

Male infertility, female fertility and extrapair copulations

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(Received 5 June 2008; revised 11 November 2008; accepted 19 November 2008)

ABSTRACT

Females that are socially bonded to a single male, either in a social monogamy or in a social polygyny, are often sexually polyandrous. Extrapair copulations (EPC) have often been suggested or rejected, on both empirical and theoretical grounds, as an important mechanism that enables females to avoid fertility risks in case their socially bonded male is infertile. Here, we explore this possibility in two steps. First, we present a mathematical model that assumes that females have no precopulatory information about male fertility, and shows that a female EPC strategy increases female reproductive success only if certain specific conditions are upheld in the nature of male infertility. In particular, these conditions require both (i) that fertile sperm precedence (FSP) is absent or incomplete within ejaculates of the same male (i.e. that an infertile male is, at least partly, truly infertile), and (ii) the existence of FSP among ejaculates of different males (such that infertile spermatozoa of the infertile male are at a disadvantage when competing against spermatozoa of a fertile male). Second, to evaluate their potential role in the evolution of female EPC, we review the abundance and FSP patterns of the different male infertility types. The conclusion is drawn that some common infertility types, such as poor sperm count or motility, contribute to the evolution of female EPC, whereas other common infertility types, such as sperm depletion or allocation in a social monogamy (but not in a social polygyny), and in particular male driven polyspermy, do not. Also, a deeper look at the arms race between sperm fertilization efficiency and female barriers to sperm may answer the non-trivial question: “why are some types of infertility so common?”

Key words: extra-pair copulations (EPC), infertility, direct benefits, arms race, oligospermia, polyspermy, fertile sperm precedence (FSP), sexual polyandry.

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I. INTRODUCTION

Males are notoriously known for being sexually polygynous, even in socially monogamous mating systems. The generally accepted explanation is that females are the males' limiting reproductive resource and therefore that male promiscuity often gains a significant advantage over a strict monogamy strategy by exploiting the reproductive efforts of more than one female (Trivers, 1972). However, a plethora of paternity studies in many socially monogamous species show that females are also unfaithful to their bonded mates (reviewed by Westneat & Stewart, 2003). This is what one should expect, at least in a "perfect" social monogamy, which we define as a 1:1 sex ratio, where all adult males are monogamously bonded to all adult females. In such a framework, any sexual conduct by a bonded male must be coupled with identical conduct by a bonded female. What do females gain by extrapair copulations (EPC) if, as a limiting reproductive resource, they cannot gain reproductive benefits? Or can they?

There are several answers to this question (Eberhard, 1996; Reynolds, 1996). Among answers that assume that females have a choice (i.e. are not coerced by males), two are the most common (Griffith, Owens & Thuman, 2002). First, that a female directly gains offspring by EPC if there is a fair chance that her bonded male is infertile (Wetton & Parkin, 1991; Krokene *et al.*, 1998; Fossøy, Johnsen & Lifjeld, 2006; Lifjeld *et al.*, 2007). Second, that EPC benefit females directly (more surviving offspring) or indirectly (improved offspring competitiveness, leading to a greater number of grand-offspring) because females are able to choose extrapair males whose genes are better than those of her bonded male (Jennions & Petrie, 2000).

Obviously, these two answers are not mutually exclusive. Furthermore, "infertility" and "bad genes" are not entirely two distinct causes of male incompetence as a sire, but are found along a continuum (Birkhead *et al.*, 2008). Variations in sperm "quality" may have a major impact on the sperm failure to reach eggs, to penetrate oocytes, to form viable zygotes (Sutovsky, Manandhar & Schatten, 1999; Rawe

et al., 2002), to contribute successfully to zygote survival at early or at late embryonic stages, to survival of the offspring's larval or young stages, and then to their success as competitive reproductive adults (Jennions & Petrie, 2000). Failure of sperm during the earlier stages following copulation is viewed as "male infertility", whereas sperm failure at later stages of reproduction is usually attributed to inheritance of "bad genes". The intermediate stage, early embryonic failure, starting with zygotic failure of first cell division, is often attributed to male infertility, particularly in human fertility studies (e.g. Krestner, 1997), although it clearly has genetic reasons. The fact that boundaries between "infertility" and "bad genes" are non-trivial is shown, for example, by Morrow, Arnqvist & Pitcher (2002), who describe as "infertility" any parental cause of embryonic death, including genetic causes such as the expression of recessive alleles. Furthermore, many, if not most causes of sperm failure, at any stage between copulation and birth, are heritable. In humans, semen samples that include sperm with poor DNA integrity, such as DNA breaks or fragmentations, often have abnormal motility, low concentration and abnormal morphology (Schlegel, 2006). Inbreeding in the rare scarce heath moth *Coenonympha bero* is the likely cause for both egg hatching failure and larval poor survival under stress (Cassel *et al.*, 2001). Hence, following copulation, "bad genes" can result in poor male performance as a sire at any stage of female reproduction, where many of the male-related failures are intercorrelated, and are likely to be a result of a shared causal factor (see Section IV).

For a clearer discussion, we make a distinction between two dichotomies that are commonly found in the literature, often interchangeably (e.g. Krokene *et al.*, 1998; Whitekiller *et al.*, 2000), about female EPC: (i) direct versus indirect benefits to the female, and (ii) male fertility versus good genes. These two dichotomies often overlap, but not always. Male infertility always entails direct costs to a no-EPC female. Bad genes, however, may entail either direct or indirect costs or both to female fertility. A male can inherit bad or incompatible genes that produce sick or too weak embryos (or "offspring" – where exactly does one put the

line?) to survive and/or reproduce, which “directly” reduces female reproductive success. This is sometimes attributed to male “infertility” (e.g. Morrow *et al.*, 2002) and sometimes to “bad genes” (Krokene *et al.*, 1998; Jennions & Petrie, 2000; Birkhead & Pizzari, 2002). At the other end of the spectrum are genetic causes of male sterility, which (i) cannot be inherited if a mutant of full sterility is coded by a dominant allele, or (ii) they can be intermittently inherited if they result in only partial sterility, or (iii) they may be partially inherited when coded by recessive alleles, or (iv) they may be inherited by females only if male infertility is produced by mtDNA mutations (see Section IV.2c). This review concerns infertility resulting from either heritable or environmental causes, or both. We generally do not address any indirect consequences of a cause of infertility on female reproduction. Correspondingly, we examine the evolution of female EPC as a consequence of male failure at the stages that begin with copulation attempts and end with the first zygotic cell division, i.e. of what is traditionally viewed as male “infertility”. This corresponds to a recent definition of fertilization by Birkhead *et al.* (2008, p. 508): “. . .the events that occur between penetration of the ovum by sperm and fusion of the male and female pronuclei (also referred to as syngamy).” We thereby exclude cases such as genetic incompatibility or homozygosity, but include cases such as centriole failure and polyspermy (the penetration of more than one spermatozoon into an egg, which may cause zygote mortality).

We first present a straightforward mathematical model that is based on direct payoffs of female sexual strategies, assuming variations in male fertility. This model shows that the fertility argument is not trivial. Indeed, the model was motivated by the confusing and sometimes inconsistent views found in the literature. For example, Lifjeld *et al.* (2007) make no assumption about a female’s ability to recognise male infertility, yet propose that: “If the frequency of male infertility is rather low in the population, say, 1%, a female mated to an infertile male can ensure egg fertility by mating with just one extra-pair male, as this male will be fertile with a probability of 0.99” (p. 268). However, if females have no information about male fertility [an implicit assumption throughout Lifjeld *et al.* (2007)], then for any female bonded to an infertile male and with a 99% chance of gaining fertility by EPC, there are 99 females bonded to fertile males that stand a 1% chance of losing fertility by EPC. Our first model (Case I) deals with a situation like this and concludes that, in the absence of biases towards fertile males or sperm, and regardless of the frequency or intensity of male infertility, on average females cannot gain a fertility advantage by EPC (see also Arnqvist & Kirkpatrick, 2005).

We also develop the model (Case II) to allow for biases that are potentially inherent in male infertility. We find that Lifjeld *et al.*’s (2007) proposition can be true, even in the absence of information about male fertility, but only if certain assumptions hold regarding the actual nature of male infertility, hence also of sperm competition. The model is relevant to the understanding of female EPC since it shows that only particular kinds of male infertility make female sexual polyandry an evolutionarily stable strategy (ESS). The model is based on the assumptions of a socially

monogamous species with random precopulatory female choice with respect to male fertility. As the contribution of male infertility types to the evolution of female EPC is found to be a function of both their abundance and the biases they inherently create in sperm competition, we follow the model below with a review of different male infertility types in birds and mammals, emphasising their relevance to these two factors. We conclude that some types of male infertility can play an important role in the evolution of female EPC, whereas others may be irrelevant. Finally, we use both the model’s conclusions and the review to examine other potential biases in female EPC that may give females fertility advantages, and to understand why male-related infertility is so common.

II. MODELS OF MALE INFERTILITY

Our model assumes that each female is socially bonded to a single male. For simplicity we model a socially monogamous mating system, although the model’s conclusions hold equally for social polygyny. This is because the model makes no distinction between a social monogamy, where a female is the only female who is socially bonded to the male, and social polygyny, where at least one other female is socially bonded to the same male. In both cases each female is socially bonded to a single male who can make a phenotypic contribution to her offspring, and the female is capable of making sexual choices. The model also makes an important assumption that during precopulatory choice, females cannot perceive differences in male fertility (but see Section V), and that, therefore, with respect to fertility, a female can either randomly select an extrapair sexual partner, or mate exclusively with her bonded male.

The model is based on the following four parameters: (A) Female potential fertility, M , defined as the per female number of eggs that are ready to be fertilized. (B) The proportion t of fertile males in the male population ($0 \leq t \leq 1$). (C) The intensity μ of female involvement in extrapair copulations ($0 \leq \mu \leq 1$). (D) The male fertility f ($0 \leq f \leq 1$), representing the male’s ability to use all of the female’s reproductive potential when the male is a single inseminator.

More specifically, the model assumes two types of males: “fertile” males are represented by $f = 1$, and “infertile” males who can be partially infertile ($0 < f < 1$) or fully infertile ($f = 0$). The parameter μ should be regarded as the proportion of extrapair sperm in the total sperm found in the female reproductive tract at the time of egg fertilization, and represents the component of female choice in EPC. Females control μ first by making a discrete number of extrapair copulations. They can then fine-tune the effective proportions of extrapair sperm by increasing the total number of copulations, by selecting the order and the exact timing of copulations with each male prior to egg maturation, by controlling the duration of each copulation, and by selectively expelling different proportions of sperm received from the two males (Parker, 1990a, b; Wetton & Parkin, 1991; Colegrave, Birkhead & Lessells, 1995).

Separating competing ejaculates into different storage compartments can give females even greater control over μ (Bakst, 1998; Hellriegel & Ward, 1998). We can therefore treat μ as a continuous variable, and define $\mu = 0$ for no EPC and $\mu = 1$ when all sperm in the female tract is extrapair. We therefore make a clear distinction between female mating decisions, μ , and male fertility, f .

(1) Case I: infertile and fertile sperm are equally competitive

(a) Cost-free EPC

The parameter f may also be viewed as the chance of an egg to be fertilized by a male’s ejaculate when the male is a single inseminator. In Case I of the model, we assume that the proportions of in-pair and extra-pair spermatozoa are controlled fully by the female (via μ), and that fertile and infertile spermatozoa are equally competitive and have an equal chance to access and control eggs in a fair raffle sperm competition (Parker, 1990a). Consequently, semen or sperm of an infertile male can prevent fertile spermatozoa, including those within his own ejaculate if there are any, from fertilizing a female’s eggs. That fertile sperm has no precedence is an important assumption in the model, and we leave its biological interpretation for later. It is represented formally in the model as follows: if a female copulates with a single male, she will have Mf fertilized eggs. Given that some males are infertile, a female bonds with a fertile male ($f = 1$) with a probability t , and with an infertile male with a probability $1 - t$. Consequently, the payoff F_N for a female who copulates exclusively with her bonded male (“No EPC”) is, on average,

$$F_N = M[(1 - t)f + t]. \tag{1}$$

Male infertility reduces the female’s total productivity, and females who copulate exclusively with their bonded male lose, on average, $M - M[(1 - t)f + t] = M(1 - t)(1 - f)$ eggs. This apparent loss leads to the intuitive conclusion that such females may cut some of their losses by copulating with other males. In order to see if this argument is valid, we now compare payoffs of the faithful female (F_N) with those of females who practice EPC (F_{EPC}).

For simplicity, it is assumed that when a female uses the EPC strategy, she copulates with a single extrapair sexual male partner from whom she receives a proportion μ of her total ejaculates. The remainder, $1 - \mu$, is received from her bonded male. Given the assumption that females cannot detect male fertility, a female selects an infertile extrapair sexual male partner with a probability of $1 - t$, and a fertile one with a probability of t . Therefore, the average fitness of a female who uses the EPC strategy is given by:

$$F_{EPC} = M[(1 - \mu)(1 - t)f + (1 - \mu)t + \mu(1 - t)f + \mu t], \tag{2}$$

$$= M[(1 - t)f + t] = F_N.$$

The first two expressions within the square brackets represent the average fertile share of the bonded male, and the last two, the average fertile share of the extrapair male.

As shown, Equation 2 collapses to Equation 1, and therefore, $F_{EPC} = F_N$.

Perhaps better biological intuition is gained through the following cost-benefit considerations which will be helpful again shortly. As shown above, it is seen from Equation 1 that due to male infertility, a female who exclusively copulates with her bonded male loses, on average, $M(1 - t)(1 - f)$ eggs. By engaging in EPC there is a probability t (if the extrapair male is fertile) that she saves a proportion μ of this loss. She thus gains, on average, $G = M(1 - t)(1 - f)t\mu$. However, if her bonded male is fertile (with a probability t), by engaging in EPC she therefore risks, with a probability $1 - t$ (that the extrapair male is infertile), losing a proportion $\mu(1 - f)$ of her M eggs. Her average loss is $L = Mt(1 - t)\mu(1 - f)$. Hence, the change in fitness, Δ , or the net balance of benefits minus costs from EPC, is given by

$$\Delta = G - L = M(1 - t)(1 - f)t\mu - Mt(1 - t)\mu(1 - f) = 0 \tag{3}$$

and

$$F_{EPC} = F_N + \Delta = F_N. \tag{4}$$

Therefore, on average, and given the assumption that females cannot detect differences in male fertility, a female replaces by EPC the same number of infertile eggs with fertile eggs, when her bonded male is infertile, as she does fertile eggs with infertile eggs if her bonded male is fertile. Metaphorically speaking, by engaging in EPC a female draws new cards at random, using them to replace randomly cards in her hand, which themselves were also picked at random. On the average, it does not matter. The frequency of fertility (t), the intensity of infertility (f) or the intensity of involvement in EPC (μ) have no effect on the female extrapair sexual strategy.

(b) EPC entails cost

The model thus far assumes no costs to the female as a result of her EPC activity. EPC could be costly, however, if, for example, it lowers her fertility from M to M' , where $M' < M$. This may be a result of (i) energetic costs involved with the search for an extrapair male, (ii) decreased paternal efforts by cuckolded bonded males, (iii) infection by social diseases, or (iv) missed copulation opportunities with the bonded male. Although the change in M has no effect on Δ in Equation 3 (which remains equal to zero), it affects F_{EPC} . A faithful female’s payoffs remain $F_N = M[(1 - t)f + t]$ (Equation 1), whereas payoffs for a female who uses a costly EPC strategy become $F'_{EPC} = M'[(1 - t)f + t] < F_N$. This makes EPC an inferior strategy rather than a neutral one.

(2) Case II: fertile sperm precedence (FSP)

The Case I model assumes, like Arnqvist & Kirkpatrick (2005), that fertile and infertile sperm have equal opportunities to access female eggs. Hence, the sperm fertility coefficient f results in a proportion of f fertilized eggs of the $(1 - \mu)M$ “share” of the bonded infertile male, and f fertilized eggs of the μM “share” of the extrapair infertile

male. (A male’s “share” is shorthand for the expected proportion of eggs controlled by a male’s sperm, assuming a fair raffle sperm competition.) This calculation makes an important implicit assumption: fertile sperm does not take the place of infertile sperm in the race to the eggs. This assumption, of no fertile sperm precedence (no FSP), critically affects the model’s conclusions. We now violate it and assume, instead, that fertile sperm is more mobile and, when competing with sperm of an infertile male, gains access to a greater number of eggs than the fertile male’s share. For now, we treat both options, FSP and no FSP, as purely theoretical options, and leave the consideration of the actual nature of sperm infertility and sperm competition to Section III.

(a) Cost-free EPC

To include FSP we now assume that infertile sperm loses access to some of its share of eggs due to sluggishness or to other traits that hinder sperm competitiveness when pitted against more fertile sperm. Therefore, when a fertile bonded male competes against an infertile extrapair male, he fertilizes all of his own share of eggs, $(1 - \mu)M$, plus $D_B\mu M$ eggs taken from the extrapair male’s share (see Table 1). Here D_B is the proportion of the infertile extrapair male’s share of eggs which are fertilized by the bonded male due to FSP. Similarly, when a fertile extrapair male competes against a bonded infertile male, he fertilizes his own share, μM eggs, plus $D_{EP}(1 - \mu)M$ eggs taken from the bonded male’s share. Likewise, D_{EP} is the proportion of an infertile bonded male’s share of eggs, $(1 - \mu)M$, actually fertilized by a fertile extrapair male as a consequence of FSP (see Table 1).

It now remains to specify D_B and D_{EP} . To do so, we formalise the action of FSP between ejaculates of two males by introducing a sperm lethargy coefficient, l . Let $0 \leq l \leq 1$ be the potential proportion of an infertile male’s share of eggs lost to its fertile male rival in sperm competition, due to its sperm’s lethargy. We determine $l > 0$ for an infertile male whose sperm competes against sperm of a fertile male, and $l = 0$ (no eggs transferred to a rival male) otherwise. However, the infertile male’s loss, l , is likely not to be a fixed value, but frequency dependent. If sperm of the fertile male is only available in small quantities, it will not be able to exploit fully the infertile sperm’s lethargy. Hence, l does not necessarily measure actual gain of the fertile male, only its potential. Actual gains of a fertile male competitor depend, therefore, on the proportion of his ejaculates in the total pool of ejaculates in the female reproductive tract: $(1 - \mu)$ if

he is the bonded male; μ if he is the extrapair male. We therefore denote D_B and D_{EP} as actual gains by the fertile male, and assume, for simplicity, they are linear functions of μ . Let $D_B = l(1 - \mu)$ be the proportion of the infertile extrapair male’s share of eggs, μM , actually monopolized (i.e. fertilized) by a fertile bonded male. Similarly, we let $D_{EP} = l\mu$ be the proportion of an infertile bonded male’s share of eggs, $(1 - \mu)M$, actually fertilized by a fertile extrapair male.

If a female exclusively copulates with her bonded male (no EPC), her productivity is still represented by Equation 1. However, given the current model’s assumptions, when she also copulates with an extrapair male, she cannot lose and has the potential to win: if both males are either equally fertile or equally infertile (i.e. when $l = 0$), her gains and losses by EPC are identical, as before. Unlike the Case I model above (which assumes no FSP), however, if her bonded male is infertile and her extrapair male fertile, then EPC and sperm infertility and lethargy combined increase her gains by $M(1 - \mu)(1 - f)D_{EP}$ fertilized eggs. If her bonded male is fertile and her extrapair male infertile, EPC with sperm infertility and lethargy combined cut her losses by $M\mu(1 - f)D_B$ eggs. Hence, Equation 4 is replaced by:

$$F_{EPC} = F_N + \Delta' \tag{5}$$

$$\begin{aligned} \text{where } \Delta' &= M(1 - t)t(1 - \mu)(1 - f)D_{EP} \\ &+ Mt(1 - t)\mu(1 - f)D_B \\ &= 2Mt(1 - t)\mu(1 - \mu)(1 - f)l. \end{aligned} \tag{6}$$

$$\text{Thus } F_{EPC} = F_N + 2Mt(1 - t)\mu(1 - \mu)(1 - f)l > F_N \tag{7}$$

for any $l > 0, f < 1, 0 < \mu, t < 1$.

We can now conclude that, unlike Case I, sperm infertility that gives precedence to fertile sperm increases average productivity of females engaged in EPC (F_{EPC}). Since $F_{EPC} > F_N$, then the EPC strategy is an ESS. Under the present assumptions, Equations 5–7 show that F_{EPC} is a linear (positive) function of the fertile sperm’s lethargy, l , and of male infertility, $1 - f$, and peaks at $\mu = 0.5, t = 0.5, f = 0, l = 1$ (where infertility is most severe, and fertile sperm has exclusive access to the female’s eggs). In fact, at these most optimal conditions for EPC, and given that D_B and D_{EP} are linear functions of μ , we obtain $F_N = 0.5M$, and $F_{EPC} = 0.625M$ (i.e. a net gain of 25%).

Table 1. The contribution of the bonded and extrapair males to egg fertilization when one of them is fertile and the other is infertile

No. of eggs fertilized:	by the bonded male	by the extrapair male
<u>When:</u> bonded male is fertile and extrapair male is infertile	$(1 - \mu)M + D_B\mu M$	$\mu M - D_B\mu M$
bonded male is infertile and extrapair male is fertile	$(1 - \mu)M - D_{EP}(1 - \mu)M$	$\mu M + D_{EP}(1 - \mu)M$

μ , intensity of female involvement in extrapair copulations; M , female potential fertility; D_B , proportion of infertile extrapair male’s share of eggs fertilized by bonded male due to fertile sperm precedence (FSP); D_{EP} , proportion of an infertile bonded male’s share of eggs fertilized by fertile extrapair male due to FSP.

This model shows that the evolution of female EPC requires no variation in male genetic quality, nor the existence of female perception of differences in male fertility. Such female perception, if it exists, is expected to change the optimal female strategy, μ , and to increase F_{EPC} due to a greater precision of the use of EPC (see Section V).

(b) *EPC entails cost*

As before, we introduce cost by assuming that EPC lower female fertility, M . Let $M' = (1-\varepsilon)M$, where $0 \leq \varepsilon \leq 1$, and where $\varepsilon = 0$ represents no cost, and $\varepsilon = 1$ represents full reproductive cost. Equation 5 is now replaced by:

$$F'_{\text{EPC}} = (1 - \varepsilon)(F_{\text{N}} + \Delta'), \quad (8)$$

where, as before, F'_{EPC} represents female payoffs for a female performing EPC when costs are included, and Δ' is taken from Equation 6. Given that it is costly, EPC can nevertheless be an ESS, when $F'_{\text{EPC}} > F_{\text{N}}$, which gives:

$$(1 - \varepsilon)(F_{\text{N}} + \Delta') > F_{\text{N}} \quad (9)$$

or

$$\varepsilon < \hat{\varepsilon} = \frac{\Delta'}{F_{\text{N}} + \Delta'} = \frac{2t(1-t)\mu(1-\mu)(1-f)l}{(1-t)f + t + 2t(1-t)\mu(1-\mu)(1-f)l}, \quad (10)$$

where $\hat{\varepsilon}$ is the maximum cost allowed for the evolution of EPC, i.e. the female cost at the point where $F'_{\text{EPC}} = F_{\text{N}}$. For any $\varepsilon < \hat{\varepsilon}$, EPC is an ESS. Note that $\hat{\varepsilon}$ also represents the net female gain by EPC relative to her total fitness. Fig. 1 is derived from Equation 10 and shows the effect of t and f

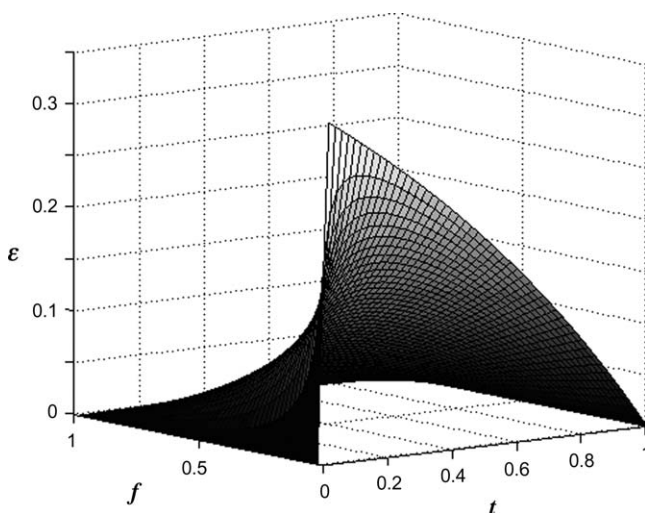


Fig. 1. Female extrapair copulations evolve when its cost ε (as a proportion of total reproduction M) is below the value $\hat{\varepsilon}$ indicated by the surface. $\hat{\varepsilon}$ is computed by using Equation 10 to show the effect of male fertility, f , and of its frequency, t at optimal conditions for EPC, i.e. when the intensity of female involvement in extrapair copulations $\mu = 0.5$, and the sperm lethargy coefficient $l = 1$.

on $\hat{\varepsilon}$. Fig. 2 shows that $\hat{\varepsilon}$ monotonically decreases when l , sperm lethargy, decreases, such that when $l = 0$, $\hat{\varepsilon} = 0$. $l = 0$ brings us back to the first model (Case I), and to the conclusion that without FSP, females cannot gain by EPC.

Our model uses a conservative approach, and assumes that females have no information about male fertility. This assumption has an immediate consequence: female direct (reproductive) gain by EPC with FSP, Δ' , becomes limited. This is largely due to the fact that Δ' is frequency dependent jointly on μ , $(1-\mu)$, t and $(1-t)$ (see Equation 6), and thus has a maximum of $\Delta' = M/8$, at $t = 0.5$, $\mu = 0.5$, $f = 0$, and $l = 1$. However, given that payoffs of a faithful female, F_{N} , get smaller as t gets smaller (chances that the bonded male is fertile are smaller, Equation 1), the relative fertility benefit to a female performing EPC (or maximal cost allowed, $\hat{\varepsilon}$) where FSP exists, tends to a maximum ($1/3$) when t tends to 0 (Fig. 1).

Note that the treatment of the optimal proportion of extrapair sperm, represented by μ , is simplified. Using the model's assumptions, an optimum at $\mu = 0.5$ is a result of females minimising fertility risks when copulating with two males in a system that lacks precopulatory information about fertility. This may change when the frequency dependence in displacement of lethargic sperm with fully fertile sperm is non-linear, when costs of EPC are a function of μ rather than fixed, or when females copulate with more than one extrapair male. The latter possibility is particularly interesting: FSP creates positive but diminishing fertility returns for EPC with each additional male. This indicates benefits to performing each extrapair copulation with a different male, as well as benefits to doing this with many males. The number of extrapair males eventually should be determined by the combination of diminishing fertility

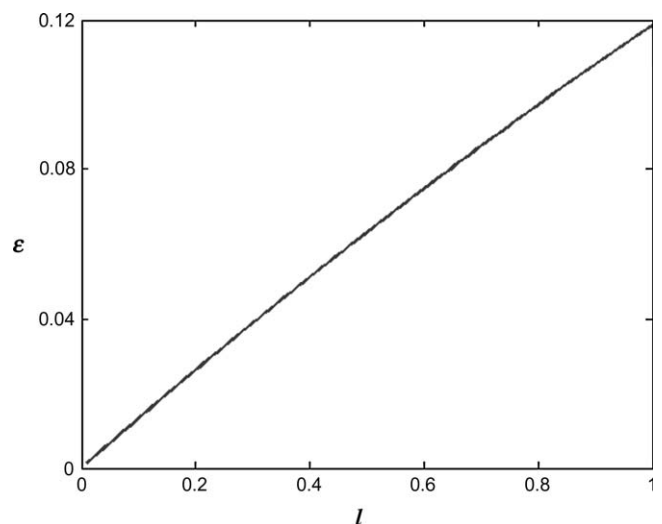


Fig. 2. Female extrapair copulations (EPC) evolve when its cost ε (as a proportion of total reproduction M) is below the value $\hat{\varepsilon}$ indicated by the line. $\hat{\varepsilon}$ is computed by using Equation 10 (where the intensity of female involvement in EPC is $\mu = 0.5$, the frequency of male infertility $t = 0.5$, and male fertility $f = 0.3$) and increases monotonically but not linearly with the sperm lethargy coefficient l .

returns for additional EPC, with non-diminishing costs. Such costs are a result of time and energy that may be required to find each additional extrapair male, and when each additional male increases risk of infection by sexually transmitted diseases, or risk of being exposed and penalised by the bonded male. Consequently, an optimal proportion of extrapair copulations should become separate from an optimal number of sexual partners, and females may repeatedly copulate with the same extrapair males. This may result in an apparent precopulatory preference, even when “choice” becomes purely a matter of convenience (i.e. of avoiding costs). Finally, the optimal value of μ should also change when females are capable of precopulatory choice based on male fertility, depending on certainty assigned to signals and cues (*sensu* Hasson, 1994) of fertility of the competing males.

III. FSP PATTERNS AND FEMALE FERTILITY

The proposition that when some males are infertile, females can increase their own fertility by EPC, even in the absence of precopulatory choices based on male infertility, is both common (Wetton & Parkin, 1991; Gray, 1997; Lifjeld *et al.*, 2007), and easily dismissed (Jennions & Petrie, 2000, Arnqvist & Kirkpatrick, 2005; 2007). The model presented here shows how important for this proposition is the assumption that sperm of fertile males has precedence to eggs over sperm of infertile males. It is therefore surprising how little effort has been made to study the effect of different sperm pathologies and other causes of male infertility on the potential direct female benefits by EPC. Clearly, as emphasised by Eberhard (1996), research into female EPC is strongly biased towards male strategies in sperm competition and underestimates female strategies (e.g. Wedell, Gage & Parker, 2002; Snook, 2005; Arnqvist & Kirkpatrick, 2005). Consequently, studies of sperm precedence typically compare fertilization success of healthy males based on the order in which they copulate with a female, rather than on the basis of variations in their fertility (but see García-González, 2004).

Previous suggestions that EPC help to ensure females' fertility have not been explicit about assumptions of sperm precedence to eggs, probably due to the absence of an adequate theoretical framework. Confusion may arise by the fact that FSP has two opposing effects on the female's direct benefits from EPC, one operating within males, another operating between competing males.

(1) FSP within males

Male fertility, f , is defined in the model as the male efficiency of using the full reproductive potential of a fertile female, when he is a sole inseminator. Hypothetically, however, an ejaculate with even one fertile sperm per egg with full precedence over infertile spermatozoa of the same male, may be sufficient to fertilize all of the female's eggs.

Given that males produce a vast number of spermatozoa, intra-male FSP could lead to complete fertilization.

Male infertility ($f < 1$), which is required for the evolution of female EPC based on male variations in fertility, is therefore generated by either of the following two cases: (i) no fertile spermatozoa are transferred to the female, (ii) full intra-male FSP does not exist (i.e. infertile sperm, both within and between ejaculates of the same male, reach and control some of the female eggs). Either of these two options describes the inability of fertile spermatozoa of a single male to reach and fertilize all of the female eggs if he is the sole inseminator, or all of his share of eggs, if the female copulates with more than one male. Either way, some eggs of the infertile male's “share” remain unfertilized.

(2) FSP between males

The model shows that when this happens, females are able, on average, to fertilize more eggs by EPC, making EPC an ESS. However, this is only true if the male infertility is of a type that also results in inter-male FSP (precedence to fertile sperm between ejaculates of competing males). Sperm lethargy of infertile sperm, $l > 0$, represents this bias towards spermatozoa of a fertile male. Infertile sperm of a bonded male can then be replaced by fertile sperm of an extrapair male [in the proportion of D_{EP} of the $(1 - \mu)M$ infertile bonded male's sperm], while risks of replacing fertile sperm of a bonded male with infertile sperm of an extrapair male are reduced (because a proportion D_B of the μM infertile extrapair male's share of eggs is actually fertilized by the fertile bonded male's sperm).

IV. MALE INFERTILITY TYPES AND FSP PATTERNS

Our model shows that in the absence of precopulatory biases toward fertile males, both lack of intra-male FSP and the existence of inter-male FSP are prerequisites for the evolution of female EPC based on variations in male fertility. However, it is not intuitively obvious that male infertility can simultaneously induce both effects. Therefore, to appreciate the possible role of male infertility in the evolution of female EPC under the assumption of no a priori female recognition of male infertility, we review the occurrence of relevant male sexual dysfunctions and sperm pathologies at the stages of sexual reproduction where they are traditionally viewed as “infertility” problems. Specifically, we are interested in their effect on intra- and inter-male FSP. Any male-related failure in performance that occurs only later in the offspring life, starting at the early embryonic stage, is made after eggs are fertilized, and is attributed to “bad genes”. Our search for FSP patterns in the different male sexual dysfunctions, and for a general understanding of why some of them remain at relatively high levels begins, therefore, with male failure to

inseminate, and ends in egg penetration by sperm that fails to form viable zygotes.

(1) First step: insemination and sperm rejection

(a) Insemination failure and azoospermia

Azoospermia is a complete lack of sperm in ejaculates. Insemination failure relates here to copulation attempts that fail to transfer sperm to the female reproductive organs as a result of male sexual dysfunctions, such as mechanical infertility (García-González, 2004). Males who fail to inseminate may nevertheless have fully functional spermatozoa, although the effect of azoospermia and insemination failure is the same – spermatozoa are not delivered to females. If this occurs consistently for males long enough to reduce female productivity (i.e. when multiple copulation attempts with the same male are futile), both dysfunctions clearly show the combination of no intra-male FSP (because infertile males cannot provide any fertile sperm) and inter-male FSP (successful inseminations by sexually functioning males take over the infertile males' "share of eggs").

In humans, azoospermia is often the result of deletions in the Y chromosome (Krestner, 1997). Such deletions, which commonly affect spermatogenesis, can persist at low frequencies, depending on the severity of their effect, through a balance between recurrent mutation and haploid selection (Repping *et al.*, 2003). For birds, in which males are homogametic (ZZ), Lifjeld *et al.* (2007) found azoospermia in one out of 48 males in the bluethroat *Luscinia svecica*, and in two out of 50 males in the willow warbler *Phylloscopus trochilus*.

(b) Sperm depletion and sperm allocation

Recent copulations by a male, as well as differential sperm allocation to different females, can lead to poor sperm counts in ejaculates, to the point where they may reduce female fertility (reviewed in Wedell *et al.*, 2002). There is conflicting evidence and a paucity of data on the role sperm depletion plays in male infertility and sperm competition in monogamous birds, and on its effect on the proportion of extrapair offspring (Cordero, Wetton & Parkin, 1999). However, in a strict sexual monogamy, where copulation rates and sperm production tightly co-evolve, sperm depletion of a healthy bonded male should probably have only a minor effect, if any, on female fertility, relative to its role in other mating systems. Hence, although sperm depletion and allocation may contribute to the evolution of female sexual polyandry in general (Wedell *et al.*, 2002), this may not be the case in a social monogamy (Krokene *et al.*, 1998). On a regular basis, sperm depletion of a healthy male can reduce his bonded female's fertility only when extrapair copulations are common in the first place, and the male is popular (e.g. Gray, 1997; Cornwallis & Birkhead, 2007; Brouwman *et al.*, 2007). Therefore, sperm depletion and sperm allocation are not likely to be important in the initiation of the evolution of female EPC in a social monogamy,

although they may contribute to strengthening it once it is there. They may, however, contribute to EPC of bonded females in a social polygyny (e.g. Gray, 1997).

(c) Sperm rejection

Sperm may fail to remain in the female reproductive tract as a result of physical rejection executed either by the female, or by competing males. Cryptic female choice is a term describing those mechanical, physiological and chemical processes within the female that lead to rejection of sperm of certain males, often unattractive, by isolating their sperm (limiting its access to eggs), by handicapping its activity or by expelling it (Baker & Bellis, 1993; Eberhard, 1996), effectively reducing its numbers. Pizzari & Birkhead (2000) found that female feral fowl *Gallus gallus domesticus* actively eject sperm of subdominant males. However, unless we also find tight relationships between attractiveness and fertility (see Pizzari, Jensen & Cornwallis, 2003), we cannot conclude that sperm ejection is based on sperm infertility *per se*. Physiological mechanisms that maintain sperm in birds' storage tubules (Stepinska & Bakst, 2006), hint that poor sperm motility, which is often associated with infertility, increase sperm susceptibility to active ejection by the female. This, however, needs further confirmation. If infertile, slow sperm is ejected more frequently, then both conditions for the evolution of female EPC are satisfied (insufficient intra-male FSP, which leads to infertility, and inter-male FSP, due to selective ejection of infertile sperm). For now, we very cautiously conclude, by default, that sperm ejection by females has no effect on either form of FSP. Similarly, we know of no indication that active sperm replacement by competing males is based on differences in sperm fertility. Consequently, removal of previous sperm by competing males seems to be also irrelevant to FSP, and gives no fertility benefits to females.

(2) Second step: racing to the eggs

(a) Poor sperm count (oligospermia)

Variations in sperm count in ejaculates reduce some of the female control over μ , the proportion of sperm of an extrapair male. Nevertheless, when females have no information about male fertility, variations in sperm count create a symmetrical distribution of μ around a certain mean that is under female control. This alone should not have a major impact on female EPC. However, poor sperm counts can have an impact, simply because they constitute a major cause of male infertility.

Theoretically, as long as sperm count does not go down to nil, an ejaculate with a low sperm count may still provide a female with sufficient spermatozoa to fertilize all of her eggs. Indeed, ever since Parker (1982), and with a few exceptions (e.g. Birkhead Møller & Sutherland, 1993), the outstanding discrepancy between the number of oocytes available for fertilization and sperm number in an ejaculate is attributed to sperm competition, not to male infertility (Mesterton-Gibbons, 1999; Greeff & Parker, 2000). Hence, although males with high sperm counts gain reproduction

benefits over males with poor sperm counts, *via* fair raffle sperm competition, there should be no FSP of any kind as long as males, even with a poor sperm count, are fully fertile when inseminating a female alone. Hence, theoretically, variations in male sperm count should give no advantage to a female EPC strategy.

Nevertheless, data show an apparent paradox: sperm count matters. Using intrauterine inseminations (IUI), van Voorhis *et al.* (2001) found a fertility threshold in human males as high as 10 million total motile sperm count (TMSC) per ejaculate. Furthermore, human sperm counts smaller than 20 million ml⁻¹ already show low conception rate, and are categorised as oligospermia (Iammarrone *et al.*, 2003). In humans (van Voorhis *et al.*, 2001), red deer *Cervus elaphus* (Malo *et al.*, 2005) and domestic pigs *Sus scrofa* (Martinez *et al.*, 2002), egg fertilization using artificial inseminations was found to be dependent on TMSC. For the pig, a TMSC lower than 5 million significantly decreased egg fertilization despite the use of deep intrauterine insemination, which artificially increases the number of spermatozoa close to the eggs. Accordingly, oligospermia does not describe the case where there are only a few spermatozoa in an ejaculate, but the case where there are not enough spermatozoa in an ejaculate to fertilize efficiently all oocytes available.

Hence, despite a plethora of research supporting the common sense understanding that high sperm numbers evolve because they increase male success in sperm competition, sperm competition seems to be but part of the story. First, because the very large numbers of spermatozoa required for efficient fertilization of female oocytes cannot be explained by sperm competition alone. Even Parker (1982), in his seminal paper on the role of sperm competition in the evolution of many tiny spermatozoa, saw the importance of the small chance for a single spermatozoon to find an egg in the female tract, in species with internal fertilization [although Parker (1982) does not explain why female tract are so long in the first place]. Second, although a high sperm count improves male chances in sperm competition, female EPC cannot evolve based on inter-male variations in sperm numbers alone.

This seeming paradox, that sperm count matters, is, to our opinion, an important factor in understanding the evolution and maintenance of male infertility. The mere fact that sperm count matters in female fertility creates, by definition, male infertility that is based on too small a number of fertile, healthy spermatozoa, and gives room for inter-male FSP. Hence, it promotes female EPC. However, this cannot explain the strong impact of oligospermia, nor does it explain why is it so common. In order to reach a better understanding of these questions, we return to oligospermia below (Section IV-3*b*) in the discussion of its antagonistic type of male infertility, polyspermy.

In part, the paradox that sperm count matters may be explained by correlations that are commonly found between poor sperm count and other kinds of sperm dysfunction. Ruiz-Pesini *et al.* (2000*a*) discovered, in humans, a positive correlation between low sperm count and failure of mitochondrial enzyme activities that result mostly from nuclear deleterious mutations. Nakada *et al.* (2006) found in

transmitochondrial mice (mito-mice), engineered for different proportions of mutations in their mtDNA (4,696-bp deletion), that mitochondria energise male spermatogenesis in the male testis. High proportions of mtDNA mutations, with only a minor effect on females, decreased the numbers of spermatocytes, spermatids and sperm in the mice testis, pointing at a common causal factor affecting both sperm lethargy and low sperm count.

Krestner (1997) suggests other correlates of oligospermia in humans, such as gonad and sperm dysfunctions, through microdeletions in the Y chromosome, most of which probably arise *de novo*. Shared factors are also suggested for the decline in sperm count, and the increase in testis abnormalities in humans between the 1940s and the 1990s (Giwerzman *et al.*, 1993). In red deer, testes size showed significant correlations with sperm suspension volume, sperm concentration and relative sperm number. Although male fertility *via* artificial insemination experiments in red deer was only correlated with sperm velocity and with percentage of normal sperm, it is claimed there is also a strong association between sperm velocity and sperm production parameters (Malo *et al.*, 2005). If sperm count and functionality are influenced by a common causal variable, as also suggested by Ruiz-Pesini *et al.* (2000*a*), then males with infertile sperm are also expected to have a poor sperm count. This satisfies both conditions required for the evolution of female EPC based on male infertility, namely, the absence of intra-male FSP and the existence of inter-male FSP.

(b) Sperm age

There are indications that sperm quality deteriorates as it ages. Pizzari *et al.* (2008) define sperm senescence as a decline through time in the ability of a sperm cell to fertilize an egg. The reduction in fertilization efficiency can result from changes in the morphology, metabolic performance and behaviour of the sperm cell, while the reduction in zygote viability is attributed to thermodynamic damage to the DNA, and to the reduced ability of spermatozoa to repair it, due to their small cellular cytoplasmic volume (Siva-Jothy, 2000; Pizzari *et al.*, 2008). At least one form of female preference for young sperm is documented. In the kittiwake *Rissa tridactyla*, females tend to eject sperm of early copulations, which is expected to age before fertilization, and thereby gain higher hatching success (Wagner, Helfenstein & Danchin, 2004). However, it is not clear whether higher hatching success is a result of genetic effects only (the retention of early sperm also leads to poorer chick condition), or also to greater fertility of the fresher sperm. Other forms of intra- and inter-male FSP patterns are unknown when young and old sperm compete. However, even if old sperm becomes lethargic, sperm is replenished in following copulations, which is the case for the genetically monogamous kittiwake. Hence, later copulations with the same male, either bonded or extrapair, guarantee fresh sperm. There is, therefore, no a priori direct benefit to extrapair copulations over multiple copulations with the bonded male. Furthermore, harmful effects of sperm ageing can be solved by purely masculine sperm replenishment

strategies, such as masturbation or human nocturnal emission.

(c) Impaired sperm mobility (asthenozoospermia) and mitochondrial mutations

High sperm mobility gives precedence to egg fertilization in sperm competition over slow sperm (Birkhead *et al.*, 1999; Anderson & Dixon, 2002; Snook, 2005). However, to induce both no intra-male FSP and inter-male FSP, lethargic sperm should also reduce fertility even in the absence of inter-male sperm competition. Therefore, the best candidates for sperm pathologies that are likely to promote female EPC are diseases that decrease male fertility, while impeding sperm mobility. This is the case, for example, in red deer, where infertility and sperm velocity are negatively correlated (Malo *et al.*, 2005). In birds, sperm may need to swim not just long distances, but also upstream, and withstand a reversible suppression of metabolism and motility in order to remain in sperm storage tubules (Stepinska & Bakst, 2006). Sperm lethargy *per se* may therefore reduce the number of spermatozoa that remain in the female tract and reach her fertilization site, thereby creating infertility *via* effective oligospermia. Of pathologies that induce sperm lethargy, primary candidates are those caused by mitochondrial failure (Gage, 1998).

Spermatozoa have very specialized mitochondria that are exclusively located at the midpiece of the cell, where the flagellum is inserted. ATP generated from the mitochondria is delivered to the axoneme and is used for flagellar propulsion. In a carefully designed set of experiments, Ruiz-Pesini *et al.* (2000*b*) showed that motility of human spermatozoa is fully dependent on its mitochondrial functionality, and that mtDNA mutations are indeed responsible for much sperm lethargy. By looking at matrilineal and patrilineal heritability of sperm lethargy in feral domestic fowls, Pizzari *et al.* (2003) argue for an effect that derives from the DNA of both mitochondrial and cytoplasmic origin. What makes mtDNA mutations even better candidates as precursors of the evolution of female EPC is that they may be very common, as they cannot be counterbalanced by natural selection operating on males.

From an evolutionary point of view, mitochondria live in haploid asexual populations, with much reproduction wasted on males that are evolutionary dead ends. Inheritance of paternal mtDNA, on a regular basis, is an exception (see Rawson & Hilbish, 1995; Ort & Pogson, 2007). Male mitochondria usually do not enter oocytes [but see Shitara *et al.* (2000) as an exception in an experimental design in mice, and Schwartz & Vissing (2002) for a natural rare exception in humans], or the few that do remain in the small spermatid plasma are present in too small a number to survive the active elimination that occurs during embryonic development, perhaps due to competition between paternal and maternal mitochondrial clones. Because spermatid paternal mtDNA is not transferred to zygotes, or dies soon afterwards, offspring quality is not hindered at all by female acceptance of fertilization by sperm equipped with dysfunctional mitochondria, nor is it

improved by the rejection of such sperm. mtDNA mutations in males can, therefore, only hinder female fertilization success, but not offspring quality.

For practical reasons, therefore, mtDNA is exclusively inherited in matrilineal lines; harmful effects of mtDNA mutations on males at any stage in their life cycle, including complete male sterility, have no consequences on their evolution unless they also impair phenotypes of their female carriers (Frank & Hurst, 1996). If it has no effect on the female phenotype, an equilibrium frequency of a mtDNA mutation, \hat{p} , is solely determined by the equilibrium between mutation and recurrent mutation rates, and can easily be as high as 0.5, say, if rates are equal, or even higher, for $u > v$ [using the standard one-locus two-alleles model, with no selection, in a haploid population, $\hat{p} = u / (v + u)$, where u and v are mutation and recurrent mutation rates respectively (Falconer, 1981)]. Survival of males or of sperm has no effect on the mtDNA mutation frequency at equilibrium.

However, it appears that mtDNA mutations that result in sperm lethargy or total immobility also have mild effects on the individual, hence also on female carriers. Ruiz-Pesini *et al.* (2000*b*) found indications that mtDNA mutations responsible for sperm lethargy are also responsible, in humans, for rare diseases such as DIDMOAD (characterised by diabetes insipidus, diabetes mellitus, optic atrophy and deafness), and multiple sclerosis. When female fitness is affected by mtDNA mutations, even if mildly or rarely, the effect of recurrent mutation rate becomes negligible, and an equilibrium frequency of a mtDNA mutation is approximated by a mutation-selection balance, $\hat{p} = u / s_f$, where s_f is the selection coefficient operating on females alone (Falconer, 1981). The frequency of each mtDNA mutation can therefore have a very strong effect on male fertility, yet remains at a relatively high frequency if selection against it in females is mild (Frank & Hurst, 1996). This is supported, in humans, by an estimate made by Ruiz-Pesini *et al.* (2000*b*) that mtDNA mutations are a relevant contributing factor in at least 7-10% of men who suffer from infertility due to sperm lethargy.

Ruiz-Pesini *et al.* (2000*b*) also found that sperm lethargy caused by mitochondrial mutations is associated with human couples' infertility, indicating at least some degree of no intra-male FSP. The greater agility of normal sperm should result in inter-male FSP, hence a reproductive advantage for female EPC. Among other types of male infertility, sperm lethargy, especially *via* mtDNA mutations, is likely to stand as a primary contributor to the evolution of female EPC.

(3) Third step: penetrating the egg and zygote formation

(a) Failure in egg penetration

Sperm binding to an egg is a process of complex biochemical and physical reactions between a sperm and an egg (Howes & Jones, 2002; Primakoff & Miles, 2002; Bedford, 2004; Stepinska & Bakst, 2006). Fertilization failure can therefore result from mutation in any of the

male enzymes involved. Assuming that a spermatozoon that fails to penetrate the egg cannot prevent other spermatozoa from binding to an oocyte, infertility that is caused by a failure to penetrate the egg may result in both intra- and inter-male FSP. Nevertheless, if and when sperm failure to penetrate an egg affects a significant number of spermatozoa, then it has an effect similar to that of oligospermia: the male may not have enough fertile sperm to fertilize all of the female's eggs. Thus, sperm failure to penetrate oocytes, *per se*, will not contribute to the evolution of female EPC unless it is widespread within males' ejaculates (which results in effective oligospermia), and is sufficiently common among males – facts that are difficult to determine *in vivo*.

(b) Polyspermy

Polyspermy is the case where more than one sperm cell penetrates an ovum. In mammals, polyspermy unconditionally kills the zygote. In birds, polyspermy is the norm, but excessive polyspermic fertilization is lethal and results in early embryonic mortality (Bakst & Howarth, 1977; Morrow *et al.*, 2002; Fairchild & Christensen, 2005; Stepinska & Olszanska, 2003). In mammals, penetration of a sperm into an ovum triggers a reaction, probably biochemical, that blocks the zona pellucida's binding sites, reducing risk of pathological polyspermy (Primakoff & Myles, 2002; Bedford, 2004). In birds, the egg's outer perivitelline layer begins thickening immediately following ovulation, and spermatozoa can penetrate the egg during only about the first 15 min (Stepinska & Bakst, 2006). Nevertheless, zygote mortality due to polyspermy naturally occurs in both mammals and birds (Hunter, 1991, 1996; Morrow *et al.*, 2002; Bedford, 2004; Stepinska & Olszanska, 2003; Stepinska & Bakst, 2006). This strongly suggests an advantage, for females, to counterbalance increasing sperm efficiency by decreasing sperm numbers that approach the ova (Hunter, 1991, 1996; Bedford, 2004; Stepinska & Bakst, 2006). Hence, to appreciate the importance of polyspermy to male infertility there is a need for a better understanding of the tight relationships between polyspermy and oligospermia. This should also help us to appreciate better why oligospermia is so important a cause of male infertility. For this purpose, the sections below explain in some detail how the internal fertilization arms race works, and how a dynamic equilibrium is reached such that both oligospermia and polyspermy have become major causes of infertility. We then apply the model's conclusions to consider the role of male-driven polyspermy in the evolution of female EPC.

(i) *The internal fertilization arms race.* The highest possible evolutionary bid is to transmit successfully heritable copies to the next generation. Spermatozoa directly compete for highest bids, sometimes against spermatozoa of other males, and always against other spermatozoa of the same male. Either way, from the male point of view, increased spermatid efficiency is likely to be strongly selected for, and be stopped only by too great a risk of zygote mortality as a result of polyspermy. However, given that oocyte numbers are exceedingly small compared to those of spermatozoa, competition for sperm among oocytes is overwhelmingly weaker than competition for oocytes among

spermatozoa. It is more likely, therefore, that selection to increase fertilization efficiency is stronger in males than in females, whereas selection to avoid polyspermy is stronger in females than in males. The expected result is an arms race between male traits that increase sperm numbers and competitiveness on the one hand, and female traits that increase severity of barriers against sperm on the other hand (Birkhead *et al.*, 1993; Eberhard, 1996; Morrow *et al.*, 2002). This explains the evolution of chemical, anatomical and physiological female barriers that kill, divert, dilute and control sperm flow before they reach oocytes, thereby reducing risks of polyspermy. Bedford (2004), who reviews these mechanisms in detail for eutherian mammals, describes this evolutionary arms race as a series of “domino” events. Stepinska & Bakst (2006) similarly describe the centrality of polyspermy avoidance in the evolution of female reproductive mechanisms in birds.

An arms race leads to the female need to balance between two opposing evils, female infertility as a result of polyspermy, and female infertility as a result of oligospermia (insufficient sperm to overcome female barriers) (Eberhard, 1996; Morrow *et al.*, 2002). Trade-offs between them should be most evident when conditions abruptly increase or decrease sperm densities. As discussed above, the literature shows that oligospermia, a sperm count that is lower than the norm, is a major cause of male infertility. Apparently, abnormally high numbers of spermatozoa can also be detrimental to proper fertilization. There is evidence that pathological polyspermy is especially high in *in vitro* fertilization (van der Ven *et al.*, 1985; Hunter, 1991; Bakst, 1998), or after surgical deposition of sperm directly into the fallopian tubes, where spermatozoa become unnaturally abundant at the site of egg fertilization (Hunter, 1991, 1996; Fairchild & Christensen, 2005). Hunter & L'Eglise (1971) similarly reported polyspermy as high as 32.4% when the isthmus is eliminated in pigs and ampullary sperm numbers greatly increased. Matter *et al.* (1989) reported high polyspermy in humans when partial zona dissection was used to open the zona pellucida, enabling easy sperm access to the egg.

As long as sperm count and efficiency are stable, one should therefore expect a stable equilibrium between oligospermia and polyspermy. As a first approximation, one might expect this to be at the point that minimizes their sum, and where their levels are about equal (but see later why some deviations from this are expected). When male average sperm counts and fertilization efficiency change in response to either genetic or environmental influences or both, the intensity of female barriers is expected to shift in response. A review of mammalian polyspermy by Hunter (1996) implies “an extremely precise regulation of sperm numbers gaining the ampullary portion of the fallopian tubes” (p. 417), and a “sperm/egg ratios that may be close to unity during the initial stages of fertilization” (p. 417). Despite the fact that, in mammals, offspring within the same litter can be fathered by different males, the intense elimination of sperm in the female reproductive tract, and the very few spermatozoa arriving at the fertilization site, led Bedford (2004) to question even whether the main effect

of sperm competition is indeed the numbers inseminated. There are enough indications that numbers matter in sperm competition, but with such an intense sperm elimination executed by females, and despite the fact that much of it is random with respect to sperm functionality, we incline to agree with Bedford (2004) that any minute sperm dysfunctions may nevertheless be of equal importance.

In all likelihood, this equilibrium is delicate and sensitive to changes in needs. This predicts particular deviations from equilibrium in special cases. For example, the well-documented increased susceptibility of postovulatory ageing eggs to polyspermy penetration (Hunter, 1991; Matter *et al.*, 1989) could be a result of a particular change in females' needs. Mature healthy eggs age when they remain unfertilized either because there was no copulation, or because not enough fertile sperm overcame the female barriers to reach her fertilization site. Relaxing egg defences may therefore be a female adaptation in cases where plan A, "control sperm numbers", fails. Female plan B, "lower defences", enables fertilization in cases where copulations happened to be with males that have poor sperm count or mobility.

Parker (1982) explains the evolution of many tiny sperm as a result of sperm competition (i.e. an arms race among males) that operates on top of sperm numbers that ensure fertilization, given that the female tract is long and that many spermatozoa may get lost. However, in Parker (1982) the long female tract is presented as a physiological constraint rather than as an evolved female strategy. Although there is no doubt that inter-male sperm competition plays a role in the evolution of a vast number of spermatozoa in an ejaculate, currently, sperm competition alone gives no satisfactory explanation for the common occurrence of infertility as a result of oligospermia. It also gives no satisfactory answer to the question why has the female tract evolved to be so hostile to sperm, and not for maximizing fertilization efficiency? By contrast, the hostile female reproductive tract and oligospermia are necessary byproducts of the internal fertilization arms race between males and females over fertilization efficiency. This arms race seems, therefore, a more compelling explanation for the evolution of many tiny sperm. Nonetheless, insufficient understanding of the relationships between sperm size and competitiveness (García-González & Simmons, 2007) hinders both explanations.

(ii) *Why is infertility common?* The two mechanisms, sperm competition and internal fertilization arms race, make two distinct predictions: if inter-male sperm competition dominates, it is selection in males that should maintain the balance between polyspermy, as one type of male fertility failure, and decreased sperm competitiveness as another type of male infertility failure. Intense sperm competition, both within and between males, should lead males to accept higher risks of polyspermy than of oligospermia. By contrast, reproductive systems controlled by an internal fertilization arms race should have better female control, and an equilibrium between the two types of female infertility, oligospermia and polyspermy, should exist at about equal probabilities. However, when combined with sexual polyandry and inter-male sperm com-

petition, oligospermia may even be favoured: formidable barriers against sperm give an advantage to males with high sperm counts and motility, and therefore select against undesired heritable or non-heritable male deficiencies associated with poor sperm counts and motility. This may shift the oligospermia-polyspermy balance towards increased risks of oligospermia relative to risks of polyspermy, but with a better zygote quality for eggs that are fertilized. We can therefore expect oligospermia to be somewhat more common than polyspermy if females are in control of the equilibrium between polyspermy and oligospermia as a result of the internal fertilization arms race. Similarly we can expect polyspermy to be more common than oligospermia if males dominate the process, *via* the selective power of sperm competition.

One way or the other, the existence of two antagonistic infertility types is likely to result in a higher degree of total infertility than that expected by directional selection operating against each infertility type alone. Whereas the female trade-offs are mainly between oligospermia and polyspermy, the male trade-offs involve also competition among spermatozoa, which could be very intense both within and between males. Selection to avoid polyspermy is therefore weaker in males than in females. The male-female arms race can be roughly described, therefore, as a line of equilibria with a tendency to slide towards increasing efficiency in males at any evolutionary opportunity. Moreover, each evolutionary "notch" in sperm efficiency is followed by an evolution in females of increasing barriers to sperm, and a tendency to hold on to an equilibrium between polyspermy and oligospermia thereafter, which "locks" the notch. It is nevertheless an arms race because it has an inherent direction (Fig. 3).

If this description is true, then an ever-increasing sperm efficiency is limited mostly by lack of appropriate mutations and physiological constraints in males, and ever-increasing female barriers are stopped mostly by a risk of oligospermia rather than by physiological constraints operating on females. This makes it different from a typical "red queen" arms race (van Valen, 1973), such as predator and prey running efficiency in which both sides benefit by increasing efficiency, and both are limited by evolutionary constraints (i.e. lack of new appropriate mutations) and physiological trade-offs. It also explains why male fertilization, constrained by physiological trade-offs, has become very sensitive to a variety of environmental and internal factors, some of them, such as parasitic load and the immune system (Følstad & Skarstein, 1997), tend to fluctuate. Female total infertility is therefore expected to increase further due to an evolutionary lag of the female adjustments to evolutionary changes in sperm efficiency. In particular, it is expected to lag behind rapid changes in male fertilization efficiency that result from male sensitivity to fluctuations in a variety of environmental and phenotypic factors.

Finally, the importance of an internal fertilization arms race can be inferred from cases where certain biological environments inherently and constantly impose high risks of oligospermia. Such cases are expected to be followed by the evolution of special adaptations in females that maintain the polyspermy-oligospermia equilibrium by correspondingly

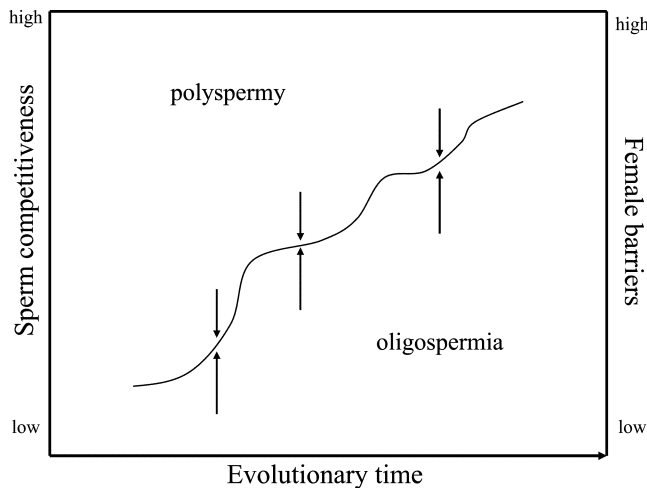


Fig. 3. A hypothetical description of an internal fertilization arms race between sperm competitiveness and female barriers. Arrows indicate balancing selection on females, which is likely to be somewhat stronger reducing polyspermy than reducing oligospermia. At each point in time, selection operating on females is stabilizing, whereas mutations that increase sperm competitiveness would tend to establish despite creating an increased risk of polyspermy, unless they constitute too strong a leap (i.e. too much polyspermy). The level of female infertility is expected to remain more or less constant throughout the process, though male sensitivity to both genetic and environmental influences should increase as female barriers increase.

and constantly increasing risks of polyspermy. In pigs, for example, where many eggs (for a mammal) are ovulated simultaneously, and therefore require more sperm at the female fertilization site, pathological polyspermy is exceptionally high [30–40%, according to Hunter (1991)]. Similarly, two biological differences in reproductive mechanisms make risks of oligospermia in birds inherently higher than in mammals: (i) the large yolky (megalechithal) avian egg adds risk because the germinal disc constitutes a relatively small target on the surface of the ovum (Bakst & Howarth, 1977; Birkhead, Sheldon & Fletcher, 1994), and (ii) the avian fertilization window is very short, estimated as 8–15 min (Bakst & Howarth, 1977; Birkhead, Veiga & Fletcher, 1995; Wishart, 1999; Stepinska & Bakst, 2006) before thickening of the outer perivitelline layer prevents further sperm penetration. To avoid such inherently high risks of oligospermia, higher sperm concentrations on the inner perivitelline surface are required (Bakst & Howarth, 1977; Birkhead *et al.*, 1994; Wishart, 1999). The regular occurrence of polyspermy in birds suggests, indeed, somewhat more relaxed female barriers than in mammals. These are accompanied by mechanisms that enable the avian oocyte to cope with a certain, limited level of multiple spermatid pronuclei (Fairchild, 2001; Stepinska & Olszanska, 2003). Such oligospermia risks were typically eliminated in eutherian reproduction, leading to a greater control over the number of spermatozoa that actually reach the egg during ovulation (Hunter, 1996; Bedford, 2004).

Polyspermy may also be affected by female sexual behaviour, which leads to an unexpected cost of female

EPC. Bedford & Kim (1993) reported that multiple mating in female rats was followed by greater ampullary sperm numbers, and an increased level of polyspermy, up to 9.3%. Fairchild & Christensen (2005) found that artificial insemination in turkeys with high-density sperm (where industrial breeders mix sperm of several males), leads to a high level of pathological polyspermy, despite the fact that female birds seem to be able to control semen flow from their sperm storage tubules (Stepinska & Bakst, 2006). Therefore, intense female involvement in EPC in birds may also increase sperm density in the female reproductive tract to a level that increases polyspermy. We have possibly found, therefore, yet another explanation for the positive within-broods association sometimes reported in birds, such as the house sparrow *Passer domesticus*, between the number of unhatched eggs in a clutch and the percentage of extrapair offspring (Wetton & Parkin, 1991; Lifjeld, 1994; Birkhead *et al.*, 1995).

In contrast to oligospermia, polyspermy is less detectable and, therefore, probably underrated. There is a high degree and variance of unexplained hatching failure in birds, much of it is attributed to oligospermia (Morrow *et al.*, 2002). A reasonable alternative is that many of these failures, as well as many cases of unexplained infertility in humans (Mak & Jarvi, 1996; Liu & Baker, 2000; van Voorhis *et al.*, 2001), might be undetected cases of polyspermy, which is likely to be about or almost as common as oligospermia.

(iii) *Effects of polyspermy on the evolution of EPC.* Nevertheless, polyspermy and oligospermia have opposite effects on the evolution of female EPC. Whereas oligospermia is favourable to the evolution of female EPC, male-driven infertility by polyspermy involves no inter-male FSP. This is because overly aggressive spermatozoa control oocytes they reach. Therefore, male-driven polyspermy seems to be a major infertility factor that cannot promote female EPC.

(c) *Early embryo mortality (excluding polyspermy)*

Early embryo mortality is common in both mammals and birds. Morrow *et al.* (2002) point out that hatching success varies in different bird species between 61% and over 95%. Studies of hatching success in the house sparrow, for example, show that much failure takes place before embryo development is noticeable (Wetton & Parkin, 1991; Cordero *et al.*, 1999; Birkhead *et al.*, 1995). Using a test designed to separate causes of egg hatching failure in the house sparrow, Birkhead *et al.* (1995) estimated that about 40% of the unhatched eggs failed after fertilization took place, but before any embryo development could be detected. Much of this may be a result of polyspermy, but there are other male dysfunctions that may cause early embryo mortality. In most mammals, for example, first zygotic cell division depends, in part, on a functional sperm centriole that is released into the oocyte cytoplasm, from which it attracts the oocyte-derived proteins of pericentriolar material and ultimately converts itself into an active zygotic centrosome (Sutovsky *et al.*, 1999; Rawe *et al.*, 2002). A dysfunctional sperm centriole therefore leads to zygote mortality as early as the first cell division (Nagy, 2000). Centriole dysfunction, however, is often associated with poor sperm motility

(Sutovsky *et al.*, 1999; Nagy, 2000; Rawe *et al.*, 2002), in which case its lethargy gives precedence to fertile sperm.

Zygote mortality at any stage of the zygotic life, including the early embryonic stage, can also be the result of different causes of genetic incompatibility between gametes. These may be shown when parents are genetically too similar (inbreeding), or when they are genetically too different (hybridization) (Morrow *et al.*, 2002; Price & Bouvier, 2002). However, whatever the cause of embryo mortality, and regardless of the stage at which it occurs after fertilization, male-driven embryo mortality cannot result in intra-male FSP. Also, here we attribute such failures to bad genes rather than to infertility.

(d) Sperm autoimmunity

Autoimmune infertility is the case where spontaneously occurring antibodies bind to antigens of the individual's gametes, and impair normal sperm activities and sperm-oocyte interactions. Antisperm antibodies (ASA) are far more frequent than oocyte antibodies (Bohring & Krause, 2003). In humans, autoimmunity is an important cause of infertility, and is found in up to 6% (Krestner, 1997) or 8% (Dohle, 2003) of patients with male infertility. Many infertile men who suffer from ASA have experienced clinical and surgical interventions in the male reproductive organs, which points to inflammation as a major cause of ASA. The most common factor for development of male antisperm antibodies is vasectomy, associated with a postoperative presence of serum antibodies in 34–74% of cases, but other surgical interventions such as accidental ligation of the vas deferens during hernial repair, can also induce sperm autoimmunity (Wald, 2005). It appears, therefore, that natural increased levels of ASA in animals may be mostly byproducts of natural diseases and infections in the male urogenital system, indicating perhaps a potential weakness in the male immune system. A correlation between autoimmunity and parasite resistance may result from phenotypic trade-offs, controlled by the immunosuppressive androgens, between the male's need to avoid parasites on the one hand, and risks of sperm autoimmunity on the other (Følstad & Skarstein, 1997).

ASA can hamper sperm activity at any stage, starting with motility, continuing in affecting acrosome reaction and zona binding, and ending with inability to form properly a zygotic pronucleus (Bohring & Krasue, 2003). The effect of ASA on sperm lethargy at the early stages following copulations can result in no intra-male FSP and inter-male FSP, both of which are required for the evolution of female EPC. The influence of ASA at the latest stages of sperm-oocyte interaction, cannot lead to female EPC, as they occur after blocking possible activities of other, more fertile sperm.

V. DISCUSSION

There has been a general disagreement whether male infertility can (Wetton & Parkin, 1991; Gray, 1997; Lifjeld

et al., 2007) or cannot (Jennions & Petrie, 2000) increase females' direct fertility benefits by EPC. Our model and review indicate that previous studies may have underappreciated the complexities of male infertility types and of the male-female evolutionary dynamics, and therefore overlooked some important consequences on the evolution of female sexual behaviour. Our Case I model resembles three earlier brief mathematical treatments that also assume no precopulatory information about male quality. Each model looked at a different quality that varies among males, and all reached the same conclusion, as Case I here, that multiple mating cannot add to female reproductive success. Yasui (1998) looked at male variations in sterile genes, Kisdí (2003) at inbreeding depression, and Arnqvist & Kirkpatrick (2005) at genetic incompatibility with the female. Case I adds variations in male infertility, regardless of their source, to this list. We can therefore generalise and argue that when no bias exists towards high-quality males, either before copulation or at any later stage (starting with the copulation itself, and ending at biases during the parental care period), a female tendency to copulate multiply may not evolve. Among these studies, only Kisdí (2003) mentions that precedence towards better sperm, if it exists, can alter this conclusion. Our Case II model and review of male infertility types elaborate on this option.

We have found that, indeed, certain male infertility types are likely to contribute to the evolution of female EPC. These are male mechanical infertility, azoospermia, sperm lethargy caused by mtDNA mutations and a variety of other infertility types that are associated with sperm lethargy, poor sperm count and some effects of sperm autoimmunity. Special attention should be paid to mtDNA mutations and oligospermia, which are expected to be fairly common, and to mechanical infertility and azoospermia, whose effect is very strong on FSP patterns, and therefore on the female benefit from EPC. By contrast, other male infertility types, such as sperm ejection, sperm age, sperm depletion, sperm allocation, polyspermy and sperm failure to form viable zygotes, usually cannot give females direct benefits from EPC, and are not likely to promote its evolution, unless they can be perceived prior to copulation. Special attention should be paid here to polyspermy as a potentially major cause of infertility that may easily be mistaken for early embryonic mortality (Birkhead *et al.*, 2008).

It seems that female control over the balance between oligospermia, which promotes female EPC, and polyspermy, which does not promote female EPC, largely determines levels of infertility. Sexual polyandry strategies can further enhance or diminish the intensity of sperm competition. Of the two antagonistic causes of infertility, it seems most sensible that females should have a tendency towards oligospermia rather than towards polyspermy, because a tendency for oligospermia is likely to result in a bias towards fertilization by the most viable, healthy males, who can produce high functional sperm counts. Diminished fertilization by poor-quality males may then be compensated for by the high sperm genetic quality of the healthy males. This tendency should be positively correlated with the intensity of additive genetic variance for viability and competitiveness, and may give females better offspring

at the expense of increased infertility. This balance, and the internal fertilization arms race that increases both sperm fertilization efficiency and female polyspermy avoidance mechanisms, might explain the relatively high infertility rates found in many species. The equilibrium between oligospermia and polyspermy, and the female inclination towards oligospermia rather than polyspermy, confine most incidences of male infertility to unhealthy, phenotypically inferior males.

Previous suggestions that male infertility can promote the evolution of female EPC have largely overlooked fertility costs that females pay, by EPC, when their bonded males are fertile (but see Yasui, 1998; Kisdí, 2003; Arnqvist & Kirkpatrick, 2005). Often, such suggestions inexplicitly assume inter-male precedence of fertile sperm. For example, Lifjeld *et al.* (2007) studied male infertility in two species of passerine birds, and concluded that “there should be an overrepresentation of clutches with all eggs sired by extra-pair males” because “in those pairs in which the male is infertile, the entire clutch should be sired by extra-pair males,” (p. 268). This conclusion clearly assumes both absolutely no intra-male FSP and full inter-male FSP (equivalent, in our model, to $l = 1$ for any $f < 1$). Not fully appreciating the importance of this assumption, Lifjeld *et al.* (2007) do not explicitly justify it in the text. Nevertheless, as the infertility of the two passerine birds in question was found to be complete azoospermia (i.e. $f = 0$, $l = 1$), their intuition was correct, and their inexplicit assumption of complete fertile sperm precedence is, indeed, justified. It does not follow, however, that as a rule, any male infertility can lead to an overrepresentation of extrapair fertilization, nor can any type of male infertility lead to the evolution of female EPC.

We can take Lifjeld *et al.*'s (2007) data, however, and use Equation 10 to calculate the maximum cost allowed for the evolution of EPC ($\hat{\epsilon}$) to ask further if their reported infertility types and rates merit the argument that variations in male fertility can drive females to EPC, given that EPC is costly. Lifjeld *et al.* (2007) report azoospermia in two out of 50 males in the willow warbler population, giving $t = 0.96$, $f = 0$, and $l = 1$, and in one out of 48 males of the bluethroat population, giving $t = 0.98$, $f = 0$, and $l = 1$. For purposes of illustration, we suppose $\mu = 0.5$, and this allows computation of the highest possible costs allowed for EPC, $\hat{\epsilon} = 0.0099M$ for the willow warbler (where M , the number of eggs ready to be fertilized, represents in the model the female full reproductive potential), and $\hat{\epsilon} = 0.005M$ for the bluethroat. Thus costs of EPC cannot exceed, at best, about 1 or 0.5%, respectively, of total female reproduction for each of the two species. These estimates assume the data is representative over an evolutionary scale, that females copulate equally with their bonded and an extrapair male, and that there are no precopulatory biases with respect to male sterility. It remains questionable whether actual costs of female EPC are indeed below this level, costs that are unknown for these two populations. Arnqvist & Kirkpatrick (2005), for example, used a different measure, selection gradients, calculated for a number of bird species, to estimate the relative direct costs of female EPC, as a result of deprivation of paternal care by

cuckolded males. They conclude that such costs are severe, at least relative to the indirect benefits that they may gain. This still leaves us with the question of whether 1% or 0.5% as maximal costs females can pay to overcome azoospermia of their bonded males in these two species is too high, given that (i) the estimates of Arnqvist & Kirkpatrick, (2005) are not directly comparable with our values, and (ii) Griffith (2007) (but see a reply by Arnqvist & Kirkpatrick, (2007), and Eliassen & Kokko (2008) identify critical factors in Arnqvist & Kirkpatrick's analysis, which should have been taken into account.

There is also much information missing in this puzzle: (i) actual costs of EPC in the willow warbler population are unknown. (ii) The only male infertility that was found in the willow warbler population was azoospermia, which is relatively easy to measure. There could be other undetected infertility types involved, which may promote female EPC further, such as oligospermia. This said, other infertility types may be more common, but their effect on female EPC should be weaker (higher f , smaller l , see Figs. 1 and 2). (iii) Precopulatory female choice patterns that may favour fertile males are unknown. Considering all this, the reported male infertility for willow warbler contributes to the evolution of female EPC in this species, but may not be sufficient to explain it.

Our model deliberately uses the worst case scenario, and looks for an explanation for the evolution of female EPC assuming females cannot exhibit precopulatory mating preference based on variations in male fertility. Furthermore, the model deals with the effects of male infertility types on female fertility only, i.e. with its pure infertility consequences, and ignores possible correlated effects such as “bad genes”. Such other effects are likely only to strengthen benefits from female EPC (see below).

The model deals with a worst case scenario also because it assumes that biases toward fertile males are specifically induced by male infertility types. However other biases are also possible. For example, a bias toward fertile sexual partners is created if females use the following simple rule of thumb: early in the breeding season mate exclusively with your bonded male, but if a first breeding attempt fails, then mate also with extrapair males. If some males are infertile, some failures will be due to male infertility. The proposed inherited EPC rule will necessarily be executed then by the female population whose chances of being bonded to an infertile male are greater than average. Unlike the model presented above, here fertility benefits are gained regardless of the male infertility type (including, say, male-driven early embryonic mortality). If benefits of such an EPC strategy are greater than its costs, it can evolve. This possibility predicts an increasing tendency toward EPC as the breeding season progresses, and in particular after hatching failures. We are currently not aware of empirical studies that show such a trend.

Bias in favour of fertile sexual partners can also be created by females that are able to perceive phenotypic correlates of male fertility (Sheldon, 1994), and prefer fertile males as their sexual partners. Female precopulatory discrimination effectively increases t (the proportion of fertile males) in the sexually preferred extrapair male

population, and decreases t among the cheated bonded male population (by expressing a lower tendency for EPC if the bonded male's phenotype is "fertile"). Furthermore, the calculated risks that females take by limiting EPC to cases where bonded males are infertile and extrapair males are fertile, can significantly reduce female EPC costs.

It is striking to see the prevalence of correlations between different male infertility types. In humans, abnormal sperm morphology becomes a good predictor of a number of sperm dysfunctions such as motility and zona pellucida binding and penetration (Mak & Jarvi, 1996; Liu & Baker, 2000). There are indications that male infertility is also associated with male morphological deformations. Mak & Jarvi (1996) provide details on correlations, in humans, between different male infertility types and various morphological deformations, including cranio-facial asymmetry. Mak & Jarvi (1996) even warn that treatment breakthroughs in human male infertility increase risk of transmitting genetic abnormalities to progeny. Semen quality was found to be negatively correlated with fluctuating asymmetry of the hand in humans (Manning, Scutt & Lewis-Jones, 1998), and with fluctuating asymmetry of the horns in three gazelle species (Gomendio, Cassinello & Roldan, 2000). For birds, Sheldon (1994) suggests that infertility can be recognised by variations in male phenotypic quality, and a recent study found indeed that carotenoid-based bill colour is correlated with sperm performance in mallards *Anas platyrhynchos* (Peters *et al.*, 2004).

It seems most probable, therefore, that male fertility is particularly sensitive to a number of phenotypic dysfunctions, which is most apparent in the case of mitochondrial dysfunctions. Mitochondria energise sperm mobility, sperm production and whole individuals. Therefore, mtDNA or chromosomal mutations that hinder mitochondrial enzymatic activities are likely to be an important causal factor for such correlations [but see Pizzari *et al.* (2003) for caution that should be taken with this argument]. Consequently, male courtship displays that drive males to extreme efforts in behavioural displays, or the production of delicate, environmentally sensitive structures such as feather decorations (Hasson, 1991) and antlers (Lagesen & Følstad, 1998; Pélabon & van Breukelen, 1998), can promote the evolution of handicaps, indices and amplifiers (Zahavi, 1975; Hasson, 1989, 1997; Harper, 2006) that enhance perception of variations in male phenotypic quality that are associated with both good genes and fertility. Both non-random precopulatory preferences for fertile extrapair sexual partners and postcopulatory biases towards fertile sperm that are inherent in some male infertility types can increase fertility of females that engage in EPC. When females are capable of expressing both precopulatory and postcopulatory biases towards fertile males and sperm, then the evolution of EPC may withstand greater costs than when each bias is expressed alone.

Our model and review of male infertility types show that some infertility types inherently promote female EPC. Whether or not this is sufficient to lead to female EPC in some species is an open question. More often, the fertile sperm precedence mechanism may work in concert with

other pre- and postcopulatory biases that benefit females engaged in EPC.

VI. CONCLUSIONS

(1) It has been frequently suggested that female extrapair copulations (EPC) have evolved because they improve females' biological success. EPC allegedly improves biological success by giving females more and/or better offspring, where "more offspring" is hypothetically the result of male variations in fertility, and "better offspring", the result of male variations in "genetic quality". We show that "fertility" and "genetic quality" are frequently inseparable, and that they may be usually found along a continuum. This paper, however, looks at the potential of female EPC to increase the number of a female's eggs that are fertilized and go through a successful first zygotic cell division, i.e. at processes that are usually attributed to "fertility" rather than to "genetic quality". In particular, we look at male causes of infertility, i.e. at variations in male attributes that have implications for female fertility. The general assumption of our model is that females have no precopulatory information about male fertility. Hence, this paper considers female gains and losses by EPC that are the result of postcopulatory processes only.

(2) The Case I model assumes that fertile and infertile sperm have equal access to eggs. It shows that a female average gain by EPC, when her bonded male is infertile and her extrapair male is fertile, is identical to her average loss when her bonded male is fertile and her extrapair male is infertile. This is regardless of the frequency or intensity of male infertility. Hence, given the assumptions of Case I, on the average EPC do not provide any overall benefits or improve female fertility.

(3) Unlike Case I, the Case II model deals with a bias that may arise in the presence of sperm competition. It assumes that sperm of an infertile male is lethargic and less competitive than sperm of a fertile male. As a result, an infertile male controls fewer eggs than its "share" in copulations. Fertile sperm precedence (FSP) decreases losses of a female who is bonded to a fertile male and copulates with an infertile extrapair male, and improves fertility of a female bonded to an infertile male and copulates with a fertile extrapair male. Given the assumptions of the Case II model, EPC improve female fertility.

(4) FSP can give fertility benefits to the female EPC strategy only if some males are truly infertile. This may happen when the infertile male produces no functional spermatozoa, or when FSP within males is weak or absent, such that when the male is the only male who copulates with a female, at least some of her eggs remain unfertilized. Hence, in order that females gain fertility benefits *via* EPC, it is required that the following two conditions are both true: (i) FSP within infertile males is absent or incomplete, and (ii) FSP among ejaculates of different males is substantial.

(5) In order to see whether these two conditions can be true simultaneously, we review male infertility pathologies in

birds and mammals. This review shows that some infertility types inherently produce no intra-male FSP while maintaining inter-male FSP, and therefore give fertility benefits to females who use EPC, whereas other infertility types do not. Important common infertility types in which EPC leads to benefits are azoospermia (no sperm is produced or transferred to the female), oligospermia (too few spermatozoa are produced) and asthenozoospermia (impaired sperm mobility). EPC fails to give females a fertility advantage in cases where sperm manages to control the eggs it reaches but fails to produce a healthy zygotic cell division. Among these, the most common and most important type is polyspermy (where more than one spermatozoon penetrates the egg), which may be a result of overly competitive sperm.

(6) Our review of male infertility necessarily raises the question “why is infertility so common?” Two important answers arise in relation to each of the three most common male infertility types, asthenozoospermia, oligospermia and polyspermy.

(7) Asthenozoospermia is frequently caused by mutations in mtDNA. Mutations in mitochondrial DNA that affect only male fertility cannot be selected against, as males are evolutionary dead ends for mitochondria anyway. They can, therefore, be selected against only if they also affect the female phenotype. Consequently, mtDNA mutations that strongly reduce male sperm production or motility but mildly affect entire phenotypes can be relatively common.

(8) Oligospermia is surprisingly common, considering the enormous amount of sperm produced by males. Apparently, the female reproductive tracts of birds and mammals constitute barriers that eject, kill, dilute, divert and weaken sperm such that close to the female fertility site, the spermatozoa to eggs ratio is close to unity. From an evolutionary point of view, this seems to be the result of an internal fertilization arms race between males and females. Overly aggressive sperm, including excessively high numbers of spermatozoa, result in a risk of polyspermy. Increased female barriers seem to have evolved to cope with such a risk but, in turn, increase the risk of oligospermia. Any step in increased male competitiveness (either genetic or environmental) must be followed by the evolution of increased female barriers. The result is an equilibrium between these two infertility types, polyspermy and oligospermia, which maintains both at relatively high frequencies. Another likely result of this arms race is male sensitivity to genetic and environmental factors, such that the best male phenotypes, which probably, on the average, have fewer deleterious mutations, are also the most fertile. Therefore, although the result of the internal fertilization arms race is that females are bound to maintain some level of infertility, they also get to be fertilized by males with better genes (including males with better mtDNA, which is not inherited by their offspring!).

(9) Correlations that are commonly found between different male infertility types, and between them and other male phenotypic characters, suggest that females can probably use male cues and signals as reliable guides for precopulatory biases (choice) towards the more fertile

males. This should increase further the female reproductive benefits by EPC.

VII. ACKNOWLEDGEMENTS

This paper benefited from many helpful comments and suggestions by Hanna Kokko and an anonymous referee. In addition we thank the editor for the many comments and copious corrections that served to improve the quality of the manuscript greatly. This research was supported by the Israel Science Foundation (Grant 5605).

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