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Nongenetic inheritance and the evolution of costly female preference

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Abstract

In species where males provide neither direct benefits nor paternal care, it is typically assumed that female preferences are maintained by indirect selection reflecting genetic benefits to offspring of preferred males. However, it remains unclear whether populations harbour sufficient genetic variation in fitness to support costly female preferences – a problem called the 'lek paradox'. Here, we ask whether indirect selection on female preferences can be maintained by nongenetic inheritance. We construct a general model that can be used to represent either genetic or nongenetic inheritance, depending on the choice of parameter values. Interestingly, we find that costly preference is most likely to evolve and persist when fitness depends on an environmentally induced factor that can be transmitted over a single generation only, such as an environment-dependent paternal effect. Costly preference can also be supported when fitness depends on a highly mutable factor that can persist over multiple generations, such as an epigenetic mark, but the necessary conditions are more restrictive. Our findings show that nongenetic inheritance provides a plausible hypothesis for the maintenance of costly female preferences in species where males provide no direct benefits to females. Nongenetic paternal inheritance of fitness can occur in species lacking conventional forms of paternal care. Indeed, transmission of paternal condition via sperm-borne nongenetic factors may be more likely to evolve than conventional forms of paternal investment because sperm-borne effects are protected from cuckoldry. Our results furnish a novel example of an interaction between genetic and nongenetic inheritance that can lead to otherwise unexpected evolutionary outcomes.

Introduction

In many species of birds, mammals, insects and other animals, females are choosy about their mates (Andersson, 1994). In some cases, the evolution and maintenance of female preferences is attributable to direct selection (Price *et al.*, 1993; Schluter & Price, 1993; Vahed, 1998). For example, males may vary in the provision of nutritious 'nuptial gifts' to females, or the probability of infecting females with parasites. Likewise, if males provide care directly to offspring and thereby enhance offspring fitness, then choosy females can benefit indirectly (i.e. through enhanced offspring fitness) by choosing mates on the basis of indicators of parental care quality (Heywood, 1989; Hoelzer, 1989; Wolf *et al.*, 1997; Iwasa & Pomiankowski, 1999). In many other cases, however, males contribute nothing more than an ejaculate to females and their offspring. In such systems, females are assumed to benefit indirectly, through the genetic benefits received by offspring of attractive males. Such systems present a theoretical conundrum, dubbed the 'lek paradox' (Borgia, 1979; Taylor & Williams, 1982; Bradbury & Gibson, 1983; Kirkpatrick & Ryan, 1991): because mutation rates are low relative to the strength of purifying selection, populations at evolutionary equilibrium are expected to

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exhibit negligible additive genetic variance in fitness. Consequently, there should be little or no difference in fitness between the offspring of attractive and unattractive males. Female mate choice is likely to be costly (Reynolds & Gross, 1990) and should therefore be rapidly lost in the absence of compensating benefits.

Many theoretical studies have aimed to identify the mechanisms that could maintain sufficient genetic variation in fitness to support costly female preferences (reviewed in Radwan, 2008). Several mechanisms are based on the idea that an evolutionary equilibrium is never attained because of variable selection or gene flow. Continual variation in selection could result from coevolution between parasites and hosts (Hamilton & Zuk, 1982), or antagonistic coevolution between sexes (Iwasa & Pomiankowski, 1995; Gavrilets *et al.*, 2001; Gavrilets & Hayashi, 2006). Zeh & Zeh (2008) also argued that exclusively maternal transmission of mitochondria, which precludes any response to selection for nuclear–mitochondrial co-adaptation in males, could maintain heritable variation in fitness. Likewise, if gene flow occurs between subpopulations or host-races occupying distinct environments, then indirect selection could maintain female preferences for locally adapted males (Day, 2000). Similarly, genetic variation in fitness could be maintained if genotype–environment interactions are strong, such that different alleles confer high fitness in different ambient conditions or microenvironments (Bussiere *et al.*, 2008; Kokko & Heubel, 2008; Ingleby *et al.*, 2010). Other mechanisms posit developmental-genetic architectures that amplify additive genetic variance for fitness, or slow its depletion. Pomiankowski & Møller (1995) suggested that nonlinear effects of secondary sexual trait expression on male mating success could drive the evolution of modifiers that increase variance in signalling traits. Rowe & Houle (1996) argued that condition-dependent sexual displays reflect genetic variation at numerous loci, providing a sufficient mutational target to maintain additive genetic variance in fitness. The depletion of additive genetic variance could be slowed by indirect genetic effects on the expression of male displays (Wolf *et al.*, 1997; Miller & Moore, 2007). Even at realistic mutation rates, it has been suggested that female preferences could provide a benefit to sexual populations, relative to asexual ones, by reducing the mutation load (Siller, 2001). It has also been shown that, in small populations, or under biased mutation rates, heterozygosity may be 'heritable', and this can maintain female preferences for outbred males (Neff & Pitcher, 2008; Fromhage *et al.*, 2009).

The empirical evidence bearing on the efficacy of these mechanisms is mixed. Many traits exhibit abundant additive genetic variance (Pomiankowski & Møller, 1995) and, in some populations, fitness is subject to strong genotype × environment interactions (Zhou *et al.*, 2008). However, studies on laboratory populations of *Drosophila* suggest that multivariate additive genetic variation in the direction of sexual selection is scant (Blows *et al.*, 2004; Hine *et al.*, 2004; Van Homrigh *et al.*, 2007). Moreover, a growing body of evidence suggests that many genes have sexually antagonistic effects on fitness (Chippindale *et al.*, 2001; Fedorka & Mousseau, 2004; Brommer *et al.*, 2007; Foerster *et al.*, 2007; Bonduriansky & Chenoweth, 2009; Innocenti & Morrow, 2010). Attractive males may thus typically sire poor-quality daughters, negating any indirect benefits of mate choice for females (Pischedda & Chippindale, 2006).

Here, we ask whether costly preference can be maintained by nongenetic inheritance – a set of inheritance mechanisms mediated by the transmission from parents to offspring of components of the parental phenotype or environment that influence gene expression and development in the offspring (Bonduriansky & Day, 2009; Danchin *etal.*, 2011). Nongenetic inheritance encompasses mechanisms such as transgenerational epigenetic inheritance (i.e. transmission of DNA methylation or chromatin structure variants), somatic inheritance (i.e. transmission of components of the parental soma, such as glandular secretions), behavioural inheritance (i.e. influences of parents on offspring behaviour via learning) and environmental inheritance (i.e. parental influence on the ambient environment experienced by the offspring). Such mechanisms of inheritance operate in parallel with Mendelian genetic inheritance and influence many aspects of the phenotype (Jablonka & Lamb, 1995, 2005, 2010; Bonduriansky & Day, 2009; Danchin *et al.*, 2011). Empirical evidence suggests that some nongenetically transmitted factors, such as epigenetic (DNA methylation) patterns (epialleles), can be transmitted over multiple generations like alleles, although they can be subject to a higher mutation rate than is typically observed (or assumed in models) under genetic inheritance (Vaughn *et al.*, 2007; Johannes *et al.*, 2009; Teixeira *et al.*, 2009; Roux *et al.*, 2011). Such semi-stable factors can apparently arise spontaneously via a mutation-like process (Vaughn *et al.*, 2007), or in response to an environmental inducer (Anway *et al.*, 2005). Many other examples of nongenetic inheritance involve factors that appear to wane over one or two generations following environmental induction, unless re-induced in subsequent generations (Mousseau & Dingle, 1991; Rossiter, 1996; Magiafoglou & Hoffmann, 2003; Anderson *et al.*, 2006; Bonduriansky & Head, 2007; García-González & Simmons, 2007; Ng *et al.*, 2010; Seong *et al.*, 2011). A potential role for nongenetic inheritance in the maintenance of heritable variation in fitness and the evolution of female preferences has been suggested previously (Bonduriansky & Head, 2007; Zeh & Zeh, 2008; Pizzari & Bonduriansky, 2010), but this possibility has not been verified in a formal model.

Nongenetic inheritance has the potential to generate and maintain heritable variation in fitness, both via the high mutability of nongenetically transmitted factors

such as epialleles (Vaughn *et al.*, 2007) and via the potential for nongenetic mechanisms of inheritance to mediate the transmission of environmental effects (i.e. 'acquired traits') from parents to offspring (Jablonka & Lamb, 1995; Bonduriansky & Day, 2009; Danchin *et al.*, 2011). Importantly, such variation can be transmitted paternally. This can occur via conventional forms of paternal care, such as the provision of resources or protection to offspring (e.g. Hunt & Simmons, 2000). However, such effects can also occur in the absence of conventional forms of paternal care, via mechanisms such as transgenerational epigenetic inheritance, RNA-mediated inheritance or the transmission of tiny quantities of compounds in the gamete cytoplasm or accessory gland products (reviewed in Bonduriansky & Head, 2007; Bonduriansky & Day, 2009). For example, in mammals, there is evidence that environmental factors can alter the patterns of DNA methylation in sperm and thereby influence gene expression in offspring (Guerrero-Bosagna *et al.*, 2010; Stouder & Paoloni-Giacobino, 2010; Feil & Fraga, 2012; Manikkam *et al.*, 2012). Moreover, there is evidence that offspring development can be affected by factors transferred to the zygote via the sperm cytoplasm or membranes (Diaz & Esponda, 2004; Rassoulzadegan *et al.*, 2006, 2007) or via compounds present in ejaculate fluids (Ying *et al.*, 1998; Chow *et al.*, 2003). Such mechanisms might mediate observed effects of paternal diet on offspring physiology (Anderson *et al.*, 2006; Ng *et al.*, 2010). Likewise, in insects, DNA methylation patterns are affected by environmental factors such as diet (Field *et al.*, 2004). In *Drosophila melanogaster*, it has also been shown that males can transmit stress-induced changes in chromatin structure that affect offspring phenotype (Seong *et al.*, 2011). Male accessory gland products can also affect offspring development (García-González & Simmons, 2007; Ivy, 2007; Simpson & Miller, 2007; Priest *et al.*, 2008). A male's larval diet has been shown to affect the phenotype of its offspring in species lacking conventional forms of paternal investment, such as the flies *Drosophila melanogaster* and *Telostylinus angusticollis* (Bonduriansky & Head, 2007; Vijendravarma *et al.*, 2010; Valtonen *et al.*, 2012), although the molecular mechanisms involved are unknown. Our analysis is based on the biology of *T. angusticollis*, in which males reared on a nutrient-rich larval diet develop into large, high-condition adults, and also produce larger offspring (Bonduriansky & Head, 2007). Males in high condition are preferred by females as mates (C. Fricke, M. Adler, R. Brooks and R. Bonduriansky, unpublished manuscript).

As a factor in signaller–receiver coevolution, nongenetic inheritance of paternal condition can be modelled in a similar way to environmentally generated variation in the quality of paternal care or direct benefits to females that have been shown in previous studies to support the evolution of costly female preferences (Heywood, 1989; Hoelzer, 1989; Price *et al.*, 1993; Schluter & Price, 1993; Iwasa & Pomiankowski, 1999). However, our analysis differs from previous studies in several important ways. First, previous models assumed that condition was advertised via specialized and costly ornaments, whereas we assume that condition affects male phenotypic traits (such as body size) that can be assessed directly by females. This assumption allows us to simplify our analysis and focus on the potential for nongenetic paternal effects to support costly female preferences. Second, previous studies considered systems with conventional forms of paternal care, whereas we focus on systems involving paternal effects that are not conventionally classified as paternal care, but which appear to be far more taxonomically widespread. Third, as a corollary of our focus on such paternal effects, we consider the consequences for sexual coevolution of allowing paternal condition to be transmitted over a single generation or multiple generations. This is an important and novel question in relation to some types of paternal effects, such as those mediated by epigenetic variation.

Our analysis also diverges in key assumptions from earlier work on the role of condition-dependent male signals as 'handicaps' (costly signals of male genetic quality). It was noted that Zahavi's (1975) handicap principle was most plausible if the handicap reflected nonheritable environmental variation in condition, and only served to 'test' and reveal the fitness of the remainder of the paternal genome (Maynard Smith, 1976; Dominey, 1983). Males bearing large handicaps would then pass on 'good genes' to their offspring without also passing on the costly handicap trait, and females would thereby derive indirect benefits by mating with males bearing large handicaps. In contrast, we assume that environmentally acquired paternal condition is itself transmissible to offspring, whereas we do not assume the existence of any genetic variation in fitness ('good genes'). Rather, in our model, costly female preferences are maintained because offspring of high-condition males receive a fitness benefit via a nongenetic paternal effect.

To examine the conditions whereby nongenetic inheritance can generate sufficient indirect selection to maintain female preferences, we first construct a general model that can be used to explore both genetic and nongenetic modes of inheritance with an appropriate choice of parameters. We then use this model to examine three main scenarios. First, we investigate the case of strictly genetic inheritance, where condition is determined by the allele at a single locus. Second, we explore a case where condition is determined by a semi-stable nongenetic factor, such as an epiallele, which is subject to a much higher mutation rate than a genetic locus. This case differs from the standard genetic, single-locus model only in the assumption of a much higher mutation rate, and can therefore also represent a case where condition is affected by many loci that jointly provide a large mutational target (Rowe &

Houle, 1996). Third, we explore a case where the inherited nongenetic factor can be induced *de novo* by the environment, such that offspring condition reflects an environment-dependent paternal effect.

In our models, selection on female preference is entirely indirect. The distinction between direct and indirect selection is important because, under reasonable assumptions, indirect selection is generally weak in absolute terms (Kirkpatrick & Barton, 1997) and weaker than direct selection (Kirkpatrick, 1996). Selection on preference is generally considered to be 'direct' when preference affects the female's own viability or fecundity, and 'indirect' when preference only affects the fitness of the offspring (e.g. see Price *et al.*, 1993; Schluter & Price, 1993; Kirkpatrick, 1996). In our model, variation in male condition has no effect on female viability or fecundity and thus can only generate indirect selection on female preferences via effects on offspring fitness. Indirect selection on female preferences has typically been envisaged in the context of 'good genes' and thus expected to result in a genetic correlation between preference alleles and genetic quality (Kirkpatrick, 1996). Under nongenetic paternal effects, instead, a covariance can be established between the preference allele and the nongenetic factor conferring high offspring fitness.

Our analysis furnishes several novel insights. We show that several forms of nongenetic inheritance can support costly female preferences in the absence of genetic variation in fitness. We also show, counterintuitively, that female preferences are most readily maintained when fitness depends on an environment-induced paternal effect that persists for a single generation only. Our analysis suggests that nongenetic inheritance of fitness provides an alternative to genetic variation as a hypothesis for the maintenance of costly preferences in species where males contribute nothing except small ejaculates. Moreover, we argue that paternal effects conveyed by the sperm (such as transgenerational epigenetic effects, or RNA-mediated effects) and, to a lesser extent, effects conveyed by the ejaculate fluids may be more likely to evolve than conventional forms of paternal investment because they are more likely to benefit a male's own offspring (see Discussion). Finally, our findings provide a novel example of how the interaction between genetic and nongenetic inheritance can lead to unanticipated evolutionary outcomes.

Model structure

We model a sexual haploid organism with a 50 : 50 sex ratio, using the framework of Day & Bonduriansky (2011). The model assumes that male condition is determined by a single locus (or nongenetic factor) with two possible alleles (or states): *C*, which results in high condition, and *c*, which results in low condition. Female mate preference is also determined by a single genetic locus with two segregating alleles: *A*, which causes females to prefer high-condition males, and *a*, which causes females to mate randomly. The frequency of high-condition males in the population is denoted by q, and the frequency of choosy females in the population is denoted by p (see Table 1 for notation). Each haploid individual therefore carries either *C* or *c* and either *A* or *a* at the condition and preference loci, respectively. Because the organisms are haploid, the allele/factor present at each locus is inherited, with equal probability, from either the mother or the father. For simplicity, we assume that the condition locus (or factor) is expressed only in males and that the preference locus is expressed only in females (Fig. 1).

We consider three models of the inheritance of condition (Fig. 1). First, we investigate a case where the condition-determining factor is a single locus with two segregating alleles (genetic model). Second, we consider a case where the condition-determining factor is a single locus with two segregating epialleles (e.g. distinct DNA methylation patterns) that differentially affect condition (epigenetic model). We assume that the epiallele at this locus is unaffected by the environment, but subject to a high mutation rate. This case can also represent a genetic model with many loci influencing condition. Third, we consider a nongenetic model where condition is determined by the environment (environmental induction model). This case can represent any type of environment-dependent paternal effect, including a transgenerational epigenetic effect where environment induces epigenetic changes that affect condition. The derivations and analysis described below are detailed in an annotated Mathematica notebook (see Appendix S1).

Under the environmental induction model, males are distributed randomly among good/bad quality environments (i.e. resource patches) in each generation. With a certain induction probability, ι, environment quality induces a male's condition (i.e. determines whether the male carries *C* or *c*). With the complement probability,

Table 1 A list of notation.

Parameter	Interpretation
p	Frequency of the female preference allele
q	Frequency of the male condition allele or factor
d	Linkage disequilibrium between preference and condition components
w_m	Viability of high-condition males ($w_m > 1$); low-condition males are standardized to 1
w_f	Viability of females with the preference allele ($w_f < 1$); nonchoosy females are standardized to 1
ε	Fraction of environmental patches that are of high quality
ι	Probability of induction of male condition by the environment
φ	Relative attractiveness of high-condition males ($\varphi > 1$); low-condition males are standardized to 1
μ	Mutation probability from high condition to low condition during offspring production

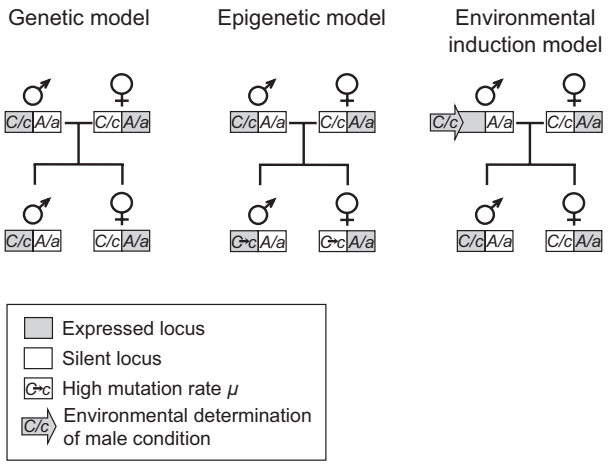

Fig. 1 Models of the inheritance of condition examined in the analysis: Organisms are sexual and haploid. Boxes represent genetic loci or types of nongenetically transmitted factors. Each locus/factor has one of two possible alleles or nongenetic states: *C*/*c* confer high/low condition in males, and *A*/*a* confer preference/nonpreference in females. Shaded boxes represent expressed loci/factors, whereas white boxes represent nonexpressed loci/factors. In the epigenetic model, a higher mutation rate is assumed to be possible, whereas in the environmental induction model, male condition can be induced by the quality of the environment (see text for further explanation).

$1-\iota$, the male's condition is unaffected by the environment (and thus continues to reflect the factor, *C* or *c*, inherited from its mother or father). Males then pass on their factor (*C* or *c*) to their offspring, and this factor affects offspring viability (probability of surviving to adulthood). If they survive to adulthood, the sons are then likewise distributed randomly among high-/low-quality environments, and their adult condition either reflects environment quality (with probability ι) or the factor (*C* or *c*) inherited from their mother or father (with probability $1-\iota$). Note that by setting the probability of induction to unity, we obtain a model in which the nongenetically inherited factor is transmissible over a single generation only (unlike an allele or semi-stable epiallele) because adult condition is determined *de novo* in every generation by the environment. We now describe each stage in the life cycle of the model in more detail.

Sex determination

Haploid, newborn individuals develop as males or females with equal probability, and in a way that is independent of the genetic or nongenetic factors that they carry.

Viability selection

We assume that viability selection occurs prior to adulthood (reproduction). In males, probability of survival to adulthood (viability) depends on the allele or factor (*C* or *c*) inherited from the mother or father. Individuals carrying *c* have a viability standardized to unity, whereas individuals carrying *C* have a viability of $w_m > 1$. In females, probability of survival to adulthood is determined by the allele (*A* or *a*) carried at the preference locus. Preference is assumed to impose a viability cost, with *a* (nonchoosy) individuals having a viability standardized to unity and *A* (choosy) individuals having a viability of $w_f < 1$.

Sexual maturation

Males and females that survive viability selection then undergo sexual maturation. In the absence of environmental induction, male condition is determined by the allele/factor *C* or *c*. If environmental induction occurs (subject to induction probability ι), then the male's adult condition is induced to match the quality of its environment, such that a high-quality environmental patch induces a *C* and a low-quality environmental patch induces a *c*. We assume that high- and low-quality environment patches occur with frequencies ε and $1-\varepsilon$, respectively, and that males settle randomly in the environment. We assume that females are uninfluenced by the environment in which they develop.

Mating

After sexual maturation, mating occurs and females carrying the *A* allele exert a mate preference for high-condition (*C*) males. This preference operates as follows (also see Kirkpatrick, 1982). Each male is assigned an attractiveness score based on its condition, with *c* males having a score standardized to unity and *C* males having a score of $\phi > 1$. The probability that a female chooses a given type of male is then equal to the frequency of this type of male at the time of mating, weighted by its score, divided by the average score in the population at the time of mating. For example, if q' is the frequency of high-condition males at the time of mating, then the fraction of matings by *A* females with such males is given by $q'\phi/(q'\phi + 1\text{-}q')$.

Mutation and segregation

Mating produces diploid zygotes which then segregate into haploid offspring. Segregation is assumed to occur independently for the preference locus and condition locus or factor. Furthermore, prior to mating, the high-condition allele or factor is assumed to mutate to low condition with probability μ. In the nongenetic cases, such 'mutation' might reflect a change in DNA methylation pattern from the high-condition epiallele to the low-condition epiallele, or it might reflect disease or parasitism that prevents a high-condition male from transmitting the viability-enhancing factor to his offspring. We assume that no mutation occurs at the female preference locus.

Given the above formulation, the genetic model is obtained by setting the induction probability to zero ($\iota = 0$), whereas the opposite extreme, in which condition inheritance is stable for a single generation only, is obtained by setting the induction probability to unity ($\iota = 1$). In both cases, we obtain the following three equations for the evolutionary dynamics of the preference frequency, p, the condition frequency, q, and the covariance (i.e. linkage disequilibrium), d, between the two:

$$\begin{aligned}\Delta p &= p(1-p)\beta_f + d\beta_m \\ \Delta q &= q(1-q)\beta_m + d\beta_f + E[\delta q] \\ \Delta d &= G(p,q,d)\end{aligned} \tag{1}$$

where β_m is the selection gradient on the condition factor, β_f is the selection gradient on the preference locus, $E[\delta q]$ is the fitness-weighted average change in condition between a parent and its offspring over all individuals in the population, and G specifies the dynamics of disequilibrium. Each of these elements takes on a different form depending on whether or not condition is induced by the environment (i.e. the value of ι).

Genetic and Epigenetic Model (no environmental induction, $\iota = 0$)

When condition is genetically or epigenetically determined but there is no environmental induction, the selection gradients are given by

$$\beta_f = \frac{1}{2}\frac{w_f - 1}{(1 - p + pw_f)} \tag{2a}$$

and

$$\beta_m = \frac{1}{2}\frac{(1-p')(w_m - 1) + p'(w_m\sigma_C - \sigma_c)}{1 - q + qw_m}. \tag{2b}$$

where

$$\sigma_C = \frac{\phi}{q'\phi + 1 - q'}$$

$$\sigma_c = \frac{1}{q'\phi + 1 - q'}$$

and

$$p' = \frac{pw_f}{1 - p + pw_f}$$

$$q' = \frac{qw_m}{1 - q + qw_m}.$$

The quantity $E[\delta q]$ represents the fitness-weighted expected change in condition through mutation and is given by

$$E[\delta q] = -q\left(\frac{1}{2}w_m\frac{1 - p' + p'\sigma_C}{1 + (w_m - 1)q} + \frac{1}{2}\frac{pw_f + 1 - p - \frac{d}{q}(1 - w_f)}{1 - p + pw_f}\right)\mu \tag{3}$$

We omit the expression for the dynamics of disequilibrium as it is very lengthy and difficult to interpret.

Environmental Induction Model (complete induction, $\iota = 1$)

For the nongenetic model with complete environmental induction, the selection gradient on female preference is the same, but the selection gradient on male condition is given by

$$\beta_m = \frac{1}{2}\frac{w_m - 1}{(1 - q + qw_m)}. \tag{4}$$

The quantity $E[\delta q]$ now represents the fitness-weighted expected change in condition through both mutation and environmental induction, and is given by

$$\begin{aligned}E[\delta q] = q\Bigg(&\frac{1}{2}w_m\frac{-(1-p')(\varepsilon\mu + 1 - \varepsilon) - p'\left(\hat{\sigma}_C\varepsilon\mu + \hat{\sigma}_c(1-\varepsilon)\right)}{1 + (w_m - 1)q} \\ &+ \frac{1}{2}\frac{pw_f + 1 - p - \frac{d}{q}(1 - w_f)}{1 - p + pw_f}(-\mu)\Bigg) \\ &+ (1-q)\left(\frac{1}{2}\frac{(1-p')\varepsilon(1-\mu) + p'\hat{\sigma}_C\varepsilon(1-\mu)}{1 - q + qw_m}\right)\end{aligned} \tag{5}$$

where

$$\hat{\sigma}_C = \frac{\phi}{\varepsilon\phi + 1 - \varepsilon}$$

$$\hat{\sigma}_c = \frac{1}{\varepsilon\phi + 1 - \varepsilon}$$

Analysis

We explore the potential for female preference to evolve by first letting male condition reach an equilibrium in the absence of female preference, and then introducing a rare preference allele. A general analysis is cumbersome, but simplifies greatly if we assume the dynamics of disequilibrium are fast relative to allele frequency dynamics. Numerical results suggest that this approximation is reasonable under a wide variety of parameter values (results not shown).

For the genetic and epigenetic case (no environmental induction), the female preference allele will spread if the following inequality is satisfied:

$$\frac{1 + w_f}{2} + \frac{w_f(1+\mu)\mu(2 - (1-\mu)(w_m + 1))}{\left(4 + (1-\mu)\left(-3 - \mu + w_m(-4 + w_f(1-\mu))\right)\right)(2 - 2\phi + (1-\mu)(-2 + \phi + w_m\phi))}(\phi - 1) > 1 \tag{6}$$

Inequality (6) is difficult to interpret, but it simplifies greatly at the upper and lower limits of mutation rate, μ. At intermediate mutation rates, the model's behaviour can be investigated numerically.

For the nongenetic case with environmental induction, the female preference allele will spread if inequality (7) is satisfied:

$$\frac{1+w_f}{2}+\frac{w_f(w_m-1)(1-\varepsilon)\varepsilon(1+\mu)(1-\mu)}{2(4-w_f(1-\mu))(2+(-1+(w_m-1)\varepsilon)(1-\mu))(1+\varepsilon(\phi-1))}(\phi-1)>1 \tag{7}$$

When the mutation rate goes to zero (i.e. approximates a realistic mutation rate for a genetic locus), inequality (6) simplifies to $(1 + w_f)/2 > 1$. Because female preference is costly ($w_f < 1$), this condition is never satisfied (Fig. 2a), indicating that female preference cannot evolve in the low mutation limit. When mutation is low, female preference cannot evolve precisely because of the lek paradox: there is not enough genetic variation in condition maintained to give utility to female choice.

Intermediate mutation rate (e.g. in the range of 15–60% under the parameter combination in Fig. 2a) represents the epigenetic (noninduction) case (i.e. a spontaneously mutating epiallele). It can also represent a genetic case where condition is determined jointly by many loci. This case can support the evolution of female preference (Fig. 2a). However, for this to occur, the strength of selection on male condition, w_m, must effectively balance the mutation rate, μ, in order to generate sufficient variation in condition at equilibrium. This is indicated by the positive slope of the light region in Fig. 2a.

When the mutation rate approaches one, inequality (6) simplifies to $1/2 > 1$, which is never satisfied. Under very high mutation rates, female preference cannot evolve because there is virtually no correlation between the condition of a chosen male and the condition of his offspring.

When the mutation probability goes to one, this becomes $(1 + w_f)/2 > 1$ which, again, is never satisfied. When mutation probability goes to zero, however, we get

$$\frac{1+w_f}{2}+\frac{w_f(w_m-1)(1-\varepsilon)\varepsilon}{2(4-w_f)(2+(-1+(w_m-1)\varepsilon))(1+\varepsilon(\phi-1))}(\phi-1)>1 \tag{8}$$

which can be readily satisfied provided there is some environmental variation (i.e. $\varepsilon \neq 0$ and $\varepsilon \neq 1$). Figure 2b shows the parameter space supporting the evolution of female preference under the same conditions as the genetic model and with $\varepsilon = 0.5$. In general, for a given set of parameter values, there is some range of environmental variability that supports the evolution of female preference. Also note that it is no longer necessary for selection, w_m, to balance mutation, μ, for preference to evolve. When male condition is environmentally induced selection can no longer erode the variation because the environmental heterogeneity remains unaltered by selection from one generation to the next.

Finally, we can also explore the evolutionary dynamics when the nongenetic factor is not completely induced by the environment anew each generation, but

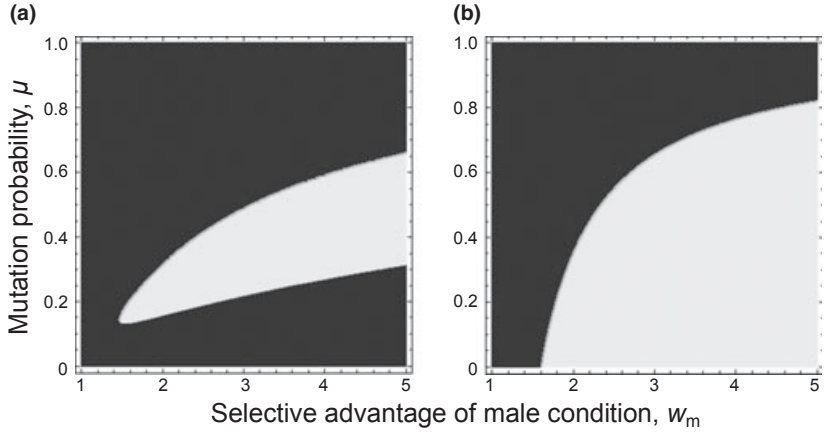

Fig. 2 Regions of parameter space supporting the evolution of costly female preference. Preference evolves and is maintained in the light region, but not in the dark region. (a) Genetic and nongenetic model without environmental induction (parameter values: $\iota = 0$, $w_f = 0.94$, $\varphi = 5$). (b) Nongenetic model with complete environmental induction (parameter values: $\iota = 1$, $w_f = 0.94$, $\varphi = 5$, and $\varepsilon = 0.5$).

rather has some degree of permanence from one generation to the next. This case can represent transgenerational epigenetic inheritance of semi-stable epialleles that can be induced by the environment, and it is modelled by using an intermediate value of the induction probability, ι. In this case, the region of parameter space supporting the evolution of female preference is intermediate between the genetic and completely induced nongenetic cases (results not shown).

Discussion

How could costly female preferences be maintained in species where males provide nothing but small ejaculates? Most previous attempts to answer this question have endeavoured to resolve the 'lek paradox' by asking how additive genetic variation in fitness can be maintained in the face of persistent directional selection favouring alleles that confer high fitness (Borgia, 1979; Hamilton & Zuk, 1982; Taylor & Williams, 1982; Kirkpatrick & Ryan, 1991; Pomiankowski & Møller, 1995; Rowe & Houle, 1996; Miller & Moore, 2007), or whether nonadditive genetic variation (Neff & Pitcher, 2008; Fromhage *et al.*, 2009) or nuclear–mitochondrial interactions (Zeh & Zeh, 2008) could play an equivalent role. In contrast, we show that nongenetic inheritance of fitness can support costly female preferences in such species in the absence of genetic variation in fitness. Our findings suggest that nongenetic inheritance of fitness provides an alternative to genetic variation in fitness as a potential mechanism for the maintenance of costly female preferences.

We develop a general model that can be adapted, by modifying parameter values, to examine genetic inheritance or various forms of nongenetic inheritance. To begin with, we use this model to show that genetic inheritance ($\iota = 0$) can never support costly preference when a realistic single-locus mutation rate is assumed. This illustrates the classic lek paradox.

Next, we investigate the epigenetic model ($\iota = 0$), which allows for a much higher mutation rate than that conventionally assumed for a genetic locus (see Fig. 2a). This can represent a situation where fitness depends on a single, semi-stable epiallele that is not subject to environmental induction (see Vaughn *et al.*, 2007). We find that an 'intermediate' mutation rate (see Fig. 2a) can support costly preference, but only if the mutation rate balances net selection strength on male condition, so that heritable variation in fitness is maintained at equilibrium. The epigenetic model might also be interpreted as a situation where male condition has a multilocus genetic architecture that furnishes a large mutational target (Rowe & Houle, 1996).

Finally, we investigate cases where the nongenetically transmitted factor is subject to environmental induction ($\iota > 0$). Induction can occur in every generation (complete induction, $\iota = 1$), or with some intermediate probability ($0 < \iota < 1$). Such a case is analogous to many examples of environment-dependent parental effects that wane over one or a few generations in the absence of the inducing environmental factor (Mousseau & Dingle, 1991; Rossiter, 1996; Magiafoglou & Hoffmann, 2003; Anderson *et al.*, 2006; Bonduriansky & Head, 2007; García-González & Simmons, 2007; Ng *et al.*, 2010; Seong *et al.*, 2011). Interestingly, of all the genetic and nongenetic cases examined, we find that costly preference is most likely to evolve under complete environmental induction, where the fitness-determining factor can be transmitted over a single generation only. This somewhat counterintuitive result occurs because, under complete induction, all of the heritable variation in fitness is re-generated in every generation by environmental heterogeneity, and thus cannot be depleted by selection. Because indirect selection on preference is proportional to the magnitude of heritable variation in fitness, complete induction therefore allows for maximum selection strength on preference.

In our models, we made the simplifying assumption that paternal condition affects sons only. Assuming that daughters' fitness is also influenced by the paternal effect would generate stronger indirect selection on female preferences. However, this assumption does not alter our conclusions qualitatively because it only affects the quantitative advantage gained by choosy females. We also assumed that male condition could be assessed directly by females. We did so because empirical studies show that condition often affects male traits (such as body size) that are not likely to impose viability costs, and can probably be assessed directly by females (Bonduriansky & Head, 2007; C. Fricke, M. Adler, R. Brooks and R. Bonduriansky, unpublished manuscript). Furthermore, the evolution of costly indicator traits has already been investigated in numerous theoretical studies (Iwasa & Pomiankowski, 1999; Kokko *et al.*, 2003; Radwan, 2008), and avoiding this added complication allowed us to focus our analysis on the more novel question of the role of nongenetic paternal effects in the evolution of female preferences.

The paternal effects that we model must be costly and condition dependent in order for male condition to provide an honest signal of offspring fitness. As with other condition-dependent traits, the high costs of expressing the most advantageous phenotype are assumed to prevent 'cheating' (Zahavi, 1975). In the noninduction case, we assume that a spontaneously mutating epiallele affects both a male's own condition (which females can assess) and the viability of his offspring. Males lacking the appropriate epigenetic variant are assumed to be incapable of expressing the high-condition phenotype. In the environmental induction case, environment quality is assumed to determine both a male's own condition and the viability of his offspring. The costs of molecular mechanisms that mediate paternal effects are unknown, but are likely to

be nontrivial. Intracellular processes such as DNA repair are highly complex, requiring precise regulation and biosynthesis of numerous compounds (Park & Gerson, 2005), and are believed to be costly (Breivik & Gaudernack, 2004). Such costs are, for example, central to theory on the evolution of ageing (Kirkwood & Rose, 1991). It is thus reasonable to assume that DNA methylation and processes involved in gamete formation and biosynthesis of accessory gland products are costly, and likely to exhibit condition-dependent expression. Indeed, environmental effects on condition are reflected in patterns of gene expression throughout the genome (Wyman *et al.*, 2010).

Condition-dependent paternal effects might evolve as a cryptic form of paternal care (Bonduriansky & Head, 2007), if the net benefit of investing resources into such effects exceeds the net benefit of investing those resources into searching and competing for mates (Kokko & Jennions, 2008). A key impediment to the evolution of paternal investment is uncertainty of paternity and the possibility of cuckoldry (Trivers, 1972). As a result of this impediment, little paternal investment is predicted to occur in polygamous species, where males can gain more fitness by investing their resources in competition for additional mates (Kokko, 1999). Interestingly, this impediment may be relaxed for paternal investment mediated by some mechanisms of nongenetic inheritance. For example, if males transmit their condition to offspring via transgenerational epigenetic inheritance (i.e. variation in DNA methylation or chromatin structure), the investment cannot go to another male's offspring because it is inseparable from the sire's own DNA. Likewise, investment mediated by factors transmitted in the sperm cytoplasm, such as RNA (Rassoulzadegan *et al.*, 2006, 2007; Suter & Martin, 2009), may be tied to fertilization, and protected from the possibility of cuckoldry, unless females can digest the sperm and allocate beneficial compounds to the offspring of other males. To a lesser extent, factors transmitted via ejaculate fluids (e.g. accessory gland products) may also be less vulnerable to cuckoldry than conventional forms of male investment, such as nuptial feeding of females or even the provision of care directly to offspring. Thus, paternal investment via epigenetic or cytoplasmic inheritance, and perhaps other mechanisms involving ejaculate-borne factors, may be more likely to evolve than other forms of paternal investment.

Males might also transmit the effects of pathologies, stress or toxins via paternal effects (Anway *et al.*, 2005; Ryabokon & Goncharova, 2006; Seong *et al.*, 2011). Such cases are exactly analogous to paternal effects that evolve as a form of paternal investment, in that females are selected to reject low-condition males because such males produce offspring of lower fitness.

Our analysis suggests that the evolution and maintenance of costly female preferences in species where males provide no direct benefits to females, and no conventional form of paternal care to offspring, could potentially be explained by nongenetic paternal inheritance of fitness. Our findings are similar to those of analyses based on conventional forms of paternal investment or direct effects of male condition on female fitness, in that such sources of variation in male mate quality can readily select for costly female preferences (e.g. Dominey, 1983; Heywood, 1989; Hoelzer, 1989; Price *et al.*, 1993; Schluter & Price, 1993; Iwasa & Pomiankowski, 1999). Our analysis goes beyond this earlier work, however, by also showing that costly female preferences are most likely to evolve when paternal effects do not persist beyond the F1 generation, such that variation in fitness is completely re-generated in every generation by environmental heterogeneity. Our analysis thus encompasses recently discovered mechanisms of nongenetic paternal transmission of fitness, such as transgenerational epigenetic inheritance.

Our findings suggest that nongenetic inheritance provides a general alternative to genetic variation in fitness as a hypothetical mechanism maintaining costly female preferences in such species. This hypothesis can be tested by manipulating environmental parameters that affect condition, and testing for the transmission of environmentally generated variation in condition from fathers to offspring (e.g. see Hunt & Simmons, 2000; Bonduriansky & Head, 2007; Vijendravarma *et al.*, 2010; Seong *et al.*, 2011; Valtonen *et al.*, 2012). A corollary of our findings is that laboratory studies performed under carefully standardized environmental conditions may fail to detect heritable variation in fitness because much nongenetically heritable variation may depend on the environmental variation to which individuals are exposed.

Our analysis differs in an important way from analyses based on indirect genetic effects (Wolf *et al.*, 1997; Miller & Moore, 2007). Under indirect genetic effects, male display is condition dependent and reflects the quality of the environment experienced by the male. However, environmental quality reflects genetic quality, and it is the additive genetic variation in fitness that drives the evolution of female preferences. Although indirect genetic effects may slow the rate at which genetic variation is depleted (Miller & Moore, 2007), it is unlikely that they can prevent the eventual fixation of alleles conferring high fitness. In contrast, we show that nongenetically heritable variation that is uncorrelated with genetic variation can maintain indirect selection on female preferences, such that costly preferences can be maintained without genetic variation for fitness.

Our model furnishes a novel example of the complex interactions that can occur between genetic and nongenetic inheritance, and the potential for such interactions to influence the course of evolution. The best-known example of such an interaction is the evolution of lactase persistence in humans, where alleles that allow adults to digest the milk sugar lactose have

evidently spread through cattle-herding populations via their interaction with the nongenetically inherited, cultural practice of milk use in the adult diet (Feldman & Cavalli-Sforza, 1989; Swallow, 2003; Gerbault *et al.*, 2009). This example is of particular interest because the evolutionary outcome – the evolution of lactase persistence – is highly improbable in the absence of nongenetic inheritance, thereby illustrating the potential importance of nongenetic inheritance in evolution (Jablonka & Lamb, 2005; Richerson & Boyd, 2005; Bonduriansky & Day, 2009; Laland *et al.*, 2010). It is less clear whether such outcomes are possible in nonhumans and, more generally, in organisms lacking any form of cultural transmission (although see Avital & Jablonka, 2000). A putative example of the interaction between genetic and nongenetic inheritance systems in nonhumans is furnished by sexual imprinting in birds, which can be regarded as a nonhuman form of cultural transmission (Freeberg, 2000). Laland (1994) showed that such nongenetic inheritance of preferences can influence the evolution of genetically inherited male displays, although the consequences of nongenetic inheritance are relatively subtle, and the persistence of culture through uniparental transmission has been questioned (Enquist *et al.*, 2010). In contrast, we model a noncultural system where female preference is genetically determined but male display is transmitted nongenetically, and show that the presence of nongenetic inheritance can lead to an evolutionary outcome – the evolution of preference – that is unlikely under purely genetic inheritance.

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Supporting information

Additional Supporting Information may be found in the online version of this article:

Appendix S1 This notebook derives the general model and then presents each of the special cases.

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