© 2024. Published by The Company of Biologists Ltd | Journal of Experimental Biology (2024) 227, jeb245569. doi:10.1242/jeb.245569

COMMENTARY

Mitochondrial function and sexual selection: can physiology resolve the 'lek paradox'?

Ondi L. Crino[1,2,*], Megan L. Head[2], Michael D. Jennions[2,3] and Daniel W. A. Noble[2]

ABSTRACT

Across many taxa, males use elaborate ornaments or complex displays to attract potential mates. Such sexually selected traits are thought to signal important aspects of male 'quality'. Female mating preferences based on sexual traits are thought to have evolved because choosy females gain direct benefits that enhance their lifetime reproductive success (e.g. greater access to food) and/or indirect benefits because high-quality males contribute genes that increase offspring fitness. However, it is difficult to explain the persistence of female preferences when males only provide genetic benefits, because female preferences should erode the heritable genetic variation in fitness that sexually selected traits signal. This 'paradox of the lek' has puzzled evolutionary biologists for decades, and inspired many hypotheses to explain how heritable variation in sexually selected traits is maintained. Here, we discuss how factors that affect mitochondrial function can maintain variation in sexually selected traits despite strong female preferences. We discuss how mitochondrial function can influence the expression of sexually selected traits, and we describe empirical studies that link the expression of sexually selected traits to mitochondrial function. We explain how mothers can affect mitochondrial function in their offspring by (a) influencing their developmental environment through maternal effects and (b) choosing a mate to increase the compatibility of mitochondrial and nuclear genes (i.e. the 'mitonuclear compatibility model of sexual selection'). Finally, we discuss how incorporating mitochondrial function into models of sexual selection might help to resolve the paradox of the lek, and we suggest avenues for future research.

KEY WORDS: Additive genetic variation, Cellular respiration, Condition, Development, Maternal effects, Sexual traits

Introduction

Across many taxa, animals use sexually selected traits – such as elaborate ornaments and complex behavioural displays – to attract potential mates. Sexually selected traits have more often evolved in males than females as a result of the lower benefit of parental care for males (Fromhage and Jennions, 2016), and because anisogamy (see Glossary) creates an asymmetry in the benefits of increasing fertilization success when sperm outnumber eggs (Lehtonen et al., 2016). The question of why females prefer males with greater expression of elaborate sexual traits has been widely debated. In some species, females gain benefits from their mates in the form of material resources that increase their lifetime reproductive output (reviewed in Kokko et al., 2003). In these species, sexually selected traits are hypothesized to signal the benefits that females will receive from mates; thus, females benefit from choosing males with greater relative expression of sexually selected traits (Iwasa and Pomiankowski, 1999; Kirkpatrick, 1996). However, in many species, females do not appear to receive any material benefits from mate choice (Andersson, 1994; Moller, 1994). Therefore, a key challenge has been to explain the persistence of female mating preferences when there are no apparent material benefits to female choice (i.e. when males only transfer sperm to females).

In standard 'Fisherian runaway' scenarios, females that choose males with more attractive sexually selected traits produce sons that also have more attractive sexually selected traits as adults (i.e. 'sexy sons'). Consequently, the sons of choosy females are more successful than average at acquiring mates. In this scenario, female mating preferences will be favoured through indirect selection if genes for a female mating preference co-occur with genes for preferred traits in males that are under direct selection (Kokko et al., 2006). In other words, non-random mating as a result of female choice increases the co-occurrence (linkage disequilibrium) of genes for mating preferences and genes for beneficial sexually selected traits (Kuijper et al., 2012). One challenge to explain the evolution of mating preferences is that female choice should generate strong directional selection on males that erodes additive genetic variation (see Glossary) in preferred sexually selected traits and, hence, reduces variation in trait expression (Fig. 1A–C). If males do not vary in the fitness benefits they provide to offspring, then females no longer gain a genetic benefit from their mating preferences and selection will eliminate female mating preferences if choosiness is costly (Bonduriansky and Day, 2013; Kokko et al., 2015; Kotiaho et al., 2008; Tobler et al., 2011; Vitousek et al., 2007). However, female mating preferences and heritable variation in sexually selected male traits persist in many species. This so-called 'paradox of the lek' is most obvious in lek mating systems (see Glossary) where, compared with other mating systems, female choice is strongest, male sexual traits are most elaborate and choosy females only appear to gain genetic benefits from males (Kirkpatrick and Ryan, 1991). The lek paradox has puzzled biologists since it was originally described by R. A. Fisher (Fisher, 1930; Kirkpatrick and Ryan, 1991; Petrie, 2021; Pomiankowski and Moller, 1995; Taylor and Williams, 1982), and it has inspired a plethora of hypotheses and experimental tests that seek to resolve it (e.g. Bonilla et al., 2016; Pomiankowski and Moller, 1995; Tomkins et al., 2004).

One challenge in distinguishing between competing hypotheses that try to resolve the lek paradox is a poor understanding of the physiological mechanisms that underlie the development and

[1]School of Science and Engineering, Flinders University, Bedford Park, SA 5042, Australia. [2]Division of Ecology and Evolution, Research School of Biology, The Australian National University, Canberra, ACT 2600, Australia. [3]Stellenbosch Institute for Advanced Study (STIAS), Wallenberg Centre, 10 Marais Street, Stellenbosch 7600, South Africa.

*Author for correspondence (ondi.crino@anu.edu.au)

O.L.C., 0000-0001-5700-1387; M.L.H., 0000-0002-8123-7661; M.D.J., 0000-0001-9221-2788; D.W.A.N., 0000-0001-9460-8743

Journal of Experimental Biology (2024) 227, jeb245569. doi:10.1242/jeb.245569

Glossary

Additive genetic variation
Genetic variation that arises through additive genetic effects whereby specific alleles at a locus tend to either increase or decrease the expression of a trait when they are present. Many loci can have additive effects on a focal trait.

Anisogamy
Reproductive systems where female and male gametes are different sizes (Lehtonen et al., 2016).

Bet hedging
In general, a strategy where risk is diversified to decrease variance in success. In evolutionary biology, bet hedging is a strategy that lowers mean fitness for some individuals (arithmetic mean), but increases long-term fitness (geometric mean) across generations through lower variance in fitness among individuals.

Body condition
Often refers to energy reserves (i.e. fat or protein). Body condition or condition is often viewed as a measure of individual quality and is a key concept that underpins many hypotheses in behaviour, ecology and evolution. Here, we refer to a more contemporary definition of condition that emphasizes an animal's ability to convert food or stored energy into cellular energy (adenosine triphosphate, ATP). Focusing on energy utilization rather than energy availability highlights the role of mitochondria in determining the amount of energy available for animals to allocate to fitness-related traits (Hill, 2011).

Epistasis
Interactions between alleles at two or more loci such that the effect of an allele at one locus varies depending on the allele at another locus. Epistatic interactions affect trait expression through changes in the magnitude or direction of gene effects.

Good genes
Nuclear genes that increase the expression of fitness-related traits. Good genes show additive genetic effects and will increase in frequency in a population in response to directional selection (Neff and Pitcher, 2005).

Lek mating system
A reproductive strategy where males gather at specific sites (known as leks) to perform elaborate displays to attract females for mating. In lek mating systems, males do not provide direct resources to females or contribute to parental care (Kirkpatrick and Ryan, 1991).

Non-additive genetic effects
Genes can affect trait expression through non-additive effects where: (a) alleles interact within a locus (dominance) so the effect an allele has on a trait depends on the identity of the other allele at the locus; (b) there are interactions between loci (epistasis) that affect trait expression.

Maternal effects
The effects of a mother's phenotype on her offspring beyond the transmission of genetic material. These include maternal care and the maternal environment (e.g. egg yolk, maternal condition, hormone status; Mousseau and Fox, 1998).

Mitochondrial antioxidants
Mitochondrial compounds that degrade reactive oxygen species (ROS) that are produced during cellular respiration and protect mitochondria from oxidative damage (Mailloux, 2018; Murphy, 2012).

Mitonuclear complexes
Proteins that are part of the electron transport system (ETS; the site of OXPHOS) that are composed of subunits encoded by the mitochondrial (13 subunits) and nuclear (~73 subunits) genomes (McKenzie et al., 2007). Incompatibilities between the mitochondrial and nuclear subunits affect structural and biochemical properties and decrease the efficiency of the ETS (Wolff et al., 2014).

Respiratory control ratio (RCR)
A measurement of mitochondrial efficiency that is often calculated as the ratio of oxygen consumed as a result of maximal respiration to oxygen consumed through proton leak (state 3/state 4). High RCR values indicate that mitochondria have a high capacity to produce ATP and a low proton leak (Brand and Nicholls, 2011).

Oxidative phosphorylation (OXPHOS)
The final biochemical pathway of aerobic respiration involved in the production of ATP. The OXPHOS system is composed of ~85 proteins that are embedded in the inner mitochondrial membrane and are encoded by both the mitochondrial and nuclear genomes (Smeitink et al., 2001).

Uncoupling proteins
Proteins located in the inner mitochondrial membrane that act as proton carriers. Increased activation of uncoupling proteins (UCP1 – found in brown adipose tissue) increases proton leak across the inner mitochondrial membrane, which reduces the number of protons that flow through ATP synthase, resulting in decreased ATP production and increased heat production (Echtay, 2007; Rousset et al., 2004).

expression of sexually selected traits. Most hypotheses have focused on generic physiological traits that mediate sexual trait expression, such as body condition (see Glossary), immune function and hormone levels (e.g. Folstad and Karter, 1992; Gusdon et al., 2007; Hamilton and Zuk, 1982; McGraw et al., 2010; Morehouse, 2014; Tomkins et al., 2004). Broadly speaking, empirical studies have shown some associations between these physiological traits and sexual trait expression, but the direction of the associations is inconsistent, and the underlying mechanisms that regulate such relationships remain largely unresolved. Consequently, very few hypotheses have specified how the genetic basis of physiological mechanisms that affect sexually selected traits contributes to the maintenance of additive genetic variation for these traits (but see Hill, 2018, discussed below).

Here, we argue that understanding the proximate mechanisms that determine mitochondrial function could help explain the maintenance of additive genetic variation in sexually selected traits, despite the erosive effect of directional female mate choice. Mitochondria play a central role in the conversion of food into cellular energy, and variation in their efficiency affects the amount of energy that eukaryotic organisms can allocate to competing life processes (Box 1). Recent theoretical work has proposed that mitochondrial function affects the expression of sexually selected traits and could play a critical role in the evolution of mating preferences (Hill, 2014, 2018; Koch and Hill, 2018; Koch et al., 2017). We combine this theoretical work with an idea proposed by Miller and Moore (2007) that indirect genetic effects maintain variation in the expression of sexually selected traits through maternal effects (see Glossary).

Specifically, we propose that greater expression of sexually selected traits indicates superior mitochondrial function. Given that mitochondrial function is often influenced by the maternal

Journal of Experimental Biology (2024) 227, jeb245569. doi:10.1242/jeb.245569

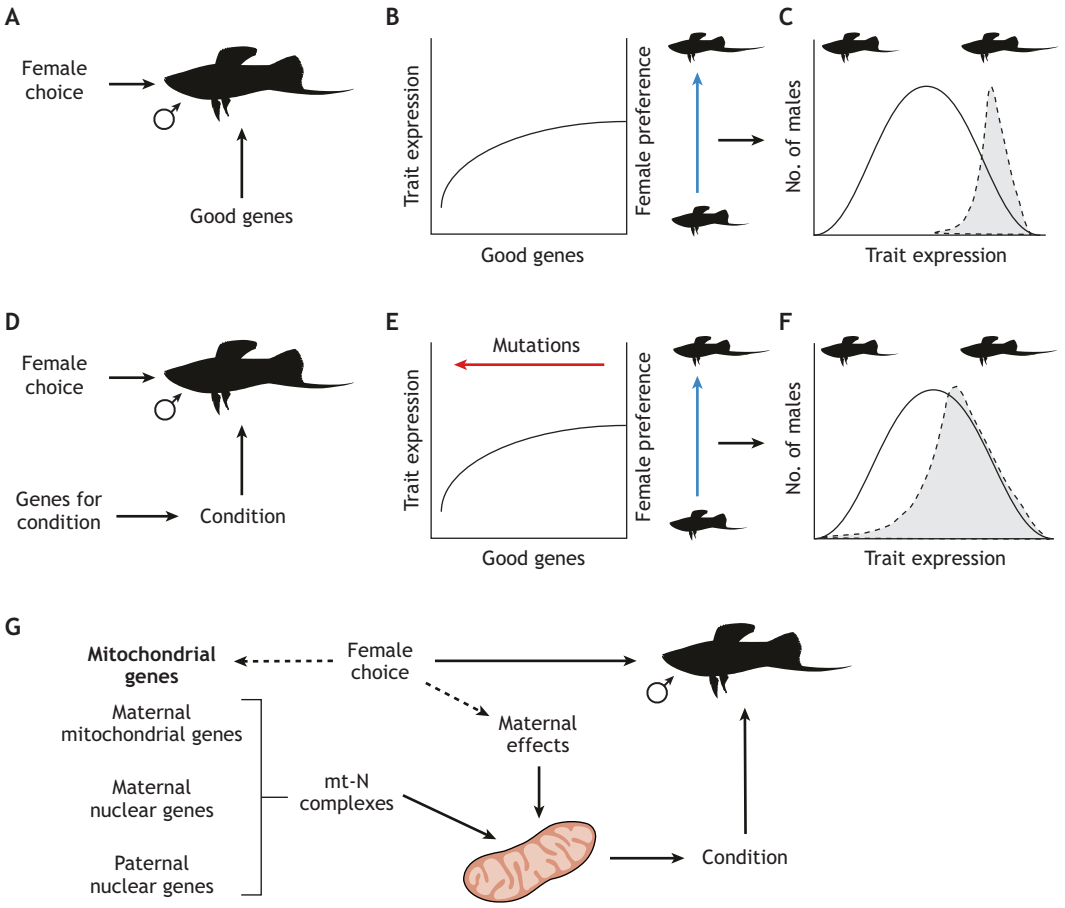

Fig. 1. Hypothesized pathways for how female preferences can affect tail length (a sexually selected trait) in male swordtail fish. (A) In this hypothetical example, females choose males based on tail length, and tail length is controlled additively by alleles at many loci (good genes). (B) Males with alleles that contribute positively to the expression of tail length (good genes) have longer tails, and females prefer males with the longest tails (blue arrow). (C) Female preference should reduce additive genetic variation and, consequently, should reduce variance in trait expression in a population, as only males with long tails are chosen (narrower dashed/grey distribution). However, genetic variation persists despite directional selection and variance in tail length is maintained (i.e. the lek paradox; solid line). (D) The 'genic capture' hypothesis proposes that additive genetic variation can persist when the expression of tail length is dependent on a male's condition. (E) Genes at many loci contribute to condition, making it more likely that new mutations that affect condition are 'captured' (red arrow; the 'genic capture' hypothesis; Rowe and Houle, 1996; Tomkins et al., 2004). (F) The loss of genetic variation is reduced, and more variance in tail length is maintained in the population. (G) An individual's condition is influenced by their ability to convert resources into ATP and, hence, mitochondrial function plays a central role in determining condition (Hill, 2011, 2014). Mitochondrial function is affected by developmental conditions such as those influenced by maternal effects (e.g. Armitage et al., 2005; Bruce and Hanson, 2010; Crino et al., 2022). Additionally, interactions between mitochondrial and nuclear genes affect mitochondrial function through mitonuclear (mt-N) complexes (Hill, 2019). Mothers can affect the expression of sexually selected traits in their offspring through maternal effects (Miller and Moore, 2007) and through their choice of a mate (who supplies half the nuclear genes for mitochondrial function; Hill and Johnson, 2013). In species where most females breed, females of poor quality will provide a poor-quality developmental environment for their offspring, resulting in sons with reduced mitochondrial function and poor-quality sexually selected traits. Here, a female can be viewed as choosing mates that will provide her with genes for daughters that will provide high-quality developmental environments for their future offspring and/or nuclear genes that will complement her mitochondrial genes and produce offspring with highly functional mt-N complexes (dashed arrows). Genetic variation of sexually selected traits can be maintained indirectly through genes that determine maternal performance and/or through selection for compatible mt-N complexes. Female choice for superior mitochondrial function could therefore maintain variance in tail length within a population. The fish image silhouette is from PhyloPic (www.phylopic.org) and was contributed by T. Michael Keesey (2023; CC0 1.0).

environment (see below), a male with superior sexual trait expression provides information to females about his mother's quality (which will be inherited by a choosy female's daughters if there is additive genetic variation in maternal effects). In this scenario, phenotypic variation in male sexually selected traits arises from two non-mutually exclusive sources that are both under female control: (1) variation in maternal effects that influence mitochondrial function in sons; and (2) the combination of mitochondrial and nuclear genes that a female provides to her offspring through her own genetic contribution and by her choice of mate (i.e. the 'mitonuclear compatibility model of sexual selection'; Hill and Johnson, 2013; see below). In both cases, choosy females can gain genetic benefits because being choosy improves mitochondrial function of their offspring in ways that increase: (a) the expression of sexually selected traits in their sons and (b) the fitness of their daughters, whose own offspring receive better maternal effects.

In this Commentary, we describe how indirect genetic effects for good 'maternal' genes can maintain genetic variation in condition-dependent, sexually selected male traits despite strong female preferences (Miller and Moore, 2007). We then discuss how mitochondrial function can link maternal effects to the expression of sexually selected traits and review empirical studies that link mitochondrial function to the expression of sexually selected

Journal of Experimental Biology (2024) 227, jeb245569. doi:10.1242/jeb.245569

Box 1. Mitochondrial bioenergetics and the role of mitonuclear complexes in determining mitochondrial efficiency

Mitochondria produce ∼90% of the adenosine triphosphate (ATP) used by eukaryotic animals (Lane and Martin, 2010). However, mitochondria vary in the rate and efficiency with which they convert metabolic substrates to ATP (Brand and Nicholls, 2011; Smeitink et al., 2001). Mitochondrial efficiency is influenced by mitonuclear complexes and regulated by cellular mechanisms that are affected by environmental conditions such as temperature and nutrition (Bennett et al., 2022; Zhu et al., 2014). The efficiency of mitochondria in converting substrates into ATP ultimately determines how much energy is available to allocate to competing life-history processes, such as reproduction and self-maintenance (Lane, 2011). Mitochondria also vary in the amount of heat and reactive oxygen species (ROS) they produce as by-products of cellular metabolism. ROS can cause oxidative damage and have been linked to increased senescence, telomere shortening and reduced longevity (Lushchak, 2014; Metcalfe and Olsson, 2022; Shields et al., 2021). Given that mitochondrial function affects both reproduction and survival through the production of ATP and ROS, variation in mitochondrial function is thought to underlie life-history strategies and to be inherently linked to processes such as sexual selection and speciation (Chou and Leu, 2010; Chung et al., 2018; Gangloff et al., 2020; Gershoni et al., 2009; Hill, 2015, 2018; Sloan et al., 2017).

Mitochondrial metabolic rate and efficiency are partly determined by the ability of mitochondria to establish and maintain a proton gradient between the intermembrane space and the mitochondrial matrix (i.e. membrane potential; Brand and Nicholls, 2011). Mitochondria establish membrane potential by the active pumping of protons from the matrix to the intermembrane space (using complexes I, II and IV). The energy needed to transport protons is harnessed from the movement of electrons across electron transport system (ETS) complexes as they move to positions of lower energy (Lane, 2015). During oxidative phosphorylation, ATP is produced by the ETS when protons interact with ATP synthase (complex V) as they cross from the intermembrane space (high proton concentration) to the mitochondrial matrix (low proton concentration; Brand and Nicholls, 2011). Mitonuclear gene interactions affect the fit of subunits on the inner mitochondrial membrane and can reduce electron flow, resulting in decreased ATP production (Hill, 2019; Lane, 2015). Poor compatibility between mitonuclear gene complexes can also affect gene replication, transcription and translation, as well as increase ROS and mitochondrial oxidative damage (Burton, 2022; Gusdon et al., 2007; Moreno-Loshuertos et al., 2006). For example, in a *Drosophila* model, flies with incompatible mitochondrial and nuclear genes have decreased mitochondrial oxygen consumption, increased ROS production and increased mitochondrial copy number compared with flies with compatible genes (Pichaud et al., 2019). At an organismal level, incompatibilities between mitochondrial and nuclear genes can affect both reproductive success and longevity (Healy and Burton, 2020; Hill et al., 2019a; Wolff et al., 2014).

ornaments (carotenoid coloration) and displays (bird song). We highlight how maternal effects and interactions between the mitochondrial and nuclear genomes drive variation in mitochondrial function. We then discuss the implications of these processes for mate choice and consider whether including measurements of mitochondrial function in models of sexual selection can help to resolve the lek paradox. Finally, we propose directions for future studies to test the strength of the proposed links between mitochondrial function, maternal effects, sexually selected traits and female mating preferences.

Resolving the lek paradox for condition-dependent sexually selected traits – a potential role of maternal effects

Sexually selected traits often exhibit more phenotypic variation than other traits under natural selection (Pomiankowski and Moller, 1995), even though directional sexual selection due to female mate choice should reduce additive genetic variation. All else being equal, a decline in phenotypic variation in sexual trait expression should make it harder for females to discriminate among males (Fig. 1A–C). Phenotypic and additive genetic variation in sexually selected traits could be maintained more easily if genes at many loci contribute to the expression of sexual traits. With more loci, there is a greater likelihood that mutations generate phenotypic variation and a lower likelihood that alleles reach fixation as a result of directional selection (Houle, 1992; Pomiankowski and Moller, 1995). The maintenance of additive genetic variation in this way is particularly likely for sexually selected traits whose expression is dependent on condition. Theory proposes that condition-dependent sexually selected traits are costly to produce and/or maintain (e.g. Jennions et al., 2001; but see Kotiaho, 2001). Consequently, individuals in good condition that pay smaller marginal costs can afford to invest more heavily in these traits compared with individuals in poor condition (Kuijper et al., 2012). An individual's condition is likely to depend on many loci (Houle, 1992; Tomkins et al., 2004). Therefore, additive genetic variation in sexually selected traits is more likely to be maintained, despite strong directional selection, if they are condition dependent, because a large proportion of the genome affects condition, creating a bigger 'mutational target'. Consequently, mutations in genes that affect condition are 'captured' and additive genetic variation in sexually selected traits is maintained (the 'genic capture' hypothesis; Rowe and Houle, 1996; Tomkins et al., 2004; Fig. 1D–F).

In most models of mate choice evolution that invoke body condition, it is assumed that sexually selected traits signal additive genetic variation that directly elevates a male's condition and, thus, his ability to express and maintain elaborate ornaments or displays. Miller and Moore (2007) put a twist on this logic by pointing out that mothers can affect the phenotype of their offspring beyond their own genetic contribution through maternal effects (Mousseau and Fox, 1998). Their rationale is clever and, in their own words, 'simple': a mother can affect the environment her offspring experience during development and her ability to do so can be influenced by her genes. Specifically, Miller and Moore (2007) acknowledged that mothers often play a fundamental role in shaping the developmental environment, which can have strong effects on the body condition of their offspring (e.g. Cunningham and Russell, 2000; Love et al., 2005; but see Merila et al., 2001). Maternal effects can thus affect the expression of sexually selected traits in her sons through developmentally induced changes that influence their condition as adults. Crucially, the magnitude of maternal effects can vary, in part, as a result of genes that the mother bears (e.g. genes for better parental care or more egg yolk). Therefore, when a female chooses a male based on sexually selected traits, she obtains genes for daughters who are better mothers that provide a higher-quality developmental environment for their offspring and not just genes for sexy sons. A choosy female will therefore have fitter grandsons (i.e. increased sexual trait expression in grandsons) because of the superior maternal environment that her daughters provide.

Condition-dependent sexually selected traits – potential links to mitochondrial function

A shared component of the genic capture hypothesis and the ideas proposed by Miller and Moore (2007) is that variation in condition among males generates variation in sexually selected traits. Historically, 'body condition' has been defined as the total pool of resources available to an individual to allocate to fitness-related traits (Miller and Moore, 2007; Rowe and Houle, 1996; Tomkins

et al., 2004). A fundamental challenge to empirical studies that seek to link variation in body condition to variation in the expression of sexually selected traits is that body condition (as defined above) is difficult to quantify and often obligates the use of morphometric indices (reviewed in Jakob et al., 1996; Kraft et al., 2019; Peig and Green, 2010). Many common body condition indices are reflective of body fat (Kraft et al., 2019). However, they may not be biologically relevant depending on the natural history of the focal species, the environmental conditions individuals experience or the life history stage of individuals. As such, they do not always predict fitness (Barnett et al., 2015; Sánchez et al., 2018; Wilder et al., 2016).

Recent theoretical work that discusses condition in terms of not just energy availability but also energy utilization may offer a resolution (e.g. Hill, 2011). In eukaryotes, energy utilization depends on cellular respiration, including the process of oxidative phosphorylation (see Glossary) where mitochondria convert substrates derived from food into adenosine triphosphate (ATP; Box 1). Importantly, mitochondrial structure and function are determined by gene products that are encoded by both the mitochondrial and nuclear genomes (Poyton and McEwen, 1996). It has been proposed that mitochondrial function provides the most fundamental information about a potential mate's quality to females because of the role of mitochondria in the production of ATP and overall organismal function (Hill, 2011; Hill and Johnson, 2013). However, females cannot directly evaluate the mitochondrial function of their potential mates. Instead, sexually selected traits that are energetically expensive to produce and maintain are hypothesized to signal a potential mate's mitochondrial function (Hill, 2011; Hill and Johnson, 2013; Koch and Hill, 2018). Including energy utilization in the definition of condition provides a physiological mechanism (mitochondrial function) that links genes to condition and, ultimately, to the expression of sexually selected traits (Fig. 1G).

Empirical evidence supports a mechanistic link between mitochondrial function and the expression of sexually selected traits. For example, male house finches (*Haemorhous mexicanus*) with redder feathers are more attractive to females (Hill, 1990, 1991), and Hill et al. (2019b) found that plumage redness was positively correlated with the efficiency of liver mitochondria. Specifically, redder males had higher respiratory control ratios (see Glossary), increased mitochondrial membrane potential and less proton leak than duller-coloured birds (Hill et al., 2019b). Other studies have found that drug treatments that increase mitochondrial antioxidants (see Glossary) increase bill redness (Cantarero and Alonso-Alvarez, 2017; Cantarero et al., 2020), further supporting a mechanistic link between mitochondrial function and carotenoid-coloured ornaments. Mitochondrial function has also been invoked to explain the evolution of sexually selected behaviours, such as bird song (Koch and Hill, 2018). For example, male zebra finches (*Taeniopygia castanotis*) with lower mitochondrial respiration capacity and less efficient mitochondria have lower-frequency songs (a trait important for mate choice; Mikula et al., 2021) than males with greater mitochondrial performance (Crino et al., 2022). Mitochondrial function could affect behavioural displays through effects on neurogenesis that determine an individual's ability to learn and perform complex cognitive tasks, or by limiting the energy available to perform displays or to allocate to morphological structures used in displays (Crino et al., 2022; Koch and Hill, 2018).

Variation in mitochondrial function – the role of maternal effects and compatible mitonuclear complexes

Mitochondria vary in the rate and efficiency of ATP production and in the production of metabolic by-products such as reactive oxygen species (ROS). One source of variation in mitochondrial function arises from the conditions that individuals experience during development through maternal effects. In support of this claim, there is a rich biomedical literature from studies with mammals that link maternal diet and body condition during gestation to long-term metabolic consequences for offspring due to changes in mitochondrial function (reviewed in Armitage et al., 2005; Bruce and Hanson, 2010; Fernandez-Twinn and Ozanne, 2010). In addition to maternal nutrition, studies have identified other developmental conditions that induce long-term effects on mitochondrial function. For example, Stier et al. (2022) found that adult Japanese quail (*Coturnix japonica*) that experienced an elevated incubation temperature (38.4°C) had higher mitochondrial respiration rates than quail that were incubated at a cooler temperature (37.0°C). Similarly, studies in zebra finches found that exposure to elevated temperatures during development has sustained effects on mitochondrial function that persist into adulthood (Pacheco-Fuentes et al., 2023; Ton et al., 2021; Udino et al., 2021). Exposure to elevated temperatures during development could cause sustained changes in mitochondrial function through epigenetic modifications that affect the expression of mitochondrial uncoupling proteins (see Glossary) (Argyropoulos and Harper, 2002; Gao et al., 2021). Alternatively, higher temperatures could cause changes in mitochondrial function through elevation of glucocorticoid hormones. Glucocorticoids are steroid hormones that regulate mitochondrial function and play a role in the vertebrate 'stress' response (McEwen and Wingfield, 2003; Picard et al., 2018). Nestling birds exposed to elevated rather than control levels of glucocorticoids have less-efficient mitochondria as adults (Casagrande et al., 2020; Crino et al., 2022), and these effects are thought to be regulated through sustained changes in the neuroendocrine pathway that regulates glucocorticoid release, which, in turn, affects mitochondrial function into adulthood (Crino et al., 2022). Together, these studies demonstrate that conditions during development can have long-term effects on mitochondrial function.

Another potential source of variation in mitochondrial function arises from variable compatibility between nuclear and mitochondrial genes. Processes such as mitochondrial gene replication, transcription and translation, and the generation of ATP through oxidative phosphorylation depend on interactions between mitochondrial and nuclear genes (Burton and Barreto, 2012). For example, mitochondrial genes code for proteins that combine with proteins encoded by nuclear genes to form complexes that are integral to the process of oxidative phosphorylation. These mitonuclear complexes (see Glossary) must 'fit' together precisely for mitochondria to function efficiently. Incompatibilities between mitochondrial and nuclear genes reduce the fit of mitonuclear complexes and, consequently, the ability of electrons to move through the electron transport system (Hill, 2019; Lane, 2015). A poorly fitting complex reduces ATP production and increases the production of ROS (Brand and Nicholls, 2011). Empirical studies suggest that selection strongly favours compatible mitonuclear complexes (Healy and Burton, 2020; Pereira et al., 2021). A female contributes all her mitochondrial genome to her offspring but only half of her nuclear genome, and she can potentially increase offspring fitness by choosing a mate whose nuclear genes are more likely to be compatible with her mitochondrial genes (Hill, 2018; Hill and Johnson, 2013).

Mitochondrial function, mate choice and the lek paradox

If condition-dependent sexual traits signal mitochondrial function, a key question that follows is how this information might benefit

choosy females. The mitochondrial genome is transmitted from mothers to their offspring, so a father's mitochondrial genes will not affect his offspring. However, we propose that females can benefit from choosing a mate that demonstrates superior mitochondrial function in scenarios where mitochondrial function is influenced by maternal effects and/or indicative of compatible mitochondrial and nuclear genes (i.e. the 'mitonuclear compatibility model of sexual selection').

Maternal effects and mitochondrial function – 'nurturing daughters' rather than 'sexy sons'

Miller and Moore (2007) proposed that when maternal effects have a heritable basis, a female will benefit from choosing males with high-quality sexually selected traits because her daughters are likely to inherit genes for superior maternal care. Briefly, they point out that sexual traits can signal the quality of the maternal environment that a male received. Factors that maintain additive genetic variation in maternal effects therefore also maintain variation in the genetic benefits signalled by sexual traits. We expand on this idea by suggesting that mitochondrial function is the key physiological mechanism that links maternal effects to the expression of sexual traits. Given that females are more likely than males to reproduce, regardless of their quality (Arnold, 1994; Janicke et al., 2016), there will be persistent variation in the sexual traits of sons due to variation in maternal effects. Even so, sons of low-quality mothers are unlikely to breed because of their low attractiveness, thereby eliminating some genes for poor maternal effects. In contrast, daughters of low-quality mothers will still tend to reproduce, so the consequences of genes for poor maternal effects are partially hidden from selection, thereby slowing erosion of additive genetic variation in sexual traits.

Mitonuclear compatibility and mitochondrial function – the search for compatible mates

Mitochondrial function can also be influenced by mate choice that results in compatible mitonuclear complexes for offspring. The mitonuclear compatibility model of sexual selection shifts the focus of female choice away from 'good genes' (see Glossary) to nuclear genes that create compatible mitonuclear complexes (Hill, 2018; Hill and Johnson, 2013). The logic is reminiscent of non-additive models of sexual selection that propose that females seek out 'genetically compatible' mates, such that the combination of maternal and paternal genes increases offspring fitness (i.e. non-additive genetic effects; see Glossary; Aguirre et al., 2016; Zeh and Zeh, 1996, 1997). Here, the expression of sexually and naturally selected traits in offspring, and hence their fitness, is determined by interactions between paternal and maternal genes (e.g. increased heterozygosity can elevate fitness; Fromhage et al., 2009; Mays and Hill, 2004). Non-additive models of sexual selection propose that variation in sexual traits can be maintained by interactions between alleles at a locus (dominance) or through epistasis (see Glossary) when population size, mutation rates and allele frequencies are low (Neff and Pitcher, 2008). In these scenarios, non-additive gene actions can explain persistent genetic variation in sexually selected traits (Neff and Pitcher, 2008, 2009; Puurtinen et al., 2009). The mitonuclear compatibility model of sexual selection proposes that genetic variation in sexually selected traits can be maintained because new combinations of mitonuclear genes arise each generation, providing an extensive source of variation despite directional selection (Hill, 2018). This hypothesis proposes a novel explanation for how mitochondrial and nuclear genes interact at an individual level to generate genetic variation in sexually selected traits. Future studies that use mathematical models could test what factors determine the extent to which mitonuclear compatibility maintains genetic variation in sexually selected traits.

Female choice for compatible mitonuclear complexes is only useful if males with better mitochondrial function transfer nuclear genes that increase the likelihood of siring offspring with above-average mitochondrial function. A male may signal that his mitonuclear complexes are highly compatible through the expression of sexually selected traits, but that does not guarantee that his nuclear genes will be compatible with a choosy female's mitochondrial and nuclear genes, and result in offspring with highly compatible complexes. However, there are far fewer mitochondrial than nuclear genes, which suggests that most of the variation in mitochondrial function is due to nuclear rather than mitochondrial genes (Hill, 2019). As such, a female who chooses a male that demonstrates highly compatible mitonuclear complexes is more likely to receive nuclear genes for her offspring that produce compatible complexes than is a female who mates randomly. One way to envisage this is to take the extreme example of a species with only two mitochondrial haplotypes. In this scenario: (a) a female is more likely to transmit the more common haplotype to her offspring; and (b) a male is more likely to have this haplotype. As such, a male whose ornaments reveal that he has superior mitochondrial function is signalling that he has nuclear genes that are likely to be compatible with the mitochondrial haplotype that the female will transmit to her offspring. By extension of this argument, as mitochondrial genetic variation increases, females are less likely to receive nuclear genes from a mate that are compatible with her mitochondrial genes.

An alternative possibility is that females increase the chance that some of their offspring have highly compatible mitonuclear complexes through an adaptive bet-hedging strategy (see Glossary). For example, if a female mates with multiple males, some males will provide nuclear genes that are more compatible with her mitochondrial genes than others, even if all the males have high-quality sexual traits. In this scenario, some of her offspring will perform well and some will not. Conversely, if a female mates singly, then either all or none of her offspring will receive nuclear genes that produce highly compatible mitonuclear complexes and all or none of her offspring will perform well. In this way, mating multiply increases variation in mitochondrial function within a female's offspring and may decrease her fitness compared with mating singly. However, selection will favour such a bet hedging strategy compared with an invariant strategy if it lowers variance in fitness among females and increases long-term fitness across generations (Philippi and Seger, 1989; Seger and Brockmann, 1987; Starrfelt and Kokko, 2012). In general, models that test the adaptive value of bet-hedging strategies, such as multiple mating, find that the parental fitness gains from lower variation in offspring fitness are small, present under restricted circumstances (e.g. small populations, high environmental variation) and can easily be outweighed by even small costs imposed by bet hedging (e.g. sexual diseases in the case of multiple matings; Holman, 2016; Starrfelt and Kokko, 2012; Yasui, 2001). However, the potential benefits of a bet hedging strategy in relation to mitonuclear compatibility have yet to be formally modelled.

Conclusions and future directions

The core of the lek paradox is that choosy females obtain genes that enhance mean offspring fitness through a combination of naturally and sexually selected benefits for sons and/or daughters by mating with males that invest more in sexually selected traits (Kokko et al.,

2006). This benefit of choosiness requires that there is genetic variation in fitness even though choosiness should deplete variation. Here, we have proposed that additive genetic variation in sexually selected traits can be maintained through indirect selection on maternal effects that influence the expression of sexually selected traits through changes in mitochondrial function. We have expanded on proposals that variation in mitochondrial function underlies variation in the expression of sexually selected traits (Hill, 2011, 2014, 2018, 2022; Hill and Johnson, 2013; Koch and Hill, 2018; Koch et al., 2017). Variation in mitochondrial function among individuals can arise both from the quality of the environment experienced during development (i.e. maternal effects) and from mitonuclear gene interactions. Both sources of variation are strongly influenced by the maternal genotype. In species where most females breed, some females provide a poor-quality developmental environment and/or transfer mitochondrial and nuclear genes that increase the likelihood of incompatible mitonuclear complexes. Males produced by such mothers will have reduced mitochondrial function and, consequently, lower expression of sexually selected traits.

The pathway we propose provides a partial resolution to the lek paradox that captures the salient points of other widely accepted hypotheses and suggests avenues for future research. First, mitochondrial function determines an individual's ability to convert food resources to cellular energy and is therefore intrinsically linked to condition-dependent sexually selected traits which underpin the 'genic capture' hypothesis (Houle, 1992). Second, we are building on ideas proposed by Miller and Moore (2007) by identifying a physiological mechanism (mitochondrial function) that links maternal genes (i.e. genes for good maternal effects) to the expression of sexually selected traits in sons. Finally, these ideas are compatible with hypotheses that invoke genetic compatibility to explain the maintenance of female preferences (i.e. non-additive genetic benefits; Aguirre et al., 2016; Zeh and Zeh, 1996, 1997), by noting that females should choose mates with nuclear genes that increase mitonuclear compatibility (and, hence, mitochondrial function) in their offspring (Hill, 2018). Overall, our ideas highlight the need to look more closely at the sources of variation in mitochondrial function in model study systems for sexual selection.

There has been a recent boom in evolutionary and ecological studies of mitochondrial bioenergetics (Koch et al., 2021), and there is compelling evidence that links mitochondrial function to the expression of sexually selected traits (e.g. Crino et al., 2022; Hill et al., 2019b). Even so, this area of research is in its infancy. Future studies need to test the extent to which mitochondrial function affects a wider range of sexually selected ornaments and displays. Additionally, we need to disentangle the different physiological pathways that influence mitochondrial function. Studies that experimentally alter mitochondrial function using drugs such as 2,4-dinitrophenol (DNP), mitoquinone mesylate (MitoQ; e.g. Cantarero and Alonso-Alvarez, 2017; Stier et al., 2014) or mitochondria-targeted antioxidants (Jiang et al., 2020; Zinovkin et al., 2023) could be particularly informative. New methodologies now allow for non-terminal sampling of mitochondrial function from small animals (Stier et al., 2017). Such advances allow for longitudinal studies in many model systems and open promising avenues of research. Finally, formal mathematical models are needed to validate verbal arguments that sexually selected traits in males signal additive genetic variation in nuclear genes that affect mitochondrial function, be this through the nuclear and mitochondrial genes they express or because of maternally inherited genes that affect the expression of the bearer's sexual traits. Identifying the benefits females gain from choosing males with specific traits is crucial to understanding the process of sexual selection. Thus, we will improve our understanding of fundamental principles in sexual selection by considering the role that females play in driving the expression of sexually selected traits in sons through effects on mitochondrial function.

Acknowledgements
We thank three anonymous reviewers for suggestions that improved the quality of this manuscript. O.L.C. thanks H. Jane Brockman and the participants in her Animal Behaviour reading group at the University of Florida from 2005 to 2008, especially Matt D. Smith.

Competing interests
The authors declare no competing or financial interests.

Funding
This research was funded by an Australian Research Council grant to D.W.A.N. (DP210101152).

References
Aguirre, J. D., Blows, M. W. and Marshall, D. J. (2016). Genetic compatibility underlies benefits of mate choice in an external fertilizer. *Am. Nat.* **187**, 647-657. doi:10.1086/685892

Andersson, M. (1994). *Sexual Selection*. Princeton, NJ: Princeton University Press.

Argyropoulos, G. and Harper, M. E. (2002). Uncoupling proteins and thermoregulation. *J. Appl. Physiol.* **92**, 2187-2198. doi:10.1152/japplphysiol.00994.2001

Armitage, J. A., Taylor, P. D. and Poston, L. (2005). Experimental models of developmental programming: consequences of exposure to an energy rich diet during development. *J. Physiol.* **565**, 3-8. doi:10.1113/jphysiol.2004.079756

Arnold, S. J. (1994). Batemans's principles and the measurement of sexual selection in plants and animals. *Am. Nat.* **144**, S126-S149. doi:10.1086/285656

Barnett, C. A., Suzuki, T. N., Sakaluk, S. K. and Thompson, C. F. (2015). Mass-based condition measures and their relationship with fitness: in what condition is condition? *J. Zool.* **296**, 1-5. doi:10.1111/jzo.12213

Bennett, C. F., Latorre-Muro, P. and Puigserver, P. (2022). Mechanisms of mitochondrial respiratory adaptation. *Nat. Rev. Mol. Cell Biol.* **23**, 817-835. doi:10.1038/s41580-022-00506-6

Bonduriansky, R. and Day, T. (2013). Nongenetic inheritance and the evolution of costly female preference. *J. Evol. Biol.* **26**, 76-87. doi:10.1111/jeb.12028

Bonilla, M. M., Zeh, J. A. and Zeh, D. W. (2016). An epigenetic resolution of the lek paradox. *BioEssays* **38**, 355-366. doi:10.1002/bies.201500176

Brand, M. D. and Nicholls, D. G. (2011). Assessing mitochondrial dysfunction in cells. *Biochem. J.* **435**, 297-312. doi:10.1042/BJ20110162

Bruce, K. D. and Hanson, M. A. (2010). The Developmental Origins, Mechanisms, and Implications of Metabolic Syndrome. *J. Nutr.* **140**, 648-652. doi:10.3945/jn.109.111179

Burton, R. S. (2022). The role of mitonuclear incompatibilities in allopatric speciation. *Cell. Mol. Life Sci.* **79**, 103. doi:10.1007/s00018-021-04059-3

Burton, R. S. and Barreto, F. S. (2012). A disproportionate role for mtDNA in Dobzhansky-Muller incompatibilities? *Mol. Ecol.* **21**, 4942-4957. doi:10.1111/mec.12006

Cantarero, A. and Alonso-Alvarez, C. (2017). Mitochondria-targeted molecules determine the redness of the zebra finch bill. *Biol. Lett.* **13**, 20170455. doi:10.1098/rsbl.2017.0455

Cantarero, A., Andrade, P., Carneiro, M., Moreno-Borrallo, A. and Alonso-Alvarez, C. (2020). Testing the carotenoid-based sexual signalling mechanism by altering CYP2J19 gene expression and colour in a bird species. *Proc. R. Soc. B Biol. Sci.* **287**, 20201067. doi:10.1098/rspb.2020.1067

Casagrande, S., Stier, A., Monaghan, P., Loveland, J. L., Boner, W., Lupi, S., Trevisi, R. and Hau, M. (2020). Increased glucocorticoid concentrations in early life cause mitochondrial inefficiency and short telomeres. *J. Exp. Biol.* **223**, jeb222513. doi:10.1242/jeb.222513

Chou, J. Y. and Leu, J. Y. (2010). Speciation through cytonuclear incompatibility: insights from yeast and implications for higher eukaryotes. *BioEssays* **32**, 401-411. doi:10.1002/bies.200900162

Chung, D. J., Healy, T. M., McKenzie, J. L., Chicco, A. J., Sparagna, G. C. and Schulte, P. M. (2018). Mitochondria, temperature, and the pace of life. *Integr. Comp. Biol.* **58**, 578-590. doi:10.1093/icb/icy013

Crino, O. L., Falk, S., Katsis, A. C., Kraft, F. and Buchanan, K. L. (2022). Mitochondria as the powerhouses of sexual selection: testing mechanistic links between development, cellular respiration, and bird song. *Horm. Behav.* **142**, 105184. doi:10.1016/j.yhbeh.2022.105184

Cunningham, E. J. A. and Russell, A. F. (2000). Egg investment is influenced by male attractiveness in the mallard. *Nature* **404**, 74-77. doi:10.1038/35003565

Echtay, K. S. (2007). Mitochondrial uncoupling proteins - What is their physiological role? *Free Radic. Biol. Med.* **43**, 1351-1371. doi:10.1016/j.freeradbiomed.2007.08.011
Fernandez-Twinn, D. S. and Ozanne, S. E. (2010). Early life nutrition and metabolic programming. *Year Diabetes Obes.* **1212**, 78-96.
Fisher, R. A. (1930). *The Genetical Theory of Natural Selection*. Oxford: Claredon Press.
Folstad, I. and Karter, A. J. (1992). Parasites, bright males, and the immunocompetence handicap. *Am. Nat.* **139**, 603-622. doi:10.1086/285346
Fromhage, L. and Jennions, M. D. (2016). Coevolution of parental investment and sexually selected traits drives sex-role divergence. *Nat. Commun.* **7**, 12517. doi:10.1038/ncomms12517
Fromhage, L., Kokko, H. and Reid, J. M. (2009). Evolution of mate choice for genome-wide heterozygosity. *Evolution* **63**, 684-694. doi:10.1111/j.1558-5646.2008.00575.x
Gangloff, E. J., Schwartz, T. S., Klabacka, R., Huebschman, N., Liu, A. Y. and Bronikowski, A. M. (2020). Mitochondria as central characters in a complex narrative: linking genomics, energetics, pace -of -life, and aging in natural populations of garter snakes. *Exp. Gerontol.* **137**, 110967. doi:10.1016/j.exger.2020.110967
Gao, W., Liu, J. L., Lu, X. and Yang, Q. (2021). Epigenetic regulation of energy metabolism in obesity. *J. Mol. Cell Biol.* **13**, 480-499. doi:10.1093/jmcb/mjab043
Gershoni, M., Templeton, A. R. and Mishmar, D. (2009). Mitochondrial bioenergetics as a major motive force of speciation. *BioEssays* **31**, 642-650. doi:10.1002/bies.200800139
Gusdon, A. M., Votyakova, T. V., Reynolds, I. J. and Mathews, C. E. (2007). Nuclear and mitochondrial interaction involving mt-Nd2 leads to increased mitochondrial reactive oxygen species production. *J. Biol. Chem.* **282**, 5171-5179. doi:10.1074/jbc.M609367200
Hamilton, W. D. and Zuk, M. (1982). Heritable True Fitness and Bright Birds - a Role for Parasites. *Science* **218**, 384-387. doi:10.1126/science.7123238
Healy, T. M. and Burton, R. S. (2020). Strong selective effects of mitochondrial DNA on the nuclear genome. *Proc. Natl. Acad. Sci. U.S.A.* **117**, 6616-6621. doi:10.1073/pnas.1910141117
Hill, G. E. (1990). Female house finches prefer colorful males - sexual selection for a condition-dependent trait. *Anim. Behav.* **40**, 563-572. doi:10.1016/S0003-3472(05)80537-8
Hill, G. E. (1991). Plumage coloration is a sexually selected indicator of male quality. *Nature* **350**, 337-339. doi:10.1038/350337a0
Hill, G. E. (2011). Condition-dependent traits as signals of the functionality of vital cellular processes. *Ecol. Lett.* **14**, 625-634. doi:10.1111/j.1461-0248.2011.01622.x
Hill, G. E. (2014). Cellular respiration: the nexus of stress, condition, and ornamentation. *Integr. Comp. Biol.* **54**, 645-657. doi:10.1093/icb/icu029
Hill, G. E. (2015). Mitonuclear ecology. *Mol. Biol. Evol.* **32**, 1917-1927. doi:10.1093/molbev/msv104
Hill, G. E. (2018). Mitonuclear mate choice: a missing component of sexual selection theory? *BioEssays* **40**. doi:10.1002/bies.201700191
Hill, G. E. (2019). *Mitonuclear Ecology*. Oxford University Press.
Hill, G. E. (2022). Evolution: the biochemistry of honest sexual signaling. *Curr. Biol.* **32**, R1005. doi:10.1016/j.cub.2022.09.001
Hill, G. E. and Johnson, J. D. (2013). The mitonuclear compatibility hypothesis of sexual selection. *Proc. R. Soc. B Biol. Sci.* **280**, 20131314. doi:10.1098/rspb.2013.1314
Hill, G. E., Havird, J. C., Sloan, D. B., Burton, R. S., Greening, C. and Dowling, D. K. (2019a). Assessing the fitness consequences of mitonuclear interactions in natural populations. *Biol. Rev.* **94**, 1089-1104. doi:10.1111/brv.12493
Hill, G. E., Hood, W. R., Ge, Z. Y., Grinter, R., Greening, C., Johnson, J. D., Park, N. R., Taylor, H. A., Andreasen, V. A., Powers, M. J. et al. (2019b). Plumage redness signals mitochondrial function in the house finch. *Proc. R. Soc. B Biol. Sci.* **286**, 20191354. doi:10.1098/rspb.2019.1354
Holman, L. (2016). Bet hedging via multiple mating: a meta-analysis. *Evolution* **70**, 62-71. doi:10.1111/evo.12822
Houle, D. (1992). Comparing evolvability and variability of quantitative traits. *Genetics* **130**, 195-204. doi:10.1093/genetics/130.1.195
Iwasa, Y. and Pomiankowski, A. (1999). Good parent and good genes models of handicap evolution. *J. Theor. Biol.* **200**, 97-109. doi:10.1006/jtbi.1999.0979
Jakob, E. M., Marshall, S. D. and Uetz, G. W. (1996). Estimating fitness: a comparison of body condition indices. *Oikos* **77**, 61-67. doi:10.2307/3545585
Janicke, T., Haderer, I. K., Lajeunesse, M. J. and Anthes, N. (2016). Darwinian sex roles confirmed across the animal kingdom. *Sci. Adv.* **2**, e100983. doi:10.1126/sciadv.1500983
Jennions, M. D., Moller, A. P. and Petrie, M. (2001). Sexually selected traits and adult survival: a meta-analysis. *Q Rev. Biol.* **76**, 3-36. doi:10.1086/393743
Jiang, Q., Yin, J., Chen, J., Ma, X., Wu, M., Liu, G., Yao, K., Tan, B. and Yin, Y. (2020). Mitochondria-targeted antioxidants: a step towards disease treatment. *Oxid. Med. Cell. Longev.* **2020**, 8837893. doi:10.1155/2020/8837893
Kirkpatrick, M. (1996). Good genes and direct selection in evolution of mating preferences. *Evolution* **50**, 2125-2140. doi:10.2307/2410684
Kirkpatrick, M. and Ryan, M. J. (1991). The evolution of mating preferences and the paradox of the lek. *Nature* **350**, 33-38. doi:10.1038/350033a0
Koch, R. E. and Hill, G. E. (2018). Behavioural mating displays depend on mitochondrial function: a potential mechanism for linking behaviour to individual condition. *Biol. Rev.* **93**, 1387-1398. doi:10.1111/brv.12400
Koch, R. E., Josefson, C. C. and Hill, G. E. (2017). Mitochondrial function, ornamentation, and immunocompetence. *Biol. Rev.* **92**, 1459-1474. doi:10.1111/brv.12291
Koch, R. E., Buchanan, K. L., Casagrande, S., Crino, O., Dowling, D. K., Hill, G. E., Hood, W. R., McKenzie, M., Mariette, M. M., Noble, D. W. A. et al. (2021). Integrating mitochondrial aerobic metabolism into ecology and evolution. *Trends Ecol. Evol.* **36**, 321-332. doi:10.1016/j.tree.2020.12.006
Kokko, H., Brooks, R., Jennions, M. D. and Morley, J. (2003). The evolution of mate choice and mating biases. *Proc. R. Soc. B Biol. Sci.* **270**, 653-664. doi:10.1098/rspb.2002.2235
Kokko, H., Jennions, M. D. and Brooks, R. (2006). Unifying and testing models of sexual selection. *Ann. Rev. Ecol. Evol. Syst.* **37**, 43-66. doi:10.1146/annurev.ecolsys.37.091305.110259
Kokko, H., Booksmythe, I. and Jennions, M. D. (2015). Mate sampling costs and sex sons. *J. Evol. Biol.* **28**, 259-266. doi:10.1111/jeb.12532
Kotiaho, J. S. (2001). Costs of sexual traits: a mismatch between theoretical considerations and empirical evidence. *Biol. Rev.* **76**, 365-376. doi:10.1017/S1464793101005711
Kotiaho, J. S., Lebas, N. R., Puurtinen, M. and Tomkins, J. L. (2008). On the resolution of the lek paradox. *Trends Ecol. Evol.* **23**, 1-3. doi:10.1016/j.tree.2007.09.012
Kraft, F. O. H., Driscoll, S. C., Buchanan, K. L. and Crino, O. L. (2019). Developmental stress reduces body condition across life-history stages: a comparison of quantitative magnetic resonance data and condition indices. *Gen. Comp. Endocrinol.* **272**, 33-41. doi:10.1016/j.ygcen.2018.11.008
Kuijper, B., Pen, I. and Weissing, F. J. (2012). A guide to sexual selection theory. *Ann. Rev. Ecol.Evol. Syst.* **43**, 287. doi:10.1146/annurev-ecolsys-110411-160245
Lane, N. (2011). Mitonuclear match: optimizing fitness and fertility over generations drives ageing within generations. *BioEssays* **33**, 860-869. doi:10.1002/bies.201100051
Lane, N. (2015). *The Vital Question: Energy, Evolution, and the Origins of Complex Life*. New York: WW Norton & Company.
Lane, N. and Martin, W. (2010). The energetics of genome complexity. *Nature* **467**, 929-934. doi:10.1038/nature09486
Lehtonen, J., Parker, G. A. and Scharer, L. (2016). Why anisogamy drives ancestral sex roles. *Evolution* **70**, 1129-1135. doi:10.1111/evo.12926
Love, O. P., Chin, E. H., Wynne-Edwards, K. E. and Williams, T. D. (2005). Stress hormones: a link between maternal condition and sex-biased reproductive investment. *Am. Nat.* **166**, 751-766. doi:10.1086/497440
Lushchak, V. I. (2014). Free radicals, reactive oxygen species, oxidative stress and its classification. *Chem-Biol. Interact.* **224**, 164-175. doi:10.1016/j.cbi.2014.10.016
Mailloux, R. J. (2018). Mitochondrial antioxidants and the maintenance of cellular hydrogen peroxide levels. *Oxid Med Cell Longev* **2018**, 7857251. doi:10.1155/2018/7857251
Mays, H. L. and Hill, G. E. (2004). Choosing mates: good genes versus genes that are a good fit. *Trends Ecol. Evol.* **19**, 554-559. doi:10.1016/j.tree.2004.07.018
McEwen, B. S. and Wingfield, J. C. (2003). Reproduction and resistance to stress: when and how. *J. Neuroendocrinol.* **15**, 711-724. doi:10.1046/j.1365-2826.2003.01033.x
McGraw, K. J., Cohen, A. A., Costantini, D. and Horak, P. (2010). The ecological significance of antioxidants and oxidative stress: a marriage between mechanistic and functional perspectives. *Funct. Ecol.* **24**, 947-949. doi:10.1111/j.1365-2435.2010.01772.x
McKenzie, M., Lazarou, M., Thorburn, D. R. and Ryan, M. T. (2007). Analysis of mitochondrial subunit assembly into respiratory chain complexes using Blue Native polyacrylamide gel electrophoresis. *Anal. Biochem.* **364**, 128-137. doi:10.1016/j.ab.2007.02.022
Merila, J., Kruuk, L. E. B. and Sheldon, B. C. (2001). Natural selection on the genetical component of variance in body condition in a wild bird population. *J. Evol. Biol.* **14**, 918-929. doi:10.1046/j.1420-9101.2001.00353.x
Metcalfe, N. B. and Olsson, M. (2022). How telomere dynamics are influenced by the balance between mitochondrial efficiency, reactive oxygen species production and DNA damage. *Mol. Ecol.* **31**, 6040-6052. doi:10.1111/mec.16150
Mikula, P., Valcu, M., Brumm, H., Bulla, M., Forstmeier, W., Petruskova, T., Kempenaers, B. and Albrecht, T. (2021). A global analysis of song frequency in passerines provides no support for the acoustic adaptation hypothesis but suggests a role for sexual selection. *Ecol. Lett.* **24**, 477-486. doi:10.1111/ele.13662
Miller, C. W. and Moore, A. J. (2007). A potential resolution to the lek paradox through indirect genetic effects. *Proc. R. Soc. B Biol. Sci.* **274**, 1279-1286. doi:10.1098/rspb.2006.0413
Moller, A. P. (1994). *Sexual Selection and the Barn Swallow*. Oxford: Oxford University Press.
Morehouse, N. I. (2014). Condition-dependent ornaments, life histories, and the evolving architecture of resource-use. *Integr. Comp. Biol.* **54**, 591-600. doi:10.1093/icb/icu103

Moreno-Loshuertos, R., Acin-Perez, R., Fernandez-Silva, P., Movilla, N., Perez-Martos, A., De Cordoba, S. R., Gallardo, M. E. and Enriquez, J. A. (2006). Differences in reactive oxygen species production explain the phenotypes associated with common mouse mitochondrial DNA variants. *Nat. Genet.* **38**, 1261-1268. doi:10.1038/ng1897

Mousseau, T. A. and Fox, C. W. (1998). The adaptive significance of maternal effects. *Trends Ecol. Evol.* **13**, 403-407. doi:10.1016/S0169-5347(98)01472-4

Murphy, M. P. (2012). Mitochondrial thiols in antioxidant protection and redox signaling: distinct roles for glutathionylation and other thiol modifications. *Antioxid Redox Signal.* **16**, 476-495. doi:10.1089/ars.2011.4289

Neff, B. D. and Pitcher, T. E. (2005). Genetic quality and sexual selection: an integrated framework for good genes and compatible genes. *Mol. Ecol.* **14**, 19-38. doi:10.1111/j.1365-294X.2004.02395.x

Neff, B. D. and Pitcher, T. E. (2008). Mate choice for non-additive genetic benefits: A resolution to the lek paradox. *J. Theor. Biol.* **254**, 147-155. doi:10.1016/j.jtbi.2008.05.019

Neff, B. D. and Pitcher, T. E. (2009). Mate choice for nonadditive genetic benefits and the maintenance of genetic diversity in song sparrows. *J. Evol. Biol.* **22**, 424-429. doi:10.1111/j.1420-9101.2008.01646.x

Pacheco-Fuentes, H., Ton, R. and Griffith, S. C. (2023). Short- and long-term consequences of heat exposure on mitochondrial metabolism in zebra finches (*Taeniopygia castanotis*). *Physiol. Ecol.* **201**, 637-648.

Peig, J. and Green, A. J. (2010). The paradigm of body condition: a critical reappraisal of current methods based on mass and length. *Funct. Ecol.* **24**, 1323-1332. doi:10.1111/j.1365-2435.2010.01751.x

Pereira, R. J., Lima, T. G., Pierce-Ward, N. T., Chao, L. and Burton, R. S. (2021). Recovery from hybrid breakdown reveals a complex genetic architecture of mitonuclear incompatibilities. *Mol. Ecol.* **30**, 6403-6416. doi:10.1111/mec.15985

Petrie, M. (2021). Evolution by sexual selection. *Front. Ecol. Evol.* **9**, 786868. doi:10.3389/fevo.2021.786868

Philippi, T. and Seger, J. (1989). Hedging ones evolutionary bets, revisited. *Trends Ecol. Evol.* **4**, 41-44. doi:10.1016/0169-5347(89)90138-9

Picard, M., McEwen, B. S., Epel, E. S. and Sandi, C. (2018). An energetic view of stress: focus on mitochondria. *Front. Neuroendocrinol.* **49**, 72-85. doi:10.1016/j.yfrne.2018.01.001

Pichaud, N., Bérubé, R., Côté, G., Belzile, C., Dufresne, F., Morrows, G., Tanguays, R. M., Rands, D. M. and Blier, P. U. (2019). Age dependent dysfunction of mitochondrial and ROS metabolism induced by mitonuclear mismatch. *Front. Genet.* **10**, 130. doi:10.3389/fgene.2019.00130

Pomiankowski, A. and Moller, A. P. (1995). A resolution of the Lek Paradox. *Proc. R. Soc. B Biol. Sci.* **260**, 21-29. doi:10.1098/rspb.1995.0054

Poyton, R. O. and McEwen, J. E. (1996). Crosstalk between nuclear and mitochondrial genomes. *Annu. Rev. Biochem.* **65**, 563-607. doi:10.1146/annurev.bi.65.070196.003023

Puurtinen, M., Ketola, T. and Kotiaho, J. S. (2009). The Good-Genes and Compatible-Genes Benefits of Mate Choice. *Am. Nat.* **174**, 741-752. doi:10.1086/606024

Rousset, S., Alves-Guerra, M., Mozo, J., Miroux, B., Cassard-Doulcier, A., Bouillaud, F. and Ricquier, D. (2004). The biology of mitochondrial uncoupling proteins. *Diabetes* **53**, S130-S135. doi:10.2337/diabetes.53.2007.S130

Rowe, L. and Houle, D. (1996). The lek paradox and the capture of genetic variance by condition dependent traits. *Proc. R. Soc. B Biol. Sci.* **263**, 1415-1421. doi:10.1098/rspb.1996.0207

Sánchez, C. A., Becker, D. J., Teitelbaum, C. S., Barriga, P., Brown, L. M., Majewska, A. A., Hall, R. J. and Altizer, S. (2018). On the relationship between body condition and parasite infection in wildlife: a review and meta-analysis. *Ecol. Lett.* **21**, 1869-1884. doi:10.1111/ele.13160

Seger, J. and Brockmann, H. J. (1987). *What is Bet-Hedging?* Oxford, UK: Oxford University Press.

Shields, H. J., Traa, A. and Van Raamsdonk, J. M. (2021). Beneficial and detrimental effects of reactive oxygen species on lifespan: a comprehensive review of comparative and experimental studies. *Front. Cell Dev. Biol.* **9**, 628157. doi:10.3389/fcell.2021.628157

Sloan, D. B., Havird, J. C. and Sharbrough, J. (2017). The on-again, off-again relationship between mitochondrial genomes and species boundaries. *Mol. Ecol.* **26**, 2212-2236. doi:10.1111/mec.13959

Smeitink, J., Van Den Heuvel, L. and Dimauro, S. (2001). The genetics and pathology of oxidative phosphorylation. *Nat. Rev. Genet.* **2**, 342-352. doi:10.1038/35072063

Starrfelt, J. and Kokko, H. (2012). Bet-hedging - a triple trade-off between means, variances and correlations. *Biol. Rev.* **87**, 742-755. doi:10.1111/j.1469-185X.2012.00225.x

Stier, A., Bize, P., Roussel, D., Schull, Q., Massemin, S. and Criscuolo, F. (2014). Mitochondrial uncoupling as a regulator of life-history trajectories in birds: an experimental study in the zebra finch. *J. Exp. Biol.* **217**, 3579-3589. doi:10.1242/jeb.103945

Stier, A., Romestaing, C., Schull, Q., Lefol, E., Robin, J. P., Roussel, D. and Bize, P. (2017). How to measure mitochondrial function in birds using red blood cells: a case study in the king penguin and perspectives in ecology and evolution. *Methods Ecol. Evol.* **8**, 1172-1182. doi:10.1111/2041-210X.12724

Stier, A., Monaghan, P. and Metcalfe, N. B. (2022). Experimental demonstration of prenatal programming of mitochondrial aerobic metabolism lasting until adulthood. *Proc. R. Soc. B Biol. Sci.* **289**, 2021679. doi:10.1098/rspb.2021.2679

Taylor, P. D. and Williams, G. C. (1982). The lek paradox is not resolved. *Theor. Popul. Biol.* **22**, 392-409. doi:10.1016/0040-5809(82)90052-1

Tobler, M., Schlupp, I. and Plath, M. (2011). Costly interactions between the sexes: combined effects of male sexual harassment and female choice? *Behav. Ecol.* **22**, 723-729. doi:10.1093/beheco/arr044

Tomkins, J. L., Radwan, J., Kotiaho, J. S. and Tregenza, T. (2004). Genic capture and resolving the lek paradox. *Trends Ecol. Evol.* **19**, 323-328. doi:10.1016/j.tree.2004.03.029

Ton, R., Stier, A., Cooper, C. E. and Griffith, S. C. (2021). Effects of heat waves during post-natal development on mitochondrial and whole body physiology: an experimental study in zebra finches. *Front. Physiol.* **12**, 661670. doi:10.3389/fphys.2021.661670

Udino, E., George, J. M., McKenzie, M., Pessato, A., Crino, O. L., Buchanan, K. L. and Mariette, M. M. (2021). Prenatal acoustic programming of mitochondrial function for high temperatures in an arid-adapted bird. *Proc. R. Soc. B* **288**, 20211893. doi:10.1098/rspb.2021.1893

Vitousek, M. N., Mitchell, M. A., Woakes, A. J., Niemack, M. D. and Wikelski, M. (2007). High costs of female choice in a lekking lizard. *PLoS One* **2**, e567. doi:10.1371/journal.pone.0000567

Wilder, S. M., Raubenheimer, D. and Simpson, S. J. (2016). Moving beyond body condition indices as an estimate of fitness in ecological and evolutionary studies. *Funct. Ecol.* **30**, 108-115. doi:10.1111/1365-2435.12460

Wolff, J. N., Ladoukakis, E. D., Enriquez, J. A. and Dowling, D. K. (2014). Mitonuclear interactions: evolutionary consequences over multiple biological scales. *Philos. Trans. R. Soc. B Biol. Sci.* **369**, 0130443. doi:10.1098/rstb.2013.0443

Yasui, Y. (2001). Female multiple mating as a genetic bet-hedging strategy when mate choice criteria are unreliable. *Ecol. Res.* **16**, 605-616. doi:10.1046/j.1440-1703.2001.00423.x

Zeh, J. A. and Zeh, D. W. (1996). The evolution of polyandry I: intragenomic conflict and genetic incompatibility. *Proc. R. Soc. B Biol. Sci.* **263**, 1711-1717. doi:10.1098/rspb.1996.0250

Zeh, J. A. and Zeh, D. W. (1997). The evolution of polyandry .2. Post-copulatory defences against genetic incompatibility. *Proc. R. Soc. B Biol. Sci.* **264**, 69-75. doi:10.1098/rspb.1997.0010

Zhu, C. T., Ingelmo, P. and Rand, D. M. (2014). GxGxE for lifespan in : mitochondrial, nuclear, and dietary interactions that modify longevity. *PLoS Genet.* **10**, e1004354. doi:10.1371/journal.pgen.1004354

Zinovkin, R. A., Lyamzaev, K. G. and Chernyak, B. V. (2023). Current perspectives of mitochondria-targeted antioxidants in cancer prevention and treatment. *Front. Cell Dev. Biol.* **11**, 1048177. doi:10.3389/fcell.2023.1048177