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A MODEL FOR GENOMIC IMPRINTING IN THE SOCIAL BRAIN: ELDERS

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Genomic imprinting refers to the process whereby genes are silenced when inherited via sperm or egg. 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 was originally developed in the context of fitness interactions within nuclear families, to understand intragenomic conflict in the embryo and infant, but it has recently been extended to encompass interactions within wider social groups, to understand intragenomic conflict over the social behavior of juveniles and adults. Here, we complete our model of genomic imprinting in the social brain by considering age-specific levels of expression in a society were generations overlap, to determine how intragenomic conflict plays out in older age. We determine the role of sex bias in juvenile dispersal, reproductive success, and adult mortality in mediating the direction and intensity of conflict over the competing demands of parental and communal care as the individual ages. We discover that sex-specific asymmetries in these demographic parameters result in intragenomic conflict at early age but this conflict gradually decays with age. Although individuals are riven by internal conflict in their youth and middle age, they put their demons to rest in later life.

KEY WORDS: **Autosomal genes, communal care, grand-maternal care, grand-paternal care, kin selection, overlapping generations, reproductive success, sex-biased dispersal, survival, viscosity.**

Genomic imprinting is broadly defined as the asymmetric expression of genes inherited via eggs and via sperm (Reik and Walter 2001). The term often refers to the extreme case of genes that are either silenced when paternally inherited (PI) but expressed when maternally inherited (MI) (henceforth maternally expressed genes), or silenced when MI but expressed when PI (henceforth paternally expressed genes) (Reik and Walter 2001). There is direct evidence of imprinting in almost a hundred genes in humans and mice (Morison et al. 2005). However, recent work measuring allele-specific expression in the mouse brain identified 824 genes showing strong bias toward the expression of one of the two copies (Gregg et al. 2010). Thus genomic imprinting seems to be more widespread than previously believed.

Since its discovery, a little more than a quarter century ago, genomic imprinting has captured the interest of molecular and evolutionary biologists. Over the years a large number of theories for the evolution of genomic imprinting have been put forward (see Wilkins and Haig (2003) for a review). Of these, the "kinship theory of genomic imprinting" (henceforth the "kinship theory") is the most widely accepted, and has shown the strongest predictive power (Tycko and Morison 2002; Wilkins and Haig 2003; Burt and Trivers 2006; Moore and Mills 2008). The kinship theory argues that genes with different parental origin may come into conflict over their combined level of expression (Wilkins and Haig 2003; Grafen 2006). This conflict manifests in genes whose phenotype affects the kin of their carrier, and where the carrier

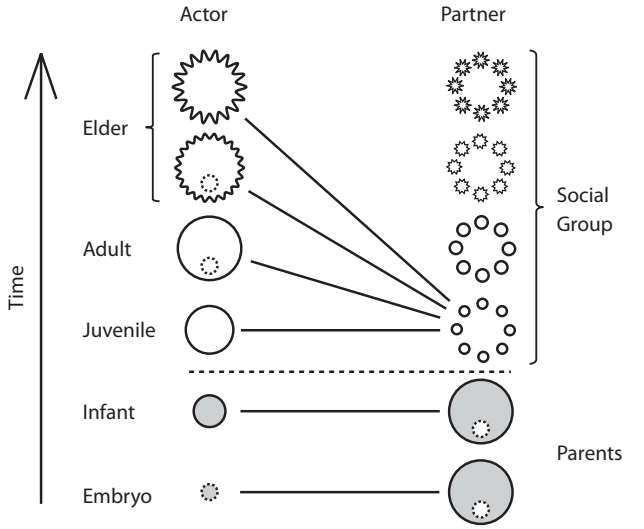

Figure 1. **Interactions between individuals in a patch. Below the dashed line, we represent interactions between an embryo/infant and her parents (scenario in which the kinship theory was originally developed). Above the dashed line, we represent social interactions beyond the nuclear family. Age increases from bottom to top. As individuals age, they transition from receiving resources from their nuclear family (via placenta or maternal milk) to exchanging resources with members of their natal patch. Three developmental stages of the focal individual are considered: juveniles, young adults, and older adults (elder) all interacting with local juveniles.**

is differentially related to the target kin via her MI versus her PI genes (Hamilton 1970; Haig 1997). The outcome is self-imposed silencing of the gene that is selected for a lower level of expression, the so-called "loudest voice prevails principle" (Haig 1996; Úbeda and Haig 2003).

Because imprinted genes were first discovered in relation to embryonic and infant growth, the kinship theory was originally formulated in terms of conflict between genes expressed in an offspring that determine the allocation of maternal resources between current and future offspring (Moore and Haig 1991). In a similar vein, the theory has also been extended to the allocation of paternal resources (Úbeda 2008). This work suggests that imprinting will evolve when parents change reproductive partners over their lifetime, such that maternal siblings need not be paternal siblings and vice versa (Haig 1997; Úbeda 2008). Thus, siblings are valued differently by the individual's MI and PI genes (Haig 1997). This logic applies through the embryonic and infant stages, when offspring depend upon parental investment for their development, but not to the postinfant stages after cessation of parental care (juveniles and adults) (Úbeda and Gardner 2010, 2011) (Fig. 1).

However, there is growing evidence that genes show imprinted expression in the postinfant brain (Lefebvre et al. 1998; Goos and Silverman 2001; Plagge et al. 2005; Gregg et al. 2010; Garfield et al. 2011). Close to 43% of the genes showing allele-specific expression in the mouse brain (372 genes) do so in the adult brain (Gregg et al. 2010). The formulation of the kinship theory in the context of fitness interactions within the nuclear family (mother, father and siblings) cannot explain the role of imprinted genes in postinfants. However, a formulation of the kinship theory in the context of fitness interactions between members of a social group (neighbors) may be able to do so (Haig 2000; Isles et al. 2006) (Fig. 1). Úbeda and Gardner (2010, 2011) provide a mathematical model for the evolution of genomic imprinting in this wider social context (see also Brandvain 2010; Van Cleve et al. 2010).

A comprehensive formulation of the kinship theory in a social context requires that all postinfant stages are considered. The juvenile stage, occurring between cessation of parental investment and initiation of own reproduction, has already been considered (Úbeda and Gardner 2010). This work shows that genes underlying altruistic and egoistic interactions among neighboring juveniles are expected to be imprinted when dispersal or variance in reproductive success (RS) is sex-biased. The adult stage, occurring after the initiation of own reproduction but prior to any generational overlap, has also been considered (Úbeda and Gardner 2011). This work shows that genes underlying parental and communal care of neighboring juveniles, are expected to be imprinted when dispersal or variance in RS are sex-biased.

Here we complete our formulation of the kinship theory within a social context, by elaborating a general evolutionary demographic model for the expression of age-specific genes with different parental origin in aging adults (henceforth "elders") in a society were generations overlap. We consider genes with different parental origin, expressed at different stages in life, that underlie parental and communal care of neighboring juveniles. Recent work has modeled overlapping generations with genes showing age-specific expression but not parent of origin-specific expression (Johnstone and Cant 2010), and genes showing parent of origin-specific expression but not age-specific expression (Van Cleve et al. 2010). This is the first work to consider parent of origin and age-specific expression that allows us to explore how patterns of genomic imprinting change in aging adults.

Our model makes specific predictions about the extent of the intragenomic conflict between MI and PI genes (i.e., the difference between the level of expression favored by each genetic faction) and the direction of the imprint (i.e., whether a gene is maternally or paternally expressed). Moreover, by considering that genes may adjust their level of expression at different ages, our model is the first to investigate how the conflict between MI and PI genetic factions changes over a lifetime. This allows us to question whether silent genes are under selective pressure to be expressed again later in lifetime.

Model and Results

Our model is based upon the island model of population structure (Wright 1931),which has become a standard setting for kin selection analysis (e.g., Taylor 1992; Rousset 2004). In particular, we build upon the models of Gardner (2010) and Úbeda and Gardner (2010, 2011).

We consider an infinite population of diploid individuals structured into neighborhoods. Each neighborhood contains juveniles (nonreproductive individuals), and adults (reproductive individuals) of different ages. Adults engage in social interactions that mediate the survival of juveniles. In particular, we consider that elder mothers can provide care to either their own offspring (maternal care) or else to all juveniles in the neighborhood (communal care) (König 1997; Haig 2010; Johnstone and Cant 2010). For simplicity, we focus upon maternal versus communal care provided by elder females. However, a symmetric model applies to paternal versus communal care provided by elder males. We assume that greater provision of maternal care reduces the amount of communal care that elder females can provide, and vice versa. Let "maternal-care genes" refer to genes whose greater expression in an elder female results in a greater investment into her own offspring, and "communal-care genes" refer to genes whose greater expression in an elder female results in a greater investment into all local juveniles.

Upon reaching adulthood, young adults disperse with probabilities d_f and d_m for females and males, respectively, or else they remain in their natal neighborhood with probabilities $l_f = 1 - d_f$ and $l_m = 1 - d_m$, respectively (Fig. 2). Older adults do not disperse, and survive to the next generation with probabilities $1 - \mu_f$ and $1 - \mu_m$ for females and males, respectively, or die with probabilities μ_f and μ_m, respectively (Fig. 2). Young adults occupy vacant reproductive spots left by deceased older adults, at random. Adults of all ages mate within their neighborhood, producing the next generation of juveniles (Fig. 2). We assume an even sex ratio at birth.

We allow for a wide range of mating systems, by using α to denote the probability that two juveniles, randomly chosen from the same neighborhood, share the same mother (probability of maternal sibship), and by using β to denote the probability that they share the same father (probability of paternal sibship) (Fig. 2). Parameters α and β capture the inequity in RS among females and males, respectively (see Gardner 2010; Úbeda and Gardner 2010, 2011 for further discussion).

We use this model to determine the extent to which MI and PI autosomal genes, expressed in adult females of age t and underlying communal care, value the survival of neighboring juveniles relative to the survival of their own offspring: that is the "potential for communal care" (Grafen 1984; Frank 1998; Gardner 2010; Van Dyken 2010; Úbeda and Gardner 2010, 2011) of the

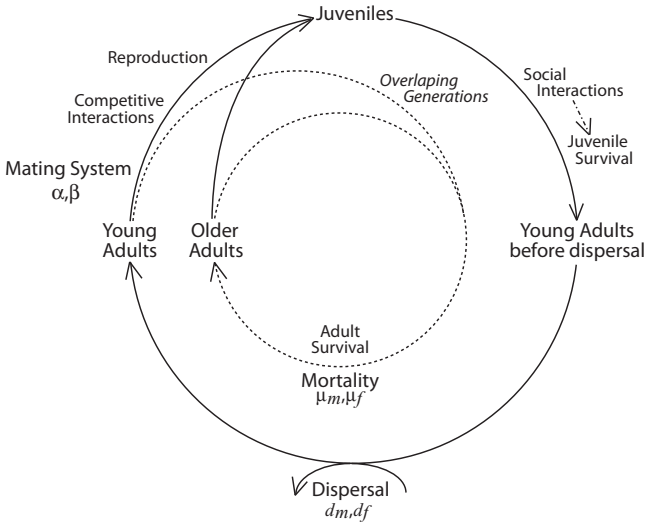

Figure 2. Life cycle of individuals in a patch. Individuals of different ages overlap in their natal patch. Adults engage in social interactions with juveniles that determine juvenile survivorship to adulthood. Young adult females and males remain in their natal patch with probability $1 - d_f$ and $1 - d_m$, respectively. Older adult females and males survive to the next generation with probability $1 - \mu_m$ and $1 - \mu_f$. Young adults compete among themselves for one of the reproductive spots left vacant by deceased adults. The mating system considered determines the probability of maternal and paternal sibship α and β, respectively. Once the reproductive spots have been claimed young and older adults reproduce at random, leading to the new generation of juveniles. After their first reproduction young adults join the population of older adults.

MI and PI genes in an adult female of age t (see the Appendix and Table 2 for its derivation in terms of the variables of the model). These potentials for communal care are given by the expressions

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

and

$$Q_{\bar{P}|t} = \frac{(1-a)r_{\bar{P}|t}}{1 - ar_{\bar{P}|t}}, \tag{2}$$

where subscripts $\bar{M}$ and $\bar{P}$ refer to MI and PI genes, respectively, and t refers to the age of their carrier. The compound parameter $a = \frac{1}{2}(l_f^2 + l_m^2)$ accounts for the intensity of local competition between juveniles (Frank 1998; Gardner 2010). The compound parameters $r_{\bar{M}|t}$ and $r_{\bar{P}|t}$ are the coefficients of relatedness between an adult female of age t and a neighboring juvenile, from the perspective of the former's MI and PI genes, respectively.

The difference in the valuation of the relative survival of neighboring juveniles made by the MI and PI genes in a female of age t, defines the "potential for intragenomic conflict"

Table 1. Variables and parameters in the model. Dropping a subindex represents the average of that variable.

Symbol	Definition
d_ξ, l_ξ	Juvenile dispersal in sex ξ, and juvenile nondispersal
μ_ξ	Adult mortality in sex ξ
α, β	Probability of sibship in males and females
t	Age (measured in generations)
K	Number of diploid individuals in a patch
S	Juvenile survival
x_t, y_t, z_t	Investment into communal care at age t by the mother of the focal individual, the average female in the patch, and the average female in the population
c, b	Cost to the focal individual of providing communal care in terms of juvenile survival, and benefit of receiving communal care
C, B	Cost to the focal individual of providing communal care in terms of juvenile fitness, and benefit of receiving communal care
w_ξ, $\bar{w}_\xi$, W_ξ	Fitness of a juvenile, average fitness of all juveniles, and fitness of a juvenile relative to the fitness of all juveniles
γ_ξ	Expected number of reproductive spots that arise each generation in a patch
π_t	Proportion of adult females of age t
g_χ	Level of expression of the χ-inherited copy of gene g
$\tilde{g}_\chi$, $\tilde{g}'_\chi$	Genic value of the mother of the focal individual, and genic value of a female in the patch of the focal individual
$p_{S\chi\|t}$, $p_{M\chi\|t}$, $p_{X\chi\|t}$, $p_{A\chi\|t}$, $p_{U\chi\|t}$, $p_{AA\chi\|t}$, $p_{AU\chi\|t}$, $p_{UU\chi\|t}$	Consanguinity to self through the χ-inherited copy when the female is of age t, consanguinity mother-juvenile, juvenile–juvenile, female-juvenile, male-juvenile, female–female, female–male, and male–male
a	Scale of competition between individuals in the same patch
$r_{\chi\|t}$	Relatedness female-juvenile through the χ-inherited copy when the female is of age t
$Q_{\chi\|t}$, $P_{\chi\|t}$	Potential for communal care of a female through the χ-inherited copy when the female is of age t, and potential for maternal care
I_t	Potential for conflict of an age t individual
$\xi = \{f, m\}$, $\chi = \{\bar{M}, \bar{P}\}$	Subscripts corresponding to male and female, and to MI and PI gene copies

(Úbeda and Gardner 2010, 2011) in an age-t female. This potential for intragenomic conflict is given by

$$I_t = Q_{\bar{M}|t} - Q_{\bar{P}|t} = \frac{(1-a)(r_{\bar{M}|t} - r_{\bar{P}|t})}{(1-ar_{\bar{M}|t})(1-ar_{\bar{P}|t})}, \qquad (3)$$

Let the level of expression at a locus in an age-t female be g_t. This corresponds to the combined level of expression of her MI ($g_{\bar{M}|t}$) and PI ($g_{\bar{P}|t}$) gene copies, that is $g_t = g_{\bar{M}|t} + g_{\bar{P}|t}$.

When the potential for intragenomic conflict is positive $I_t > 0$, the evolutionarily stable level of expression g_t^* is greater for the MI copy than for the PI copy, that is $g^*_{t\prec\bar{M}} > g^*_{t\prec\bar{P}}$. The antagonistic coevolution of the levels of expression of the MI and PI copies results in the expression of the copy selected for higher expression (the MI copy) at its evolutionarily stable level of expression $g^*_{\bar{M}|t}$, and the silencing of the copy selected for lower expression (the PI copy), that is $\{g_{\bar{M}|t}, g_{\bar{P}|t}\} = \{g^*_{t\prec\bar{M}}, 0\}$ (Haig 1996). When the potential for intragenomic conflict is negative $I_t < 0$, g_t^* is lower for the MI than the PI copy, that is $g^*_{t\prec\bar{M}} < g^*_{t\prec\bar{P}}$. The antagonistic coevolution of the levels of expression of the MI and PI copies results in the expression of the PI copy at its evolutionarily stable level of expression $g^*_{t\prec\bar{P}}$, and the silencing of the MI copy, that is $\{g_{\bar{M}|t}, g_{\bar{P}|t}\} = \{0, g^*_{t\prec\bar{P}}\}$ (Haig 1996). This corner solution is referred to as the "loudest voice prevails principle" (Haig 1996). When the potential for intragenomic conflict is zero, $I_t = 0$, g_t^* is equal for the MI and the PI copies, that is $g^*_{t\prec\bar{M}} = g^*_{t\prec\bar{P}} = g^*_{t\prec\bar{M}\bar{P}}$. There is no antagonistic coevolution of the MI and PI copies and they remain at their unimprinted state $\{g_{\bar{M}|t}, g_{\bar{P}|t}\} = \{\frac{1}{2}g^*_{t\prec\bar{M}\bar{P}}, \frac{1}{2}g^*_{t\prec\bar{M}\bar{P}}\}$.

We investigate how demographic factors mediate the evolution of genomic imprinting (Fig. 3). The potential for intragenomic conflict in young adult females is positive ($I_{Q|t} > 0$) when the probability of sibship is female biased ($\alpha > \beta$), and negative ($I_{Q|t} < 0$) when the probability of sibship is male biased ($\beta > \alpha$) (all other demographic factors being equal in both sexes) (Fig. 3A.1). The potential for intragenomic conflict in young adult females is positive ($I_{Q|t} > 0$) if either dispersal ($d_m > d_f$) or mortality ($\mu_m > \mu_f$) are male biased, and negative ($I_{Q|t} < 0$) if either dispersal ($d_f > d_m$) or mortality ($\mu_f > \mu_m$) are female biased (all other demographic factors being equal in both sexes) (Fig. 3A.2, A.3). When several demographic factors are

Table 2. **Potential for conflict and coefficients of consanguinity from top to bottom, we provide the definition of a term as a function of the set of terms defined immediately below. Each set of terms is separated by a continuous line, i.e., $r_{\chi|t}$ requires the definition of $p_{A\chi|t}$, $p_{M\chi|t}$, and these in turn require the definition of $p_{AA\chi|t}$, $p_{AU\chi|t}$, p_S. A close definition of the recursions I_t, $r_{\chi|t}$, $p_{M\chi|t}$, $p_{A\chi|t}$, $p_{AA\chi|t}$, $p_{AU\chi|t}$ requires: (a) solving the system of equations p_S, p_X, p_A, p_U, p_{AA}, p_{AU}, p_{UU} to get the expression of p_S, p_{AA}, p_{AU}, p_{UU} in terms of the demographic parameters α, β, l_m, l_f, μ_m, μ_f, (b) defining the initial conditions $p_{AA\chi|1}$, $p_{AU\chi|1}$ in terms of demographic parameters (which requires solving the system of equations $p_{S\chi}$, $p_{M\chi}$, $p_{X\chi}$, $p_{A\chi}$, $p_{U\chi}$, $p_{AA\chi}$, $p_{AU\chi}$, $p_{UU\chi}$ to get the expression of $p_{X\chi}$, $p_{A\chi}$, $p_{U\chi}$ in terms of demographic parameters).**

Term	Definition	Equation
Potential for conflict	$I_t = \frac{(1-a)(r_{\tilde{M}\mid t} - r_{\tilde{P}\mid t})}{(1-ar_{\tilde{M}\mid t})(1-ar_{\tilde{P}\mid t})}$	(3)
Relatedness juvenile-female (age t, χ-inherited copy)	$r_{\chi\mid t} = \frac{p_{A\chi\mid t}}{p_{M\chi\mid t}}$	(A.69,A.70)
Consanguinity juvenile-female (age t, χ-inherited copy)	$p_{A\chi\mid t} = \frac{1}{2}[\alpha p_S + (1-\alpha)p_{AA\chi\mid t}] + \frac{1}{2}p_{AU\chi\mid t}$	(A.39,A.41)
Consanguinity juvenile-Mother (age t, χ-inherited copy)	$p_{M\chi\mid t} = \frac{1}{2}[p_S + p_{AU\chi\mid t}]$	(A.34,A.37)
Consanguinity female–female (age t, χ-inherited copy)	$p_{AA\chi\mid t} = (1-\mu_f)p_{AA\chi\mid t-1} + l_f\mu_f p_{A\chi\mid t-1}$	(A.51,A.55)
Consanguinity female–male (age t, χ-inherited copy)	$p_{AU\chi\mid t} = (1-\mu_m)p_{AU\chi\mid t-1} + l_m\mu_m p_{A\chi\mid t-1}$	(A.61,A.65)
Consanguinity to Self	$p_S = \frac{1}{2} + \frac{1}{2}p_{AU}$	(A.15)
Consanguinity female–male	$p_{AU} = (1-\mu_f)(1-\mu_m)p_{AU} + (1-\mu_f)\mu_m l_m p_A + \mu_f(1-\mu_m)l_f p_U + \mu_f\mu_m l_f l_m p_X$	(A.32)
Consanguinity juvenile-female	$p_A = \frac{1}{2}[\alpha p_S + (1-\alpha)p_{AA}] + \frac{1}{2}p_{AU}$	(A.20)
Consanguinity juvenile-male	$p_U = \frac{1}{2}[\beta p_S + (1-\beta)p_{UU}] + \frac{1}{2}p_{AU}$	(A.22)
Consanguinity juvenile–juvenile	$p_X = \frac{1}{4}[\alpha p_S + (1-\alpha)p_{AA}] + \frac{1}{2}p_{AU} + \frac{1}{4}[\beta p_S + (1-\beta)p_{UU}]$	(A.23)
Consanguinity female–female	$p_{AA} = (1-\mu_f)^2 p_{AA} + 2\mu_f(1-\mu_f)l_f p_A + \mu_f^2 l_f^2 p_X$	(A.27)
Consanguinity male–male	$p_{UU} = (1-\mu_m)^2 p_{UU} + 2\mu_m(1-\mu_m)l_m p_U + \mu_m^2 l_m^2 p_X$	(A.28)

simultaneously sex-biased, the predictions of our model depend on the relative importance of each demographic effect (results not shown).

Sex-specific demographic asymmetries result in intragenomic conflict and the evolution of genomic imprinting at least in young adults. As adult females age, the potential for intragenomic conflict tends toward zero ($I_{Q\mid t\to\infty} = 0$), even when the probability of sibship, dispersal, or survival (or combinations of them) is sex-biased (Fig. 3). The potential for intragenomic conflict decays over time toward its age-equilibrium 0 (see Appendix for derivation).

Discussion

In our previous analyses of genomic imprinting in the social brain, we showed that imprinting evolves in genes underlying altruism and egoism expressed in juveniles and adults, driven by sex-bias in RS and/or dispersal (Úbeda and Gardner 2010, 2011). Here, we extend our analysis to consider "elders," showing that imprinting can evolve in age-specific genes underlying parental care and communal care expressed in adult life. We find that sex bias in adult mortality provides an additional source of asymmetry that drives the evolution of genomic imprinting. Moreover, we find that the intragenomic conflict between an individual's MI and PI genes fades away as she ages.

Our current model shows that genomic imprinting evolves not only when there is a sex bias in dispersal and/or variance in RS but also when there is sex bias in adult mortality. This is consistent with the findings of Úbeda and Gardner (2010, 2011) and Van Cleve et al. (2010). The intuition for our results is that lower adult mortality or dispersal of one sex translates into greater relatedness to social partners with respect to genes inherited from that sex. Natural selection favors the expression of genes encoding

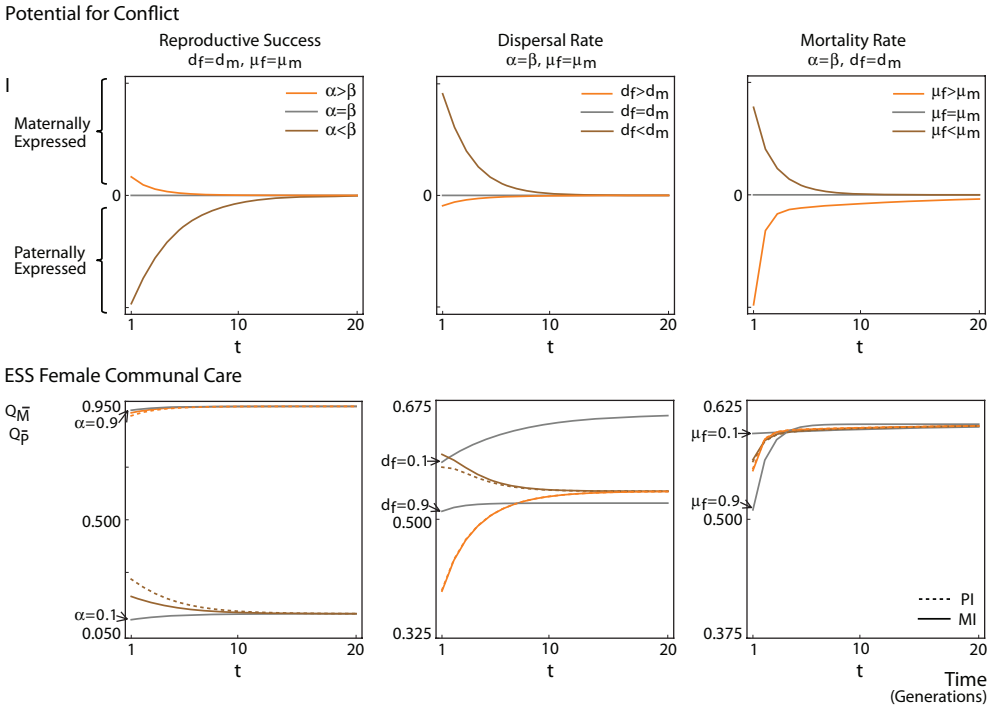

Figure 3. **Potential for conflict and ESS female communal care as a function of time. Panel A depicts the potential for conflict over time. The horizontal axis represents time (in generations). The vertical axis represents the potential for conflict between the MI and PI genes underpinning female communal care. Positive and negative values indicate selection in favor of maternally expressed (paternally silenced) and paternally expressed (maternally silenced) genes, respectively. Panel B depicts the ESS level of female communal care over time. The horizontal axis represents time (in generations). The vertical axis represents the optimal level of expression for the MI and PI genes underpinning female communal care. Color code and parameter use are equivalent to the previous panel. Panels A.1 and B.1 represent the case of sex-bias in reproductive success (RS) when there is no difference in dispersal and mortality between the sexes, in particular $\{\alpha, \beta\} = \{0.1, 0.9\} \times \{0.1, 0.9\}$ while $d_f = d_m = \mu_f = \mu_m = \frac{1}{2}$. Panels A.2 and B.2 represent the case of sex-bias in dispersal when there is no difference in RS and mortality between the sexes, in particular $\{d_f, d_m\} = \{0.1, 0.9\} \times \{0.1, 0.9\}$ while $\alpha = \beta = \mu_f = \mu_m = \frac{1}{2}$. Panels A.3 and B.3 represent the case of sex-bias in mortality when there is no difference in RS and dispersal between the sexes, in particular $\{\mu_f, \mu_m\} = \{0.1, 0.9\} \times \{0.1, 0.9\}$ while $\alpha = \beta = d_f = d_m = \frac{1}{2}$. In all cases the potential for conflict decreases steadily toward zero. The ESS level of expression increases over time in all cases except when either $(\alpha, \beta) = (0.1, 0.9)$ or $(d_f, d_m) = (0.1, 0.9)$ all other parameters being equal for both sexes in both cases. Lines are obtained by numerically iterating equations I_t, $Q_{\chi|t}$, $r_{\chi|t}$, $p_{M\chi|t}$, $p_{A\chi|t}$, $p_{AA\chi|t}$, and $p_{AU\chi|t}$.**

communal care in elders when they are derived from the longer lived but less-dispersing sex, and it favors the expression of genes encoding parental care when they are derived from the shorter lived but more dispersing sex (Fig 3A.2, A.3). Higher variance in RS among individuals of one sex translates into greater relatedness to social partners with respect to genes inherited from that sex. Natural selection favors the expression of genes encoding communal care in elders 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. 3A.1).

Although discussed in terms of parental versus communal care, our results apply to genes underlying any behavior of an individual leading to a variation in her own fertility compensated by a variation of the opposite sign in the fertility of her neighbors (e.g., including behaviors of solitary individuals making use of a shared limiting resources). Therefore, the evolution of genomic imprinting is more likely but not limited to species in which there is an opportunity for communal care.

The evolution of genomic imprinting in elders requires sex-asymmetries in demographic variables. Such asymmetries are the norm in the natural world. In general, males show greater variance in RS among birds and mammals, and dispersal is female biased among birds and hominids, and male biased among non-hominid mammals (see Úbeda and Gardner (2010) for a review). Furthermore, adult mortality differs between females and males. In general, mortality is male biased among mammals (Clutton-Brock and Isvaran 2007), and female biased among birds (Owens and Bennett 1994; Liker and Szekely 2005).

Regarding ancestral humans, we assume that they exhibited male-biased variance in RS, in conformity with most mammals (Clutton-Brock 2007). It is debated whether ancestral humans

exhibited patrilocality (women moving to their husband's natal patch) or a more balanced dispersal rate between the sexes (see Úbeda and Gardner 2010, for a review). Henceforth we assume the prevalent view that ancestral humans were patrilocal although a more balanced dispersal does not change qualitatively our conclusions. Concerning adult mortality, women live longer than males (Owens 2002; Maklakov 2008). However, women are unique among mammals (except whales) in that fertility ceases well before death (i.e., they experience menopause) (Johnstone and Cant 2010). We did not explicitly include menopause in our model but, because women that do not reproduce cease to contribute to the observed increase in relatedness, we expect the impact of menopause on the evolution of genomic imprinting to mirror the impact of female mortality. Thus, from this perspective, humans group with species that exhibit female-biased mortality.

Upon the basis of these assumptions, we can predict the taxonomic distribution of imprinting patterns in the elder brain. Genes underlying communal care are predicted to be paternally expressed in birds and humans, taxa that in general show male-biased RS and female-biased dispersal and mortality. Some variability is expected among the corresponding mammalian genes. These are predicted to be paternally expressed in nonhominid mammals when the effect of male-biased RS dominates the effects of male-biased dispersal and mortality, but maternally expressed otherwise. Genes expressed in hominids (excluding humans) are predicted to be paternally expressed when the effect of male-biased RS and female-biased dispersal dominates the effects of male-biased mortality, but maternally expressed otherwise.

Interestingly, our model shows that there is a steady decrease in the level of intragenomic conflict between MI and PI genes in aging individuals: the different levels of expression favored by natural selection acting upon the MI and PI genes converge as the individual ages. The intuitive reason for this is that, in the absence of inbreeding, a mother is equally related to her direct descendants via her MI and PI genes, whereas she is differentially related to nondescendant relatives via her MI and PI genes. As a young mother ages, there is a greater probability that the juveniles in her local neighborhood are her direct descendants (offspring, grandoffspring, etc) rather than nondescendant relatives (nieces, nephews, great-nieces, great-nephews, etc). The presence of inbreeding somewhat dampens this effect, but does not eliminate it entirely.

This reduction in the level of intragenomic conflict has two implications. First, genomic imprinting is more likely to evolve in young adults than in older adults. Although conflict between the MI and PI copies does not disappear over any finite-time interval, conflict declines with age. Thus, selection for the evolution of genomic imprinting in young adults is stronger than selection acting upon older adults. It is important to notice that, in our model, age is given in generations not in years. For example the mouse lemur (*Microcebus murinus*) dies at the age of 14 years roughly overlapping with 14 other younger generations, while humans (*Homo sapiens*) die at the age of 80 years roughly overlapping with 4 other younger generations (Cant and Jonhstone 2008). For the purposes of determining the level of intragenomic conflict at old ages in these two species, the mouse lemur is of age 14 when it dies but humans are much younger when they die (of age 4). Therefore, all other life-history parameters being equal (although they are not), humans are more likely to have imprinted genes expressed in their senescence than mouse lemur do.

Second imprinted genes in young adults might be biallelic ally expressed in older adults. In general, silenced copies are not expected to be reactivated when conflict between MI and PI copies disappears. The reason why, is that selection favoring biallelic expression is in general very small (Spencer 1997). However, when the conflict between MI and PI copies becomes negligible, at older ages, silent copies might be reactivated if the combined level of expression that is favored by natural selection increases over time. Our model shows that the combined level of expression that is favored by natural selection at a locus coding for communal care changes over time (Fig. 3). When the combined level of expression is under selective pressure to decrease ($\alpha < \beta$ and $d_f < d_m$ in panels B.1 and B.2 of Fig. 3), the only way to achieve this reduction is by downregulating the expressed copy, but not by reactivating the silenced copy. When the combined level of expression is under selective pressure to increase ($\alpha \geq \beta$ and $d_f \geq d_m$ in panels B.1–3 of Fig. 3), this increment in expression can be achieved either by upregulating the expressed copy or by reactivating the silenced copy. Reactivating the silenced copy might be the default mechanism for achieving an increment in expression due to mechanistic reasons. The most common way of silencing imprinted genes is by methylating their promoter and methylation patterns must be actively maintained during cell division. Failure to do so results in passive reactivation of imprinted genes over time (Wilkins 2005).

Our analysis lends further support to the idea that the social brain is split between two genetic factions (set of genes that share a common pattern of inheritance and relatedness), namely the MI and PI genomes (Haig 2006; Grafen 2006; Úbeda and Gardner 2010, 2011). Each of these factions are selected to influence the behavior of their host regarding care in the opposite direction with the MI faction under selection for less communal care and the PI fraction for more communal care, leading to an inner turmoil that cannot be understood from the perspective of "the good of the individual". However, we have also found that the conflict of interests between MI and PI factions settles down with increasing age. So, although we predict that individuals are riven by internal conflict in their youth and middle age, we suggest that they will put their demons to rest in later life.

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Appendix

We consider an infinite population structured into patches. Each patch contains a large constant number K of diploid juveniles

with an even sex ratio, and some adults. We derive the results for adult females but symmetric results apply to adult males.

JUVENILE SURVIVAL

The behavior of adult females determines survival of juveniles in each patch. The probability of survival for a focal juvenile is given by $S(x, y)$ where x is the investment into communal care of her mother, y is the average investment into communal care of females in her group (i.e., the individual's "aunts"). Because we consider that resources invested into communal care are taken away from a female's own offspring, we assume that females with more offspring make absolutely greater contributions to communal care than do females with fewer offspring, all else being equal. Hence, y is a weighted average of the proportional investments made by the breeding females in the patch, the weights being equal to their relative fecundities. We assume vanishingly small variation in x and y, ensuring linearity of survival effects in the region occupied by the population at any time (Taylor and Frank 1996).

We assume that the juvenile's survival is a monotonically decreasing function of her mother's communal care (owing to the concomitant reduction in parental care):

$$\frac{\partial S}{\partial x} = -c < 0, \tag{A.1}$$

and is a monotonically increasing function of her aunts' communal care:

$$\frac{\partial S}{\partial y} = b > 0. \tag{A.2}$$

JUVENILE 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 mother and her aunts. Note that, in taking a neighbor modulated fitness approach to kin-selection analysis (Hamilton 1964, Taylor and Frank 1996), we assign fitness to recipients (i.e., juveniles) and not to actors (i.e., adults).

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 $l_f = 1 - d_f$). Adult females die with probability μ_f and are replaced by random females that recently matured. The probability of breeding success is inversely proportional to the number of females competing within the patch upon which she finds herself after the dispersal phase (Gardner 2010). Newly matured females that fail to secure a breeding spot die. Adults then produce a large number K of diploid juveniles, these being shared potentially unequally among the adults in each patch. For example, if there are two adult females in each patch, we might choose one at random and assign her a share s of the juveniles, so she has fecundity sK and her neighbor has fecundity $(1 - s)K$. Hence, the probability that two of the juveniles, chosen at random, share the same mother is $\alpha = s^2 + (1 - s)^2$, and this may take any value in the range $\frac{1}{2} \leq \alpha \leq 1$. More generally, if there are n adult females per patch, then the probability of maternal sibship may take any value in the range $\frac{1}{n} \leq \alpha \leq 1$. This returns the model to the beginning of the life cycle (see Fig. 2).

The fitness of a juvenile female (the expected probability that a juvenile female survives, disperses, and earns a breeding spot in her patch) is:

$$w_f = S(x, y)\left[l_f \frac{1}{l_f S(y, y)\frac{1}{2}K + (1 - l_f)S(z, z)\frac{1}{2}K} + (1 - l_f)\frac{1}{S(z, z)\frac{1}{2}K} \right]\gamma_f \tag{A.3}$$

where z is the average investment into communal care of females in the population, and γ_f is the expected number of adult female vacancies that arise each generation in the patch.

The average fitness over all juvenile females in the population is $\bar{w}_f = 2\gamma_f/K$ and the relative fitness of a juvenile female $W_f = w_f/\bar{w}_f$ is

$$W_f = S(x, y)\left[\frac{l_f}{l_f S(y, y) + (1 - l_f)S(z, z)} + \frac{1 - l_f}{S(z, z)} \right]. \tag{A.4}$$

Similarly the relative fitness of a juvenile male is

$$W_m = S(x, y)\left[\frac{l_m}{l_m S(y, y) + (1 - l_m) S(z, z)} + \frac{1 - l_m}{S(z, z)} \right]. \tag{A.5}$$

Natural selection maximizes the average of female and male relative fitness weighted by class reproductive value (Fisher 1930; Price and Smith 1972; Taylor 1990) which is a half under diploidy, that is

$$W = \frac{1}{2}W_f + \frac{1}{2}W_m. \tag{A.6}$$

For an individual with genic value g at a particular locus, the quantities x and y are random variables, and to calculate the expected fitness for this individual we would need to average over the probability distributions for these random variables. We assume that, owing to vanishing genetic variation and vanishing impact of social interaction on fitness, we recover the expected fitness by treating x and y as the expected values of these random variables.

AGE-CONDITIONAL COOPERATION

We allow adult females modify their investment into communal care as they age. Age is measured in generations, with $t = 1$ denoting those newly matured adult females who are engaging in their first bout of social interactions, $t = 2$ denoting those adult females who are a generation older than these, and so on.

The investment of an individual's mother if she is of age t is denoted by x_t. The expected investment of the focal individual's mother is:

$$x = \sum_{t=1}^{\infty} \pi_t x_t, \tag{A.7}$$

where π_t is the proportion of adult females who are of age t.

The investment of adult females in the focal individual's group is:

$$y = \sum_{t=1}^{\infty} \pi_t y_t. \tag{A.8}$$

NATURAL SELECTION

Consider a gene that impacts upon the investment strategy of an age-T individual. The condition for natural selection to favor an increase in investment into communal care when the gene is inherited from parent $\chi = \{\bar{M}, \bar{P}\}$ is $dW/dg_\chi > 0$ where:

$$\frac{dW}{dg_\chi} = \sum_{t=1}^{\infty} \frac{\partial W}{\partial x_t} \frac{dx_t}{d\tilde{g}_\chi} \left(\frac{d\tilde{g}_\chi}{dg_\chi} \right)_t + \sum_{t=1}^{\infty} \frac{\partial W}{\partial y_t} \frac{dy_t}{d\tilde{g}'_\chi} \left(\frac{d\tilde{g}'_\chi}{dg_\chi} \right)_t, \tag{A.9}$$

where $\tilde{g}_\chi$ is the genic value of the focal individual's mother and $\tilde{g}'_\chi$ is the genic value of a random adult female on the focal individual's patch.

Note that the genic value of the mother only impacts upon her investment phenotype if she is of age T, that is $dx_T/d\tilde{g}_\chi = dy_T/d\tilde{g}'_\chi = 1$ but $dx_t/d\tilde{g}_\chi = dy_t/d\tilde{g}'_\chi = 0$ for all $t \neq T$. Let $p_{M\chi|t} = (d\tilde{g}_\chi/dg_\chi)_t$ be the consanguinity between an age-t mother and her offspring $(d\tilde{g}_\chi/dg_\chi)_t$, and $p_{A\chi|t}$ be the consanguinity between an age-t female and a random juvenile on the same patch $(d\tilde{g}'_\chi/dg_\chi)_t$.

Thus the condition for natural selection favoring a gene increasing communal care at age T is:

$$\begin{aligned} \frac{\partial W}{\partial x} \pi_T p_{M\chi|T} + \frac{\partial W}{\partial y} \pi_T p_{A\chi|T} > 0 \\ \frac{\partial W}{\partial x} p_{M\chi|T} + \frac{\partial W}{\partial y} p_{A\chi|T} > 0. \end{aligned} \tag{A.10}$$

From equations (A.4) to (A.6), the expected fitness cost $-C = \partial W/\partial x|_{x=y=z}$ and benefit $B = \partial W/\partial y|_{x=y=z}$ experienced by a juvenile due to the communal care provided by her mother and her aunts are:

$$\frac{\partial W}{\partial x} = -\frac{c}{S}, \tag{A.11}$$

and

$$\frac{\partial W}{\partial y} = \frac{b - a(b-c)}{S}, \tag{A.12}$$

respectively. Where partial derivatives and probability of survival are evaluated in $x = y = z$, $-c = \partial S/\partial x|_{x=y=z}$ is the expected survival cost, $b = \partial S/\partial y|_{x=y=z}$ is the expected survival benefit, and $a = \frac{1}{2}(l_f^2 + l_m^2)$ is the scale of competition between juvenile patch mates (Frank 1998; Gardner 2010).

Substituting into equation (A.10) yields

$$\begin{aligned} -\frac{c}{S} p_{M\chi|T} + \frac{b - a(b-c)}{S} p_{A\chi|T} > 0 \\ -c + (b - a(b-c)) r_{\chi|T} > 0, \end{aligned} \tag{A.13}$$

where $r_{\chi|T} = p_{A\chi|T}/p_{M\chi|T}$ is the relatedness between a juvenile and her age-T aunt via the latter's χ-inherited gene (Bulmer 1994).

Therefore, investment in communal care is favored by χ-inherited genes in age-T females when

$$\begin{aligned} \frac{c}{b} < \frac{(1-a) r_{\chi|T}}{1 - a r_{\chi|T}} \\ \frac{c}{b} < Q_{\chi|T}, \end{aligned} \tag{A.14}$$

where $Q_{\chi|T}$ is the potential for communal care of a χ-inherited gene in an age-T female. Potential $Q_{\chi|T}$ can be interpreted as the valuation made by a χ-inherited gene in an age-T female of the survival of juveniles in the same patch relative to the survival of its carrier's own offspring. Investment in communal care by an χ-inherited gene in age-T females is disfavored when $c/b > Q_{\chi|T}$.

Maternal care (as opposed to communal care) is favored by χ-inherited genes in age-T females when $c/b < P_{\chi|T}$ where $P_{\chi|T} = 1/Q_{\chi|T}$ is the potential for maternal care of a χ-inherited gene in an age-T female. Investment in maternal care by χ-inherited genes in age-T females is disfavored when $c/b > P_{\chi|T}$.

RELATEDNESS

We need to define the relatedness between a juvenile and a female of age t via the χ-inherited copy ($r_{\chi|t}$). The definition of $r_{\chi|t}$ is not straightforward, and calls for the recursive definition of several coefficients of consanguinity until a system of equations, that can be solved in terms of the demographic parameters of the model, is found. Table 2 provides a summary of this process. The solution for each coefficient of consanguinity is not simple, this is the reason why in this Appendix we provide the recursive definition of each coefficient, but not its explicit solution.

Consanguinity to self

The consanguinity of an individual to itself is:

$$p_S = \tfrac{1}{2} + \tfrac{1}{2} p_{AU}, \tag{A.15}$$

where p_{AU} is the average consanguinity between a breeding female (an "aunt") and a breeding male (an "uncle") over time, that is $p_{AU} = \sum_{t=1}^{\infty} \pi_t p_{AU|t}$, where $p_{AU|t}$ is the consanguinity

between a female of age t and her mating partner (this is equivalent to the consanguinity between mating partners, φ in Úbeda and Gardner (2011)). The probability of a reproductive female being of age t is the probability that a reproductive female in her patch did not die in the last $t-1$ generations but did die in the generation before, that is $\pi_t = \mu_f(1-\mu_f)^{t-1}$. Notice that p_S is independent of age.

Consanguinity between juvenile and mother

The consanguinity between a juvenile and her mother of age t is the probability that a gene drawn from a juvenile is identical by descent to another gene drawn from her mother of age t, that is:

$$p_{M|t} = \tfrac{1}{2}p_S + \tfrac{1}{2}p_{AU|t}. \quad \text{(A.16)}$$

Replacing p_S by its definition yields

$$p_{M|t} = \tfrac{1}{4}[1 + p_{AU} + 2p_{AU|t}]. \quad \text{(A.17)}$$

On average, the consanguinity between a juvenile and her mother $p_M = \sum_{t=1}^{\infty}\pi_t p_{M|t}$ is:

$$p_M = \tfrac{1}{4}[1 + 3p_{AU}]. \quad \text{(A.18)}$$

Consanguinity between juvenile and female

The consanguinity between a juvenile and a breeding female of age t born in the same patch is

$$p_{A|t} = \tfrac{1}{2}[\alpha p_S + (1-\alpha)p_{AA|t}] + \tfrac{1}{2}p_{AU|t}, \quad \text{(A.19)}$$

where $p_{AA|t}$ is the consanguinity between two breeding females of age t on the same patch.

On average, the consanguinity between a juvenile and a female $p_A = \sum_{t=1}^{\infty}\pi_t p_{A|t}$ is:

$$p_A = \tfrac{1}{2}[\alpha p_S + (1-\alpha)p_{AA}] + \tfrac{1}{2}p_{AU}. \quad \text{(A.20)}$$

Consanguinity between juvenile and male

The consanguinity between a juvenile and a breeding male of age t born in the same patch is

$$p_{U|t} = \tfrac{1}{2}[\beta p_S + (1-\beta)p_{UU|t}] + \tfrac{1}{2}p_{AU|t}, \quad \text{(A.21)}$$

where $p_{UU|t}$ is the consanguinity between two breeding males of age t on the same patch.

On average, the consanguinity between a juvenile and a male $p_U = \sum_{t=1}^{\infty}\pi_t p_{U|t}$ is:

$$p_U = \tfrac{1}{2}[\beta p_S + (1-\beta)p_{UU}] + \tfrac{1}{2}p_{AU}. \quad \text{(A.22)}$$

Consanguinity between juveniles

The consanguinity between juveniles born in the same patch is:

$$p_X = \tfrac{1}{4}[\alpha p_S + (1-\alpha)p_{AA}] + \tfrac{1}{2}p_{AU} + \tfrac{1}{4}[\beta p_S + (1-\beta)p_{UU}]. \quad \text{(A.23)}$$

Consanguinity between females

The consanguinity between breeding females on the same patch when the focal one is of age 1 is:

$$p_{AA|1} = l_f[l_f\mu_f p_X + (1-\mu_f)p_A], \quad \text{(A.24)}$$

(when the focal female is of age 1, she was a new born in the previous generation but we need to average over all possibilities for the other female) when she is of age 2 is:

$$p_{AA|2} = (1-\mu_f)p_{AA|1} + l_f\mu_f p_{A|1}, \quad \text{(A.25)}$$

(when the focal female is of age 2, with probability $1-\mu_f$ the other female was not a new born in the previous generation and their consanguinity is the same as it was in the previous generation, with probability μ_f the other female was a new born in the previous generation and their consanguinity is p_A if she did not disperse) and when she is of age t is:

$$p_{AA|t} = (1-\mu_f)p_{AA|t-1} + l_f\mu_f p_{A|t-1}. \quad \text{(A.26)}$$

On average, the consanguinity between two breeding females $p_{AA} = \sum_{t=1}^{\infty}\pi_t p_{AA|t}$ is:

$$\begin{aligned} p_{AA} &= \sum_{t=1}^{\infty}\mu_f(1-\mu_f)^{t-1}[(1-\mu_f)p_{AA|t-1} + l_f\mu_f p_{A|t-1}] \\ &= (1-\mu_f)^2 p_{AA} + 2\mu_f(1-\mu_f)l_f p_A + \mu_f^2 l_f^2 p_X. \end{aligned} \quad \text{(A.27)}$$

When $\mu_f = 1$ then $p_{AA} = l_f^2 p_X$ and we recover the result in Úbeda and Gardner (2011).

Consanguinity between males

On average, the consanguinity between two breeding males on the same patch is:

$$p_{UU} = (1-\mu_m)^2 p_{UU} + 2\mu_m(1-\mu_m)l_m p_U + \mu_m^2 l_m^2 p_X. \quad \text{(A.28)}$$

When $\mu_m = 1$ then $p_{UU} = l_m^2 p_X$ and we recover the result in Úbeda and Gardner (2011).

Consanguinity between female and male

The consanguinity between a breeding female and a breeding male (i.e., mating partners) on the same patch when the focal female is of age 1 is:

$$p_{AU|1} = l_f[l_m\mu_m p_X + (1-\mu_m)p_U], \tag{A.29}$$

when she is of age 2 is:

$$p_{AU|2} = (1-\mu_m)p_{AU|1} + l_m\mu_m p_{A|1}, \tag{A.30}$$

and when she is of age t is:

$$p_{AU|t} = (1-\mu_m)p_{AU|t-1} + l_m\mu_m p_{A|t-1}. \tag{A.31}$$

On average

$$\begin{aligned} p_{AU} = {} & (1-\mu_f)(1-\mu_m)p_{AU} + (1-\mu_f)\mu_m l_m p_A \\ & + \mu_f(1-\mu_m)l_f p_U + \mu_f\mu_m l_f l_m p_X. \end{aligned} \tag{A.32}$$

When $\mu_f = \mu_m = 1$ then $p_{AU} = l_f l_m p_X$ and we recover the result in Úbeda and Gardner (2011).

Solving the system of equations (A.18), (A.15), (A.23), (A.20), (A.22), (A.27), (A.28), (A.32) we get expressions for p_S, p_{AU}, p_{AA}, and p_{UU} in terms of the model parameters.

IMPRINTED GENES

Consanguinity between juvenile and mother

Via mother's MI copy. The consanguinity between a juvenile and her mother of age t via her mother's MI copy, is the probability that a gene drawn from a juvenile in generation t is identical by descent to the MI gene of her mother, that is:

$$p_{M\bar{M}|t} = \tfrac{1}{2}p_S + \tfrac{1}{2}p_{AU\bar{M}|t}, \tag{A.33}$$

where subscripts $\bar{M}$ and $\bar{P}$ denote consanguinity via the MI and PI genes, respectively. Notice that p_S is the same via the MI and PI copies of an individual, that is $p_{S\bar{M}} = p_{S\bar{P}} = p_S$.

Replacing p_S by its definition in (A.15) yields

$$p_{M\bar{M}|t} = \tfrac{1}{4}[1 + p_{AU} + 2p_{AU\bar{M}|t}]. \tag{A.34}$$

On average

$$p_{M\bar{M}} = \tfrac{1}{4}[1 + p_{AU} + 2p_{AU\bar{M}}]. \tag{A.35}$$

Via mother's PI copy. The consanguinity between a juvenile and her mother of age t via her mother's PI copy is:

$$p_{M\bar{P}|t} = \tfrac{1}{2}p_S + \tfrac{1}{2}p_{AU\bar{P}|t}. \tag{A.36}$$

Replacing p_S by its definition in (A.15) yields

$$p_{M\bar{P}|t} = \tfrac{1}{4}[1 + p_{AU} + 2p_{AU\bar{P}|t}]. \tag{A.37}$$

On average

$$p_{M\bar{P}} = \tfrac{1}{4}[1 + p_{AU} + 2p_{AU\bar{P}}]. \tag{A.38}$$

Consanguinity between juvenile and female

Via female's MI copy. The consanguinity between a juvenile and a breeding female of age t born in the same patch via the focal female's MI copy is:

$$p_{A\bar{M}|t} = \tfrac{1}{2}[\alpha p_S + (1-\alpha)p_{AA\bar{M}|t}] + \tfrac{1}{2}p_{AU\bar{M}|t}. \tag{A.39}$$

On average

$$p_{A\bar{M}} = \tfrac{1}{2}[\alpha p_S + (1-\alpha)p_{AA\bar{M}}] + \tfrac{1}{2}p_{AU\bar{M}}. \tag{A.40}$$

Via female's PI copy. The consanguinity between a juvenile and a breeding female of age t born in the same patch via the focal female's PI copy is:

$$p_{A\bar{P}|t} = \tfrac{1}{2}[\alpha p_S + (1-\alpha)p_{AA\bar{P}|t}] + \tfrac{1}{2}p_{AU\bar{P}|t}. \tag{A.41}$$

On average

$$p_{A\bar{P}} = \tfrac{1}{2}[\alpha p_S + (1-\alpha)p_{AA\bar{P}}] + \tfrac{1}{2}p_{AU\bar{P}}. \tag{A.42}$$

Consanguinity between juvenile and male

Via male's MI copy. The consanguinity between a juvenile and a breeding male of age t born in the same patch via the focal male's MI copy is:

$$p_{U\bar{M}|t} = \tfrac{1}{2}[\beta p_S + (1-\beta)p_{UU\bar{M}|t}] + \tfrac{1}{2}p_{AU\bar{M}|t}. \tag{A.43}$$

On average

$$p_{U\bar{M}} = \tfrac{1}{2}[\beta p_S + (1-\beta)p_{UU\bar{M}}] + \tfrac{1}{2}p_{AU\bar{M}}. \tag{A.44}$$

Via male's PI copy. The consanguinity between a juvenile and a breeding male of age t born in the same patch via the focal male's PI copy is:

$$p_{U\bar{P}|t} = \tfrac{1}{2}[\beta p_S + (1-\beta)p_{UU\bar{P}|t}] + \tfrac{1}{2}p_{AU\bar{P}|t}. \tag{A.45}$$

On average

$$p_{U\bar{P}} = \tfrac{1}{2}[\beta p_S + (1-\beta)p_{UU\bar{P}}] + \tfrac{1}{2}p_{AU\bar{P}}. \tag{A.46}$$

Consanguinity between juveniles

Via juveniles's MI copy. The consanguinity between juveniles born in the same patch via the focal juvenile's MI copy is:

$$p_{X\bar{M}} = \tfrac{1}{2}[\alpha p_S + (1-\alpha)p_{AA}] + \tfrac{1}{2}p_{AU}. \tag{A.47}$$

Via juveniles's PI copy. The consanguinity between juveniles born in the same patch via the focal juvenile's PI copy is:

$$p_{X\bar{P}} = \tfrac{1}{2}[\beta p_S + (1-\beta)p_{UU}] + \tfrac{1}{2}p_{AU}. \quad \text{(A.48)}$$

Consanguinity between females

Via female's MI copy. The consanguinity between breeding females in the same patch via the MI copy of a focal female of age 1 is:

$$p_{AA\bar{M}|1} = l_f[l_f\mu_f p_{X\bar{M}} + (1-\mu_f)p_{A\bar{M}}], \quad \text{(A.49)}$$

when she is of age 2 is:

$$p_{AA\bar{M}|2} = (1-\mu_f)p_{AA\bar{M}|1} + l_f\mu_f p_{A\bar{M}|1}, \quad \text{(A.50)}$$

and when she is of age t is:

$$p_{AA\bar{M}|t} = (1-\mu_f)p_{AA\bar{M}|t-1} + l_f\mu_f p_{A\bar{M}|t-1}. \quad \text{(A.51)}$$

On average

$$p_{AA\bar{M}} = (1-\mu_f)^2 p_{AA\bar{M}} + 2\mu_f(1-\mu_f)l_f p_{A\bar{M}} + \mu_f^2 l_f^2 p_{X\bar{M}}. \quad \text{(A.52)}$$

When $\mu_f = \mu_m = 1$ then $p_{AA\bar{M}} = l_f^2 p_{X\bar{M}}$ and we recover the result in Úbeda and Gardner (2011).

Via female's PI copy. The consanguinity between breeding females in the same patch via the PI copy of a focal female of age 1 is:

$$p_{AA\bar{P}|1} = l_f[l_f\mu_f p_{X\bar{P}} + (1-\mu_f)p_{A\bar{P}}], \quad \text{(A.53)}$$

when she is of age 2 is:

$$p_{AA\bar{P}|2} = (1-\mu_f)p_{AA\bar{P}|1} + l_f\mu_f p_{A\bar{P}|1}, \quad \text{(A.54)}$$

and when she is of age t is:

$$p_{AA\bar{P}|t} = (1-\mu_f)p_{AA\bar{P}|t-1} + l_f\mu_f p_{A\bar{P}|t-1}. \quad \text{(A.55)}$$

On average:

$$p_{AA\bar{P}} = (1-\mu_f)^2 p_{AA\bar{P}} + 2\mu_f(1-\mu_f)l_f p_{A\bar{P}} + \mu_f^2 l_f^2 p_{X\bar{P}}. \quad \text{(A.56)}$$

When $\mu_f = 1$ then $p_{AA\bar{P}} = l_f^2 p_{X\bar{P}}$ and we recover the result in Úbeda and Gardner (2011).

Consanguinity between males

Via male's MI copy. The average consanguinity between breeding males in the same patch via the MI copy of a focal male is:

$$p_{UU\bar{M}} = (1-\mu_m)^2 p_{UU\bar{M}} + 2\mu_m(1-\mu_m)l_m p_{U\bar{M}} + \mu_m^2 l_m^2 p_{X\bar{M}}. \quad \text{(A.57)}$$

When $\mu_f = \mu_m = 1$ then $p_{UU\bar{M}} = l_m^2 p_{X\bar{M}}$ and we recover the expression in Úbeda and Gardner (2011).

Via male's PI copy. The average consanguinity between breeding males in the same patch via the PI copy of a focal male is:

$$p_{UU\bar{P}} = (1-\mu_m)^2 p_{UU\bar{P}} + 2\mu_m(1-\mu_m)l_m p_{U\bar{P}} + \mu_m^2 l_m^2 p_{X\bar{P}}. \quad \text{(A.58)}$$

When $\mu_f = \mu_m = 1$ then $p_{UU\bar{P}} = l_m^2 p_{X\bar{P}}$ and we recover the expression in Úbeda and Gardner (2011).

Consanguinity between female and male

Via female's MI copy. The consanguinity between a female and her mating partner via the MI copy in a focal female of age 1 is:

$$p_{AU\bar{M}|1} = l_f[l_m\mu_m p_{X\bar{M}} + (1-\mu_m)p_{U\bar{M}}], \quad \text{(A.59)}$$

when she is of age 2 is:

$$p_{AU\bar{M}|2} = (1-\mu_m)p_{AU\bar{M}|1} + l_m\mu_m p_{A\bar{M}|1}, \quad \text{(A.60)}$$

and when she is of age t is:

$$p_{AU\bar{M}|t} = (1-\mu_m)p_{AU\bar{M}|t-1} + l_m\mu_m p_{A\bar{M}|t-1}. \quad \text{(A.61)}$$

On average

$$p_{AU\bar{M}} = (1-\mu_f)(1-\mu_m)p_{AU\bar{M}} + (1-\mu_f)\mu_m l_m p_{A\bar{M}} + \mu_f(1-\mu_m)l_f p_{U\bar{M}} + \mu_f\mu_m l_f l_m p_{X\bar{M}}. \quad \text{(A.62)}$$

When $\mu_f = \mu_m = 1$ then $p_{AU\bar{M}} = l_f l_m p_{X\bar{M}}$ and we recover the result in Úbeda and Gardner (2011).

Via female's PI copy. The consanguinity between a female and her mating partner via the PI copy in a focal female of age 1 is:

$$p_{AU\bar{P}|1} = l_f[l_m\mu_m p_{X\bar{P}} + (1-\mu_m)p_{U\bar{P}}], \quad \text{(A.63)}$$

when she is of age 2 is:

$$p_{AU\bar{P}|2} = (1-\mu_m)p_{AU\bar{P}|1} + l_m\mu_m p_{A\bar{P}|1}, \quad \text{(A.64)}$$

and when she is of age t is:

$$p_{AU\bar{P}|t} = (1-\mu_m)p_{AU\bar{P}|t-1} + l_m\mu_m p_{A\bar{P}|t-1}. \quad \text{(A.65)}$$

On average

$$p_{AU\bar{P}} = (1-\mu_f)(1-\mu_m)p_{AU\bar{P}} + (1-\mu_f)\mu_m l_m p_{A\bar{P}} + \mu_f(1-\mu_m)l_f p_{U\bar{P}} + \mu_f\mu_m l_f l_m p_{X\bar{P}}. \quad \text{(A.66)}$$

When $\mu_f = \mu_m = 1$ then $p_{AU\bar{P}} = l_f l_m p_{X\bar{P}}$ and we recover the result in Úbeda and Gardner (2011).

Solving the system of equations p_S in (A.15), $p_{M\bar{M}}$ in (A.35), $p_{X\bar{M}}$ in (A.47), $p_{A\bar{M}}$ (A.40), $p_{U\bar{M}}$ in (A.44), $p_{AA\bar{M}}$ in (A.52), $p_{AU\bar{M}}$ in (A.62), and $p_{UU\bar{M}}$ in (A.57) we get expressions for $p_{X\bar{M}}$, $p_{A\bar{M}}$, and $p_{U\bar{M}}$ as functions of p_{AA} and p_{AU}, and the model parameters. Solving the system of equations p_S in (A.15), $p_{M\bar{P}}$ in (A.38), $p_{X\bar{P}}$ in (A.48), $p_{A\bar{P}}$ in (A.42), $p_{U\bar{P}}$ in (A.46), $p_{AA\bar{P}}$ in (A.56), $p_{AU\bar{P}}$ in (A.66), and $p_{UU\bar{P}}$ in (A.58) we get expressions for $p_{X\bar{P}}$, $p_{A\bar{P}}$, and $p_{U\bar{P}}$ as functions of p_{UU} and p_{AU}, and the model parameters.

Relatedness between juvenile and aunt

Age 1

Via female's PI copy. The relatedness between a juvenile and a female via the MI copy in a focal female of age 1 is:

$$r_{\bar{M}|1} = \frac{p_{A\bar{M}|1}}{p_{M\bar{M}|1}}. \tag{A.67}$$

To calculate this value we need to know $p_{A\bar{M}|1}$ defined in (A.39) and $p_{M\bar{M}|1}$ defined in (A.34). Consanguinities $p_{A\bar{M}|1}$ and $p_{M\bar{M}|1}$ require us to know $p_{AA\bar{M}|1}$ defined in (A.49) and $p_{AU\bar{M}|1}$ defined in (A.59) together with p_S and p_{AU} defined in terms of the system parameters in the previous section. Consanguinities $p_{AA\bar{M}|1}$ and $p_{AU\bar{M}|1}$ require us to know $p_{X\bar{M}}$ defined in (A.47), $p_{A\bar{M}}$ defined in (A.40), and $p_{U\bar{M}}$ defined in (A.44). Consanguinities $p_{X\bar{M}}$, $p_{A\bar{M}}$, and $p_{U\bar{M}}$ are already defined in terms of p_{AA} and p_{AU} and these, in turn, are defined in terms of the system parameters in the previous section.

Via female's PI copy. The relatedness between a juvenile and a female via the PI copy in a focal female of age 1 is:

$$r_{\bar{P}|1} = \frac{p_{A\bar{P}|1}}{p_{M\bar{P}|1}}. \tag{A.68}$$

To calculate this value, we need to know $p_{A\bar{P}|1}$ defined in (A.41) and $p_{M\bar{P}|1}$ defined in (A.37). Consanguinities $p_{A\bar{P}|1}$ and $p_{M\bar{P}|1}$ require us to know $p_{AA\bar{P}|1}$ defined in (A.53) and $p_{AU\bar{P}|1}$ defined in (A.63) together with p_S and p_{AU} already defined in terms of the system parameters. Consanguinities $p_{AA\bar{P}|1}$ and $p_{AU\bar{P}|1}$ require us to know $p_{X\bar{P}}$ defined in (A.48), $p_{A\bar{P}}$ defined in (A.42), and $p_{U\bar{P}}$ defined in (A.46). Consanguinities $p_{X\bar{P}}$, $p_{A\bar{P}}$, and $p_{U\bar{P}}$ are already defined in terms of p_{AA} and p_{AU} and these, in turn, are defined in terms of the system parameters.

Thus we can calculate the initial coefficient of relatedness via the MI and the PI copies

Age t

Via female's MI copy. The relatedness between a juvenile and a female via the MI copy in a focal female of age t is:

$$r_{\bar{M}|t} = \frac{p_{A\bar{M}|t}}{p_{M\bar{M}|t}}. \tag{A.69}$$

To calculate this value we need to know $p_{A\bar{M}|t}$ defined in (A.39) and $p_{M\bar{M}|t}$ defined in (A.34). Consanguinities $p_{A\bar{M}|t}$ and $p_{M\bar{M}|t}$ require us to know $p_{AA\bar{M}|t}$ defined in (A.51) and $p_{AU\bar{M}|t}$ defined in (A.61) together with p_S and p_{AU} already defined in terms of the system parameters. Consanguinities $p_{AA\bar{M}|t}$ and $p_{AU\bar{M}|t}$ require us to know $p_{A\bar{M}|t-1}$, $p_{AA\bar{M}|t-1}$, and $p_{AU\bar{M}|t-1}$ defined recursively.

Via female's PI copy. The relatedness between a juvenile and a female via the PI copy in a focal female of age t is:

$$r_{\bar{P}|t} = \frac{p_{A\bar{P}|t}}{p_{M\bar{P}|t}}. \tag{A.70}$$

To calculate this value we need to know $p_{A\bar{P}|t}$ defined in (A.41) and $p_{M\bar{P}|t}$ defined in (A.37). Consanguinities $p_{A\bar{P}|t}$ and $p_{M\bar{P}|t}$ require us to know $p_{AA\bar{P}|t}$ defined in (A.55) and $p_{AU\bar{P}|t}$ defined in (A.65) together with p_S and p_{AU} already defined in terms of the system parameters. Consanguinities $p_{AA\bar{P}|t}$ and $p_{AU\bar{P}|t}$ require us to know $p_{A\bar{P}|t-1}$, $p_{AA\bar{P}|t-1}$, and $p_{AU\bar{P}|t-1}$ defined recursively.

Potential for communal care and potential for conflict

Given the coefficient of relatedness at age t we can define the potential for communal care when the focal female is of age t via her MI copy:

$$Q_{\bar{M}|t} = \frac{(1-a)r_{\bar{M}|t}}{1 - a r_{\bar{M}|t}} \tag{A.71}$$

and via her PI copy:

$$Q_{\bar{P}|t} = \frac{(1-a)r_{\bar{P}|t}}{1 - a r_{\bar{P}|t}}. \tag{A.72}$$

The potential for conflict when the focal female is of age t results from the difference between the potential for communal care via her MI and PI copies

$$I_t = Q_{\bar{M}|t} - Q_{\bar{P}|t}. \tag{A.73}$$

POTENTIAL FOR CONFLICT AT EQUILIBRIUM OVER AGE

We are interested in characterizing the age-equilibrium value of the potential for conflict I_t. The consanguinities that depend iteratively on their own value are the consanguinity between females $p_{AA|t}$ and the consanguinity between female and male

$p_{AU|t}$. At equilibrium they do not change any longer, that is $p_{K|t} = p_{K|t-1} = \hat{p}_K$.

MI Copy. Replacing the steady-state conditions $p_{AA\bar{M}|t} = p_{AA\bar{M}|t-1} = \hat{p}_{AA\bar{M}}$ and $p_{A\bar{M}|t} = p_{A\bar{M}|t-1} = \hat{p}_{A\bar{M}}$ into $p_{AA\bar{M}|t}$ defined in (A.51) yields:

$$\hat{p}_{AA\bar{M}} = (1-\mu_f)\hat{p}_{AA\bar{M}} + \mu_f l_f \hat{p}_{A\bar{M}} \tag{A.74}$$

and solving for $\hat{p}_{AA\bar{M}}$ results in:

$$\hat{p}_{AA\bar{M}} = l_f \hat{p}_{A\bar{M}}. \tag{A.75}$$

Replacing the steady-state conditions $p_{AU\bar{M}|t} = p_{AU\bar{M}|t-1} = \hat{p}_{AU\bar{M}}$ and $p_{A\bar{M}|t} = p_{A\bar{M}|t-1} = \hat{p}_{A\bar{M}}$ into $p_{AU\bar{M}|t}$ defined in (A.61) yields:

$$\hat{p}_{AU\bar{M}} = (1-\mu_m)\hat{p}_{AU\bar{M}} + \mu_m l_m \hat{p}_{A\bar{M}} \tag{A.76}$$

and solving for $\hat{p}_{AU\bar{M}}$ results in:

$$\hat{p}_{AU\bar{M}} = l_m \hat{p}_{A\bar{M}}. \tag{A.77}$$

Substituting the equilibrium values $\hat{p}_{AA\bar{M}}$ given by (A.75) and $\hat{p}_{AU\bar{M}}$ given by (A.77) in $p_{A\bar{M}|t}$ given by (A.39) yields:

$$\hat{p}_{A\bar{M}} = \tfrac{1}{2}[\alpha p_S + (1-\alpha) l_f \hat{p}_{A\bar{M}}] + \tfrac{1}{2} l_m \hat{p}_{A\bar{M}} \tag{A.78}$$

and solving for $\hat{p}_{A\bar{M}}$ results in:

$$\hat{p}_{A\bar{M}} = \frac{\alpha}{2-(1-\alpha)l_f - l_m} p_S. \tag{A.79}$$

Substituting the equilibrium value $\hat{p}_{AU\bar{M}}$ given by (A.77) in $p_{M\bar{M}|t}$ given by (A.33) yields:

$$\hat{p}_{M\bar{M}} = \tfrac{1}{2} p_S + \tfrac{1}{2} l_m \hat{p}_{A\bar{M}}. \tag{A.80}$$

Replacing the equilibrium value $\hat{p}_{A\bar{M}}$ derived in (A.79) in the above expression yields:

$$\begin{aligned} \hat{p}_{M\bar{M}} &= \tfrac{1}{2} p_S + \tfrac{1}{2} l_m \frac{\alpha}{2-(1-\alpha)l_f - l_m} p_S \\ &= \frac{2-(1-\alpha)(l_f + l_m)}{2-(1-\alpha)l_f - l_m} \tfrac{1}{2} p_S. \end{aligned} \tag{A.81}$$

PI Copy. Replacing the steady-state conditions $p_{AA\bar{P}|t} = p_{AA\bar{P}|t-1} = \hat{p}_{AA\bar{P}}$ and $p_{A\bar{P}|t} = p_{A\bar{P}|t-1} = \hat{p}_{A\bar{P}}$ into $p_{AA\bar{P}|t}$ defined in (A.55) yields:

$$\hat{p}_{AA\bar{P}} = (1-\mu_f)\hat{p}_{AA\bar{P}} + \mu_f l_f \hat{p}_{A\bar{P}} \tag{A.82}$$

and solving for $\hat{p}_{AA\bar{P}}$ results in:

$$\hat{p}_{AA\bar{P}} = l_f \hat{p}_{A\bar{P}}. \tag{A.83}$$

Replacing the steady-state conditions $p_{AU\bar{P}|t} = p_{AU\bar{P}|t-1} = \hat{p}_{AU\bar{P}}$ and $p_{A\bar{P}|t} = p_{A\bar{P}|t-1} = \hat{p}_{A\bar{P}}$ into $p_{AU\bar{P}|t}$ defined in (A.65) yields:

$$\hat{p}_{AU\bar{P}} = (1-\mu_m)\hat{p}_{AU\bar{P}} + \mu_m l_m \hat{p}_{A\bar{P}} \tag{A.84}$$

and solving for $\hat{p}_{AU\bar{P}}$ results in:

$$\hat{p}_{AU\bar{P}} = l_m \hat{p}_{A\bar{P}}. \tag{A.85}$$

Substituting the equilibrium values $\hat{p}_{AA\bar{P}}$ in (A.83) and $\hat{p}_{AU\bar{P}}$ (A.85) in $p_{A\bar{P}|t}$ given by (A.41) yields:

$$\hat{p}_{A\bar{P}} = \tfrac{1}{2}[\alpha p_S + (1-\alpha) l_f \hat{p}_{A\bar{P}}] + \tfrac{1}{2} l_m \hat{p}_{A\bar{P}} \tag{A.86}$$

and solving for $\hat{p}_{A\bar{P}}$ results in:

$$\hat{p}_{A\bar{P}} = \frac{\alpha}{2-(1-\alpha)l_f - l_m} p_S. \tag{A.87}$$

Substituting the equilibrium value $\hat{p}_{AU\bar{P}}$ given by (A.85) in $p_{M\bar{P}|t}$ given by (A.37) yields:

$$\hat{p}_{M\bar{P}} = \tfrac{1}{2} p_S + \tfrac{1}{2} l_m \hat{p}_{A\bar{P}}. \tag{A.88}$$

Replacing the equilibrium value $\hat{p}_{A\bar{P}}$ derived in (A.87) in the above expression yields:

$$\begin{aligned} \hat{p}_{M\bar{P}} &= \tfrac{1}{2} p_S + \tfrac{1}{2} l_m \frac{\alpha}{2-(1-\alpha)l_f - l_m} p_S \\ &= \frac{2-(1-\alpha)(l_f + l_m)}{2-(1-\alpha)l_f - l_m} \tfrac{1}{2} p_S. \end{aligned} \tag{A.89}$$

At equilibrium there is no difference in the consanguinities juvenile-female and juvenile-mother via the MI and PI copies, that is $\hat{p}_{A\bar{M}} = \hat{p}_{A\bar{P}}$ and $\hat{p}_{M\bar{M}} = \hat{p}_{M\bar{P}}$. Thus there is no difference in relatedness juvenile-aunt via the MI and PI copies:

$$\hat{r}_{\bar{M}} = \hat{r}_{\bar{P}} = \frac{\alpha}{1-(1-\alpha)\frac{1}{2}(l_f + l_m)}, \tag{A.90}$$

or in potential for communal care via the MI and PI copies:

$$\begin{aligned} \hat{Q}_{\bar{M}} = \hat{Q}_{\bar{P}} &= \frac{(1-a)\alpha}{1-(1-\alpha)\frac{1}{2}(l_f + l_m) - \alpha a} \\ &= \frac{(1-l_f^2+1-l_m^2)\alpha}{1-l_f+1-l_m+((1-l_f)l_f + (1-l_m)l_m)\alpha} \end{aligned} \tag{A.91}$$

and the potential for conflict becomes 0, that is $\hat{I} = 0$. Notice that the equilibrium values $\hat{r}$ and $\hat{Q}$ are independent of the probability of survival μ and the probability of paternal sibship β.