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Author for correspondence:

Benjamin Bossan

e-mail: b.bossan@googlemail.com

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We were all young once: an intragenomic perspective on parent–offspring conflict

Benjamin Bossan, Peter Hammerstein and Arnulf Koehncke

Institute for Theoretical Biology, Humboldt-Universität zu Berlin, Germany

Parent–offspring conflict (POC) describes the evolutionary conflict between offspring and their parents over parental resource allocation. Offspring are expected to demand more resources than their parents are willing to supply because these offspring are more related to their own than to their siblings' offspring. Kin selection acts to limit these divergent interests. Our model departs from previous models by describing POC as an intragenomic conflict between genes determining life-history traits during infancy or parenthood. We explain why a direct fitness approach that measures the total fitness effect during exactly one generation is required to correctly assess POC in interbrood rivalry. We find that incorrect assumptions in previous models led to an overestimation of the scope of POC. Moreover, we show why the degree of monogamy is more important for POC than previously thought. Overall, we demonstrate that a life-history-centred intragenomic approach is necessary to correctly interpret POCs. We further discuss how our work relates to the current debate about the usefulness of inclusive fitness theory.

1. Introduction

Kin selection theory [1] was an eye-opener for researchers studying cooperation in nature. Hamilton's famous rule, $r \cdot b > c$, describes the condition for cooperation between related individuals. Potential helpers only help if the benefit b to the helped, weighted by the coefficient of relatedness r from helper to helped, exceeds the cost c to the helper. However, Hamilton's rule can also be used to understand when it pays to harm related individuals. As Trivers noted [2], individuals benefit from selfishly harming related individuals as long as the cost to the harmed, weighted by the coefficient of relatedness from self to harmed, does not exceed the benefit to self. The resulting potential for selfishness among related individuals creates a conflict within families, for which Trivers coined the term *parent–offspring conflict* (POC).

In essence, POC suggests that optimal resource allocation strategies differ between parents and offspring [2,3]. Kin selection predicts this conflict to occur systematically because parents are equally related to all their offspring, whereas each individual offspring's coefficient of relatedness to itself is twice as high as to its full siblings. Therefore, each individual offspring is expected to demand more resources than its parents are willing to supply. At the same time, kin selection limits offspring selfishness [2,3]. This is because when selfish offspring skew resource distribution in their favour, they leave parents with fewer resources, which in turn reduces the parents' prospect of producing more offspring. The resulting loss of genetically related siblings represents an opportunity cost to selfish offspring that needs to be balanced by the benefits from increased parental provision.

In order to assess the extent to which harming an actor's relatives still increases the actor's fitness, one has to account for all benefits and costs generated by this selfish behaviour. In Trivers's original work [2], he gave the example of a caribou calf that could extract more resources from its mother than the mother deemed fit. The extra resources would increase the viability of this calf at the detriment of potential future siblings. Trivers assumed explicitly that benefits and costs would subsume all potential effects caused by greater offspring demand, most prominently the potential cost of lost future siblings. From a simple kin

selection perspective, this opportunity cost is half as big for the offspring as it is for the mother because the offspring is twice as related to itself as to its future full siblings, whereas the mother is equally related to all her offspring.

Although the assumption that all costs and benefits be included was practical for Trivers to prove his point, it leaves open the question of which specific costs and benefits should be taken into consideration. Alexander [4] noted that a mutation causing the mentioned shift would later affect its bearer as an adult. Part of the bearer's offspring would behave selfishly as well, thereby erasing all potential benefits. In effect, he argued, costs and benefits would be identical for the offspring and the mother; the optimal level of parental care would thus be identical, and hence there would be no POC. It was never conclusively discussed, however, which factors have to be included in parent and offspring fitness for a sensible comparison between the two. For instance, should one consider the effects of selfish offspring behaviour on a focal offspring plus one potential additional offspring (as Mock & Parker did [5])? Models that also included grandchildren showed that POC persists [6–8], and it has been argued that this is evidence against Alexander's claim [5]. But why include grandchildren in the first place? Or, conversely, why not go even further?

The goal of this paper is to clarify these open issues regarding the fitness costs and benefits of POC, and, as a consequence, the general scope of POC. To achieve this, we take a 'gene's eye' view, because, as noted by Alexander [4], offspring will eventually become parents themselves. As a result, the true conflict is not between offspring and parents, but between genes affecting the amount of parental care given to offspring (supply) and genes affecting the amount of parental care requested by offspring (demand). We thus deal with an intragenomic conflict between supply function genes and demand function genes.

Looking at POC from this intragenomic perspective allows us to deal with the second problem of which factors to include in the fitness functions. For a correct assessment, it is crucial that all effects of a mutation are accounted for once, but none twice or more. This is best achieved by focusing on one generation. When a mutant allele produces more copies on average than a wild-type allele, the mutant will spread. To keep the model simple, we assume that increasing parental care has only two effects: first an increase in offspring survival probability (offspring quality), and second a decrease in parent survival probability (and thus offspring quantity [9]). The model thus deals with interbrood conflict, as in Trivers's original work [2]. Making all assumptions about benefits and costs explicit and directly calculating fitness over a consistent time interval, our model allows us to find the true scope of POC and to compare this scope with results from previous research.

2. Model

We propose a simple life-history model of inter-brood rivalry to study POC [10]. Owing to its intricacy, the formal derivation of our model is presented in the electronic supplementary material, S1 and S2. In this section, we will focus on the most important conceptual points that are needed to understand our model. Additionally, we will give a glimpse at how we derived the model equations.

Females produce only one offspring per period and are the only providers of parental care. Of the resources at her disposal in each period, a female allocates some to her offspring, thereby increasing the offspring's survival probability, and the rest to herself, thereby increasing her own probability to survive to the next period and produce another offspring. As long as the female survives, the cycle repeats, without any ageing effects. If a female is heterozygous for a dominant mutation increasing offspring demand, then offspring inheriting this mutation receive more resources, but leave the mother with less, whereas offspring inheriting the wild-type allele receive the normal resource allocation (figure 1*a*). Mutations that increase offspring demand thus decrease the prospects of all future siblings (figure 1*b*). Our life-history model allows us to evaluate all possibilities of an individual's life-history, and thus to determine how a mutation's allele fitness (i.e. the number of copies it produces) compares with that of the wild-type.

The fitness benefit and cost of a mutation increasing offspring demand—the increased survival of that mutant offspring, as well as the reduced probability of mother's survival, and hence of her having additional offspring—depends on the number of mutant offspring a female has. If only one mutant offspring is produced, this offspring reaps the benefit of receiving more parental care. At the same time, as there are no future siblings that carry the mutation, there will be no opportunity costs associated with the mutation. For each additional mutant offspring, however, benefit and cost occur once more. The occurrence ratio of benefits to costs thus approaches one to one as more mutant offspring are produced. Consequently, the advantage of bearing such a mutation will shrink when many offspring are produced. If a model focuses only on the case of one mutant offspring with exactly one future sibling, it implicitly assumes that the benefits of the mutation manifest twice, but the cost only once. This will probably lead to an overestimation of the average benefits generated by the mutation, which is avoided when all potential future siblings are considered.

Our model allows us to determine exactly how many mutant offspring are produced. For a female carrying a mutation that increases offspring demand, the average number of mutant offspring she has increases with the amount of available resources. For male mutants, only the number of mutants produced with a given *specific* female matters because the potentially reduced survival probability of a male's current mate does not matter once the male switches females. Consequently, the monogamy rate of the given mating system plays an important role in the fitness costs and benefits of POC. If the mating system is highly promiscuous, a mutant male produces at most one mutant offspring with a given female, and the average benefit of a demand increasing mutant is large. If the system is monogamous, however, the average number of mutant offspring produced is much higher, and the average benefit of having such a mutation shrinks. By explicitly accounting for male and female fitness, as well as the mating system's monogamy rate, our model takes this problem into account.

Finally, the specific effect of the mutation itself has an influence on which fitness function is appropriate. This is because a mutation that causes the offspring to demand a lot more resources will reduce the expected number of future siblings more severely than a mutation that only

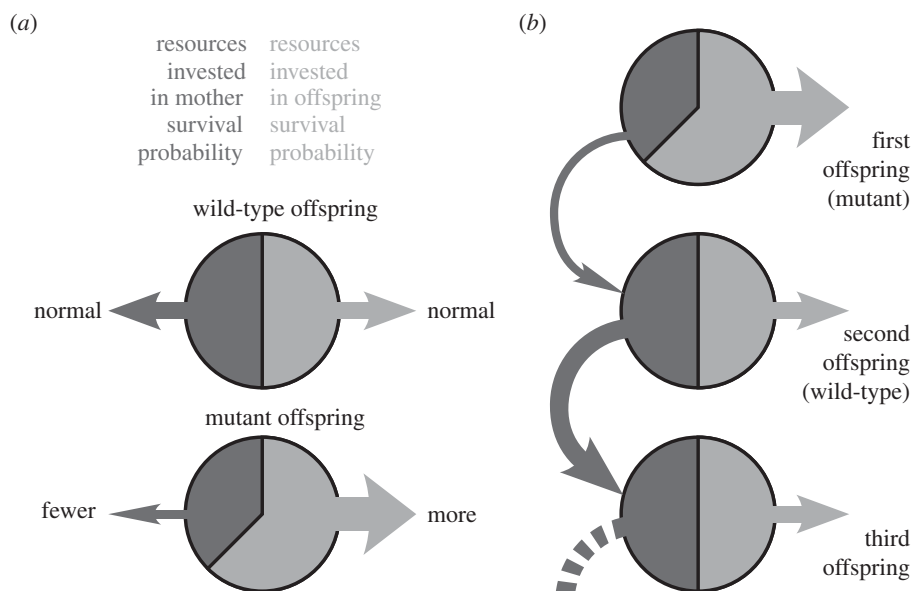


Figure 1. Life-history model of POC. (a) Effect of mutation on resource allocation. In each period, a female has a certain amount of resources (circles) that she can use to improve her own chance of surviving, or that of her offspring. The more she allocates to herself (dark grey part of the circle), the more likely it becomes that she survives to the next period and produces another offspring (offspring quantity). The more she allocates to her offspring (light grey part of the circle), the more likely the offspring is to survive to maturity (offspring quality). If the offspring is a wild-type, the resources will be allocated as usual. If the offspring inherited a mutation that causes higher demand, fewer resources are allocated to the mother and more to the offspring. (b) A possible life-history of a female. In this example, the first offspring bears the mutation, and hence has a higher survival probability (thick arrow), but makes it less likely for its mother to make it to the next period (thin arrow). The second offspring is a wild-type, so resource allocation is not skewed, leading to normal mother and offspring survival probability (medium-sized arrows).

leads to a moderate increase in demand. Therefore, the number of potential future siblings varies with the effect of the mutation. This needs to be taken into account to correctly assess mutant allele fitness.

As a result, estimating the fitness benefits of a mutation increasing offspring demand is far from being trivial, and has to be done from the perspective of both females and males. Consider a mutant allele present in a particular female, for instance. The number of copies produced by this allele needs to be summed over all mating periods that this female lives to see. Within any particular mating period, the expected number of allele copies depends on the female's current mating partner because the latter may pass on alleles to the offspring that change offspring resource demand, and thus female survival. After each mating period, the following three options must be considered. The female can die, with her survival probability depending on her previous offspring's phenotype, and future contributions to allele fitness are zero. She can survive, stay with the same male, and re-enter the loop of reproduction and survival with this male (though potentially with different genotypes and thus offspring and maternal survival rates if either of the two parents is heterozygous). Or, finally, she can change mates and re-enter the loop of mating, reproduction and survival with another male with potentially different genotype (see figure 2 for an illustration). Estimating mutant allele fitness from the perspective of males follows similar reasoning, but with males mating with a new female if the current partner dies (see electronic supplementary material S1 and S2 for further details).

Estimating the fitness benefits of a mutation increasing resource supply towards offspring is achieved in a similar fashion. There is, however, a crucial difference. When a female carries a dominant allele that increases supply, all her offspring will enjoy the increase in maternal care, regardless of the genotype of the offspring. Therefore, it does not matter which supply alleles the father carries, as they

cannot affect maternal supply. All offspring of this mother are treated equally. This contrasts with mutations that increase demand, since those mutations are only beneficial to offspring that actually inherit the mutation. Mutations in demand are thus discriminatory, whereas mutations in supply are not. This difference explains why there is a scope for conflict at all. As a consequence, there will be two optima, one for demand and one for supply; these optima constitute the boundaries of the 'battleground' of POC [5].

The specific behavioural traits underlying POC are best understood as reaction norms [11]. This is why our model assumes parents to express supply function genes that regulate resource supply to offspring and that offspring express demand function genes that regulate resource demand from parents [12]. Supply and demand are mediated by some independent variable, such as offspring begging intensity [13]. By focusing only on the *outcome* of parental supply and offspring demand, we can disregard the precise form of the supply and demand function, and consider only the resulting net resource allocation.

We use a standard evolutionarily stable strategy (ESS) approach [14] embedded into a diploid model, class-structured into females and males, to study how rare mutations fare against the wild-type allele. We assume there to be no spatial or temporal structure. Instead of calculating inclusive fitness by applying Hamilton's rule [1,15], we use a direct fitness approach that explicitly considers every outcome of the mutant allele, weighted by the corresponding probability of occurrence.

A detailed formal description of our model and the derivation of all our results can be found in electronic supplementary material S1 and S2. To explore in more detail how certain parameters affect our results, refer to electronic supplementary material S3. This document, which contains all interactive figures, can be viewed by installing the free CDF player available at <http://www.wolfram.com/cdf-player>.

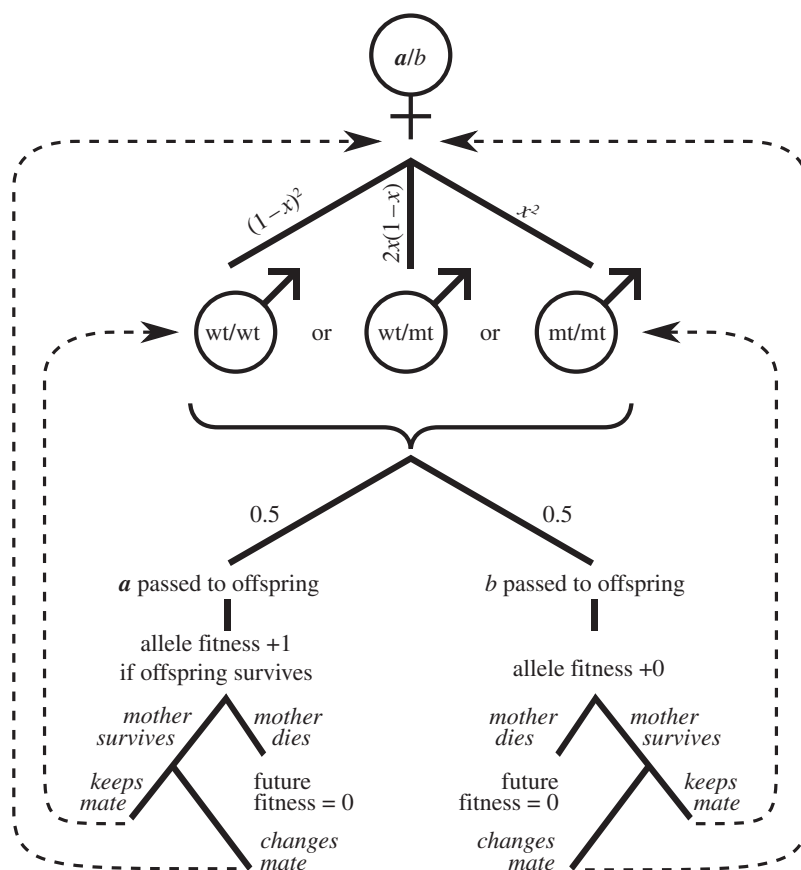


Figure 2. Estimation of mutant allele fitness. The game tree of a female life cycle lets us determine the fitness of wild-type (wt) and mutant (mt) alleles accumulated through females. Probabilities are shown in italics; x is the frequency of the mutant allele in the population. The mother carries two alleles, neutrally called a and b , which have to be replaced by the mutant or wild-type allele when making the actual calculations. At the beginning of the life cycle, a female encounters a mate, which can be a homozygous wild-type, a homozygous mutant or a heterozygote (with probabilities $(1-x)^2$, x^2 and $2x(1-x)$, respectively). In each mating period, the fitness of the a allele only increases if a is passed down to the offspring by the mother *and* if this offspring survives. Offspring survival probability is higher, but maternal survival probability lower, if the current offspring carries a mutation that increases resource demand. After producing an offspring, the mother may or may not survive, and she may or may not change her mate. If she survives and keeps her mate, she is in the same spot as *after* having encountered her mate for the first time. If the female survives and changes mates, she is in the same spot as *before* having encountered her previous mate. See §2 and electronic supplementary material S1 and S2 for further details.

All calculations were performed with WOLFRAM MATHEMATICA v. 8.0 (Wolfram Research).

3. Results

Fitness depends on quantity and quality of offspring, these two traits being traded off against each other. For analytical purposes, one of the traits can be treated as dependent on the other trait, this choice being arbitrary. For our purposes, we chose offspring quality (i.e. offspring survival probability) as the independent trait.

Our analyses show that for a given parameter set and for the assumed trade-off relationship between offspring quantity and quality, there is always exactly one value for offspring quality that is evolutionarily stable (see figure 3; also electronic supplementary material S1, and interactive figure S1 of electronic supplementary material S3). For demand function genes, this value is always greater or equal to $\frac{2}{3}$ but below $\frac{4}{5}$. It increases with the amount of available resources and decreases with higher monogamy rates (see interactive figure S2 of electronic supplementary material S3). The minimum is reached when there is strict monogamy (i.e. every individual has only one mating partner) and the maximum is

reached when there is complete polygamy (i.e. no individual mates with the same partner twice).

We show that there is also exactly one evolutionarily stable value for supply function genes (figure 3). This value is independent of the resources available to females and the monogamy rate. All else held constant, the mother supplies a fixed amount of resources to offspring survival (i.e. offspring quality) and the excess resources to her own survival, and hence production of future offspring (i.e. offspring quantity). This finding replicates that of a much earlier study [9], and thus lends support to our model.

For the given assumptions, the evolutionarily stable value for supply function genes—the genes that regulate maternal resource supply to offspring—is such that offspring have a survival probability of $\frac{1}{2}$. This value maximizes the expected number of total offspring and, since mutant supply function alleles cannot discriminately channel benefits to copies of themselves, also maximizes the expected total number of copies of the mutant allele.

Taken together, the evolutionarily stable value of offspring survival probability of supply function genes is $\frac{1}{2}$, compared with at least $\frac{2}{3}$ for demand function genes (see figure 3; also interactive figure S3 of electronic supplementary material S3). The evolutionary optima for these two groups of

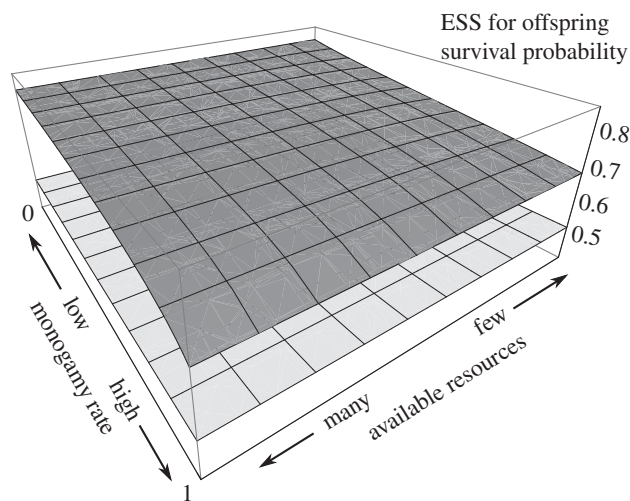


Figure 3. The battleground of POC. Evolutionarily stable values of genes affecting maternal care (light grey surface) and genes affecting offspring demand (dark grey surface) as a function of available resources and monogamy rate. Supply function genes evolve so as to guarantee a constant offspring survival probability of 50 per cent. In contrast, demand function genes evolve to reach an offspring survival probability between 66 and 80 per cent. Demand function genes thus aim at a higher offspring quality and lower offspring quantity, especially when there are many available resources and when the monogamy rate is low. As the evolutionary optima do not overlap, there is always potential for conflict between the two groups of genes. Parameter values: resource level (R) varies from 4 to 20, monogamy rate (p) from 0 to 1. See also interactive figure S3 of electronic supplementary material S3.

genes thus never overlap, meaning that there is always scope for conflict over parental resource allocation. In this sense, our results confirm previous findings that POC exists, refuting Alexander's [4] claim to the contrary.

Our model differs from previous approaches to POC [3,5] in its intragenomic perspective and the accurate incorporation of life-history-dependent effects. We examine these differences using a formula derived by Mock & Parker [5] that gives the condition for the spread of a 'conflicting tendency' (i.e. a mutant demanding more resources). We use Mock & Parker's formula because their book is the most comprehensive and widely read work on this topic. In our model's terms, where maternal care increases offspring survival probability, but not fecundity, Mock & Parker's condition for the spread of the conflicting tendency is that the fitness benefit of the focal offspring in terms of survival probability must exceed the loss of fitness through the subsequent sibling given that said sibling shares the mutant allele, the probability of which is given by the coefficient of relatedness. An offspring only ceases to demand more when the benefit and cost cancel out exactly.

To compare results, we assumed that for heterozygous offspring, the mutant allele is inherited with equal probability from mother as from father. However, mutant mothers but not mutant fathers have, on average, a decreased lifespan, so that mutations leading to increases in offspring demand are slightly more likely to be inherited from fathers than from mothers. The approximation of equal probabilities of inheritance is valid, however, if we assume mutations to have small effects.

Over almost the entire parameter space, our model predicts offspring to demand fewer resources than Mock & Parker's model does (figure 4). Mock & Parker's model

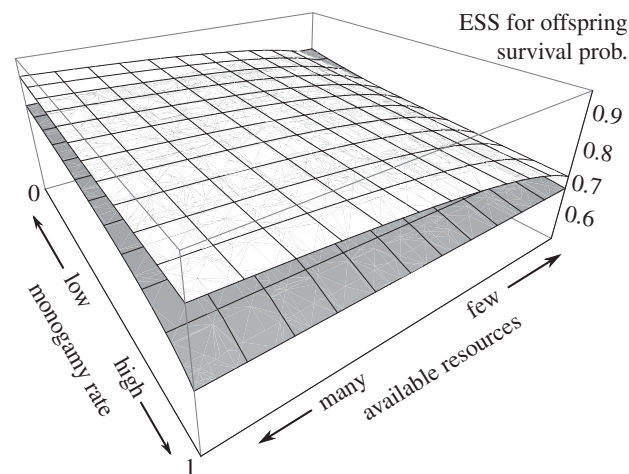


Figure 4. Evolutionarily stable value for offspring demand derived from two approaches. ESS value of offspring demand as derived from our model (dark grey surface) and as derived from Mock & Parker's model [5] (white surface) as a function of available resources and monogamy rate. Dependence on resource level is low and dependence on monogamy rate high. Mock & Parker's formula yields higher values for optimal offspring demand than our approach, except for very low resource levels and monogamy rates. The marginal impact of the monogamy rate is lower in their approach. Parameter values: resource level (R) varies from 4 to 20, monogamy rate (p) from 0 to 1. See also interactive figure S5 of electronic supplementary material S3.

only *overestimates* the costs of demanding more resources when the resource level and monogamy rate are very low. This is because a demand-increasing mutation has lower costs when fewer mutant offspring are produced, and the number of mutant offspring produced is in turn lower in this section of the parameter space. Furthermore, optimal offspring demand according to Mock & Parker's model can vary between 0 and 1, whereas our model predicts bounds between $\frac{2}{3}$ and $\frac{4}{5}$ (see electronic supplementary material S1). These divergent results originate because Mock & Parker assumed that one mutant offspring and one further sibling are produced, while our model takes into account all potential future offspring. We show that the more offspring are taken into consideration, the more the predicted demand level approaches the results derived from our model (see electronic supplementary material S1). Conversely, taking fewer offspring into account leads to an overestimation of optimal demand. Completely including all possibilities of an individual's life history is thus required for a correct fitness estimate.

Generally, Mock & Parker (as well as our approach) find the total amount of resources to only have a modest impact on the evolutionarily stable value of offspring demand, especially for high monogamy rates (figure 4). While the impact of monogamy rates on optimal offspring demand is negative in both models, it is larger in ours. This is because our approach correctly considers the expected number of future mutant siblings produced, which strongly depends on the monogamy rate.

Although the qualitative predictions are the same for our model as for that of Mock & Parker, quantitative differences can be substantial. Assuming that the conflict is resolved in favour of demand function genes, then a moderate amount of available resources and a monogamy rate of 50 per cent leads to an expected number of offspring per individual of

1.5 according to our calculation, and of approximately 1.25 according to Mock & Parker's calculation, a 16.5 per cent difference (see electronic supplementary material S1). For twice the amount of resources and strict monogamy, the expected number of offspring per individual would be approximately 3.6 according to our calculation, and only approximately 2.2 according to Mock & Parker's calculation, a difference of 39 per cent. These quantitative differences in reproductive success have highly significant evolutionary effects—a hypothetical mutation with a starting frequency of 0.1 per cent and a 16.5 per cent fitness advantage over the wild-type would displace said wild-type in less than 100 generations, versus one with a 39 per cent advantage in less than 50 generations (simulations of standard replicator dynamics not shown). The quantitative differences between the two models can be explored in more detail in interactive figures S4 and S5 of electronic supplementary material S3.

4. Discussion

We analyse POC from an intragenomic perspective. Resource allocation between offspring and mother affects the trade-off between offspring quality and quantity. From the point of view of genes affecting parental supply of care towards offspring, we find that these genes should aim at a fixed offspring survival probability, independent of the available resources or the monogamy rate. The remainder of the resources should be used to produce more offspring. From the point of view of genes affecting offspring demand for parental care, we find the evolutionarily stable value to be strictly greater than that for parental supply. A potential for POC is therefore always present. In this sense, our results resemble the 'battleground' of classical POC theory. Moreover, the need for parental care from the side of demand function genes is predicted to increase with the amount of available resources and decrease with higher monogamy rates. This means that POC is expected to be strongest in polygamous species that produce a high average number of offspring.

We find that, because previous models of interbrood competition did not incorporate the organisms' entire life history, they misestimated the total benefits and costs of skewing resource allocation. We also find the monogamy rate to play a more important role than previously thought. Our revised theoretical findings predict offspring to demand less than previously expected, with potentially large effects on lifetime reproductive success. These findings may at least partly explain why empiricists have found less evidence for conflict between parents and offspring than suggested by theory [16].

In the established terms of POC theory, our model lays out the battleground of a conflict over resource allocation in a new light. As always, solving the conflict would require additional assumptions. A resolution model of our battleground portrayal could be conceived by incorporating signalling [3,5,17] or developmental limits imposed on the form of the demand and supply function [11,18,19].

(a) The benefits of the direct fitness approach in contrast to the use of Hamilton's rule

Hamilton's rule, $r \cdot b > c$, is captivating in its elegance and purity. Though the formula's simplicity may suggest that

its components are straightforward to assess, this is often not the case. When POC occurs in species with more than one brood, for instance, each subsequent brood has a lower probability of having been sired by the same father. Therefore, the coefficient of relatedness r between siblings of different broods declines geometrically. This, however, violates the simple linearity of Hamilton's rule. Although models that make use of Hamilton's rule can extend it to take such effects into account (this leads to the same result as our direct fitness approach, see electronic supplementary material S1), it is debatable whether this more complicated formula can still be justly called 'Hamilton's rule'.

These findings, together with previous similar results [20], could be taken as evidence for the claim [21] that the direct fitness approach is superior to the inclusive fitness approach. However, we would not go so far as to claim this to be universally true. Our analysis of POC shows that conceptual disparities between direct compared with inclusive fitness models can lead to different quantitative predictions, but that the qualitative predictions made by the latter are still true. Instead of dismissing the inclusive fitness approach altogether, we believe that Hamilton's rule still provides a useful short cut to understanding the evolution of social traits. Our results serve as a reminder, however, that researchers should check carefully for each particular case as to whether this short cut may lead them astray.

(b) Outlook and conclusion

Our results demonstrate that a better understanding of POC can be achieved not from the perspective of resource allocation quarrels between parents and offspring, but from the point of view of genes involved in resource supply and demand. However, this conflict manifests itself during a time of life when the supply function genes are expressed in parents and the demand function genes are expressed in offspring. At the behavioural level, this intragenomic conflict thus still appears to be a conflict between parents and offspring.

Though the quantitative differences between our and previous models may seem small—but far from insignificant—the conceptual progress is large. After all, our model takes into account the intragenomic nature of POC and makes clear that direct fitness is the most straightforward method to assess the potential for POC. For the first time (to our knowledge), the costs and benefits of selfish offspring behaving 'against the will of their parents' are determined accurately in an interbrood rivalry setting.

The intragenomic reinterpretation of POC needs to be integrated into future empirical and theoretical studies, not only to ensure that the fitness costs and benefits of selfish offspring behaviour are assessed correctly, but also to clarify that we are not looking for winning offspring or losing parents, but for conflicting groups of genes in evolutionary equilibrium. In the end, all parents have also been offspring themselves—we were all young once.

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