancel the effects of high relatedness in the simplest models of cooperative behavior in viscous populations (Taylor 1992), this is not generally true of models in which the sexes differ in their rates of dispersal. This is because although the intensity of local competition is symmetric in terms of male and female dispersal, the dispersal of males and females can have differing impact upon the relatedness structure of a population (Gardner 2010). Hence, although local competition and relatedness have opposing impact upon the potential for cooperation, these effects need not exactly cancel out in models incorporating sex-biased dispersal.

If providing female communal care incurs an overall survival cost to a female's own offspring c_f and confers an overall survival benefit to all juveniles in her neighborhood b_f, then MI genes are selected to promote female communal care when $\frac{c_f}{b_f} < Q_{\bar{M}}$, and PI genes are selected to promote female communal care when $\frac{c_f}{b_f} < Q_{\bar{P}}$. When MI and PI genes differ in their valuation of the survival of neighboring juveniles relative to the survival of their own offspring there is intragenomic conflict between genes of different parental origin. The potential for intragenomic conflict in females regarding communal care is given by

$$I_Q = Q_{\bar{M}} - Q_{\bar{P}} = \frac{(1-a)(r_{\bar{M}} - r_{\bar{P}})}{(1-ar_{\bar{M}})(1-ar_{\bar{P}})}. \tag{5}$$

Intragenomic conflict manifests as genes being selected for different levels of combined expression. Let $g^*_{\bar{M}}$ and $g^*_{\bar{P}}$ be the evolutionarily stable level of combined expression for MI and PI genes expressed in adult females. If the locus underlies female communal care, then the gene with greater potential for female communal care is selected for a higher level of combined expression; if $I_Q > 0$ then $g^*_{\bar{M}} > g^*_{\bar{P}}$, and if $I_Q < 0$ then $g^*_{\bar{M}} < g^*_{\bar{P}}$. 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: if $I_Q > 0$ then $\{g_{\bar{M}}, g_{\bar{P}}\} = \{g^*_{\bar{M}}, 0\}$, and if $I_Q < 0$ then $\{g_{\bar{M}}, g_{\bar{P}}\} = \{0, g^*_{\bar{P}}\}$. In the event of no difference in potential for altruism, we predict that GI does not evolve: $I_Q = 0$ then $\{g_{\bar{M}}, g_{\bar{P}}\} = \{\frac{1}{2}g^*, \frac{1}{2}g^*\}$ where $g^* = g^*_{\bar{M}} = g^*_{\bar{P}}$.

The results of our model are presented in Figure 1, and the implications for imprinting of genes underlying maternal and female communal care are summarized in Figure 2. Imprinting is expected when there is a sex bias in dispersal ($d_f \neq d_m$) and/or RS ($\alpha \neq \beta$), as well as an opportunity for female communal care.

Assuming equal variance in RS in the two sexes ($\alpha = \beta$), the potential for female communal care is greater for the gene derived from the least dispersing sex, so that a gene underlying female communal care will evolve to be maternally expressed if dispersal is male biased ($\{g^*_{\bar{M}}, 0\}$ if $d_f < d_m$) but paternally expressed if dispersal is female biased ($\{0, g^*_{\bar{P}}\}$ if $d_f > d_m$) (Fig. 1). Assuming equal dispersal of the two sexes ($d_f = d_m$), the potential for female communal care is greater for the gene derived from the sex with the greatest variance in RS, so that a gene underlying female communal care will evolve to be maternally expressed if females have greater variance in RS ($\{g^*_{\bar{M}}, 0\}$ if $\alpha > \beta$) but paternally expressed if males have greater variance in RS ($\{0, g^*_{\bar{P}}\}$ if $\alpha < \beta$) (Fig. 1).

When there is a sex bias both in dispersal and variance in RS, the predictions of our model are more complex. A gene underlying female communal care will evolve to be maternally expressed if dispersal is male biased and variance in RS is female biased ($\{g^*_{\bar{M}}, 0\}$ if $d_f < d_m$ and $\alpha > \beta$), and paternally expressed if dispersal is female biased and variance in RS is male biased ($\{g^*_{\bar{P}}, 0\}$ if $d_f > d_m$ and $\alpha < \beta$) (Figs. 1 and 2). However if dispersal and variance in RS are biased toward the same sex (both greater in females or both greater in males) the direction of imprinting depends on

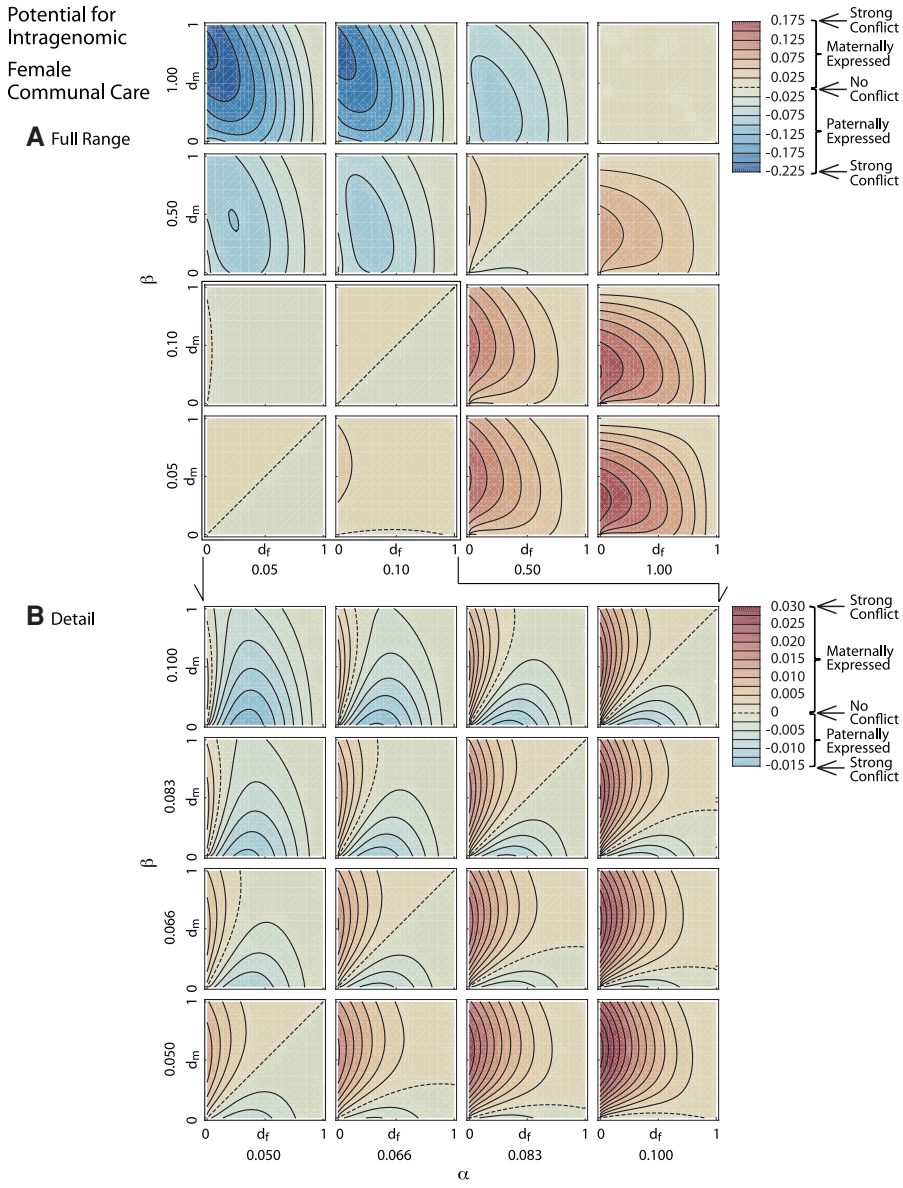

Figure 1. Potential for intragenomic conflict regarding female communal care. (A) Full range. Grid with four values of the probability of maternal sibship (α) and four values of the probability of paternal sibship (β) covering the range [0,1]. Given a pair of values (α,β) the contour lines in each figure represent the potential for intragenomic conflict between genes with different parental origin regarding communal care (I_Q) for values of dispersal in females (d_f) and males (d_m). We use a continuous line for contours in which there is conflict ($I_Q \neq 0$), and a dashed line for contours in which there is no conflict ($I_Q = 0$). We use red color to represent regions of the parameter space in which imprinted genes underlying maternal care are expected to be maternally expressed ($I_Q > 0$) and blue color to represent regions of the parameter space in which imprinted genes are expected to be paternally expressed ($I_Q < 0$). (B) Detail. Grid covering the range [0,0.1] corresponding to the more realistic case of low probability of sibship.

the specific values taken by these demographic parameters (Figs. 1 and 2).

The above analysis can be repeated for genes encoding maternal care. We calculate the extent to which genes underlying maternal care value the survival of own offspring relative to the survival of neighboring juveniles: the potential for maternal care. When the genes considered are maternally-inherited and PI, the potentials are $P_{\bar{M}} = \frac{1}{Q_{\bar{M}}}$ and $P_{\bar{p}} = \frac{1}{Q_{\bar{p}}}$ respectively (see Appendix).

Assuming equal variance in RS in the two sexes ($\alpha = \beta$), the potential for maternal care is greater for the gene derived from the more dispersing sex, so that a gene underlying maternal care will evolve to be paternally expressed if dispersal is male biased ($\{0, g^*_{\bar{p}}\}$ if $d_f < d_m$) but maternally expressed if dispersal is female biased ($\{g^*_{\bar{M}}, 0\}$ if $d_f > d_m$). Assuming equal dispersal of the two sexes ($d_f = d_m$), the potential for maternal care is greater for the gene derived from the sex with the lowest variance in RS, so

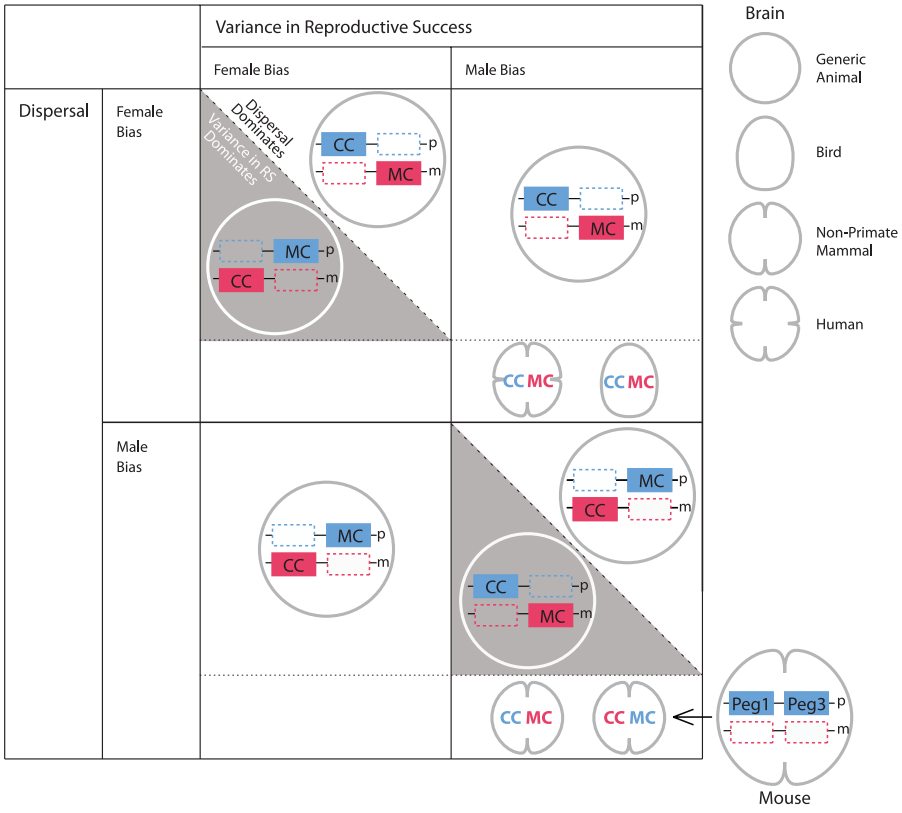

Figure 2. Predicted pattern of expression of genes underlying maternal care. Predicted pattern of expression of genes underpinning maternal care and female communal care when there is a sex bias in dispersal and/or sex-differences in the variance in RS. MC and CC stand for genes underlying maternal care and female communal care, respectively. Blue and red stand for paternally inherited and maternally inherited genes. Solid and dotted rectangles correspond to expressed and silenced genes. Specific predictions (birds, mammals, hominids) are made according to the broad taxonomic distribution of dispersal and variance in RS patterns. The prediction corresponding to paternally expressed genes in mouse underpinning maternal care (*Peg1* and *Peg3*) is indicated.

that a gene underlying maternal care will evolve to be paternally expressed if females have greater variance ($\{0, g^*_{\bar{p}}\}$ if $\alpha > \beta$) but maternally expressed if males have greater variance ($\{g^*_{\bar{M}}, 0\}$ if $\alpha < \beta$).

When there is a sex bias both in dispersal and variance in RS, the predictions of our model are more complex. A gene underlying maternal care to her offspring will evolve to be maternally expressed if dispersal is male biased and variance in RS is female biased ($\{g^*_{\bar{M}}, 0\}$ if $d_f > d_m$ and $\alpha < \beta$) and paternally expressed if dispersal is female biased and variance in RS is male biased ($\{0, g^*_{\bar{p}}\}$ if $d_f < d_m$ and $\alpha > \beta$) (Figs. 1 and 2). However if dispersal and variance in RS are biased toward the same sex, the direction of imprinting depends on the specific values taken by the demographic parameters (Figs. 1 and 2).

Discussion

Our model predicts when GI will evolve in genes underlying parental care and communal care. This is when there is a sex bias in dispersal and/or variance in RS, in addition to a social structure that allows for communal care. Intuitively, lower dispersal in one sex translates into higher relatedness among social partners with respect to genes inherited from that sex. Hence, natural selection favors the expression of genes encoding communal care when they are derived from the more philopatric sex, and it favors the expression of genes encoding parental care when they are derived from the more dispersing sex (Fig. 2). Also, higher variance in RS among individuals of one sex translates into higher relatedness among social partners with respect to genes inherited from that sex. Hence, natural selection favors the expression of genes encoding communal care when they are derived from the sex with higher variance in RS, and it favors the expression of genes encoding parental care when they are derived from the sex with lower variance in RS (Fig. 2).

Unequal dispersal and variance in RS of the sexes is the norm in the birds and mammals (reviewed by Úbeda and Gardner (2010)). In general, males have greater variance in RS, and dispersal is male biased among nonhominid mammals but female biased among birds and hominids. There is some debate as to whether ancestral humans showed female-biased dispersal or a more

balanced dispersal of both sexes (see Úbeda and Gardner 2010 for evidence in favor and against each argument). Henceforth, we assume that ancestral humans showed female-biased dispersal. However, owing to male bias in RS, our qualitative predictions are unchanged if we consider that there is no sex bias in dispersal rates.

Communal care is observed in all major avian and mammalian taxa, including humans (Riedman 1982; Packer et al. 1992; Solomon and Getz 1997; Hrdy 2009). The two extreme cases are despotic societies in which only the dominant female breeds and subordinates help to rear the young, and egalitarian societies in which most or all females (and or males) breed and cooperate on the rearing of the young (König 1997). Our predictions are more focused upon the latter scenario, in which females rear their own offspring and simultaneously provide care for the young in their group. Breeding females are often able to discriminate between their own versus alien young, and consequently preferentially invest in the former (König 1997). Communal care includes behaviors such as feeding (including suckling), grooming, babysitting, nest building, and assistance in thermoregulation (König 1997).

An example of communal care in birds is provided by the groove-billed ani (*Crotophaga sulcirostris*). This ani forms groups of monogamous pairs that construct a single nest, with both sexes participating to raise a joint clutch (Vehrencamp et al. 1986). In particular, they contribute to nestling care, incubation, and defense (Vehrencamp et al. 1986). Before reaching adulthood juveniles migrate, with females dispersing more than males (Bowen et al. 1989). Thus anis form a society with all the ingredients for the evolution of GI in the adult brain, namely: there is no sex bias in variance in RS but female-biased dispersal, and communal care is an option.

The house mouse (*Mus musculus*) provides an example of communal care in mammals. Male mice defend a territory where they mate with multiple females. Females giving birth at the same time form a communal nest and participate in the care of the young (Manning et al. 1995; Hayes 2000; Weber and Olsson 2008). In particular, they contribute to nestling care, thermoregulation, and nursing (Manning et al. 1995; Hayes 2000; Weber and Olsson 2008). Before reaching adulthood, juveniles migrate with males dispersing more than females (Pocock et al. 2005). Thus mice form a society that meets all the requirements for the evolution of GI in the adult brain, namely: there is male bias in variance in RS and male-biased dispersal, and communal care is an option.

Compared to other hominids, humans show a distinctively short interbirth interval resulting in the overlap of nursing and weaned (but nutritionally dependent) offspring. As a result, mothers present a deficit in the production of nutrients for their offspring, that can only be balanced by the surplus of other group members (Hill and Hurtado 2009; Hrdy 2009). There is general agreement that the evolution of short interbirth intervals was possible only after humans became cooperative breeders (Robson and Wood 2008; Sear and Mace 2008; Hill and Hurtado 2009; Hrdy 2009). What is strongly debated is whether supplemental food was contributed by prereproductive and reproductive males (Hill and Hurtado 2009) or by prereproductive and postreproductive females in the group (Sear and Mace 2008; Hrdy 2009). Our model allows for these resources to be contributed by females and/or males.

Given this taxonomic pattern of sex-biased dispersal and variance in RS, our model can be used to predict the taxonomic pattern of GI in the adult brain. Genes underlying communal care are expected to be paternally expressed among birds and hominids, and some diversity is expected among nonhominid mammals: paternally expressed if the effect of dispersal dominates but maternally expressed if the effect of variance in RS dominates (Fig. 2). Conversely genes underlying parental care are expected to be maternally expressed among birds and hominids, and some diversity is expected among nonhominid mammals: maternally expressed if the effect of dispersal dominates but paternally expressed if the effect of variance in RS dominates (Fig. 2).

Understanding the direction of the imprint allows predictions of the clinical phenotype of neurological disorders arising as a consequence of mutations of imprinted genes in humans. Although a normal individual will show a balance between parental care and communal care, mutations tilting the balance toward genes that are normally paternally expressed (deletions or loss-of-function mutations when MI, and epimutations such as loss of maternal imprint or paternal disomy) are expected to result in pathologies related to an excess of communal care (hypercommunal brain). Conversely, mutations tilting the balance toward genes that are normally maternally expressed (deletions and loss-of-function mutations when PI, and epimutations such as loss of paternal imprint or maternal disomy) are expected to result in pathologies related to an excess of parental care (hyperparental brain) (Fig. 3). Hypercommunal and hyperpaternal brains would often represent major disruptions at the level of proximate mechanisms underlying social behavior, and not well-honed adaptations that would function for the good of either MI and PI genes (Úbeda and Gardner 2010).

Assessment of this theory is made difficult by the sparseness of available data. However, some support is provided by studies of gene knockouts in the house mouse. First, we consider imprinted genes underlying maternal care. Mouse genes *Peg1* and *Peg3* are paternally expressed in the adult brain and underlie maternal care (Kaneko-Ishino et al. 1995; Lefebvre et al. 1998; Li et al. 1999). A loss-of-function mutation of genes *Peg1* and *Peg3* results in deficient maternal behavior (Lefebvre et al. 1998; Li et al. 1999): females who carry the mutant *Peg3* allele take longer to initiate nest building and gather their pups together, and show deficiencies in lactation (Li et al. 1999). This observation has posed a

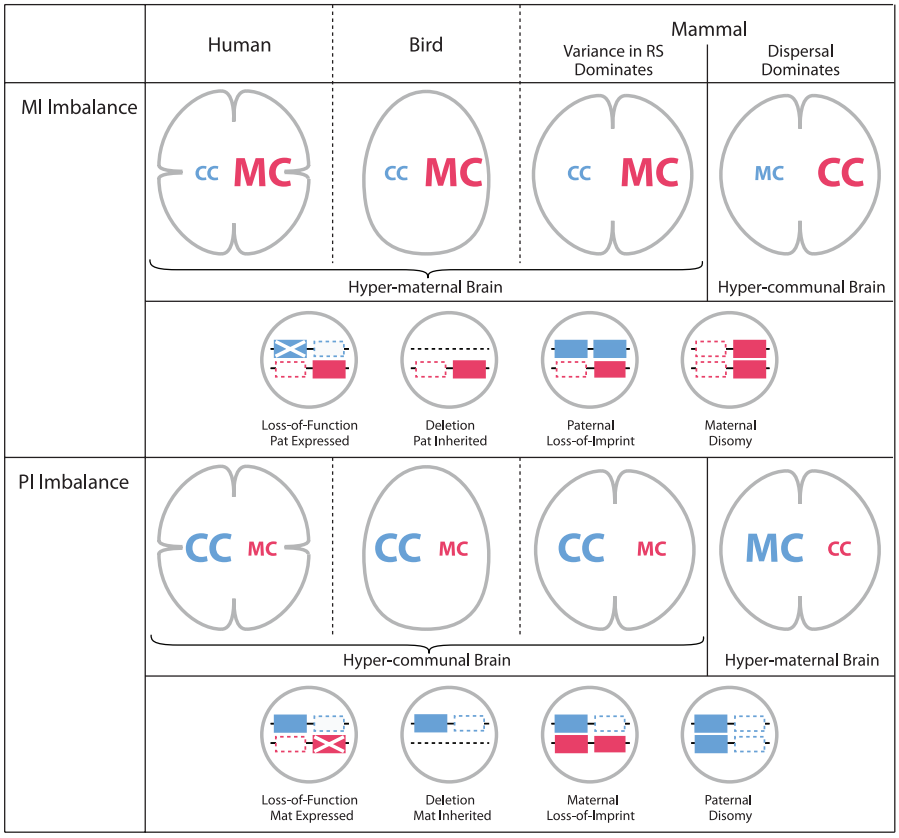

Figure 3. Predicted phenotypes linked to mutations in imprinted genes. Predicted phenotype of mutations (loss-of-function, loss-of-imprint, deletion, uniparental disomy) tilting the balance toward paternally expressed and maternally expressed genes in birds, mammals, hominids, and mouse.

challenge to the kinship theory that, in its original formulation, cannot readily explain imprinting in genes encoding maternal care (Hurst and Mcvean 1998; Hurst et al. 2000; Wilkins and Haig 2003a). In an outbred population, MI and PI genes expressed in a mother do not come into conflict over the allocation of maternal resources (Hurst and Mcvean 1998; Hurst et al. 2000).

Other authors have attempted to explain imprinting for parental care genes. For instance, Wilkins and Haig (2003a) show that in an inbred population however, MI genes can come into conflict with PI genes over the allocation of maternal resources when the rates of matrilineal and patrilineal inbreeding differ and alter over the mother's reproductive lifetime. When this is the case, the valuation of offspring produced at an early age relative to those produced later differs for MI and PI genes expressed in the mother (Wilkins and Haig 2003a). In particular, if the rate of patrilineal inbreeding declines faster than the rate of matrilineal inbreeding, genes underlying maternal care are expected to be paternally expressed, as indeed are *Peg1* and *Peg3*. A loss-of-function mutation of these genes is expected to result in deficient maternal care toward offspring produced at an early age but normal care of offspring produced later in mother's reproductive life (Wilkins and Haig 2003a).

In contrast, our model extends the kinship theory beyond the nuclear family within which mothers provide care to their own offspring, to a social context where adults provide care to juveniles. The house mouse (*M. musculus*) exhibits communal care, with multiple females breeding in each group and participating in the care of young (Manning et al. 1995; Hayes 2000). The house mouse also exhibits male-biased dispersal and male-biased variance in RS thus making predictions on the direction of imprinting conditional on the relative strength of each of these demographic factors (Pocock et al. 2005). If the effect of male-biased dispersal dominates the effect of male-biased variance in RS, our model predicts paternal expression of genes underlying maternal care as observed in *Peg1* and *Peg3*. A loss-of-function mutation of these genes is expected to result in deficient maternal care toward own offspring but normal communal care. Although the predictions in Wilkins and Haig (2003a) are clearly different from the prediction of our model, these models are not mutually exclusive.

Future research should pay attention to the effects of mutations in genes *Peg1* and *Peg3* within a social context. For example, if we introduce a *Peg1* or *Peg3* knockout mother in a communal nest will she show deficiencies in retrieving her own pups but

show more investment in defending the nest? The answer should be yes if these genes account for maternal care.

We turn now to imprinted genes underlying communal care. The mouse gene *Gnas* is maternally expressed but *Gnasxl* is paternally expressed in the adult brain and underlie thermoregulation (Frontera et al. 2008). A loss-of-function mutation of gene *Gnas* results in excess of fat deposition and hypometabolism that inhibits thermogenesis (Frontera et al. 2008). On the contrary, a loss-of-function mutation of gene *Gnasxl* results in moderate fat deposition and hypermetabolism that enhances thermogenesis (Frontera et al. 2008). Mice are born ectothermic and have poor thermoregulatory abilities up to 2–3 weeks of age (Lynch and Possidente 1978; Wilkinson and Baker 1988). Their survival often depends on heat produced by adults that create a microclimate in the nest chamber (König 1997; Hayes 2000). Thermogenesis involves relatively high energy costs for adult mice (König 1997; Hayes 2000). The energetic cost incurred by adults benefits not only the mother's own offspring but all other offspring in the nest thus thermogenesis is a form of communal care for individuals that nest communally (Haig 2008).

Haig (2008) argues that genes controlling thermogenesis in a huddle of littermates are expected to be imprinted when litter mates share the same mother but different father (Haig 2008). This explains imprinting in infants. Interestingly gene *Gnasxl* is paternally expressed in brown adipose tissue of newborns but biallelicaly expressed in brown adipose tissue after weaning (Frontera et al. 2008). However *Gnasxl* is paternally expressed in the brain through adult life. The explanation provided by Haig (2008) does not apply to adults. Genes in a mother huddling with her offspring do not come into conflict over heat production as both the maternally and PI genes are equally related to all her offspring in an outbred population although not in an inbred one (Wilkins and Haig 2003a).

We are interested in the expression of *Gnas* and *Gnasxl* in the adult brain. If the effect of male-biased dispersal dominates the effect of male-biased variance in RS, our model predicts maternal expression of genes reducing thermogenesis as is the case of *Gnas* and paternal expression of genes increasing thermogenesis as is the case of *Gnasxl*.

Gene *Peg3* not only controls maternal care (as discussed above) but also communal care (Frontera et al. 2008). A loss-of-function mutation of gene *Peg3* results in excess of fat deposition and hypometabolism that inhibits thermogenesis (Frontera et al. 2008). The direction of the imprint observed in gene *Peg3* is opposite to the direction predicted by our model. One possible explanation comes from the fact that gene *Peg3* is pleiotropic and there might be selection to be imprinted in the opposite sense for each of the functions that it controls.

Our research illustrates that it is difficult to understand the evolution of GI without establishing the appropriate model for each behavior considered and actors involved. As evidence for the expression of imprinted genes in the adult brain accumulates, there has been a tendency to extrapolate the results of conflict models dealing with nuclear families to social traits, which may result in incorrect predictions. We have shown in this and our previous article (Úbeda and Gardner 2010) that inferring the direction of imprinting in the postinfant brain is not straightforward and that formal models need to be developed if we wish to accurately predict clinical phenotypes of genetic and epigenetic mutations. These models can only be useful if they are supported by reliable ecological and demographic data of animals and ancestral human societies as well as accurate molecular data on temporal patterns of imprinted genes expression.

ACKNOWLEDGMENTS

Our gratitude to K. A. Dyer, G. D. Kelsey, D. N. Tran, J. F. Wilkins, and two anonymous referees for comments on a previous version of this manuscript, and the Wissenschaftskolleg zu Berlin and the Royal Society of London for funding.

LITERATURE CITED

Bowen, B. S., R. R. Koford, and S. L. Vehrencamp. 1989. Dispersal in the communally breeding groove-billed ani (*Crotophaga-Sulcirostris*). Condor 91:52–64.

Burt, A., and R. Trivers. 2006. Genes in conflict. Harvard Univ. Press, Cambridge, MA.

Constancia, M., G. Kelsey, and W. Reik. 2004. Resourceful imprinting. Nature 432:53–57.

Davies, W., A. R. Isles, and L. S. Wilkinson. 2005. Imprinted gene expression in the brain. Neurosci. Biobehav. Rev. 29:421–430.

Davies, W., A. R. Isles, T. Humby, and L. S. Wilkinson. 2007. What are imprinted genes doing in the brain? Epigenetics 2:201–206.

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

Fisher, R. 1930. The genetical theory of natural selection. Clarendon Press, Oxford, U.K..

Frank, S. A. 1998. Foundations of social evolution. Princeton Univ. Press, Princeton, NJ.

Frontera, M., B. Dickins, A. Plagge, and G. Kelsey. 2008. Imprinted genes, postnatal adaptations and enduring effects on energy homeostasis, Pp. 41–61 *in*. J. F. Wilkins, ed. Genomic imprinting. Springer, New York, NY.

Gardner, A. 2010. Sex-biased dispersal of adults mediates the evolution of altruism among juveniles. J. Theor. Biol. 262:339–345.

Goos, L. M., and I. Silverman. 2001. The influence of genomic imprinting on brain development and behavior. Evol. Hum. Behav. 22:385–407.

Grafen, A. 1985. A geometric view of relatedness. Oxford Surv. Evol. Biol. 2:28–89.

Haig, D. 1996. Placental hormones, genomic imprinting, and maternal-fetal communication. J. Evol. Biol. 9:357–380.

———. 1997. Parental antagonism, relatedness asymmetries, and genomic imprinting. Proc. R. Soc. Lond. B 264:1657–1662.

———. 2000. Genomic imprinting, sex-biased dispersal, and social behavior. Ann. N. Y. Acad. Sci. 907:149–163.

———. 2002. Genomic imprinting and kinship. Rutgers Univ. Press, New Brunswick, NJ.

———. 2008. Huddling: brown fat, genomic imprinting and the warm inner glow. Curr. Biol. 18: R172–R174.
———. 2010. Transfers and transitions: parent-offspring conflict, genomic imprinting, and the evolution of human life history. Proc. Natl. Acad. Sci. USA 107:1731–1735.
Hamilton, W. D. 1963. Evolution of altruistic behavior. Am. Nat. 97:354–360.
———. 1964. Genetical evolution of social behaviour I & II. J. Theor. Biol. 7:1–52.
———. 1970. Selfish and spiteful behaviour in an evolutionary model. Nature 228:1218–1222.
Hayes, L. D. 2000. To nest communally or not to nest communally: a review of rodent communal nesting and nursing. Anim. Behav. 59:677–688.
Hill, K., and A. M. Hurtado. 2009. Cooperative breeding in South American hunter-gatherers. Proc. R. Soc. Lond. B 276:3863–3870.
Hrdy, S. B. 2009. Mothers and others: the evolutionary origins of mutual understanding. Harvard Univ. Press, Cambridge, MA.
Hurst, L. D., and G. T. Mcvean. 1998. Do we understand the evolution of genomic imprinting? Curr. Opin. Genet. Develop. 8:701–708.
Hurst, L. D., A. Pomiankowski, and G. T. Mcvean. 2000. *Peg 3* and the conflict hypothesis. Science 287:1167.
Kaneko-Ishino, T., Y. Kuroiwa, N. Miyoshi, T. Kohda, R. Suzuki, M. Yokoyama, S. Viville, S. C. Barton, F. Ishino, M. A. Surani, et al. 1995. Peg1/Mest imprinted gene on chromosome-6 identified by cdna subtraction hybridization. Nat. Genet. 11:52–59.
König, B. 1997. Cooperative care of young in mammals. Naturwissenschaften 84:95–104.
Lefebvre, L., S. Viville, S. C. Barton, F. Ishino, E. B. Keverne, and M. A. Surani. 1998. Abnormal maternal behaviour and growth retardation associated with loss of the imprinted gene mest. Nat. Genet. 20:163–169.
Li, L. L., E. B. Keverne, S. A. Aparicio, F. Ishino, S. C. Barton, and M. A. Surani. 1999. Regulation of maternal behavior and offspring growth by paternally expressed Peg3. Science 284:330–333.
Lynch, C. B., and B. P. Possidente. 1978. Relationships of maternal nesting to thermoregulatory nesting in house mice (*mus-musculus*) at warm and cold temperatures. Anim. Behav. 26:1136–1143.
Manning, C. J., D. A. Dewsbury, E. K. Wakeland, and W. K. Potts. 1995. Communal nesting and communal nursing in house mice, *Mus musculus domesticus*. Anim. Behav. 50:741–751.
Moore, T., and D. Haig. 1991. Genomic imprinting in mammalian development—a parental tug-of-war. Trends Genet. 7:45–49.
Moore, T., and W. Mills. 2008. Evolutionary theories of imprinting—enough already!, Pp. 116–122 *in* J. F. Wilkins, ed. Genomic imprinting. Springer, New York, NY.
Packer, C., S. Lewis, and A. Pusey. 1992. A comparative analysis of non-offspring nursing. Anim. Behav. 43:265–281.
Plagge, A., A. R. Isles, E. Gordon, T. Humby, W. Dean, S. Gritsch, R. Fischer-Colbrie, L. S. Wilkinson, and G. Kelsey. 2005. Imprinted Nesp55 influences behavioral reactivity to novel environments. Mol. Cell. Biol. 25:3019–3026.
Pocock, M. J. O., H. C. Hauffe, and J. B. Searle. 2005. Dispersal in house mice. Biol. J. Linn. Soc. 84:565–583.
Price, G. R., and C. A. B. Smith. 1972. Fishers Malthusian parameter and reproductive value. Ann. Hum. Genet. 36:1–7.
Queller, D. C. 1992. Does population viscosity promote kin selection. Trends Ecol. Evol. 7:322–324.
Reik, W., and J. Walter. 2001. Genomic imprinting: parental influence on the genome. Nat. Rev. Genet. 2:21–32.
Riedman, M. L. 1982. The evolution of alloparental care and adoption in mammals and birds. Q. Rev. Biol. 57:405–435.
Robson, S. L., and B. Wood. 2008. Hominin life history: reconstruction and evolution. J. Anatomy 212:394–425.
Rousset, F. 2004. Genetic structure and selection in subdivided populations. Princeton Univ. Press, Princeton, NJ.
Sear, R., and R. Mace. 2008. Who keeps children alive? A review of the effects of kin on child survival. Evol. Hum. Behav. 29:1–18.
Solomon, N. G., and L. L. Getz. 1997. Examination of alternative hypothesis for cooperative breeding in rodents, Pp. 199–230 *in* N. G. Solomon and J. A. French, eds. Cooperative breeding in mammals. Cambridge Univ. Press, Cambridge.
Taylor, P. D. 1990. Allele-frequency change in a class-structured population. Am. Nat. 135:95–106.
———. 1992. Altruism in viscous populations—an inclusive fitness model. Evol. Ecol. 6:352–356.
———. 1996. Inclusive fitness arguments in genetic models of behaviour. J. Math. Biol. 34:654–674.
Taylor, P. D., and S. A. Frank. 1996. How to make a kin selection model. J. Theor. Biol. 180:27–37.
Trivers, R., and A. Burt. 1999. Kinship and genomic imprinting, Pp. 1–17 *in* R. Ohlsson, ed. Genomic imprinting: an interdisciplinary approach. Springer, Heidelberg.
Tycko, B., and I. M. Morison. 2002. Physiological functions of imprinted genes. J. Cell. Physiol. 192:245–258.
Úbeda, F. 2008. Evolution of genomic imprinting with biparental care: implications for Prader-Willi and Angelman syndromes. Plos Biol. 6:1678–1692.
Úbeda, F., and A. Gardner. 2010. A model for genomic imprinting in the social brain: juveniles. Evolution: 64:2587–2600.
Úbeda, F., and D. Haig. 2003. Dividing the child. Genetica 117:103–110.
Vehrencamp, S. L., B. S. Bowen, and R. R. Koford. 1986. Breeding roles and pairing patterns within communal groups of groove-billed anis. Anim. Behav. 34:347–366.
Weber, E. M., and I. A. S. Olsson. 2008. Maternal behaviour in *Mus musculus* sp.: an ethological review. Appl. Anim. Behav. Sci. 114:1–22.
West, S. A., I. Pen, and A. S. Griffin. 2002. Conflict and cooperation—cooperation and competition between relatives. Science 296:72–75.
Wild, G., and S. A. West. 2009. Genomic imprinting and sex allocation. Am. Nat. 173: E1–E14.
Wilkins, J. F., and D. Haig. 2003a. Inbreeding, maternal care and genomic imprinting. J. Theor. Biol. 221:559–564.
———. 2003b. What good is genomic imprinting: the function of parent-specific gene expression. Nat. Rev. Genet. 4:359–368.
Wilkinson, G. S., and A. E. M. Baker. 1988. Communal nesting among genetically similar house mice. Ethology 77:103–114.
Wolf, J. B., and R. Hager. 2006. A maternal-offspring coadaptation theory for the evolution of genomic imprinting. Plos Biol. 4:2238–2243.

Associate Editor: K. Dyer

Appendix

JUVENILE SURVIVAL

The probability of survival for a focal juvenile may be written as

$$S\left(x_f, x_m, y_f, y_m\right), \tag{A1}$$

where x_f is the investment into communal care made by the individual's mother, x_m is the investment into communal care made by the individual's father, y_f is the average investment into communal care by females in the social group (i.e., made by the individual's "aunts"), and y_m is the average investment into communal care

by males in the social group (i.e., made by the individual's "uncles"). We assume that the juvenile's survival is a monotonically decreasing function of its parents' communal care (owing to the concomitant reduction in parental care) and a monotonically increasing function of its aunts' and uncles' communal care

$$\frac{\partial S}{\partial x_\sigma} = -C_\sigma < 0, \tag{A2a}$$

and

$$\frac{\partial S}{\partial y_\sigma} = B_\sigma > 0, \tag{A2b}$$

where $\sigma \in (f, m)$. We denote the average investment into communal care of all breeding adults (of the corresponding sex) in the population by z_σ.

JUVENILE NEIGHBOR-MODULATED FITNESS

We begin by calculating the expected fitness of a focal juvenile female as a function of the investment into communal care made by her parents and her aunts and uncles. If the focal female survives to adulthood, then she may either disperse (with probability d_f) or else remain in her natal patch (with probability $l_f = 1 - d_f$). Her subsequent breeding success is inversely proportional to the number of females competing within the patch upon which she finds herself after the dispersal phase (Úbeda and Gardner 2010). Expressed relative to the average for her class (i.e., all juvenile females), this is

$$W_f = S(x_f, x_m, y_f, y_m) \times \left[\frac{l_f}{l_f S(y_f, y_m, y_f, y_m) + (1 - l_f) S(z_f, z_m, z_f, z_m)} + \frac{1 - l_f}{S(z_f, z_m, z_f, z_m)} \right]. \tag{A3}$$

We now calculate the expected fitness of a focal juvenile male. If he survives to adulthood, then he may either disperse (with probability d_m) or remain in his natal patch (with probability $l_m = 1 - d_m$). His subsequent breeding success is proportional to the ratio of adult females to adult males within the patch upon which he finds himself after the dispersal phase. The expected number of offspring produced by his mates will be inversely proportional to the number of females competing within the patch upon which he finds himself after the dispersal phase (Úbeda and Gardner 2010). Thus, his expected fitness, expressed relative to the average for his class (juvenile males), is

$$W_m = S(x_f, x_m, y_f, y_m) \times \left[\frac{l_m}{l_m S(y_f, y_m, y_f, y_m) + (1 - l_m) S(z_f, z_m, z_f, z_m)} + \frac{1 - l_m}{S(z_f, z_m, z_f, z_m)} \right]. \tag{A4}$$

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). Hence, the expected fitness of a gene is given by

$$W = \frac{1}{2} W_f + \frac{1}{2} W_m, \tag{A5}$$

where the weightings of 1/2 are the class reproductive values for females and males under diploidy (Fisher 1930; Price and Smith 1972; Taylor 1990).

From equations (A3) to (A5), we can derive an expression for the expected fitness cost $-C_\sigma = \partial W/\partial x_\sigma$ and benefit $B_\sigma = \partial W/\partial y_\sigma$ experienced by a juvenile due to its parents' and its aunts' and uncles' communal care, respectively. Partial derivatives (C_σ, B_σ) are evaluated at $x_\sigma = y_\sigma = z_\sigma$.

The fitness costs due to the juvenile's mother's and father's investments into communal care are

$$-C_f = -\frac{c_f}{S}, \tag{A6a}$$

and

$$-C_m = -\frac{c_m}{S}, \tag{A6b}$$

where $c_\sigma = \partial S/\partial x_\sigma$ is the survival cost experienced by the juvenile due to its mother's or father's investment into communal care. Again, partial derivatives (c_σ) and survival (S) are evaluated at $x_\sigma = y_\sigma = z_\sigma$.

The fitness benefits due to its aunts' and uncles' investments into communal care are

$$B_f = \frac{b_f - a\left(b_f - c_f\right)}{S}, \tag{A7a}$$

and

$$B_m = \frac{b_m - a\left(b_m - c_m\right)}{S}, \tag{A7b}$$

where $b_\sigma = \partial S/\partial y_\sigma$ is the survival benefit experienced by juveniles owing to the investment into communal care made by aunts or uncles, and $a = ({l_f}^2 + {l_m}^2)/2$ is the "scale of competition" between juvenile patch mates (Frank 1998; Gardner 2010). Partial derivatives (b_σ and c_σ) and survival (S) are once again evaluated at $x_\sigma = y_\sigma = z_\sigma$.

Condition and Potential for Communal Care and Parental Care

FEMALE COMMUNAL CARE AND MATERNAL CARE

Let an individual's genetic value for "female communal care when MI" be $g_{f\bar{M}}$ and for "female communal care when PI" be $g_{f\bar{p}}$. Generically, the genetic value for "communal care when χ-inherited" is denoted $g_{f\chi}$, and the condition for increase in

this trait is $dW/dg_{f\chi} > 0$ (Hamilton 1963, 1964, 1970; Taylor and Frank 1996; Wild and West 2009), where the derivative is evaluated at $x_\chi = y_\chi = z_\chi$.

The derivative of W with respect to $g_{f\chi}$ is

$$\frac{dW}{dg_{f_\chi}} = \frac{\partial W}{\partial x_f}\frac{dx_f}{dg_{f\chi}} + \frac{\partial W}{\partial y_f}\frac{dy_f}{dg_{f\chi}}. \tag{A8}$$

Here, the partial derivatives $\partial W/\partial x_f = -C_f$ and $\partial W/\partial y_f = B_f$ are the fitness cost and benefit of female communal care. The derivative $dx_f/dg_{f\chi} = p_{M\chi}$ is the consanguinity of the focal juvenile and its mother's χ-inherited genome, that is, the probability that a gene picked at random from the juvenile is identical by descent to the gene at the same locus in mother's χ-inherited genome. The derivative $dy_M/dg_{f\chi} = p_{A\chi}$ is the consanguinity of the focal juvenile to an aunt's χ-inherited genome.

Substituting into the condition for increase, $dW/dg_{f\chi} > 0$, yields

$$-\frac{c_f}{S}p_{M\chi} + \frac{b_f - a\left(b_f - c_f\right)}{S}p_{A\chi} > 0, \tag{A9}$$

which can be rearranged as

$$-c_f + \left(b_f - a\left(b_f - c_f\right)\right)r_{A\chi} > 0, \tag{A10}$$

where $r_{A\chi} = p_{A\chi}/p_{M\chi}$ is the relatedness of a juvenile to its aunt's χ-inherited genome.

Assuming diminishing survival returns upon investment into female communal care, the ESS (level of investment such that no deviation from this level will be favored by natural selection) for female communal care satisfies equation (A10) with equality, which can be rearranged as

$$\frac{c_f}{b_f} = \frac{(1-a)r_{A\chi}}{1 - ar_{A\chi}}. \tag{A11}$$

Female communal care is favored by χ-inherited genes when $c_f/b_f < Q_{A\chi}$ and is disfavored when $c_f/b_f > Q_{A\chi}$, where

$$Q_{A\chi} = \frac{(1-a)r_{A\chi}}{1 - ar_{A\chi}} \tag{A12}$$

is the "potential for female communal care" from the perspective of the χ-inherited gene. The potential $Q_{A\chi}$ can be interpreted as the valuation made by an adult female's χ-inherited genome of the survival of same-patch juveniles relative to the survival of her own offspring. In the main text, the focus is upon female care, and hence there we drop the subscript A (for aunt) for simplicity, that is, the potential for female communal care is denoted Q_χ.

Maternal care is favored when $c_f/b_f < P_{A\chi}$ and is disfavored when $c_f/b_f > P_{A\chi}$, where $P_{A\chi} = 1/A_{A\chi}$ is the potential for the female's χ-inherited genome to invest in maternal care as opposed to female communal care. In the main text, where the focus is upon female care, we drop the subscript A (for aunt) and denote this potential for maternal care by P_χ.

MALE COMMUNAL CARE AND PATERNAL CARE

Let an individual's genetic value for "male communal care when MI" be $g_{m\bar{M}}$ and for "male communal care when PI" be $g_{m\bar{p}}$. Generically, the genetic value for "male communal care when χ-inherited" is denoted by $g_{m\chi}$, and the condition for increase in this trait is $dW/dg_{m\chi} > 0$, where the derivative is evaluated at $x_\chi = y_\chi = z_\chi$.

The derivative of W respect to $g_{m\chi}$ is

$$\frac{dW}{dg_{m_\chi}} = \frac{\partial W}{\partial x_m}\frac{dx_m}{dg_{m_\chi}} + \frac{\partial W}{\partial y_m}\frac{dy_m}{dg_{m_\chi}}, \tag{A13}$$

where the partial derivatives $\partial W/\partial x_m = -C_m$ and $\partial W/\partial y_m = B_m$ are, respectively, the fitness cost and benefit due to male communal care. The derivative $dx_m/dg_{m\chi} = p_{P\chi}$ is the consanguinity of the juvenile to its father's χ-inherited genome, and $dy_m/d\, g_{m\chi} = p_{U\chi}$ is the consanguinity of the juvenile to an uncle's χ-inherited genome.

Substituting into the condition for increase yields

$$-\frac{c_m}{S}p_{P\chi} + \frac{b_m - a\left(b_m - c_m\right)}{S}p_{U\chi} > 0, \tag{A14}$$

which can be rearranged as

$$-c_m + \left(b_m - a\left(b_m - c_m\right)\right)r_{U\chi} > 0, \tag{A15}$$

where $r_{U\chi} = p_{U\chi}/p_{P\chi}$ is the relatedness of a juvenile to its uncle's χ-inherited genome.

Assuming diminishing survival returns upon investment into helping, the ESS for male communal care satisfies equation (A15) with equality, which can be rearranged as

$$\frac{c_m}{b_m} = \frac{(1-a)r_{U\chi}}{1 - ar_{U\chi}}. \tag{A16}$$

Male communal care is favored when $c_m/b_m < Q_{U\chi}$ and is disfavored when $c_m/b_m > Q_{U\chi}$, where

$$Q_{U\chi} = \frac{(1-a)r_{U\chi}}{1 - ar_{U\chi}} \tag{A17}$$

is the "potential for male communal care" from the perspective of the χ-inherited genome. The potential $Q_{U\chi}$ can be interpreted as the valuation made by χ-inherited genes in the adult male of the survival of same-patch juveniles relative to the survival of his own offspring.

Paternal care is favored when $c_m/b_m < P_{U\chi}$ and is disfavored when $c_m/b_m > P_{U\chi}$, where $P_{U\chi} = 1/Q_{U\chi}$ is the potential for the male's χ-inherited genome to invest in paternal care as opposed to communal care.

Coefficient of Relatedness

RELATEDNESS OF JUVENILE TO AUNT

Relatedness of Juvenile to Aunt's MI Genome: $r_{A\bar{M}}$

Consanguinity of Juvenile to Mother's MI Genome: $p_{M\bar{M}}$. The consanguinity between a juvenile and its mother's maternally inherited (MI) genome is the probability that a gene drawn at random from a juvenile is identical by descent to its mother's MI gene at the same locus. This is

$$p_{M\bar{M}} = \frac{1}{2}p_S + \frac{1}{2}\varphi_{\bar{M}}, \tag{A18}$$

where p_S is the consanguinity of an individual to itself, and $\varphi_{\bar{M}}$ is the consanguinity of an individual and its mating partner's MI genome.

Note that the consanguinity of an individual to itself is

$$p_S = \frac{1}{2} + \frac{1}{2}\varphi, \tag{A19}$$

where φ is the consanguinity of mating partners.

Substituting (A19) into (A18) yields

$$p_{M\bar{M}} = \frac{1}{4} + \frac{1}{2}\left(\frac{1}{2}\varphi + \varphi_{\bar{M}}\right). \tag{A20}$$

The consanguinities of an individual to its mating partner's MI genome and paternally inherited (PI) genome are, respectively

$$\varphi_{\bar{M}} = l_f l_m p_{X\bar{M}}, \tag{A21a}$$

and

$$\varphi_{\bar{P}} = l_f l_m p_{X\bar{P}}, \tag{A21b}$$

where $p_{X\chi}$ is the consanguinity of an individual to the χ-inherited genome of a random individual born on the same patch in the same generation.

The consanguinity between mating partners is

$$\varphi = l_f l_m p_X, \tag{A22}$$

where p_X is the consanguinity of two individuals born on the same patch in the same generation.

Substituting (A21a) and (A22) into (A20) yields

$$p_{M\bar{M}} = \frac{1}{4} + \frac{1}{2}l_f l_m\left(\frac{1}{2}p_X + p_{X\bar{M}}\right). \tag{A23}$$

The consanguinities of an individual to the MI versus PI genome of an individual born in the same patch in the same generation are, respectively

$$p_{X\bar{M}} = \frac{1}{2}\left(\alpha p_S + (1-\alpha)\, l_f^2 p_X\right) + \frac{1}{2}\varphi, \tag{A24a}$$

and

$$p_{X\bar{P}} = \frac{1}{2}\left(\beta p_S + (1-\beta)\, l_m^2 p_X\right) + \frac{1}{2}\varphi. \tag{A24b}$$

The consanguinity of two individuals born in the same patch in the same generation is

$$p_X = \frac{1}{2}\left(p_{X\bar{M}} + p_{X\bar{P}}\right), \tag{A25}$$

where $p_{X\bar{M}}$ and $p_{X\bar{P}}$ are the probabilities of maternal and paternal sibship for same-patch juveniles, respectively.

Substituting (A19), (A24a), and (A24b) into (A25) yields an expression in terms of φ and p_X; substituting in (22) yields an expression in terms of p_X

$$p_X = \frac{1}{4}\left(\alpha\frac{1}{2}\left(1 + l_f l_m p_X\right) + (1-\alpha)\, l_f^2 p_X\right) + \frac{1}{2}l_f l_m p_X + \frac{1}{4}\left(\beta\frac{1}{2}\left(1 + l_f l_m p_X\right) + (1-\beta)\, l_m^2 p_X\right), \tag{A26}$$

which can be solved for p_X

$$p_X = \frac{\alpha + \beta}{8 - (4 + \alpha + \beta)\, l_f l_m - (1-\alpha)\, l_f^2 - (1-\beta)\, l_m^2}. \tag{A27}$$

Substituting (A19) and (A22) into (A24a) yields

$$p_{X\bar{M}} = \frac{1}{2}\alpha + \frac{1}{2}h_\alpha p_X, \tag{A28}$$

where $h_\alpha = l_f((1 + 1/2\alpha)l_m + (1-\alpha)l_f)$.

Substituting (A28) into (A23) yields an expression of $p_{M\bar{M}}$ in terms of p_X

$$p_{M\bar{M}} = \frac{1}{4}\left(1 + \frac{1}{2}\alpha l_m l_f\right) + \frac{1}{4}l_f l_m\left(1 + h_\alpha\right) p_X. \tag{A29}$$

Consanguinity of Juvenile to Aunt's MI Genome: $p_{A\bar{M}}$. The consanguinity between a juvenile and its aunt's MI genome is the probability that a gene drawn at random from a juvenile is identical by descent to its aunt's MI gene at the same locus. This is

$$p_{A\bar{M}} = \frac{1}{2}\left(\alpha p_{M\bar{M}} + (1-\alpha)\, l_f^2 p_{X\bar{M}}\right) + \frac{1}{2}l_f l_m p_{X\bar{M}}. \tag{A30}$$

Substituting (A28) and (A29) into (A30) yields an expression in terms of p_X

$$p_{A\bar{M}} = \frac{1}{8}\alpha\left(1 + h_\alpha\right) + \frac{1}{4}\left(\frac{1}{2}\alpha l_f l_m + h_\alpha^2\right) p_X. \tag{A31}$$

Relatedness of Juvenile to Aunt's MI Genome: $r_{A\bar{M}}$. The relatedness of a juvenile to its aunt's MI genome is

$$r_{A\bar{M}} = \frac{p_{A\bar{M}}}{p_{M\bar{M}}}. \tag{A32}$$

Substituting (A29) and (A31) into (A32) yields

$$r_{A\bar{M}} = \frac{\alpha\left(1 + h_\alpha\right) + \left(\alpha l_f l_m + 2h_\alpha^2\right) p_X}{2 + \alpha l_f l_m + 2l_f l_m\left(1 + h_\alpha\right) p_X}. \tag{A33}$$

This relatedness coefficient can now be substituted into the potential for female communal care with respect to the MI genome; from (A12), this is

$$Q_{A\bar{M}} = \frac{(1-a)\, r_{A\bar{M}}}{1 - a r_{A\bar{M}}}. \tag{A34}$$

Relatedness of Juvenile to Aunt's PI Genome: $r_{A\bar{P}}$

Consanguinity of Juvenile to Mother's PI Genome: $p_{M\bar{P}}$. The consanguinity between a juvenile and its mother's PI genome is the probability that a gene drawn at random from a juvenile is identical by descent to its mother's PI gene at the same locus. This is

$$p_{M\bar{P}} = \frac{1}{2} p_S + \frac{1}{2} \varphi_{\bar{P}}. \tag{A35}$$

Substituting (A19), (A21b), and (A22) into (A35) yields

$$p_{M\bar{P}} = \frac{1}{4} + \frac{1}{2} l_f l_m \left(\frac{1}{2} p_X + p_{X\bar{P}} \right). \tag{A36}$$

Substituting (A19) and (A22) into (A24b) yields

$$p_{X\bar{P}} = \frac{1}{4}\beta + \frac{1}{2} h_\beta p_X, \tag{A37}$$

where $h_\beta = l_m((1 + \frac{1}{2}\beta) l_f + (1-\beta) l_m)$. Substituting (A37) into (A36) yields an expression for $p_{M\bar{P}}$ in terms of p_X

$$p_{M\bar{P}} = \frac{1}{4} + \frac{1}{8}\beta l_f l_m + \frac{1}{4} l_f l_m \left(1 + h_\beta\right) p_X. \tag{A38}$$

Consanguinity of Juvenile to Aunt's PI Genome: $p_{A\bar{P}}$. The consanguinity between a juvenile and its aunt's PI genome is the probability that a gene drawn at random from a juvenile is identical by descent to its aunts' PI gene at the same locus. This is

$$p_{A\bar{P}} = \frac{1}{2} \left(\alpha p_{M\bar{P}} + (1-\alpha) l_f^2 p_{X\bar{P}} \right) + \frac{1}{2} l_f l_m p_{X\bar{P}}. \tag{A39}$$

Substituting (A37) and (A38) into (A39) yields

$$p_{A\bar{P}} = \frac{1}{8} (\alpha + \beta h_\alpha) + \frac{1}{4} \left(\frac{1}{2} \alpha l_f l_m + h_\alpha h_\beta \right) p_X. \tag{A40}$$

Relatedness of Juvenile to Aunt's PI Genome: $r_{A\bar{P}}$. The relatedness of a juvenile to its aunt's PI genome is

$$r_{A\bar{P}} = \frac{p_{A\bar{P}}}{p_{M\bar{P}}}. \tag{A41}$$

Substituting (A38) and (A40) into (A41) yields

$$r_{A\bar{P}} = \frac{\alpha + \beta h_\alpha + \left(\alpha l_f l_m + 2 h_\alpha h_\beta \right) p_X}{2 + \beta l_f l_m + 2 l_f l_m \left(1 + h_\beta\right) p_X}. \tag{A42}$$

This relatedness coefficient can now be substituted into the potential for female communal care with respect to the PI genome; from (A12), this is

$$Q_{A\bar{P}} = \frac{(1-a)\, r_{A\bar{P}}}{1 - a r_{A\bar{P}}}. \tag{A43}$$

RELATEDNESS OF JUVENILE TO UNCLE

Relatedness of Juvenile to Uncle's MI Genome: $r_{U\bar{M}}$

Consanguinity of Juvenile to Father's MI Genome: $p_{P\bar{M}}$. The consanguinity between a juvenile and its father's MI genome is the probability that a gene drawn at random from a juvenile is identical by descent to its father's MI gene at the same locus. Owing to symmetry, this is the same as the consanguinity of a juvenile to its mother's MI genome, given in (A29), that is

$$p_{P\bar{M}} = \frac{1}{4} \left(1 + \frac{1}{2} \alpha l_m l_f \right) + \frac{1}{4} l_f l_m \left(1 + h_\alpha\right) p_X. \tag{A44}$$

Consanguinity of Juvenile to Uncle's MI Genome: $p_{U\bar{M}}$. The consanguinity between a juvenile and its uncle's MI genome is the probability that a gene drawn at random from a juvenile is identical by descent to its uncle's MI gene at the same locus.

$$p_{U\bar{M}} = \frac{1}{2} \left(\beta p_{P\bar{M}} + (1-\beta) l_m^2 p_{X\bar{M}} \right) + \frac{1}{2} l_f l_m p_{X\bar{M}}. \tag{A45}$$

Substituting (A28) and (A44) into (A45) yields an expression in terms of p_X

$$p_{U\bar{M}} = \frac{1}{8} \left(\beta + \alpha h_\beta \right) + \frac{1}{4} \left(\frac{1}{2} \beta l_f l_m + h_\alpha h_\beta \right) p_X. \tag{A46}$$

Relatedness of Juvenile to Uncle's MI Genome: $r_{U\bar{M}}$. The relatedness of a juvenile to its uncles' MI genome is

$$r_{U\bar{M}} = \frac{p_{U\bar{M}}}{p_{P\bar{M}}}. \tag{A47}$$

Substituting (A44) and (A46) into (A47) yields

$$r_{U\bar{M}} = \frac{\beta + \alpha h_\beta + \left(\beta l_f l_m + 2 h_\beta h_\alpha \right) p_X}{2 + \alpha l_f l_m + 2 l_f l_m \left(1 + h_\alpha\right) p_X}. \tag{A48}$$

This relatedness coefficient can now be substituted into the potential for male communal care with respect to the MI genome; from (A17), this is

$$Q_{U\bar{M}} = \frac{(1-a)\, r_{U\bar{M}}}{1 - a r_{U\bar{M}}}. \tag{A49}$$

Relatedness of Juvenile to Uncle's PI Genome: $r_{U\bar{P}}$

Consanguinity of Juvenile to Father's PI Genome: $p_{P\bar{P}}$. The consanguinity between a juvenile and its father's PI genome

is the probability that a gene drawn at random from a juvenile is identical by descent to its father's PI gene at the same locus. Owing to symmetry, this is the same as the consanguinity of a juvenile to its mother's PI genome, given in (A38), that is

$$p_{P\bar{P}} = \frac{1}{4} + \frac{1}{8}\beta l_f l_m + \frac{1}{4} l_f l_m \left(1 + h_\beta\right) p_X. \quad \text{(A50)}$$

Consanguinity of Juvenile to Uncle's PI Genome: $p_{U\bar{P}}$. The consanguinity between a juvenile and its uncle's PI genome is the probability that a gene drawn at random from a juvenile is identical by descent to its uncle's PI gene at the same locus. This is

$$p_{U\bar{P}} = \frac{1}{2}\left(\beta p_{P\bar{P}} + (1-\beta) l_m^2 p_{X\bar{P}}\right) + \frac{1}{2} l_f l_m p_{X\bar{P}}. \quad \text{(A51)}$$

Substituting (A37) and (A50) into (A51) yields an expression for $p_{U\bar{P}}$ in terms of p_X

$$p_{U\bar{P}} = \frac{1}{8}\left(\beta + \alpha h_\beta\right) + \frac{1}{4}\left(\frac{1}{2}\beta l_f l_m + h_\alpha h_\beta\right) p_X. \quad \text{(A52)}$$

Relatedness of Juvenile to Uncle's PI Genome: $r_{U\bar{P}}$. The relatedness of a juvenile to its uncle's PI genome is

$$r_{U\bar{P}} = \frac{p_{U\bar{P}}}{p_{P\bar{P}}}. \quad \text{(A53)}$$

Substituting (A50) and (A52) into (A53) yields

$$r_{U\bar{P}} = \frac{\beta + \alpha h_\beta + \left(\beta l_f l_m + 2 h_\alpha h_\beta\right) p_X}{2 + \alpha l_f l_m + 2 l_f l_m \left(1 + h_\alpha\right) p_X}. \quad \text{(A54)}$$

This relatedness coefficient can now be substituted into the potential for allo-paternal care with respect to the PI genome; from (A17), this is

$$Q_{U\bar{P}} = \frac{(1-a) r_{U\bar{P}}}{1 - a r_{U\bar{P}}}. \quad \text{(A55)}$$

Potential for Conflict

The potential for communal care may differ for the MI and PI genomes of the same individual, leading to a potential for intragenomic conflict. We denote the potential for intragenomic conflict over female communal care by

$$I_A = Q_{A\bar{M}} - Q_{A\bar{P}} = \frac{(1-a)(r_{A\bar{M}} - r_{A\bar{P}})}{(1 - a r_{A\bar{M}})(1 - a r_{A\bar{P}})}. \quad \text{(A56)}$$

Similarly, we denote the potential for intragenomic conflict over male communal care by

$$I_U = Q_{U\bar{M}} - Q_{U\bar{P}} = \frac{(1-a)(r_{U\bar{M}} - r_{U\bar{P}})}{(1 - a r_{U\bar{M}})(1 - a r_{U\bar{P}})}. \quad \text{(A57)}$$