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Sexual selection: incorporating non-genetic inheritance

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Non-genetic inheritance (NGI) is the transmission of parental factors, other than DNA sequences, to offspring that then affect their phenotype. Within the last decade, NGI has invoked considerable interest from evolutionary biologists. Numerous models indicate that NGI could be an important contributor to processes driven by natural selection, including speciation and local adaptation. However, less attention has been given to the role of NGI in the evolution of sexually selected traits. Here, we focus on recent theoretical models to highlight how NGI that leads to offspring acquiring either adaptive or non-adaptive traits can both influence sexual selection.

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What is non-genetic inheritance?

Traditionally, evolutionary biologists have focused on how the genetic inheritance (GI) of traits that affect fitness results in evolutionary change in trait values due to selection for different DNA sequences (i.e. genes). There is, however, growing evidence that non-genetic inheritance is also widespread and could play a role in how organisms become adapted to their environment. Non-genetic inheritance (NGI) is the transmission of components of the parental environment or phenotype that influence gene expression and development in their offspring. The consistency of such transmission spans the range from a single generation to many generations [1••]. The non-genetic factors of interest to researchers include all elements of the ancestral environment that have effects on offspring. Only DNA sequences are excluded because, by definition, they involve Mendelian GI [2].

Non-genetic inheritance incorporates a wide range of mechanisms (Table 1). These mechanisms always operate alongside GI [2] but, unlike DNA sequences, NGI factors vary greatly in how long they remain heritable [3]: they tend to be far more 'mutable' with heritable changes being generated either predictably in response to identifiable environmental cues (e.g. anti-predator defences arising in the offspring of parents exposed to predator cues), or unpredictably (e.g. changes in DNA methylation patterns that we cannot yet consistently link to specific cues) [4]. NGI can be viewed as an across generation form of phenotypic plasticity whereby environmentally induced changes in a parent's phenotype affect its offspring. Consequently, an individual's phenotype depends not only on its genotype and the environment it encounters, but also upon the phenotype (and, by extension, the environment) of its recent ancestors [5,6]. It is noteworthy that NGI can result in parent-offspring resemblance, as with GI, but this need not be the case (e.g. large-bodied parents might produce more, but smaller than average, offspring; and smaller parents might produce fewer, but larger than average, offspring).

Why is non-genetic inheritance important?

Most theoretical work on NGI asks how it influences the rate and likelihood of adaptation — by which we mean the 'fit' between an organism and its environment such that well adapted individuals are better matched to their environment and thus have higher lifetime reproductive success (review: [7]). In general, theory suggests that evolutionary change can be drastically affected by NGI, most notably because it increases the amount of heritable phenotypic variation available for selection [8,9]. As such, NGI might increase the rate at which organisms adapt to their environment. This is equivalent to arguing that an increase in DNA mutation rates would accelerate the adaptive evolution of GI traits. Of course, there is the caveat that, as with GI, too high a mutation rate eliminates the inheritance of traits favoured by selection, and that mutation-selection balance determines how close a population comes to individuals expressing the optimal phenotype.

Selection favours heritable phenotypic responses to the environment that enhance reproductive success regardless of whether there is GI (e.g. genes for phenotypic plasticity) or NGI. In some situations, however, NGI allows for adaptive transgenerational effects so that traits might increase in frequency (i.e. evolve) far more rapidly than would be the case for adaptive phenotypic plasticity

Table 1

Examples of different mechanisms of Non-genetic inheritance (NGI), their properties and how they might contribute to sexual selection.

Inheritance mechanism	Examples	Persistence of effect across multiple generations?	Variation environmentally induced or spontaneous?	Effects on parent offspring resemblance?	Potential interactions with GI/Potential for selection to act on variation in phenotype?	Potential effects on sexual selection
Transmission of epigenetic state/ Transgenerational epigenetic inheritance	DNA methylation [1,2]	Varies, may persist across numerous generations	Both	Positive, negative or no effect	Often closely linked with genes and involved in interpretation of gene sequence	Supplies source of renewable phenotypic variation in condition thus maintaining benefits of choice
	Genomic imprinting [3]	May persist across numerous generations	Both	Depends on sex of parent and offspring	Acts to silence inherited alleles	Can influence sex specific inheritance, sexual antagonism and sexual conflict
Transmission of cytoplasmic or somatic factors/Somatic inheritance	Maternal transfer of hormones via eggs [4,5]	Likely to dilute over generations unless continually induced	Environmentally induced	Positive, negative or no effect	Selection acts on genetic architecture that promotes NGI, NGI generates phenotypic variation upon which selection can act potential for cross generation GxE	Can influence expression of sexual traits
	Paternal transfer of ejaculate borne substances [6]	Likely to dilute over generations unless continually induced	Environmentally induced	Positive, negative or no effect	Selection acts on genetic architecture that promotes NGI, NGI generates phenotypic variation upon which selection can act potential for cross generation GxE	Supplies source of renewable phenotypic variation in condition thus maintaining benefits of choice
Transmission of nutrients	Maternal provisioning of embryos [7]	Likely to dilute over generations unless continually induced	Environmentally induced	Positive, negative or no effect	Selection acts on genetic architecture that promotes NGI, NGI generates phenotypic variation upon which selection can act potential for cross generation GxE	Can influence expression of condition dependent traits in offspring; Can become target of mate choice
	Paternal provisioning via nuptial gift [8,9]	Likely to dilute over generations unless continually induced	Environmentally induced	Positive, negative or no effect	Selection acts on genetic architecture that promotes NGI, NGI generates phenotypic variation upon which selection can act potential for cross generation GxE	Can influence expression of condition dependent sexual traits in offspring; Can increase indirect benefits of mate choice; Can become target of mate choice
Transmission of acquired traits/transgenerational phenotypic plasticity	Transfer of acquired immunity [10], antipredator responses [11] and chemical tolerance [12]	Likely to dilute over generations unless continually induced	Environmentally induced	Positive	Selection acts on genetic architecture that promotes NGI, NGI generates phenotypic variation upon which selection can act	Can influence expression of condition dependent sexual traits in offspring Increases heritability of traits even when no genetic variation

Transmission of extra-organismal environment/ Environmental inheritance	Habitat imprinting [13]	Single generation	Environmentally induced	Positive	Increases heritability of traits even when no genetic variation	Generates assortative mating and potentially leads to reproductive isolation between lineages
	Transfer of microbiomes [14,15]	Likely to dilute over generations unless continually induced	Both (parents obtain microbiomes from environment, but change in microbiome can be spontaneous)	Positive	Has potential links with other fitness related traits e.g. immunity	Generates assortative mating and potentially leads to reproductive isolation between lineages; Could influence expression of condition dependent sexual traits
Transmission of behaviour/Behavioural inheritance	Sexual imprinting of mate preferences [16]	Single generation	Both??	Depends on mode of imprinting	Generates covariance between NGI preference and GI sexual trait	Generates assortative mating and potentially leads to reproductive isolation between lineages
	Learning of sexual traits [17]	Single generation	Both??	Positive	Can increase heritability of traits even when no genetic variation	Generates assortative mating and potentially leads to reproductive isolation between lineages

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that involves GI. This can occur if, for instance, the heritability of traits with NGI is higher (e.g. because a genetic mutation is recessive); or because, given a sudden environmental change, the time lag for selection to act can be shorter (e.g. a beneficial response to this change in the parental generation is exposed to selection which affects its frequency in the next generation; this cannot happen for selection on DNA sequences that generate phenotypic plasticity at a life history stage before the environmental change occurred). Even so, as the population mean phenotype approaches the optimum, there is likely to be strong selection for genes that reduce plasticity in the environmental responsiveness of the trait and thereby lower deviation from the optimal phenotype [8,9]. In short, both NGI and GI can play a role in adaptive processes, but their relative importance in generating a fit between an organism and its environment probably changes over time.

Intriguingly, even maladaptive traits with NGI can still affect the rate of evolution. This is because such traits can still influence how selection acts on traits with GI (review: [7]). NGI can also affect selection on DNA sequences because it is a form of plasticity that extends across generations, with similar effects to within-generation phenotypic plasticity [7]. For instance, the acquisition of behaviours through transgenerational learning can *slow* evolutionary change in traits with GI by decreasing the genetic covariance between the behaviour trait and fitness [10,11].

How does non-genetic inheritance influence sexual selection?

Sexual selection is a major force shaping variation among species and between the sexes, but researchers have given relatively little attention to how NGI influences sexual selection. This is an oversight as NGI could affect sexual selection at multiple levels. For instance, there is much research on whether female choice drives the evolution of elaborate male traits due to genetic covariation between preferences, traits and fitness [12]. NGI could alter: the expression of both sexual traits and mating preferences for these traits; the costs and benefits of mating with individuals that invest more in sexual traits; genetic covariation between sexual traits, mating preferences and fitness because NGI can change the relationship between an individual's phenotype and its genotype.

In this mini-review we present two case studies. First, we highlight recent theoretical advances in the field of sexual imprinting to illustrate the broader, seemingly uncontroversial, claim that traits with NGI that enhance fitness are more likely to evolve than those that are maladaptive or selectively neutral. Second, we discuss recent theoretical research on ejaculate borne factors that cause NGI of neutral or maladaptive traits to illustrate the claim that NGI of non-adaptive traits can influence sexual selection.

NGI increases the heritable variation upon which selection can act

Recent theoretical studies that ask how and when sexual imprinting evolves provide a good example of how selection can act on different modes of NGI to generate adaptive, rather than maladaptive, transgenerational effects. Sexual imprinting is a common mechanism of learning that affects mate choice. It occurs when a young individual acquires its mate preferences based on the phenotype of another target individual [13]. Sexual imprinting is a form of NGI, regardless of whether the target phenotype is that of an individual's mother (maternal imprinting) or father (paternal imprinting) [14]. Imprinted mate preferences often appear to be adaptive because they allow individuals to choose higher quality mates, but there is no *a priori* reason for this to occur. Recent theoretical models, which are outlined below, investigate the conditions under which different types of sexual imprinting are each most likely to evolve. They provide insight into why sexual imprinting appears to be adaptive. Generalising from these models, we expect the same is often true for other traits with NGI.

Until recently sexual imprinting theory focused on how a specific mode of imprinting evolves against a background of no imprinting (i.e. random mating or preferences with GI). Theory ignored competition between different imprinting modes. It was only recently that researchers asked which mode of imprinting is most likely to increase in frequency under selection when modes compete? To this end, Tramm and Servedio [15••] developed a two locus, population genetic model (genes for a trait and for an imprinting mode) that compared the evolution of maternal and paternal imprinting by females. They concluded that paternal imprinting was more likely to evolve. The key reason is that the targets of paternal imprinting (i.e. males from whom the preferred trait is learned) have higher fitness than the targets of maternal imprinting. The targeted males have passed through the filter of sexual selection [15••], so, by definition, they have phenotypes with greater expression of sexually selected traits than those of males that failed to mate and breed. Similar logic is used to explain the benefits of paternal genomic imprinting (another form of NGI where certain genes are only expressed when inherited from fathers) of sexually selected traits when there is intra-locus sexual conflict [16] (i.e. their expression is detrimental in females). Clearly, selection can act on traits with NGI in the same way that it acts on traits with GI, leading to the evolution of better adapted phenotypes. By extension, it seems probable that proximate modes of NGI that generate traits with greater fitness benefits are, all else being equal, more likely to evolve.

In another study, Chaffee *et al.* [17••] asked how the costs of different modes of imprinting by females influence the evolution of mate preferences. They compared the evolution of maternal and paternal imprinting when

the ability to imprint (e.g. maintaining the neurological system necessary to identify and learn preferred phenotypes) incurs fixed absolute costs or when there are relative costs that only arise when a potential mate is rejected (e.g. due to search costs or the risk of death when continuing to search for a mate). Previous studies showed that both types of costs inhibit the evolution of mating preferences with GI (e.g. [18]), but they ignored NGI. Chafee *et al.* [17••] found, in their model, that even very small fixed costs prevented the evolution of sexual imprinting, but it could still evolve in the face of mate rejection being costly. They also corroborated the findings of Tramm and Servedio [15••] that paternal imprinting can invade and replace maternal imprinting in a population; but that, in its absence, maternal imprinting can evolve [17••]. These results have two important implications: costly sensory apparatus potentially required for sexual imprinting is unlikely to evolve solely for this purpose. This implies there need to be compensatory direct benefits of mate choice that elevate offspring production (e.g. due to increased parental compatibility) Traits that are under sexually antagonistic viability selection are unlikely to be targets of sexual imprinting (because they impose direct costs on choosy females in the next generation as these females disproportionately inherit, and express, genes that lower viability).

Most theoretical studies on imprinting that show paternal imprinting is favoured over maternal imprinting, including the two studies highlighted, assume a polygynous mating system. Here daughters are assumed to accurately identify their fathers so they can imprint on males with sexual traits correlated with above average fitness (i.e. their fathers were disproportionately successful at acquiring mates). This implies that strong paternal imprinting should be the rule in nature. Why then do so many species have maternal imprinting (e.g. [19])?

Invernizzi and Gilman [20••] departed from the assumption of polygyny to examine the evolution of imprinting in a socially monogamous mating system. However, they added the essential biological twist of extra-pair paternity (EPP). Here juveniles can encounter a paternal phenotype that might not have contributed genes to them, or indeed anyone, in the next generation. Invernizzi and Gilman [20••] modelled cases in which the target trait was expressed in both sexes, and where the benefits of female choice were indirect, genetic benefits (i.e. fitter offspring) rather than direct benefits of parental care (i.e. more offspring). They showed that maternal imprinting was more likely to evolve than paternal imprinting. Intriguingly, they also showed that when imprinting is paternal (i.e. based on the phenotype of the social father), higher levels of EPP actually *increase* choosiness for social partners, even though social mates sire *fewer* offspring. This counterintuitive result arises because as EPP increases so does non-random variance in male mating success: it becomes more important that social partners possess the phenotype of successful extra-pair males (who must be someone's social partner) [20••].

Studies that ask when different modes of imprinting will evolve are interesting because the mode of imprinting affects other major evolutionary processes, most notably speciation. For instance, Yeh and Servedio [21] found that the identity of the target individual, prevalence of the target sexual trait in the population and the strength of the mating preference can dramatically alter the role of sexual imprinting in driving speciation. Maternal imprinting can mask sexual selection and retard divergence

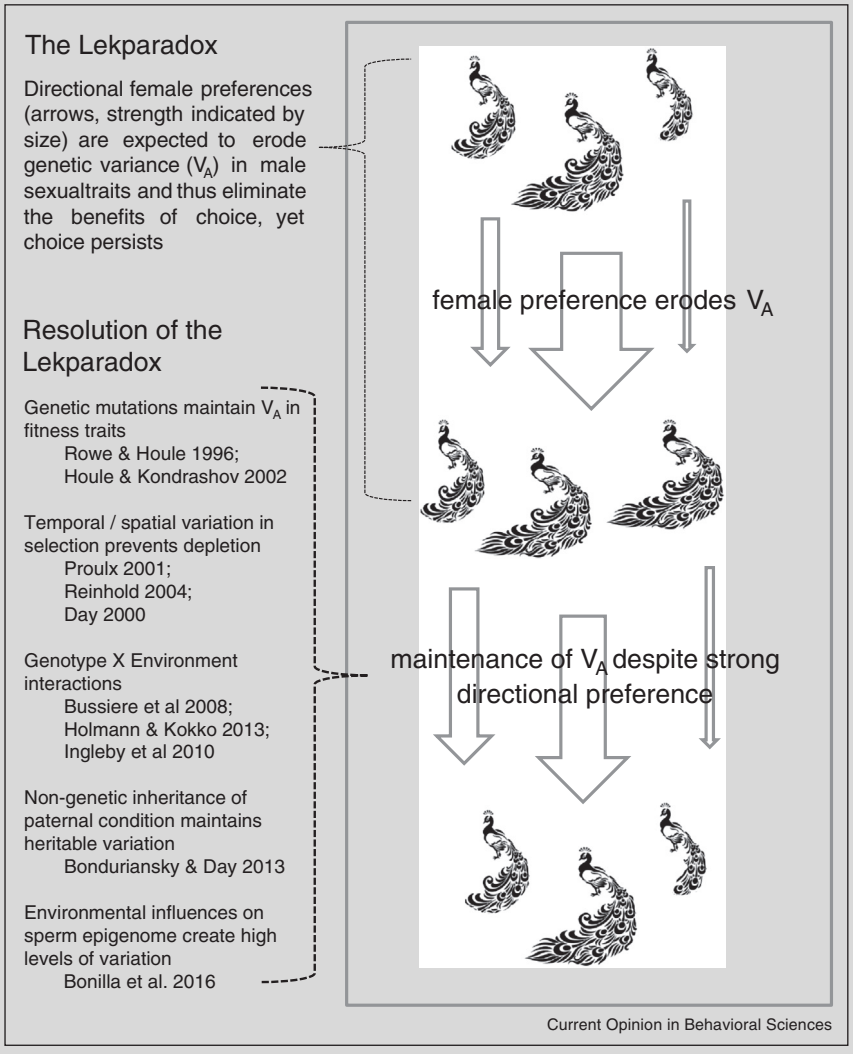
Box 1 What is the Lek Paradox?

The evolution and maintenance of exaggerated male sexual display traits is a controversial topic in evolutionary biology [37]. Female choice for these traits is expected to evolve when their bearers provide females with direct benefits (i.e. higher female life-time reproductive output through increased offspring production per breeding event and/or increased longevity and more breeding events over a lifetime) [38]. But female choice for exaggerated sexual traits presents a conundrum when males contribute only ejaculates and sperm. Choosiness is generally costly [39], and the absence of compensatory benefits should prevent the evolution of female mate choice [40] (but see [41]). In such situations it has been proposed that females that mate with showy males derive indirect benefits in the form of genes that increase offspring fitness.

In models of the evolution of female choice via indirect benefits, females select mates based on sexual characters that honestly signal male genetic quality [42] — only high quality males can afford to express costly sexual traits and by mating with more ornamented males females increase the mean fitness of their offspring [43]. However, strong directional selection on males is expected to deplete additive genetic variance for fitness. Consequently, there should be little or no difference in the fitness of offspring sired by attractive or unattractive males, eliminating the benefit of being choosy [44]. This conundrum is known as the 'lek paradox' [45] (Figure 1).

There are many mechanisms proposed to maintain additive genetic variance in fitness and resolve the lek paradox (reviewed in [46]). Some researchers argue that a high genetic mutation rate can maintain substantial additive genetic variance in fitness (e.g. [47,48]). For instance, if ornaments are condition-dependent, and many genes contribute to condition then the 'mutational target' might be large enough to maintain additive genetic variance for fitness solely due to mutations [48]. Other hypotheses invoke temporal and spatial variation in selection such that an evolutionary equilibria is never reached (e.g. [49,50,51]). For instance, Day [49] showed that female preference for locally adapted males, when combined with gene flow between environments, prevented fixation of an optimal male ornament size [49]. Likewise genotype-by-environment interactions where alleles that confer high fitness in one environment do poorly in other environments may mean that males with identical genotypes have different sexual ornament expression in different environments, thereby reducing signal reliability [52,53]. This, however, means that there is then a tension between the maintenance of variation, and the value of choice (i.e. offspring fitness is less reliably predicted by paternal sexual ornament expression).

Figure 1



Conceptual diagram of the lek paradox and a sample of popular hypotheses for its resolution.

(because it reduces the correlation between preference and preferred trait), while paternal imprinting can promote and maintain divergence when mating preferences are of moderate strength and there is already reasonably high initial divergence.

NGI can provide an endless source of phenotypic variation: could ejaculate-borne effects resolve the lek paradox?

Even when NGI seemingly promotes the persistence of non-adaptive traits it can still influence the course of adaptive evolution. The persistence of non-adaptive traits is possible when there is no, or only very short-term, heritability of traits due to NGI because selection cannot eliminate the less fit form of the trait. Can this resolve the lek paradox, which is an intriguing, unsolved puzzle in sexual selection theory? The paradox revolves around the maintenance of variation in the benefits conferred by mating with different types of males (Box 1). Recently, Bonduriansky and Day [1••] offered a possible solution to the lek paradox. They presented the first mathematical analysis of how transmission of ejaculate-borne factors other than DNA sequences (e.g. epigenetic markers, seminal proteins) could influence the evolution of male sexually selected traits. In so doing, they demonstrated the potential role of NGI in the maintenance of heritable variation in fitness.

Bonduriansky and Day [1••] asked how different modes of inheritance of male condition affect the evolution of costly female mating preferences that do not confer direct benefits (i.e. do not increase female lifetime reproductive output). In their model, females were able to directly assess male condition and selection on female preference

is entirely indirect (i.e. only through the benefit of increasing offspring fitness). Bonduriansky and Day modelled three scenarios, where high or low male condition was determined by: GI: in a two allele system with a low mutation rate; NGI: with two epigenetic states that had a high spontaneous ''mutation'' rate that was independent of environmental quality, and could persist for multiple generations; or NGI: with two environmentally induced states that depend on the quality of the environment, and are only transferred across a single generation. In all three cases a female preference for male condition was genetically determined by a single locus with two alleles (i.e. GI). The comparison of these models revealed that female choice is most probable, despite being costly, when male condition is environmentally induced and transmitted over one generation. Why? Because under this scenario heritable variation in fitness is regenerated in every generation by environmental heterogeneity and cannot be reduced by selection. Choosiness could also persist when male condition involved NGI of an environmentally-independent, epigenetic state over multiple generations, but the parameter space was more restrictive, because selection can start to eliminate lineages with low condition. By contrast, female choice did not evolve when male condition was due to GI. Although the model is based on simple genetics (i.e. it excludes a genetic correlation between male traits and fitness as in 'viability indicator' models) it nonetheless establishes a plausible role for NGI in the evolution of mating preferences [22•].

Numerous theoretical studies have modelled how paternal effects (a form of NGI) influence sexual selection (e.g. [23,24]) by assuming that paternal care has direct benefits for females and that males signal their ability to care through costly signals. The model of Bonduriansky and Day [1••] differs from these studies because the only benefits that females accrue are indirect (i.e. higher offspring fitness) and do not require GI of male traits. For example, males in high condition, which varies due to NGI, might provide more care, which increases the subsequent reproductive success of their offspring due to greater attractiveness to mates and/or survival. Indirect selection is generally considered less effective than direct selection [25], partly because it depends on the strength of the genetic correlation between mating preference and ornament. With NGI, however, there is no requirement for a genetic correlation, instead a covariance is established between the preference and the non-genetic factor that confers high offspring fitness.

The beauty of this model is in its simplicity. The factors transmitted in male ejaculates do not need to be favoured by selection, they simply transmit information about condition from father to offspring. Variation in male condition is maintained because it arises every generation (not unlike DNA mutations, but with the biologically plausible premise that environmental variation is more likely to occur than is a genetic mutation). Consequently NGI has the potential to maintain high levels of (short-term) heritability in fitness and thereby the benefit of mating with attractive males that signal their condition, even when these males only contribute ejaculates to reproduction. In sum, even non-genetic factors that only persist for a single generation could be evolutionarily important.

Bonilla *et al.* [22•] recently extended this modelling framework to suggest that environmentally mediated modification of the sperm epigenome, which consists of heritable epigenetic markers influencing gene expression (e.g. DNA methylation, histone modifications), provides a plausible and well supported mechanism whereby environmentally induced male condition can be transmitted to offspring. The sperm epigenome is sensitive to environmental influences (Reviewed in [26•]), so it has the potential to generate endless phenotypic variation in condition-dependent sexual traits.

Where to from here?

Modes of NGI that are adaptive (i.e. favour the elimination of disadvantageous phenotypes) and non-adaptive (i.e. allow disadvantageous phenotypes to persist) both appear to play an important role in sexual selection. Adaptive modes, such as sexual imprinting and many parental effects arising from maternal and paternal care [27] are advantageous because they prepare offspring for the specific environment in which they are likely to develop and compete for resources [28]. If offspring that are ''prepared'' for life in a particular environment do better, then parents who modify their offspring's phenotype accordingly will contribute more genes to future generations due to indirect selection. GI of parental effects can therefore promote the spread of adaptive forms of NGI and change the strength of selection on variation in traits that is due to GI. If we want to understand how sexual traits evolve we therefore need to consider both direct and indirect selection on genetic and non-genetic sources of variation. Of course, not all modes of NGI are adaptive (see those highlighted above in [1••]). Although non-adaptive modes of NGI may themselves be under selection and eventually eliminated, while present they can create variation in fitness and variation in other traits that intensify sexual selection.

One way to better understand how NGI contributes to sexual selection would be to conduct experiments where variation in NGI is removed or reduced and see how this influences the rate at which lineages adapt to new environments. This is a challenging task but could be achieved by, for instance, conducting experimental evolution studies in novel environments using short-lived species with parental care (e.g. Japanese Quail, Burying Beetles). In such an experiment one could employ a 2×2 factorial design looking at how the presence or absence of variation

in parental care interacts with the presence or absence of sexual selection. If NGI has important consequences for sexual selection we might expect that lineages with variation in parental care would achieve a better fit to their environment more quickly. Similarly, other creative ways to remove or enhance NGI will be needed to investigate the importance of other mechanisms of NGI in sexual selection.

We can gain much from integrating studies on the evolution of NGI with models investigating the consequences for other evolutionary processes. To date, theoretical studies of how NGI influences evolutionary processes typically assume that non-genetic effects already exist, and that NGI does not evolve [5]. Recently, researchers have begun to model the circumstances under which the mode of NGI is expected to evolve. These studies tend to show that parental effects on offspring phenotype are most likely to evolve when the environment is heterogeneous yet predictable [5] when parental and offspring environments are correlated [5,29•,30,31] (i.e. when dispersal is low [5,31]), and when transmission of information between generations is accurate [29•,31]. The fact that adaptive NGI is only likely to evolve under certain conditions could shed light on when it is most likely to affect sexual selection. But to gain a better picture of the role NGI plays in evolution we need to consider coevolutionary feedback between evolutionary processes and NGI mechanisms [32]. How does the mode of NGI affect selection on other traits, including those with GI? And how does this then affect selection on the mode of NGI?

Finally, NGI may be an important contributor to the role of sexual selection in speciation. NGI of both mating preferences (i.e. via sexual imprinting) and habitat preferences can promote assortative mating and accelerate population divergence [33,34]; and new evidence suggests that NGI of microbiomes might be yet another factor promoting assortative mating [35]. Epigenetic factors could also aid in generating reproductive isolation (regardless of how stable they are). For instance, reproductive isolation could arise if local adaptation means that immigrant males are in worse condition, which brings about epigenetic changes that reduce male fertilisation success [36]. We suggest that further investigation of how NGI promotes reproductive isolation by changing selection on sexual traits and mating preferences will be a fruitful area of future research.

Conflict of interest statement

Nothing declared.

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Papers of particular interest, published within the period of review, have been highlighted as:

- • of special interest
- •• of outstanding interest

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