Review

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Female extra-pair mating: adaptation or genetic constraint?

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Why do females of so many socially monogamous species regularly engage in matings outside the pair bond? This question has puzzled behavioural ecologists for more than two decades. Until recently, an adaptionist's point of view prevailed: if females actively seek extra-pair copulations, as has been observed in several species, they must somehow benefit from this behaviour. However, do they? In this review, we argue that adaptive scenarios have received disproportionate research attention, whereas nonadaptive phenomena, such as pathological polyspermy, *de novo* mutations, and genetic constraints, have been neglected by empiricists and theoreticians alike. We suggest that these topics deserve to be taken seriously and that future work would benefit from combining classical behavioural ecology with reproductive physiology and evolutionary genetics.

Costs and benefits of female extra-pair mating

Mating outside the social pair bond seems obviously adaptive for males, but the benefit to females is less clear because it does not increase the number of offspring that they produce. Given that active female extra-pair mating is often found, behavioural ecologists have sought explanations for this behaviour. Numerous adaptive explanations have been proposed [1,2], yet general support for these hypotheses remains limited [3–5]. Most of the research has been conducted in a framework of adaptionist thinking: the fact that females show active extra-pair mating must mean that they benefit from this behaviour. Nonadaptive explanations [6] were rapidly discarded [7,8] and then apparently forgotten ([6] was not cited in the extra-pair paternity literature between 1995 and 2011). However, several of the most powerful empirical tests of adaptive explanations have yielded puzzling results [9–11], even suggesting that female extra-pair mating behaviour is detrimental to females (behaviour we refer to as maladaptive; see Glossary). Moreover, a first empirical test of a nonadaptive explanation [6] provided support for the idea that female promiscuity could evolve even when it has negative consequences to females [12]. These two developments suggest that a broader spectrum of hypotheses should be evaluated, including models in which genetic constraints prevent the evolution of optimal behaviour.

Adaptive hypotheses for female extra-pair mating have focussed on a range of possible benefits, yet there are

Glossary

Adaptive (adaptationist) hypothesis: explanations that a particular trait has evolved to increase an individual's fitness (see 'individual fitness' below).
Antagonistic pleiotropy: when alternative genetic variants (alleles) affect multiple phenotypic traits under opposing selection pressures (e.g., an allele has beneficial effects on trait 1 but detrimental effects on trait 2); such traits can be found within one sex (intrasexual antagonistic pleiotropy), or across the sexes (intersexual antagonistic pleiotropy); in the latter, it can be the same phenotypic trait expressed in each sex that is under opposing selection.
Compatible genes: alleles that increase the fitness of an organism only when combined with a particular set of other alleles and, therefore, the fitness benefits normally are not heritable; these alleles contribute to nonadditive genetic variance (e.g., epistasis), which is part of the phenotypic variance.
Genetic constraint: limitations to the adaptation of an organism due to its genetic architecture.
Genetic correlation: correlation between two traits that arises from shared genetic effects (because the traits are affected by the same alleles); such genetic correlation can be found within one sex (within-sex genetic correlation) or across the two sexes (cross-sex genetic correlation; here, the genetically correlated traits can be the same trait expressed in males and in females).
Good genes: alleles that directly increase the fitness of an organism, so that the fitness benefits are heritable; these alleles contribute to additive genetic variance, which is part of the phenotypic variance.
Individual fitness: the contribution of an individual to the gene pool of future generations. Fitness benefits are often divided into direct (nongenetic) benefits (e.g., obtaining food) and indirect (genetic) benefits (e.g., obtaining 'good genes'). Note that, in the context of extra-pair mating, copulations do not have to lead to extra-pair fertilisations for the female to obtain direct benefits [71].
Intralocus sexual conflict: when opposing selection pressures act on allelic variation at one gene locus, because one allele enhances the fitness of males whereas the other allele enhances the fitness of females.
Linkage disequilibrium due to assortative mating: nonrandom mating with regard to phenotypes (e.g., promiscuous females mate with promiscuous males) leads to an association between alleles that influence those phenotypes (e.g., alleles for male promiscuity and alleles for female promiscuity will often be found in the same individuals).
Nonadaptive (maladaptive) hypothesis: explanations for the evolution or maintenance of a particular trait or behaviour despite the fact that it decreases the fitness of an individual (see 'individual fitness' above).
Oligospermy: male fertility condition that leads to scarcity of sperm cells, which could result in a reduced ability or inability to fertilise eggs.
Polyspermy: more than one sperm cell penetrates the egg membrane, usually due to an excessive supply of sperm. Fertilisation by more than one sperm leads to a nonviable zygote because of an abnormal copy number of chromosomes (pathological polyspermy). In birds, typically many sperm penetrate the egg membrane (referred to as physiological polyspermy) but typically the nucleus of only one sperm fuses with the nucleus of the egg (hence, the analogue of pathological polyspermy in birds is polyspermic fusion of nuclei).
Polygenic trait: a character that is controlled by numerous loci (genes); in contrast to a monogenic trait, which is controlled by one locus.
Promiscuity: used as short for an increased propensity to copulate with multiple individuals (also outside the pair bond); here not used to imply indiscriminate mating.

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Table 1. Possible benefits and costs associated with female extra-pair mating

Proposed benefits	Refs	Possible costs	Refs
Good or compatible genes (genetic benefits)	[1–4,72]	*De novo* deleterious mutations	[19,20]
Avoiding inbreeding (with related partner)	[22,23]		
Inclusive fitness gain by extra-pair mating with kin	[73]	Inbreeding depression	[73]
Fewer infertile eggs (fertility insurance)	[30]	Increased embryo mortality (polyspermy)	[37]
Avoid infanticide by other males	[46]	Punishment or aggression by social mate	[74]
Reduced harassment (convenience polyandry)	[5]	Increased harassment (Box 3)	
Increased care (by extra-pair males)	[71]	Loss of care (by social mate)	[4]
Access to resources held by neighbours	[75]		
Securing a future partner	[76]	Risk of losing the current partner	
Bet-hedging benefits via offspring diversity	[77]	Increased sibling competition	[78]
Beneficial sexually transmitted microbes	[79]	Sexually transmitted diseases	[13]

also many possible costs associated with this behaviour (Table 1). Some of these costs, such as the greater likelihood of contracting a sexually transmitted disease, have been posited for several decades [13] and, yet, have been largely ignored empirically. Most of these costs and benefits have been discussed extensively elsewhere (see the references in Table 1), so we here focus on reviewing the current support for the hitherto most widely accepted adaptive explanations. We then highlight several possible costs that have received little attention within the prevailing framework of adaptive thinking. Finally, we outline the most plausible models of genetic constraint that could explain how active female extra-pair mating could persist even if it is detrimental to female fitness.

Do females obtain genetic benefits?

'Genetic benefit models' suggest that females obtain either good genes or compatible genes from extra-pair matings, and this idea has been the focus of multiple reviews (e.g., [3,5,14–17]). Variants of the genetic benefit hypothesis have been tested in a variety of species, using one or a combination of the following approaches.

First, the occurrence of extra-pair paternity has been related to variation in adult male traits such as age, condition, immune response, and the expression of ornaments. The rationale being that female choice for partly heritable indicators of male viability and fitness provides a paternal genetic contribution to offspring survival, attractiveness, or competitive ability. The most generally reported pattern is that extra-pair sires are older and larger than males that do not sire extra-pair offspring [3,18]. However, it remains controversial whether this is necessarily indicative of a good genes benefit [19–21].

The second approach, used to test the genetic compatibility or inbreeding avoidance hypothesis, has been to investigate whether extra-pair paternity is more common when partners are genetically more similar to each other. This pattern has been found in some species (e.g., [22,23]), making the avoidance of inbreeding a likely adaptive explanation for these cases. Less clear evidence has emerged from studies testing whether the genetic similarity between the female and the extra-pair partner is lower than that between the female and her social partner (reviewed in [16], but see also [24]). For the latter kind of studies, methodological artefacts arising from paternity assignment probabilities must also be considered [25].

The third and most direct approach to test genetic benefit models is to compare fitness-related traits of extra-pair young relative to those of within-pair young. Although many studies have suggested that extra-pair offspring do better in one way or another (e.g., survive better, are more heterozygous, are in better condition, or have a better immune response), in some of the best-studied species if anything the opposite effect is found [9–11,26]. Only one out of four studies that measured lifetime fitness of offspring (arguably the best measure of female genetic benefits) found that extra-pair offspring did better [27], whereas in the three other studies, they did considerably worse [9–11]. A general problem with this approach is that such findings can be confounded by uncontrolled maternal effects (Box 1).

In sum, the available evidence raises doubts about the general applicability of the genetic benefits hypothesis and, despite much work in a variety of socially monogamous species from different taxa, the evidence for increased offspring fitness through paternal genetic contributions remains limited. When looking at the literature beyond the socially monogamous species, there is also little support for genetic benefits of mating with several males. A recent meta-analysis [28] of experimental studies revealed a weak and nonsignificant positive effect of multiple mating on offspring performance ($d = 0.12$, $P = 0.28$, $n = 16$ species) when excluding other sources of benefits (such as genetic diversity in social insects [29]).

Do females benefit from fertility insurance?

Failed support for the genetic benefit hypothesis has led to increased popularity of the 'fertility insurance hypothesis' [14,30,31]. It has often been taken for granted that extra-pair mating provides fertility insurance benefits, but a recent review [30] rightly argues that benefits are obtained only under specific circumstances, (e.g., when the partner of the female is truly infertile). However, such male infertility is expected to be a rare phenomenon (because of strong selection against infertility), and it is unlikely that infertility is detected by females from male indicator traits, such as ornamentation [32,33]. This would mean that, although all females would have to pay the potential costs of extra-pair mating, few would obtain a benefit.

Noticeably, the fertility insurance debate [30] has been centred on possible adaptive explanations for female extra-pair mating, while neglecting phenomena that might

Box 1. Do maternal effects confound paternal genetic effects?

The comparison of the fitness of within- and extra-pair offspring from the same brood or litter is the most straightforward test of the genetic benefits hypothesis, because many confounding factors that also influence fitness can be excluded. Half-siblings that grow up in the same brood differ in paternal genes or in their level of inbreeding or heterozygosity, but share the same environment, the same social parents, and the same maternal genes. However, fitness differences between within- and extra-pair offspring can still be due to parental effects, for example, if males or females invest differentially in the two types of offspring. Although this might seem unlikely [80] and might also lead to the opposite effect that extra-pair young do worse, recent work provides evidence for an important maternal effect that might lead to higher fitness of extra-pair young independent of their paternal genotype.

In many species, early-born or early-hatched offspring outperform their later born or hatched brood or littermates. An early start gives them a competitive advantage, which can lead to faster growth, better condition, and increased chances of survival. Furthermore, in birds, egg content (e.g., resources such as amount of yolk or hormones [81]) often differs depending on the laying sequence, either as a consequence of changes in female resource availability, or as a result of female reproductive decisions, and this also affects offspring performance ([82]).

There is currently no evidence that extra-pair eggs differ from within-pair eggs in size or content, but in some species extra-pair offspring are indeed more common among early laid eggs or early-hatched offspring ([83–85], but see [86,87]), and controlling for this effect reduced the observed difference between extra-pair and within-pair offspring [83,85].

Box 2. The balance between oligospermy and pathological polyspermy: a set of hypotheses (H) and tests (T)

H1: sperm numbers on the perivitelline layer (PVL) increase with numbers of copulations
T1: quantify copulations and sperm numbers following [88,89]

H2: infertility rates decrease and rates of pathological polyspermy increase with the number of sperm on the PVL (Figure 1, main text)
T2: quantify these rates in relation to sperm numbers on the PVL following [36,89]

H3: females optimise sperm uptake to maximise hatching success
T3: test whether females on average reach their optimum indicated in Figure 1 (main text)

H4: infertility rates increase and rates of pathological polyspermy decrease when preventing sperm transfer during extra-pair copulations
T4: study reproduction in communal aviaries: enforce monogamy of one focal female by fitting all extra-pair males with 'condoms' (following [90]) and study effects on fertility

H5: female responsiveness to extra-pair males increases when rates of within-pair courtship or within-pair copulations or within-pair sperm transfer decreases
T5: sterilise males, put 'condoms' or chemically castrate males (antiaphrodisiac) and study effects on extra-pair responsiveness

H6: female responsiveness to extra-pair males increases when experiencing hatching failure
T6: manipulate hatching success and study extra-pair mating behaviour following [31].

render extra-pair mating maladaptive. Although mating with multiple males can insure against infertility and oligospermy (i.e., a low concentration of sperm) of the social partner [30], it could also increase the risk of embryonic death and, hence, reduced female fecundity through pathological polyspermy (i.e., fertilisation of an egg by more than one sperm) [34–37]. When the DNA of two sperm enters the nucleus of the egg simultaneously, a triploid embryo can result, which is normally either inviable [34,36] or sterile [38].

Extra-pair copulations might increase the risk of pathological polyspermy because: (i) these copulations are additional to within-pair copulations; (ii) extra-pair copulations often transfer greater numbers of sperm than within-pair copulations [39]; and (iii) the partner might respond flexibly to a threat of sperm competition by increasing copulation frequency and potentially ejaculate size [40,41]. However, whether extra-pair copulations increase the risk of polyspermy remains to be shown (Box 2) and will depend on patterns of sperm use and storage by the female.

If low sperm numbers increase the risk of eggs not being fertilised whereas high sperm numbers increase the risk of pathological polyspermy, then females are selected to take up, or store, intermediate numbers of sperm, and reject sperm if there is too much (Figure 1 [37,42]). In egg-laying species such as birds, where the cost of laying an infertile egg should be approximately equal to the cost of laying an egg where the embryo dies of triploidy, the female optimum is expected to lie where the greatest number of surviving embryos is produced (Figure 1). Sperm numbers lower than that should be optimal for female mammals, where the cost of aborting a triploid embryo might be (much) higher than the cost of an egg not getting fertilised. However, higher sperm numbers should be optimal for males [37,42,43], creating sexual conflict over optimal rates of infertility versus polyspermy. In polyandrous species, males are under selection to stack the odds of fertilisation in their favour by inseminating large numbers of sperm, even if this is partly deleterious to female fecundity [37,43]. Current knowledge about natural rates of oligospermy and

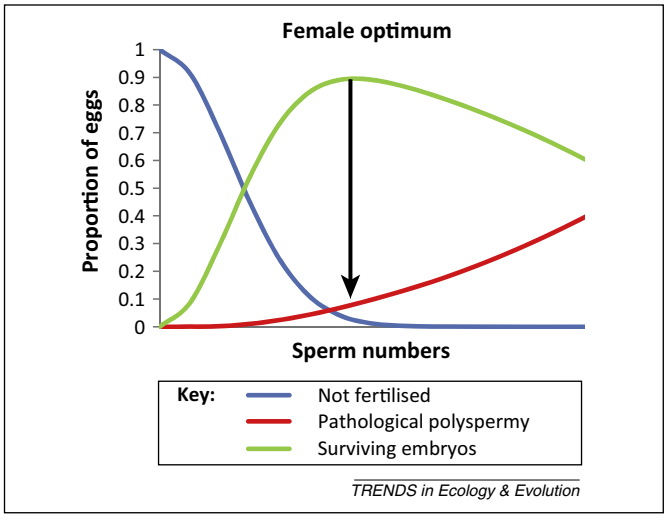

Figure 1. Hypothetical rates of infertility and polyspermy as a function of sperm numbers. The proportion of eggs that fail to be fertilised declines with sperm numbers inseminated, whereas the frequency of embryo death due to pathological polyspermy increases. The black arrow shows the optimal solution for the female (maximum of surviving embryos), if the costs of both causes of failure are the same to the female. Note that sperm competition among males will favour males that inseminate more sperm than is optimal for the female.

polyspermy is insufficient to judge whether beneficial or detrimental effects of multiple mating have the upper hand [37]. We suggest observational and experimental approaches to address this problem (Box 2).

A further issue surrounding sperm production illustrates the problem that maladaptive scenarios have received insufficient attention. In the germ line, the number of *de novo* mutations, the majority of which should be nonbeneficial, increases linearly with the number of cell divisions [44]. Therefore, old males and also males with high levels of sperm production are expected to produce sperm carrying more detrimental *de novo* mutations compared with males whose germ cells have gone through fewer mitotic divisions [19,20,44,45]. Given that successful extra-pair sires are typically older [18] and might also have higher levels of sperm production, engaging in extra-pair copulations might be detrimental for females in terms of inheriting 'bad genes' for their offspring. In other words, having a social partner with low levels of sperm production can carry a risk of some eggs not getting fertilised, yet the offspring of such males would inherit fewer *de novo* mutations because their germ cells go through fewer cell divisions. Further work is necessary to understand the likelihood and detriment to female fitness of either of these scenarios.

Extra-pair mating to avoid infanticide

In species where infanticide occurs, female extra-pair mating might have evolved to avoid infanticide by extra-pair males. Given the large direct benefits this entails, this adaptive explanation is rather uncontroversial for those systems where it applies.

In many species, offspring are vulnerable to infanticide by unrelated males. Such infanticide is adaptive for males if it increases their chances to mate with the mother of these offspring. This is often the case, because a female that loses her young will enter oestrus sooner (in mammals) or can lay a replacement clutch (e.g., in birds). Similarly, in group-living animals, offspring can be vulnerable to aggression from unrelated males, for example when competition for essential resources, such as food, shelter, or mates, is strong. Even if this does not lead to immediate death, it can negatively affect offspring fitness. In species where the risk of infanticide or aggression is high, female promiscuity might have evolved as an adaptive strategy to protect their offspring [46]. This hypothesis predicts that any male that has mated with a female will refrain from infanticide or aggression, because he might be the father of the offspring.

Although the hypothesis has not been tested directly, there is circumstantial evidence in support. For example, socially polyandrous female bank vole (*Myodes glareolus*) populations show higher recruitment compared with socially monandrous populations [47], presumably because of reduced infanticide in the former. A review of studies on mammals found that female promiscuity was more common in species in which infanticide occurred (e.g., 62% of 47 primate species) than in species where infanticide was unlikely (9% of 11 primate species) [46].

An interesting twist to the story is that dominant males might even 'encourage' female mating with subordinate males in the group, if those subordinates can help protect the offspring against aggression by immigrant males [48]. Incidentally, infanticide might also have led to the evolution of social monogamy in mammals, because biparental care reduces the period during which offspring are vulnerable to infanticide [49].

In birds, female extra-pair behaviour might also be associated with the risk of infanticide. Tree swallows (*Tachycineta bicolor*) are one of the most promiscuous birds: most broods contain extra-pair offspring and the young in a brood are often sired by several extra-pair males. Infanticide is not uncommon in tree swallows: if a new male takes over a nest after the female has started incubation, the male will wait until the eggs hatch and then remove the newly hatched offspring, one by one. Experimental work showed that if the new male arrived before the female had finished egg laying, he never committed infanticide, presumably because he copulated with the female [50]. Whether this explains the high level of extra-pair paternity is unknown, but it is plausible and would also explain why females accept copulations from many different males, including floaters [51].

The genetic constraint argument

We do not consider further the roles of male harassment and forced mating (Box 3), because our review focusses on explanations for active female involvement in extra-pair mating. Instead, we now turn to the idea that female extra-pair behaviour might be maladaptive. Are there species where females show active extra-pair mating behaviour although it is detrimental to them [9–11]? If so, how did such behaviour evolve?

Box 3. Convenience polyandry and harassment

The hypothesis of convenience polyandry [91] states that females might agree to mate with multiple males only to minimise the costs arising from male harassment. Although this cannot explain cases where females actively seek extra-pair copulations, convenience polyandry might be considered as another adaptive explanation for female extra-pair mating, because reduced resistance by females might minimise the costs of being harassed by males [5]. Although this hypothesis has received support in a range of studies on promiscuously mating insects [92], more empirical work is needed to determine whether it is applicable to systems with social monogamy and extra-pair mating, where there might be a capacity for individual recognition. If males can increase the efficiency of their pursuit of extra-pair paternity by strategically allocating their efforts to those females that have shown low levels of resistance during previous encounters, then females lowering their resistance might suffer an increase in the total amount of harassment experienced. Hence, future studies should test the extent to which males strategically invest extra-pair mating effort to different females.

In some species, such as those with intromittent copulatory organs [93], extra-pair paternity results from forced copulations. This is beyond the scope of this review, because there is no active female behaviour that requires further explanation [94]. However, in other species, it seems that females actively solicit courtship competition among males [5]. Although the resulting male behaviour resembles harassment, females might benefit by selecting the most persistent pursuer [95]. However, we note that any benefit of such behaviour in terms of producing more persistent sons is included in the fitness measures of some of the empirical studies testing 'genetic benefit models' mentioned above, although no study in such a system has measured differences in sexual behaviour among offspring.

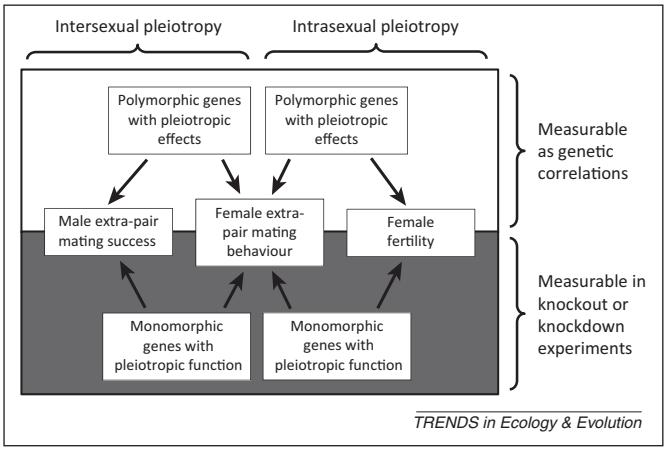

Figure 2. Pleiotropic gene effects and the existence of female extra-pair mating behaviour. Illustration of how the concepts of intra- and intersexual pleiotropy can explain the existence of female extra-pair mating behaviour as genetic corollaries of either male extra-pair mating success or female fertility.

Genetic constraint models for extra-pair mating propose that the alleles that cause maladaptive female promiscuity have additional pleiotropic effects that are beneficial and, hence, maintain the alleles in the population. To test this idea, we need to identify the beneficial side effects of such alleles. Note that this does not require knowing the specific alleles that affect female extra-pair mating. The genetic constraint of interest can be studied by estimating genetic correlations between female extra-pair mating propensity and other traits that we suspect to be affected by the same genes. In the following, we distinguish between two cases, depending on whether these other traits are expressed by the other sex (i.e., in males; intersexual pleiotropy) or by the same sex (i.e., in females; intrasexual pleiotropy).

Intersexual antagonistic pleiotropy

The hypothesis of 'intersexual antagonistic pleiotropy' refers to genes that have pleiotropic effects on the two sexes, such that they enhance fitness in one sex, while reducing it in the other. Here, the hypothesis argues that nonadaptive female extra-pair mating is caused by alleles under strong positive selection in males, because they enhance male extra-pair paternity gains (Figure 2). In other words, female and male promiscuity might be homologous traits that are influenced by the same set of genes, with alleles contributing to male extra-pair mating success and also facilitating female extra-pair behaviour (note that a similar argument has been made to explain female orgasm; Box 4).

When this hypothesis was proposed for multiple mating in general (rather than for extra-pair mating specifically) in a short commentary in 1987 [6], the hypothesis was rapidly criticised as unrealistic [8]. The main criticism was that female and male promiscuity are unlikely to be homologous traits, because the mating behaviours of the two sexes are often different. This might be valid for species where males and females indeed take different roles in mating and do not form pair bonds. However, in socially monogamous species, the behavioural repertoire of both sexes is often more similar. If pair bonding evolves *de novo*, it is likely to evolve simultaneously in the two sexes based on the same molecular mechanisms (but see, e.g., [52]). Any genetic mutation that strengthens or weakens the pair bond by modifying this molecular machinery shared between the sexes might then create similar effects in both sexes. Hence, in species where extra-pair mating is primarily a question of the strength of the pair bond, a positive genetic correlation between female and male extra-pair mating propensity might be expected.

The between-sex genetic correlation (r_{MF}) for female and male extra-pair mating has recently been estimated in a captive population of a pair-bonding species, the zebra finch (*Taeniopygia guttata*) [12]. The obtained value of r_{MF}=0.6 suggests that a substantial proportion of the additive genetic variance for male extra-pair mating success has pleiotropic effects on female extra-pair mating propensity, such that the latter could evolve largely as a by-product of strong selection on the former. Such a strong genetic correlation argues for between-sex pleiotropy, because linkage disequilibrium caused by assortative mating between promiscuous males and females should at best produce a weak positive correlation. This is because assortative mating is far from complete (due to the many within-pair young) and because the heritability of the level of extra-pair mating that leads to extra-pair paternity is small [12,53,54]. However, only a selection experiment that tries to decouple male from female promiscuity could fully reveal the degree of pleiotropy.

Strong positive estimates of r_{MF} might be the default, if the focal traits are homologous. A meta-analysis [55] found a mean r_{MF}=0.77 for behavioural traits, which is not different from that for morphological traits (r_{MF}=0.80). Somewhat lower genetic between-sex correlations might be expected for traits that are strongly sexually dimorphic (see Figure 4 in [55]). This calls for caution, because the propensity to engage in extra-pair mating is probably higher in males than in females of most species (e.g., [56,57]). This observation might also be interpreted as the result of past antagonistic selection on the expression

Box 4. The parallel debate about female orgasm

Following Lloyd's controversial book on female orgasm in humans [96], there has been a lively debate about whether female orgasm evolved as a by-product of strong selection on the male orgasm and ejaculation system [97] or serves adaptive functions of its own (e.g., pair-bond hypothesis or sire choice hypothesis, reviewed in [67]). A recent quantitative genetic study on male and female orgasmic function in humans found no significant between-sex genetic correlation [97–99], providing no support for the idea that female orgasm exists as an epiphenomenon of male orgasm.

However, the absence of a between-sex genetic correlation does not disprove the by-product hypothesis. It is possible that persistent selection on male orgasmic function keeps the genes involved in a monomorphic state (i.e., novel alleles associated with reduced orgasmic function are always driven to extinction). Given that only polymorphic loci contribute to genetic variance (and, hence, possible covariance between the sexes), the effect of these monomorphic genes cannot be quantified, although these genes still might be responsible for why females experience orgasms [67]. Only experiments where a certain gene is knocked out or its translation reduced by RNA interference might reveal such underlying pleiotropy (Figure 2, main text).

Equally problematically, if a positive genetic correlation between the sexes is found, this does not imply that the trait in one sex evolved only due to correlated selection on the trait in the other sex. Hence, female orgasm might or might not serve an adaptive function irrespective of its genetic architecture.

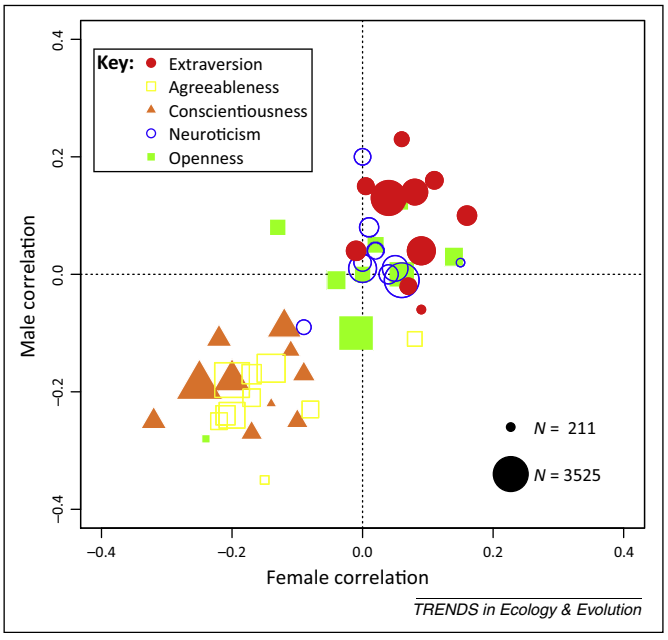

Figure 3. Phenotypic correlates of extra-pair mating behaviour in humans are shared between the sexes. The scatterplot shows male and female correlation coefficients between infidelity (lack of relation exclusivity) and the Big Five personality traits across ten different regions of the world: (1) North America (N = 3525); (2) South America (N = 622); (3) Western Europe (N = 2269); (4) Eastern Europe (N = 1923); (5) Southern Europe (N = 1074); (6) Middle East (N = 885); (7) Africa (N = 800); (8) Oceania (N = 804); (9) South and Southeast Asia (N = 211); and (10) East Asia (N = 1075). N represents the sum of male and female sample sizes. Data from [100].

of promiscuous behaviour in the two sexes, moving them further apart and bringing them closer to their sex-specific optima (monogamy for females and promiscuity for males).

Although r_{MF} has not been estimated for extra-pair mating propensity in humans, numerous studies have focussed on describing phenotypic correlates (e.g., personality traits) related to this propensity (Figure 3). It is noteworthy that these correlates go in the same direction for males and females, suggesting that much of this behavioural syndrome is shared between the sexes. It is also plausible that these correlated traits show positive between-sex genetic correlations, such that, for instance, risk-taking fathers will tend to sire risk-taking daughters.

A frequently used argument is that strong antagonistic selection will promote the evolution of sex-specific regulation of the underlying genes (leading to sexual dimorphism and possibly reducing r_{MF}). However, complex quantitative genetic traits such as personality are likely to depend on hundreds of genes [58], such that a complete sex-specific regulation of allelic effects at all these loci will be difficult, if not impossible, to achieve. The example of human infidelity (Figure 3) is particularly striking in that respect. A vast number of genes are likely to affect each personality component that will, in turn, influence the probability of engaging in extra-pair mating. However, once such a genetic correlation is in place, selection will favour females that 'make the best of a bad job', for instance by becoming more choosy and seeking good-gene benefits.

The largest handicap for measuring r_{MF} is the difficulty in obtaining good measures of an individual's propensity to engage in extra-pair mating. At the one extreme lie human questionnaire studies that seem capable of capturing this variation, as evidenced by high estimates of the heritability of this variation [59,60]. At the other extreme, field studies of birds that are limited to quantifying the realised amount of extra-pair paternity (rather than the underlying propensity, or engagement in extra-pair copulations, which in itself is rarely quantified) have found low heritability estimates [53,54]. This is expected, because paternity (from the female perspective) will additionally depend on the mating opportunities of a female, mate guarding by her partner, sperm competition and other postcopulatory processes. Hence, extra-pair paternity in a brood will only partly reflect the underlying propensity of a female to engage in extra-pair matings. Likewise, the extra-pair mating success of a male might strongly depend on the mating preferences of the available females and only to some extent on his extra-pair mating effort (his intrinsic propensity). Studies in captivity have the advantage that behavioural propensities such as the responsiveness of a female to extra-pair courtship or the extra-pair mating effort of a male can be measured directly. Higher heritabilities of these measures [12] will enable estimation of between-sex genetic correlations with smaller amounts of error.

Despite the difficulty of measuring r_{MF} in the wild, it might be worth testing whether male relatives (e.g., brothers) of females that have extra-pair offspring have a higher fitness through extra-pair paternity compared with male relatives of faithful females.

Intersexual antagonistic pleiotropy could be considered a form of indirect selection, where all the male carriers of an allele for promiscuity make up for the lower fitness of the female carriers. Note that this is different from indirect selection through 'sexy son' benefits [61,62], where the promiscuous behaviour of the female per se increases the attractiveness and, hence, fitness of her sons (leading to more grandchildren) via a paternal genetic effect. Under sexually antagonistic pleiotropy, when an evolutionary equilibrium is reached, the 'promiscuous son benefit' (adaptive promiscuous behaviour by males) will be compensated by a 'promiscuous daughter cost' (maladaptive promiscuous behaviour by females). Furthermore, the scenario allows for the female behaviour to be truly maladaptive (due to costs listed in Table 1 leading to fewer grandchildren).

Intrasexual antagonistic pleiotropy

The hypothesis of 'intrasexual antagonistic pleiotropy' argues that alleles for female extra-pair mating are maintained because these alleles have pleiotropic effects on female fecundity (Figure 2) or on female behaviours that are under positive selection, such as receptivity towards the social mate [4,12], the ability to divorce, or novelty-seeking behaviour [63].

A review on the costs and benefits of female extra-pair mating behaviour [4] suggested that alleles for female resistance towards extra-pair males do not spread in a population because these alleles also induce female resistance towards their partners, thereby leading to infertility and reduced fitness. This idea of a genetic correlation between female extra-pair and within-pair responsiveness has been tested and tentatively rejected for captive zebra

finches [12], yet the hypothesis deserves further examination, especially in species that form weaker and more ephemeral pair bonds.

Copulation frequency could evolve as a genetic corollary of female fecundity [64], because copulations might be proximately linked to stimulating reproductive processes [65,66]. Likewise, the degree of female sexual arousal might evolve with female fertility, if arousal serves the function of enhancing sperm uptake ([67], but see the issue of polyspermy discussed above). Under these scenarios, female extra-pair mating could evolve as a by-product of selection on fertility via an increased propensity to copulate.

Extra-pair mating behaviour might also result from a weakness of the social pair bond. Weaker pair bonds might evolve together with the ability to divorce, and adaptively partition reproductive investment among different partners. Elevated levels of extra-pair paternity in domesticated [12] as compared with wild-caught zebra finches [68] might be a correlate of such weakened pair bonds. Domestication might lead to weaker pair bonds because captive breeding often involves force pairing, divorce, and repairing. Such procedures might favour individuals that form only weak pair bonds and that quickly repair and reproduce after separation from the previous partner.

Finally, the incidence of extra-pair paternity has been correlated with novelty seeking and exploration behaviour [63,69], with bold and, hence, more risk-taking males more likely to sire extra-pair young. However, no such relation has so far been confirmed for females (but see Figure 3 and discussion above). Positive selection on risk-taking behaviour might enhance extra-pair mating propensities, but this would not provide a sufficient explanation for the phenomenon of extra-pair mating itself.

The female promiscuity 'syndrome': concluding remarks and future directions

Behavioural ecology provides a framework for putting adaptive and non-adaptive explanations through rigorous tests. This provides greater insight than the alternative of inventing adaptive stories to explain observations. Therefore, it is worrying that, to further our understanding of extra-pair behaviour, adaptive explanations have received a huge amount of attention (good genes, heterozygosity, or fertility insurance), whereas nonadaptive arguments (intersexual antagonistic pleiotropy, pathological polyspermy, *de novo* mutations, or sexually transmitted diseases) have been largely ignored. Potentially maladaptive behaviour deserves the special attention of behavioural ecologists.

Genetic constraint models require more emphasis on quantitative genetics [70], and a better understanding of possible constraints requires more proximate knowledge about the phenotype of interest. We argue that, to test the genetic constraint models, we need to study in greater detail the behavioural and physiological 'syndrome' of female promiscuity, especially in species that form short- or long-term monogamous pair-bonds. Genetic constraint hypotheses are still wide open for empirical testing. Quick progress can probably be made only in captive or seminatural settings, because it is easier to carry out detailed observations, standardised personality tests, and experimental manipulations.

Although studies of genetic correlations can check the plausibility of constraint arguments, long-term studies of fitness consequences from the wild are needed to assess whether and under what circumstances female extra-pair behaviour is adaptive or maladaptive.

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