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The evolution of paternal care: a role for microbes?

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Abstract

Paternal care is an evolutionary mystery. Despite extensive research, both theoretical and experimental, the reasons for its ubiquity remain unclear. Common explanations include kin selection, suggesting that the benefits to the offspring outweigh the costs to the father's future reproductive success, and limited accuracy in parentage assessment. However, these explanations do not cover the breadth of circumstances in which paternal care has been observed, particularly in conditions of uncertainty in paternity. Many recent studies presented associations between microbes and complex behavioural traits, including anxiety, depression, and autism spectrum disorders. Here we propose that microbes may play a key role in the evolution of paternal care. Using computational models, we demonstrate that microbes associated with increased paternal care could be favoured by natural selection. We find that microbe-induced paternal care could evolve under wider conditions than suggested by genetic models. Moreover, we show that microbe-induced paternal care is more likely to evolve when considering paternal care interactions that increase microbial transmission, such as feeding and grooming. Our results suggest that factors affecting the host microbiome, such as antibiotics or specific foods, could also affect paternal behaviour.

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Keywords

Microbiome, paternal care, mathematical model, extra-pair mating, sexual conflict, nongenetic inheritance

Introduction

When should a father invest in caring for its offspring, rather than looking for additional mating opportunities? This question has been broadly addressed both theoretically and experimentally. Paternal care was frequently observed among avian species[1] (~85%), and was also found in mammalian species[2] (~5%), amphibians[3], and many species of fish[4]. It is most commonly observed alongside maternal care, while exclusive paternal care is rare[4,5]. A father may demonstrate care for its offspring with several types of interactions[4,5], such as feeding, grooming, or guarding against predators. It can also provide spousal care for the female while she cares for the young[6,7]. In many species there are synergistic effects[8], significantly increasing offspring fitness when cared for by two parents and not singlehandedly by one.

A commonly proposed explanation for the prevalence of paternal care is kin selection[9,10], suggesting that paternal care would be favoured whenever the paternal contribution to offspring fitness surmounts additional mating opportunities[11,12], often limited by female availability and receptivity[12]. In some settings, such as caring for unrelated young, this explanation is insufficient, and alternative explanations are suggested[13,14]. Interestingly, studies relating paternal effort to certainty of paternity obtained mixed results[15–21], and paternal care has been observed even in cases of very high probability of extra-pair paternity (e.g. in avian species[22] where extra-pair parenthood can range up to 95% in fairywrens[23]).

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Here we consider the potential role of the microbes in host paternal care. The microbiome is a significant agent affecting host health and behaviour[24–29]. There are several proposed mechanisms for this phenomenon, broadly referenced as 'gut-brain axis'[29]. Several studies have demonstrated a possible association between microbes and social behaviour[30–35]. Certain species of microbiome have been showed to alleviate symptoms of anxiety and depression[33] and improve social interactions[36]. Microbes are highly heritable, through gestation/incubation[37,38] or parental care[39–41]. Microbes can also be transmitted horizontally in a social setting[42], through interactions[41] such as feeding, grooming and copulation. The effect of microbes on host behaviour has given rise to the idea that host manipulation by microorganisms may be driven by natural selection on the microbes[29]. Selection could drive such an effect when the induced behaviour increases microbial fitness, for example by increasing the rate of microbial transmission or proliferation[29]. Previous theoretical studies suggested that by encouraging host sociality[43] or altruism[30], the microbes can help their own propagation.

We integrated the notion of microbe-associated behaviour into a mathematical model for the evolution of paternal care. A family is a unit with a high probability of microbial transmission[44], since the members of the family partake in frequent and profound interactions. Caring for the young presents an excellent opportunity from microbial perspective, since providing care both increases odds of offspring survival[11] and establishes a higher transmission probability. Therefore, a microbial gene that is associated with host intra-family caring behaviour could be favoured by natural selection even when encouraging care towards genetically unrelated young individuals. The propagation of microbes carrying these genes may have driven the evolution of paternal care even in the absence of paternity.

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Results

Let us first examine the case where males only adopt one of two pure strategies, either paternal care or lack thereof. Offspring fitness is increased by paternal care[11], due to provision and protection from predation, by a factor of $s > 0$. There are, on average, n available mating opportunities for a male who does not provide paternal care, and the expected level of paternity is assumed to be constant across them[45]. The male procures a higher benefit from providing paternal care than from not caring for the offspring when:

$$\frac{1+s}{n} > 1 \qquad (1)$$

Where n = available mating opportunities, s = increase in offspring fitness due to paternal care.

Now, we extend the model to include microbes as a reproductive unit that can affect paternal care behaviour. For simplicity, we neglect the effect of host genetic background in the microbe model and assume that host paternal behaviour is determined by its microbes. Let us consider microbes of type α, which are associated with paternal care behaviour, and microbes of type β, which have no effect on paternal care behaviour. Microbes can be transmitted to the offspring either from the mother, with probability T_v, or from the father, with probability T_c when the father cares for the offspring. Microbes can also be transmitted from the father to the mother during mating with probability T_m, and possibly through nurture of the mother, with probability T_n. In many species, mate nurturing behaviour is more common from father to mother than vice versa[6,7]. For simplicity, we assume that each host is inhabited by a single type of microbe at a given time. A transmission probability thus includes the probabilities that a microbe transmits to a new individual, establishes, and replaces the resident microbe, encompassing the competition dynamics between different microbial

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strains. The transmission pathways and transmission probabilities of the two microbes are illustrated in Fig. 1. We consider a model where the mother cares for the offspring[46,47], and additionally can transmit microbes during gestation[38] and natally[37], so overall maternal transmission is higher than paternal transmission ($T_v > T_c$). We also assume that paternal care involves more interaction – and potential for microbe transmission – than a singular mating encounter ($T_c > T_m^{♂}$). Since the probability of transmitting microbes during mating[48] is asymmetric between the sexes, with a higher probability for male-to-female transmission, we neglect the probability of female-to-male transmission. We initially assume that males have full paternity in their brood and relax that assumption later (see Fig. 3).

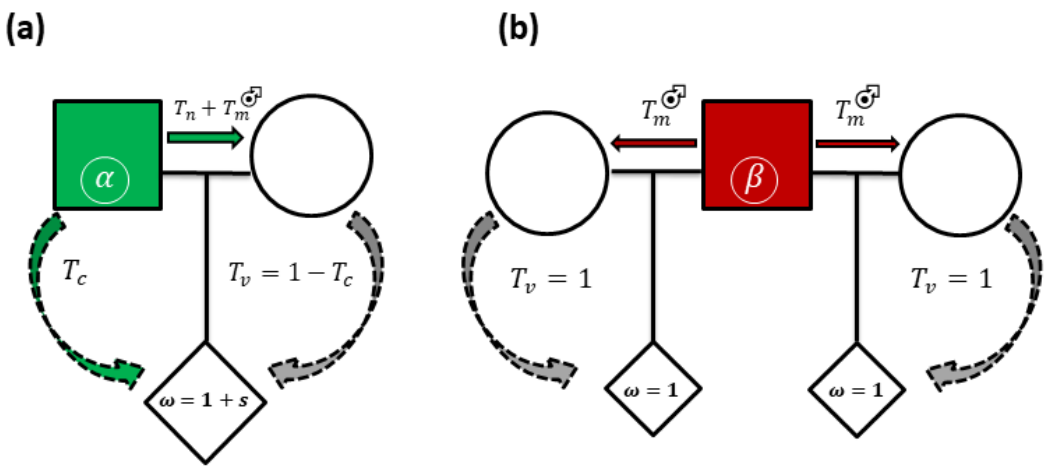

Figure 1. Illustration of microbe transmission pathways within the family. (a) where the father carries microbes of type α, inducing paternal care. (b) where the father carries microbes of type β, that have no effect on behaviour. Males carrying β do not care for the offspring and can be involved in n additional matings (illustrated is the case $n = 1$). T_v – vertical transmission probability through maternal influence (prenatal and postnatal). T_c – probability of transmission through paternal care. $T_m^{♂}$ - probability of male-to-female microbe transmission during mating. T_n – probability of transmission through male-to-female nurture.

Reproduction is divided into two stages. First is mating, where males and females randomly pair and mate. During this phase, microbes can be exchanged between the participating individuals. We assume a delay in the effect of the microbes on behaviour and neglect the

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possibility of a male altering its paternal behaviour due to contracting different microbes at the mating stage. The second phase is the transmission of microbes to the offspring. We assume $T_c + T_v = 1$, meaning that all the microbes of the offspring are obtained from its parents. In the case of lack of paternal care $T_v = 1$, meaning the offspring will receive its mother's microbes. We assume that during infancy, the offspring are interacting almost exclusively with individuals within the familial unit[49–52]. Consequently, we neglect the probability of contagion by the general population. The non-caring male paternal care has $n > 1$ mating opportunities but contributes no microbes to the offspring through care ($T_c = 0$). The fitness of an offspring whose father does not provide paternal care is $\omega_\beta = 1$, while an offspring that receives paternal care has increased fitness $\omega_\alpha = 1 + s$.

The condition for fixation of microbe type α (see Methods for full derivation) is given by:

$$\frac{\mathbf{1+s}}{\boldsymbol{n}} > \frac{\boldsymbol{T_m}}{\boldsymbol{T_c}*(\mathbf{1}-\boldsymbol{T_m})+\boldsymbol{T_m}+\boldsymbol{T_n}*(\mathbf{1}-\boldsymbol{T_c})} \qquad \textbf{(2)}$$

Fig. 2 presents the parameter range that allows for the evolution of paternal care in the model. In the genetic case, the cost-benefit isocline is given by $\frac{1+s}{n} > 1$ (Eq. 1). The range of conditions where a gene for paternal care evolves is shown by the blue area (Fig. 2). The conditions where a microbe inducing paternal care evolves can be much wider, shown by the areas below the yellow, green and red lines. The range widens with T_c, the probability of microbe transmission through paternal care (Fig. 2a) and narrows with T_m, the transmission probability during mating (Fig. 2b). These results demonstrate that paternal care can evolve even in the paradoxical case where paternal "care" decreases offspring fitness, if the overall probability of transmission through mating is sufficiently low in comparison with the probability of transmission through paternal care.

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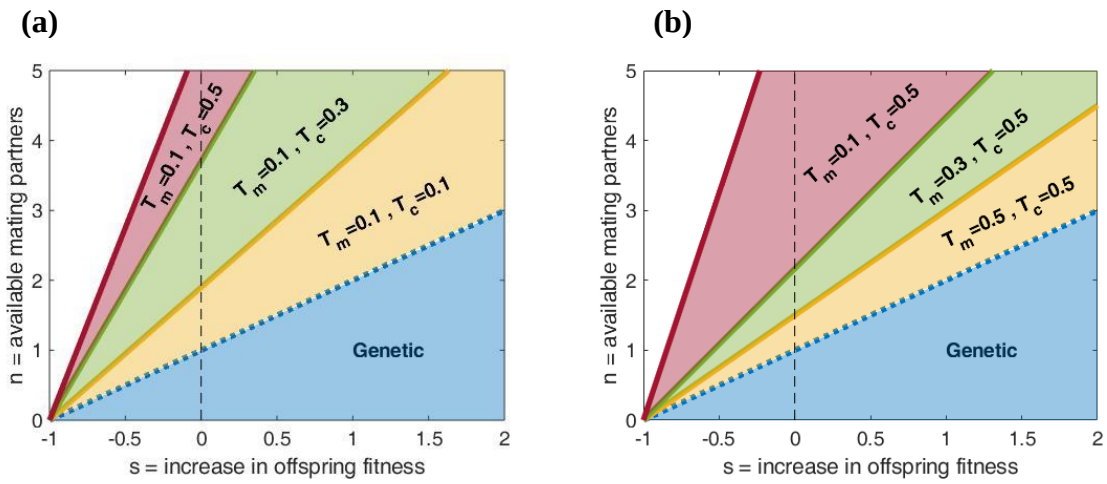

Figure 2. Microbes can expand the conditions for the evolution of paternal care. T_c – probability of microbe transmission from father to offspring through paternal care. T_m – probability of transmission from male to female during mating. The area below each graph represents the conditions allowing paternal care to evolve in the population. A microbe associated with paternal care behaviour can widen the range of conditions where paternal care prevails, and the effect increases with the transmission probability of the paternal microbes to the offspring during care. Microbe-induced paternal case can even evolve in some paradoxical cases where paternal "care" decreases offspring fitness. Other parameters: $T_n = 0$.

Now, let us consider a different social structure, where both males and females can engage in extra-pair mating, but offspring are brought up by social pairs[22,53–55]. The offspring of extra-pair mating are raised along with the rest of the mother's brood in the nest. We assume that a male has limited resources, which it distributes among its efforts to pursue additional mating opportunities and its paternal duties[12]. The more the father invests in its offspring, the fitter they will be, but the father will have fewer extra-pair progeny. The fitness of an offspring cared for by its social father is increased by a factor of $1 + s$. For each offspring in a social brood, there is a probability, P_C, that this offspring is not biologically sired by its social father. This corresponds to the availability of females interested in extra-pair matings[16], taking into account due to mate guarding, sperm competition, and cryptic female choice[56].The fitness of such an extra-pair offspring is increased by a factor of $1 + b$, due to direct or indirect benefits gained from extra-pair mating[22,57].

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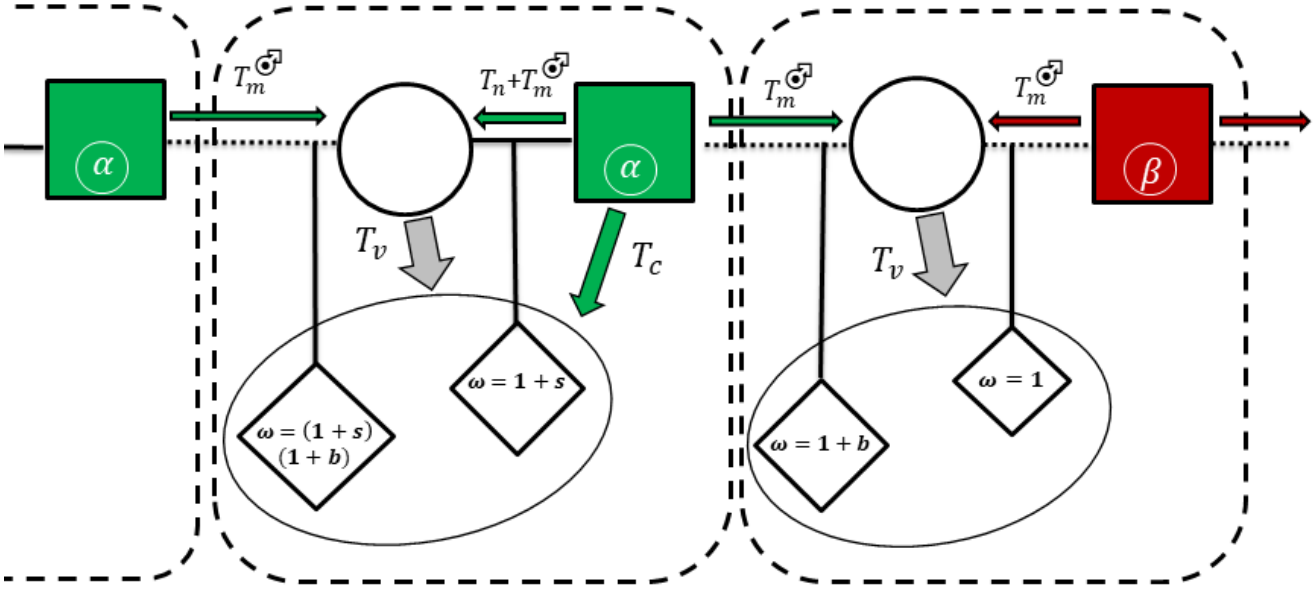

Figure 3. Illustration of microbe transmission pathways within families with extra-pair mating. Males carrying microbes of type β do not care for the offspring, while males carrying microbes of type α care for the offspring in their social nest. All males and females engage in extra-pair mating. T_v – vertical transmission probability through maternal influence (prenatal and postnatal). T_c – probability of transmission through paternal care. T_m - probability of male-to-female transmission during mating. T_n – probability of transmission through male-to-female nurture. Offspring sired by an extra-pair mate are $1 + b$ times more fit than offspring sired by the social mate. Offspring that receive paternal care are $1 + s$ times more fit than offspring that do not receive it.

Let ω_{xyz} be the fitness of an offspring with a social father of type x, a mother of type y and a biological father of type z (denoted by ω_{xy} if the social father x is also the biological father). From our assumptions $\omega_{\alpha\beta\alpha} = \omega_{\alpha\beta\beta} = \omega_{\alpha\alpha\alpha} = \omega_{\alpha\alpha\beta} = (1 + b) * (1 + s)$, $\omega_{\beta\alpha\alpha} = \omega_{\beta\alpha\beta} = \omega_{\beta\beta\alpha} = \omega_{\beta\beta\beta} = (1 + b)$, while $\omega_{\alpha\alpha} = \omega_{\alpha\beta} = (1 + s)$, and $\omega_{\beta\alpha} = \omega_{\beta\beta} = 1$.

We denote the total resources available to a male by E. These resources are allocated between its paternal care and its efforts of seeking extra-pair mates[12,58]. The amount of paternal care is given by s, the increase in offspring fitness. As paternal care increases, the caring father would be less successful in siring extra-pair offspring[53,54]. The success of a caring father in seeking additional mates is given by $n_\alpha = n * (1 - k\frac{s}{E})$, where $0 \leq ks \leq E$. The factor k

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represents the cost/benefit ratio between two effects of paternal care: the increase in offspring fitness and the decrease in paternal mating opportunities.

We find the conditions for fixation of an α gene, coding for paternal care, and similarly for the fixation of microbes of type α, inducing host paternal care (see Methods for mathematical derivations).

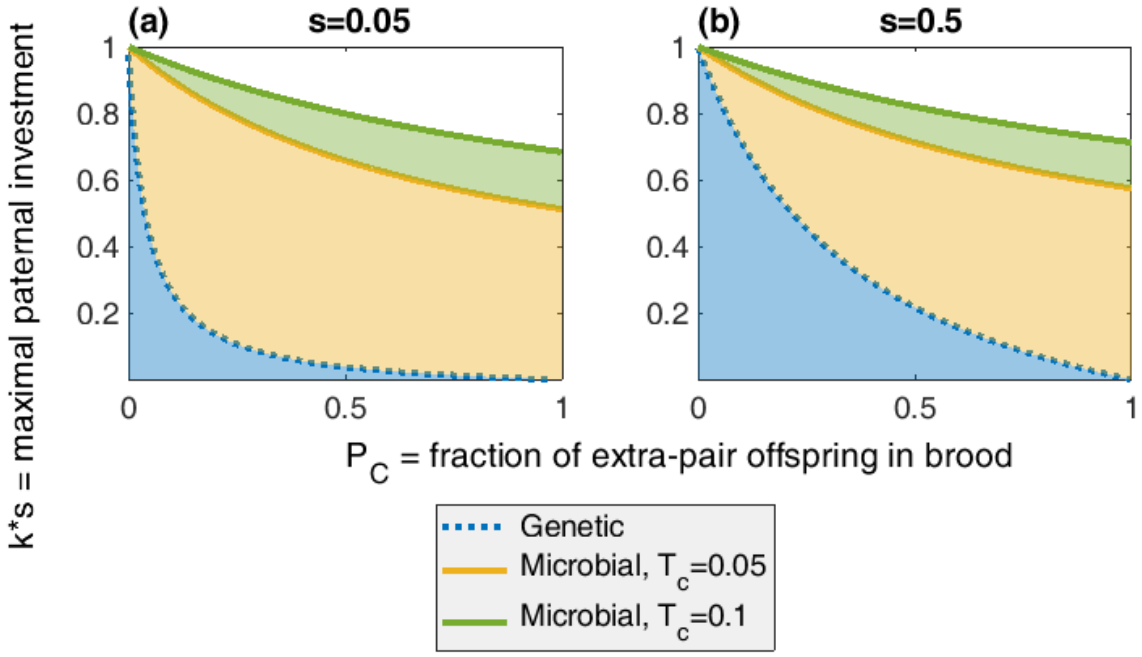

Figure 4. The evolution of paternal care in face of extra-pair offspring in brood. The figure represents the maximal paternal investment, ks, that allows for the evolution of paternal care induced by either genes or microbes. The solid lines represent the microbial case and the dotted line represents the genetic case. Generally, in the microbial case, paternal care evolves under wider conditions. The range narrows with an increase in extra-pair paternity in both the genetic and microbial cases. However, the effect is reduced when the transmission probability through paternal care (T_c) is high. The different plots (a),(b) represent different values of s, increase in offspring fitness due to paternal care. In both the microbial and the genetic cases, the range of paternal investment that allows for evolution of paternal care increases with s. In the microbial case, this effect is weaker when the ratio of transmission through caring and mating is high (T_c/T_m). Other parameters: $b = 0.2, T_m = 0.05, T_n = 0$.

Fig. 4 shows the maximal paternal investment that still allows for a gene or a microbe of type α (paternal care) to spread to fixation. A high degree of extra-pair paternity in the population

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has a dual effect in the same direction. First, it allows for more opportunities to breed as an extra-pair sire. Secondly, it reduces the genetic relatedness of the social father to the offspring in its nest, and thus the fitness benefits it receives from paternal care. This effect is stronger in the genetic case, since from microbial perspective, paternal care for a genetically unrelated young individual contributes the same fitness benefits as for a genetically related one.

The dynamics between the two microbe types (α and β) are strongly affected by the ratio between transmission probability through paternal care (T_c) and transmission probability through mating (T_m). A higher T_c allows for a wider range of conditions in which microbe-induced paternal care can evolve. An increase in offspring fitness due to paternal care (s) also widens the range of conditions where paternal care can evolve, but this effect is minor when microbial transmission through paternal care is significantly higher than transmission through mating ($T_c >> T_m$).

Discussion

In this work, we present an alternative explanation to a long-standing evolutionary conundrum: the ubiquity of paternal care. We show that when considering paternal care to be induced by microbes rather than by the father's genes, the range of conditions in which paternal care could evolve widens. We expect that microbe-induced paternal care could play a more significant role in circumstances where genetic relatedness falls short of explaining the observed degree of paternal investment, such as adoption[59], species where extra-pair paternity is common[22] and the well-known behaviour of cooperative breeding and eusociality[60,61]. Our model predicts that microbe-induced paternal care would more easily evolve when parent-offspring interactions are of high transmission ability[50,51,62–64] (high T_c, resulting from interactions corresponding to feeding, grooming).

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Previous work has discussed possible explanations for the broad existence of paternal care. Suggested explanations[60] include gaining practice in caring for young, resulting in higher chances for successful parenthood in the following year[14]; increasing indirect fitness[65,66]; and increasing chances of mating or territory acquisition[67,68]. In addition, mating may not be random with regard to the paternal trait. Since female choice is a key factor in determining male reproductive success, sexual selection may act directly on the paternal trait[10,66,69]. Another suggested explanation is inaccurate perceived paternity[70,71], or the limited accuracy of assessing offspring paternity by phenotypic signals. Lower accuracy helps preserve a stable level of paternal care[15,18,72]. However, when the cost of caring is high, and the expected level of paternity is low, selection is expected to favour more suspicious males that reduce their paternal investment with increased risk of extra-pair mating[9,18,72–75]. Nevertheless, paternal care has been demonstrated to prevail even in these cases in natural systems[22,23,54,76]. We demonstrate that the microbial perspective can explain stable levels of paternal care even under high levels of extra-pair mating, and when paternal investment is high.

Increased extra-pair mating may impose both costs and benefits on the female. In our model, the sum of costs and benefits is represented by the parameter $b > 0$, assuming that the benefits exceed the costs. The benefits of extra-pair mating for females[55,77–79] may be obtaining a higher quality or more compatible sire[78] and bet-hedging by increasing the genetic diversity of offspring[80]. The possible costs include loss of care by social mate[81], male sexual aggression[82], increased sibling competition[83,84], and the risk of contracting sexually transmitted pathogens[85]. Microbes may mediate some of these costs. As demonstrated by our results, care by the social mate prevails under wider conditions when induced by the microbes. Within-brood aggression between half-siblings[83] could also be mitigated by microbes, since relatedness among the microbes of the sibling is not expected to be significantly affected by the genetic relatedness among them.

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Our model can be extended in several ways. We examined two extremes, paternal care governed exclusively by host genes or exclusively by microbial genes. However, evolution of paternal care is likely driven by a selection on reproductive units in both levels, possibly leading to intermediate results. Additionally, it is possible to consider that when host genes and microbial genes experience conflicting selective pressures, selection on the host would drive the evolution of resistance genes to the microbial influence. In this case, we expect the host-microbe coevolution to generate oscillatory rock-paper-scissors evolutionary dynamics, that can allow the long-term maintenance of paternal care. Similar dynamics have been found by some of us with respect to microbe-induced cooperation and host resistance[86].

We may consider uneven potential reproductive opportunities among males. According to empirical studies[57,87–89], extra-pair mating success varies and has some correlation with male quality. This would have consequences on paternal strategy, where males who expect meagre success as philanderers may benefit more from increasing paternal investment. The availability of reproductive opportunities can also be restricted by the abundance of males pursuing extra-pair mating. Another extension would be allowing more female strategies. We assumed a constant level of maternal care. Yet, studies show females may reduce their care if the male provides sufficiently intensive care or increase their care to compensate for lack of male care[16,90,91]. Females may also vary the level of care offered to an offspring with the quality of its father, a strategy known as “differential allocation”[92,93]. Both these behaviours could reduce the benefits of paternal behaviour in favour of siring extra-pair offspring. In addition, we may consider maternal investment to be governed by microbial genes as well. Microbial influence over female reproductive behaviour could increase the frequency of extra-pair offspring within a brood to the extent that still allows for paternal care by the social mate.

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Our model joins the rank of previous models concerning the role of different nongenetic elements in the evolution of social traits[30,94–96]. Recent evidence suggests that microbes hold a significant role in shaping host evolution[25,30,97,98]. However, it is worth noting that the assumptions presented here are not limited to the microbiome and apply to any class of nongenetic elements that are capable of both vertical and horizontal/oblique[99,100] transfer and of influencing complex behavioural phenotypes. Examples of such elements may include epigenetic states[101,102] and culture[94,103–106].

Our theoretical results demonstrate a possible explanation to a widely studied and unresolved question in evolutionary biology – why males care for the offspring. Previous studies demonstrated hormonal regulation of paternal care[107–110], yet the mechanism by which microbes may regulate paternal behaviour is still to be experimentally validated. Nevertheless, our results suggest that factors affecting the host microbiome (for example antibiotics[111,112], probiotics[113,114], specific foods[115,116]) may also modify paternal behaviour.

Methods

Model Parameters

	Meaning	Range
$\boldsymbol{p_\alpha, p_\beta}$	frequency of individuals carrying gene/microbe of type α or β, respectively	$p_\alpha + p_\beta = 1$
$\boldsymbol{n}$	number of mating opportunities for a male that does not provide paternal care	$n > 1$
$\boldsymbol{E}$	total male resources allocated between its parental care and efforts of seeking extra-pair mates	$E = 1$
$\boldsymbol{s}$	increase in offspring fitness due to paternal care	$0 \leq k * s \leq 1$
$\boldsymbol{b}$	increase in offspring fitness due to benefits gained from extra-pair mating	$0 \leq b \leq 1$
$\boldsymbol{T_m}$	probability of male-to-female microbe	$0 \leq T_m \leq 1$

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	transmission during mating	
$\boldsymbol{T_c}$	probability of microbe transmission through paternal care	$0 \leq T_c \leq 1$
$\boldsymbol{T_n}$	probability of transmission through male-to-female nurture	$0 \leq T_n \leq 1$
$\boldsymbol{k}$	cost/benefit ratio between two effects of paternal care: the increase in offspring fitness and the decrease in paternal mating opportunities	$0 \leq k * s \leq 1$
$\boldsymbol{P_C}$	fraction of extra-pair offspring in brood (population mean)	$0 \leq P_C \leq 1$

Family structure I: full-sibs in brood, microbial case

We assume that the male who does not provide paternal care has n mating opportunities, where $n > 1$. Under random mating, the effective probabilities of the males in mating encounters are given by the following expressions:

$$\widetilde{p_\beta} = \frac{n * p_\beta}{n * p_\beta + p_\alpha}$$

$$\widetilde{p_\alpha} = 1 - \widetilde{p_\beta}$$

The frequency of microbe α in the offspring generation is given by:

$$p'_\alpha = \widetilde{p_\alpha} p_\alpha \cdot \frac{\omega_{\alpha\alpha}}{\bar{\omega}} + \widetilde{p_\alpha} p_\beta \left(T_c + T_v(T_n + T_m)\right) \frac{\omega_{\alpha\beta}}{\bar{\omega}} + \widetilde{p_\beta} p_\alpha (1 - T_m) \frac{\omega_{\beta\alpha}}{\bar{\omega}}$$

Offspring fitness increases with paternal care. An offspring that receives paternal care has a fitness of $\omega_\alpha = 1 + s$, while an offspring whose father does not provide paternal care has a fitness of $\omega_\beta = 1$. The mean offspring population fitness is given by:

$$\bar{\omega} = \widetilde{p_\alpha} p_\alpha \omega_\alpha + \widetilde{p_\alpha} p_\beta \omega_\alpha + \widetilde{p_\beta} p_\alpha \omega_\beta + \widetilde{p_\beta} p_\beta \omega_\beta$$

The change in the frequency of microbe α in the next generation is given by

$$\Delta p = p'_\alpha - p = \frac{p * (1 + s) * [(1 - p) * (T_c + T_n - T_c * T_m - T_c * T_n) + T_m]}{p * (1 + s) + n * (1 - p)} - T_m * p$$

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To find the equilibrium (p^*), we find p for which $\Delta p = 0$. The possible equilibria are $p^* = 0$ and $p^* = 1$, meaning the microbe of type α inducing paternal care either goes extinct or reaches fixation.

Stability analysis:

$$\frac{\partial \Delta p}{\partial p}|_{p^*=0} = \frac{(s + 1) * (T_c + T_m + T_n - T_c * T_m - T_c * T_n)}{n} - T_m$$

$$\frac{\partial \Delta p}{\partial p}|_{p^*=1} = T_c * T_m - T_n - T_c + T_c * T_n - \frac{T_m * (s - n + 1)}{s + 1}$$

The value of the derivative $\frac{\partial \Delta p}{\partial p}|_{p^*=0}$ and $\frac{\partial \Delta p}{\partial p}|_{p^*=1}$ can be either positive or negative, depending on the values of T_c, T_m, T_n, n, s. Hence, the equilibria may be stable, oscillatory or unstable. The condition for stable fixation of microbe α is $\frac{\partial \Delta p}{\partial p}|_{p^*=1} < 0$, which is given by the following inequality:

$$s > \left(\frac{T_m}{T_c * (1 - T_m) + T_m + T_n * (1 - T_c)}\right) * n - 1$$

Family structure II: extra-pair mating, genetic case

<u>Mating opportunities</u>

p – the proportion of hosts carrying microbes of type α before the mating season.

The success of a caring father in extra-pair matings is reduced proportionally to his paternal care (s). The parental investment is represented by ks, where k is the cost/benefit ratio between two effects of paternal care: the increase in offspring fitness and the decrease in paternal mating opportunities.

The representation of the different male types in additional mating encounters is given by the following expressions:

α males: $\frac{p \cdot (1-ks)}{p(1-ks)+1-p}$ β males: $\frac{1-p}{p(1-ks)+1-p}$

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Offspring types:

p_{xy} – proportion of offspring where the father is x and the mother is y

p_{xyz} – proportion of offspring where the social mate is x, the mother is y, and the extra-pair mate (genetic father of offspring) is z

The proportion of each offspring type is determined by the fraction of each type of parent (α/β female, α/β male) and the proportion of extra-pair offspring within a brood (P_c).

Proportion of offspring types	**Probability of α/β offspring**	**Offspring fitness**
$\boldsymbol{p_{\alpha\alpha} = p \cdot p \cdot (1 - P_c)}$	$p_o(\alpha) = 1$	$\omega = 1 + s$
$\boldsymbol{p_{\alpha\alpha\alpha} = p \cdot p \cdot P_c \cdot \frac{p \cdot (1 - k \cdot s)}{p(1 - k \cdot s) + 1 - p}}$	$p_o(\alpha) = 1$	$\omega = (1 + s) \cdot (1 + b)$
$\boldsymbol{p_{\alpha\alpha\beta} = p \cdot p \cdot p_c \cdot \frac{p \cdot (1 - k \cdot s)}{p(1 - k \cdot s) + 1 - p}}$	$p_o(\alpha) = T_c + (1 - T_m)(1 - T_c)$ $p_o(\beta) = T_m \cdot (1 - T_c)$	$\omega = (1 + s) \cdot (1 + b)$
$\boldsymbol{p_{\alpha\beta} = p \cdot (1 - p) \cdot (1 - P_c)}$	$p_o(\alpha) = T_n(1 - T_c) + T_c$ $p_o(\beta) = (1 - T_n)\,(1 - T_c)$	$\omega = 1 + s$
$\boldsymbol{p_{\alpha\beta\alpha} = p \cdot (1 - p) \cdot (1 - P_c) \cdot \frac{p \cdot (1 - k \cdot s)}{p(1 - k \cdot s) + 1 - p}}$	$p_o(\alpha) = (T_n + T_m)(1 - T_c) + T_c$ $p_o(\beta) = (1 - T_n - T_m)(1 - T_c)$	$\omega = (1 + s) \cdot (1 + b)$
$\boldsymbol{p_{\alpha\beta\beta} = p \cdot (1 - p) \cdot (1 - P_c) \cdot \frac{p \cdot (1 - k \cdot s)}{p(1 - k \cdot s) + 1 - p}}$	$p_o(\alpha) = T_c + T_n(1 - T_c)$ $p_o(\beta) = (1 - T_n)(1 - T_c)$	$\omega = (1 + s) \cdot (1 + b)$
$\boldsymbol{p_{\beta\alpha} = (1 - p) \cdot p \cdot (1 - P_c)}$	$p_o(\alpha) = (1 - T_m)$ $p_o(\beta) = T_m$	$\omega = 1$
$\boldsymbol{p_{\beta\alpha\alpha} = (1 - p) \cdot p \cdot P_c \cdot \frac{p \cdot (1 - k \cdot s)}{p(1 - k \cdot s) + 1 - p}}$	$p_o(\alpha) = 1 - T_m$ $p_o(\beta) = T_m$	$\omega = 1 + b$

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$\boldsymbol{p_{\beta\alpha\beta} = (1-p)\cdot p\cdot P_c \cdot \frac{p\cdot(1-k\cdot s)}{p(1-k\cdot s)+1-p}}$	$p_o(\alpha) = 1 - 2T_m$ $p_o(\beta) = 2T_m$	$\omega = 1 + b$
$\boldsymbol{p_{\beta\beta} = (1-p)\cdot(1-p)\cdot(1-P_c)}$	$p_o(\beta) = 1$	$\omega = 1$
$\boldsymbol{p_{\beta\beta\alpha} = (1-p)\cdot(1-p)\cdot(1-P_c) \cdot \frac{p\cdot(1-k\cdot s)}{p(1-k\cdot s)+1-p}}$	$p_o(\alpha) = T_m$ $p_o(\beta) = 1 - T_m$	$\omega = 1 + b$
$\boldsymbol{p_{\beta\beta\beta} = (1-p)\cdot(1-p)\cdot(1-P_c) \cdot \frac{p\cdot(1-k\cdot s)}{p(1-k\cdot s)+1-p}}$	$p_o(\beta) = 1$	$\omega = 1 + b$

The mean offspring fitness is given by:

$$\bar{\omega} = P_C * \left(p_{\alpha\beta\alpha} * \omega_{\alpha\beta\alpha} + p_{\alpha\beta\beta} * \omega_{\alpha\beta\beta} + p_{\alpha\alpha\alpha} * \omega_{\alpha\alpha\alpha} + p_{\alpha\alpha\beta} * \omega_{\alpha\alpha\beta} + p_{\beta\beta\alpha} * \omega_{\beta\beta\alpha} + p_{\beta\beta\beta} * \omega_{\beta\beta\beta} + p_{\beta\alpha\alpha} * \omega_{\beta\alpha\alpha} + p_{\beta\alpha\beta} * \omega_{\beta\alpha\beta}\right) + (1 - P_C) * \left(p_{\alpha\alpha} * \omega_{\alpha\alpha} + p_{\alpha\beta} * \omega_{\alpha\beta} + p_{\beta\beta} * \omega_{\beta\beta} + p_{\beta\alpha} * \omega_{\beta a}\right)$$

The frequency of the α gene in the next generation would be:

$$p'_\alpha = \frac{1}{\bar{\omega}} * P_C * \left(\frac{1}{2} * p_{\alpha\beta\alpha} * \omega_{\alpha\beta\alpha} + 0 * p_{\alpha\beta\beta} * \omega_{\alpha\beta\beta} + p_{\alpha\alpha\alpha} * \omega_{\alpha\alpha\alpha} + \frac{1}{2} * p_{\alpha\alpha\beta} * \omega_{\alpha\alpha\beta} + \frac{1}{2} * p_{\beta\beta\alpha} * \omega_{\beta\beta\alpha} + 0 * p_{\beta\beta\beta} * \omega_{\beta\beta\beta} + p_{\beta\alpha\alpha} * \omega_{\beta\alpha\alpha} + \frac{1}{2} * p_{\beta\alpha\beta} * \omega_{\beta\alpha\beta}\right) + \frac{1}{\bar{\omega}} * (1 - P_C) * \left(p_{\alpha\alpha} * \omega_{\alpha\alpha} + \frac{1}{2} * p_{\alpha\beta} * \omega_{\alpha\beta} + 0 * p_{\beta\beta} * \omega_{\beta\beta} + \frac{1}{2} * p_{\beta\alpha} * \omega_{\beta a}\right)$$

We calculate the equilibria as before, $\Delta p = p'_\alpha - p = 0$. Three equilibria points exist: $p^* = 0, p^* = 1$, and a polymorphic solution that under some conditions occurs within the range $0 < p < 1$.

We examine the derivative of Δp at $p^* = 1$ and $p^* = 0$. When $\frac{\partial \Delta p}{\partial p}|_{p^*=1} < 0$ it means there exists some critical p^* (either $p^* = 0$ or the polymorphic solution $p^* > 0$) from which the α

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gene will spread in the population to fixation, $p = 1$. When $\frac{\partial \Delta p}{\partial p}|_{p^*=0} > 0$ and $\frac{\partial \Delta p}{\partial p}|_{p^*=1} < 0$ it means that α gene will spread to fixation from $p = 0$.

$$\frac{\partial \Delta p}{\partial p}|_{p^*=1} = \frac{k * s}{2 * (1 - k * s)} - \frac{s * (1 - P_c) * (k + 1)}{2 * (P_c * b + 1) * (1 - k * s) * (s + 1)}$$

Differentiating the above expression with respect to k, we get:

$$\frac{\partial \left(\frac{\partial \Delta p}{\partial p}|_{p^*=1}\right)}{\partial k} = \frac{Pc * s * (b + 1)}{2 * (Pc * b + 1) * (k * s - 1)^2}$$

The above expression is positive for all $0 < s, Pc, b, k$. Thus, the function $\frac{\partial \Delta p}{\partial p}|_{p^*=1}$ is increasing with k. We want to find the range of k for which $\frac{\partial \Delta p}{\partial p}|_{p^*=1} < 0$. Thus, solving $\frac{\partial \Delta p}{\partial p}|_{p^*=1} = 0$ for k will give the maximal k for which this holds.

$$k^1 = \frac{1 - Pc}{Pc + s + Pc * b + Pc * b * s}$$

Now we will do the same for $\frac{\partial \Delta p}{\partial p}|_{p^*=0}$,

$$\frac{\partial \Delta p}{\partial p}|_{p^*=0} = -\frac{k * s}{2} + \frac{s * (1 - Pc) * (k + 1)}{2 * (Pc * b + 1)}$$

Differentiating the above expression in relation to k, we get:

$$\frac{\partial k \left(\frac{\partial \Delta p}{\partial p}|_{p^*=0}\right)}{\partial k} = -\frac{Pc * s * (b + 1)}{2 * (Pc * b + 1)}$$

The above expression is negative for all $0 < s, Pc, b$. Thus, the function $\frac{\partial \Delta p}{\partial p}|_{p^*=0}$ is decreasing when k is increasing. We want to find the range of k for which $\frac{\partial \Delta p}{\partial p}|_{p^*=0} > 0$. Thus, solving $\frac{\partial \Delta p}{\partial p}|_{p^*=0} = 0$ for k will give the maximal k for which this holds.

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$$k^0 = \frac{1 - Pc}{Pc \ + \ Pc * b}$$

Now, the range of k that allows for the evolution of the α gene to fixation is $0 < k < \min(k^0, k^1)$.

Let us examine the expression,

$$\delta k = k^1 - k^0 = (Pc - 1) * [\frac{1}{Pc * (1 + b)} - \frac{1}{Pc * (1 + b) + \ s * (1 + Pc)}]$$

The part in round brackets is negative, and the part in square brackets is positive. Thus, the entire expression is always negative, meaning $0 < k^1 < k^0$.

Overall,

$$Maximal\ k\ (Pc, b, s) \ = \ k^0$$

Family structure II: extra-pair mating, microbial case

Next, we introduce the microbial influence over the paternal behaviour. We define T_{sm} as the transmission probability of a microbe from a father providing paternal care to an offspring in its brood, and T_{ep} as the transmission probability of a microbe from an extra-pair mate to the offspring in the female's brood.

$$T_{sm} = \ T_c + (T_n + T_m) * (1 - T_c)$$

$$T_{ep} = \ T_m * (1 - T_c)$$

In this case, the frequency of the microbe α in the next generation is given by:

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$$p'_\alpha = \frac{1}{\bar{\omega}} * P_C * \big((T_{sm} + T_{ep}) * p_{\alpha\beta\alpha} * \omega_{\alpha\beta\alpha} + T_{sm} * p_{\alpha\beta\beta} * \omega_{\alpha\beta\beta} + p_{\alpha\alpha\alpha} * \omega_{\alpha\alpha\alpha}$$

$$+ (1 - T_{ep}) * p_{\alpha\alpha\beta} * \omega_{\alpha\alpha\beta} + T_m * p_{\beta\beta\alpha} * \omega_{\beta\beta\alpha} + 0 * p_{\beta\beta\beta} * \omega_{\beta\beta\beta}$$

$$+ (1 - T_m) * p_{\beta\alpha\alpha} * \omega_{\beta\alpha\alpha} + (1 - 2 * T_m) * p_{\beta\alpha\beta} * \omega_{\beta\alpha\beta}\big) + \frac{1}{\bar{\omega}} * (1 - P_C)$$

$$* \big(p_{\alpha\alpha} * \omega_{\alpha\alpha} + T_{sm} * p_{\alpha\beta} * \omega_{\alpha\beta} + 0 * p_{\beta\beta} * \omega_{\beta\beta} + (1 - T_m) * p_{\beta\alpha} * \omega_{\beta\alpha}\big)$$

We calculate the equilibria as for the genetic case, $\Delta p = p'_\alpha - p = 0$. Three equilibria exist: $p^* = 0, p^* = 1$, and a polymorphic solution that under some conditions exists within the range $0 < p < 1$.

For the microbial case, differentiating $\frac{\partial \Delta p}{\partial p}|_{p^*=1}$ with respect to k, we get:

$$\frac{\partial\left(\frac{\partial \Delta p}{\partial p}|_{p^*=1}\right)}{\partial k} = \frac{P_c * T_m * s * (1 - T_c) * (b + 1)}{(P_c * b + 1) * (k * s - 1)^2}$$

The expression above is positive for $0 < T_m, s, P_c, b, k, T_c < 1$. This means that $\frac{\partial \Delta p}{\partial p}|_{p^*=1}$ increases with k. We want to find the range in which $\frac{\partial \Delta p}{\partial p}|_{p^*=1} < 0$.

solving $\frac{\partial \Delta \mathrm{p}}{\partial \mathrm{p}}|_{\mathrm{p}^*=1} = 0$ for k will give the maximal k for which this holds.

$$k^1 = \frac{Tm * s * (1 + Pc * b) + (1 + s) * (1 + Pc * b) * (Tc * (1 - Tm) + Tn * (1 - Tc))}{Tm * s^2 * (1 + Pc * b) + s * (1 + s) * (1 + Pc * b) * (Tc * (1 - Tm) + Tn * (1 - Tc)) + s * (1 + s) * Pc * Tm * (1 - Tc) * (1 + b)}$$

Similarly, differentiating $\frac{\partial \Delta p}{\partial p}|_{p^*=0}$ with respect to k, we get:

$$\frac{\partial\left(\frac{\partial \Delta p}{\partial p}|_{p^*=0}\right)}{\partial k} = -\frac{P_c * T_m * s * (b + 1)}{P_c * b + 1}$$

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The above expression is negative for all $0 < b, T_c, T_n, s < 1$. Thus, the function $\frac{\partial \Delta p}{\partial p}|_{p^*=0}$ is decreasing when k is increasing. We want to find the range of k for which $\frac{\partial \Delta p}{\partial p}|_{p^*=0} > 0$. Thus, solving $\frac{\partial \Delta p}{\partial p}|_{p^*=0} = 0$ for k will give the maximal k for which this holds.

$$k^0 = \frac{(P_c * b + 1) * (s + 1) * (T_c + T_n - T_c * T_n)}{P_c * T_m * s * (b + 1)} - \frac{(P_c * b + 1) * (T_c - s + T_c * s)}{P_c * s * (b + 1)}$$

Now, the range of k that allows for the evolution of the α microbe to fixation is $0 < k < \min(k^0, k^1)$.

In this case δk can be either positive or negative, depending on the parameter values, hence the minimum needs to be calculated dynamically.

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Author Contributions

Y.G. and L.H. designed the study and formulated the model. Y.G. and O.L.-E. derived the analytical equations and implemented the code. Y.G. and L.H. analysed the results and wrote the manuscript.

Competing interests

The authors declare no competing interests.