Review

Intralocus sexual conflict

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Intralocus sexual conflict occurs when selection on a shared trait in one sex displaces the other sex from its phenotypic optimum. It arises because many shared traits have a common genetic basis but undergo contrasting selection in the sexes. A recent surge of interest in this evolutionary tug of war has yielded evidence of such conflicts in laboratory and natural populations. Here we highlight outstanding questions about the causes and consequences of intralocus sexual conflict at the genomic level, and its long-term implications for sexual coevolution. Whereas recent thinking has focussed on the role of intralocus sexual conflict as a brake on sexual coevolution, we urge a broader appraisal that also takes account of its potential to drive adaptive evolution and speciation.

A cryptic form of sexual conflict

Sexual conflict occurs because the divergent reproductive strategies of the sexes generate different selection pressures on many traits [1,2]. However, sexual conflict theory comprises two distinct modes of sexually antagonistic coevolution, each with its own history and literature [3].

Much research has focussed on interlocus sexual conflict (IRSC), antagonistic coevolution between loci that enhance male reproductive success at females' expense, and loci that enhance female resistance to male coercion [1,2]. IRSC often results in spectacular sexual 'arms races,' with striking phenotypic manifestations such as spiny genitalia or toxic ejaculates (reviewed in Ref. [3]). Far less attention has been paid to intralocus sexual conflict (IASC), the displacement of the sexes from their distinct phenotypic optima as a result of sex-specific selection on sexually homologous ('shared') traits whose expression is regulated by a shared genetic machinery [4]. Although the theoretical foundations of IASC were developed decades ago [4,5], its cryptic nature ensured its long neglect by empiricists. Phenotypic clues to the occurrence of IASC can be subtle: acute IASC is associated with a sexually monomorphic phenotype under sexually antagonistic selection, and the conflict attenuates as sexual dimorphism evolves [4]. The first clear evidence of IASC was provided in a seminal study by Chippindale and colleagues [6], initiating a growing surge of interest in IASC.

The concept of IASC encapsulates a fundamental evolutionary problem: the consequences of the sexes sharing a common genome. This problem raises a host of fascinating questions about sexual coevolution and genetics. Here we bring together key ideas and empirical evidence to identify major gaps in our understanding of IASC, clarify its potential importance in evolution and flag promising avenues of investigation.

The nature and causes of intralocus sexual conflict

IASC reflects a conflict between shared and divergent aspects of the biology of the sexes. Shared traits are assumed to be controlled, primitively, by a common genetic machinery in both sexes [4]. This is reflected in a strong, positive intersexual genetic correlation (r_{mf}), which measures the extent of similarity between the additive effects of alleles when expressed in different sexes (Box 1). However, the sexes are defined by strongly divergent reproductive strategies that generate sex-specific selection on many shared traits, favouring the evolution of sexual dimorphism [7]. In particular, shared traits such as tail feathers, colour spots or behavioural responses are often subject to sexual selection in males but not females. Sex-specific selection can also reflect ecological niche differentiation between sexes [8]. Lande [4] showed that selection on one sex can therefore cause the displacement of the other sex from its phenotypic optimum, reducing its fitness. For example, sexual selection favouring increased trait size in males can result in a correlated response in females that reduces female viability or fecundity, whereas selection opposing increased trait size in females can impede the trait's evolution in males. Alleles at a locus

Glossary

Condition: an individual's phenotypic quality, reflecting the pool of available metabolic resources, and the efficiency with which these resources can be converted into fitness.
Gender load: the extent to which population mean fitness is reduced owing to the operation of sexual conflict.
Interlocus sexual conflict (IRSC): interactions between sexually antagonistic alleles at different loci, resulting in the displacement of one or both sexes from its optimum for a phenotypic trait as a result of selection on the opposite sex.
Intersexual genetic correlation (r_{mf}): a correlation of male and female additive breeding values, measuring the extent of similarity of the additive effects of segregating alleles in each sex.
Intralocus sexual conflict (IASC): interactions between sexually antagonistic alleles within a locus, resulting in a displacement of one or both sexes from its optimum for a phenotypic trait as a result of selection on the opposite sex.
Paralogous loci: two or more loci derived from duplication of a single ancestral locus.
Sex linkage: physical location of a locus on a sex chromosome.
Sex-specific selection: any difference between sexes in the fitness surface for a phenotypic trait.
Sexually antagonistic allele: an allele that increases fitness when expressed in one sex, but reduces fitness when expressed in the other sex.
Sexually antagonistic locus: a locus segregating alleles for which the rank fitness differs between the sexes.
Sexually antagonistic selection: difference between the sexes in the sign of the covariance between a trait and fitness.
Sexually homologous trait: a trait expressed in both sexes ('shared' trait).

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Box 1. The intersexual genetic correlation, r_{mf}

The intersexual additive genetic correlation, r_{mf}, is a quantitative genetic parameter predictive of the potential for future independent evolution of the sexes within a population [4]. r_{mf} is defined as the ratio of the additive genetic covariance between the sexes (COV_{Amf}) to the geometric average of additive genetic variances of males (V_{Am}) and females (V_{Af}) for the trait (Equation I).

$$r_{mf} = \frac{cov_{Amf}}{\sqrt{V_{Am} \times V_{Af}}} \quad [I]$$

r_{mf} is usually estimated from measurements of a trait on groups of relatives [74]. Although standard breeding designs (e.g. half-sib) are most often used, r_{mf} can be estimated from complex pedigrees using the animal model [13]. Alternative breeding designs can be used to partition contributions from the autosomes versus the sex chromosomes ([36]; but see Ref. [75]). Because large sample sizes are required to obtain robust estimates of r_{mf}, hypothesis testing is usually limited to establishing whether an estimate overlaps 1, 0 or −1. Two additional caveats must be considered. First, an r_{mf} estimate applies to the specific environment within which the assay was conducted [74]. Second, statistical tools (such as the animal model) that yield estimates of V_A and r_{mf} make unrealistic simplifying assumptions, such as exponential population growth, whose consequences remain unclear [63].

What can an empirical estimate of r_{mf} realistically tell us about IASC? r_{mf} can be estimated for phenotypic traits [11], or for fitness itself [6], with fundamental differences in interpretation for each.

For fitness, the absence of IASC can be inferred when $r_{mf} = 1$, whereas compelling evidence of IASC emerges when $r_{mf} < 0$. An absolute constraint on the independent evolution of the sexes can be inferred when $r_{mf} = -1$. However, interpreting r_{mf} as a constraint assumes that all genetic variance is additive, and that variances do not differ between sexes [74]. Moreover, the constraint is not necessarily permanent, because genetic architectures can evolve so as to relax the constraint. In the range $0 \leq r_{mf} < 1$, neither the existence of sexually antagonistic alleles nor their absence can be inferred conclusively, because sex-limited rather than sexually antagonistic alleles might be responsible for reducing r_{mf} below a value of 1.

For traits under sexually antagonistic selection, $r_{mf} = 1$ reflects an absolute (but not permanent) constraint on the evolution of further sexual dimorphism, and indicates that IASC is occurring. For such a trait, values in the range of $0 < r_{mf} < 1$ suggest that some segregating alleles have sexually antagonistic effects on fitness, but further evolution of sexual dimorphism is possible (albeit slow). High r_{mf} values in already-dimorphic traits suggest that little genetic variation is available for further evolution of dimorphism.

Note that, despite its importance in studies of IASC, r_{mf} is essentially a genome-wide average across loci, whereas IASC is, by definition, a within-locus phenomenon. An r_{mf} estimate could reflect a combination of sexually antagonistic, sexually concordant and sex-limited allelic effects (Table I). To understand the contributions of particular loci to IASC, detailed dissections of genetic architecture must be combined with estimates of the effects of particular alleles on sex-specific fitness.

Table I. Types of allelic effects that can contribute to an intersexual genetic correlation

Locus	Male	Female
Sexually concordant	+ (−)	+ (−)
Sexually antagonistic	+ (−)	− (+)
Sex-limited	+ (0)	0 (+)
Sex-limited	− (0)	0 (−)

Symbols indicate allelic effects on trait expression (increasing, +; decreasing, −; no effect, 0) relative to the population mean.

affecting trait size in both sexes will thus have sexually antagonistic effects on fitness: an allele that increases trait size will enhance fitness when expressed in males, but reduce fitness when expressed in females. This can result in a negative r_{mf} for fitness itself, and suboptimal mean phenotypes in both sexes (i.e. trait size above optimum in females, but below optimum in males) [4]. IASC can occur even in the absence of genetic variation at the locus, when the single existing allele results in a more optimal phenotype in one sex than in the other. Although the most intense form of IASC occurs when the signs of selection on a trait are opposite in the sexes, IASC occurs whenever selection surfaces differ between sexes, because selection on one sex slows or impedes adaptive evolution in the other sex. Theory [4] suggests that genetic architecture evolves so as to allow independent adaptation in each sex, thus resolving IASC (Box 2).

Empirical evidence for intralocus sexual conflict

Although sex-specific selection and sex-specific genetic effects on fitness were first demonstrated decades ago [9,10], direct evidence of IASC has only recently come to light, based on estimates of selection on each sex, together with r_{mf}. We summarise the strongest evidence for IASC in Table 1, and discuss the major forms of evidence below.

Negative intersexual genetic correlation for fitness

Negative r_{mf} for fitness provides strong evidence of IASC (Box 1). Chippindale and colleagues [6] observed a negative r_{mf} for adult fitness in laboratory-adapted *Drosophila melanogaster*: genotypes that conferred high female fitness tended to confer low male fitness. Interestingly, r_{mf} for larval fitness was positive, suggesting that the evolutionary interests of the sexes are similar in the pre-reproductive phase of the life cycle. Recent reports of negative r_{mf} for fitness in several species of insects and vertebrates, and in natural populations (Table 1; Figure 1), support the reality of IASC beyond the laboratory.

Sexually antagonistic selection and genetic constraints for shared traits

Negative r_{mf} for fitness establishes that IASC is occurring, but does not reveal which shared phenotypic traits mediate the conflict. A phenotypic trait is strongly implicated in IASC when the sign of selection on the trait differs between sexes (i.e. selection is sexually antagonistic, favouring greater sexual dimorphism), but r_{mf} for the trait is positive. An apparent example of such a trait is locomotory activity in *Drosophila* [11]. Similarly, a laboratory study revealed a phenotypic signature of IASC over diet in the cricket *Teleogryllus commodus*: the sexes make similar diet choices despite having very different dietary optima [12].

We stress that, although IASC is often expected to occur during the evolution of sexual dimorphism [4], a history of IASC cannot be inferred unequivocally from existing sexual dimorphism. First, it is possible for dimorphism to evolve via completely sex-limited mutations (on the Y or W chromosome) that do not contribute to IASC. Second, sex-specific selection on one trait can result, via pleiotropy, in correlated evolution of sexual dimorphism in other

Box 2. Phases of intralocus sexual conflict

The progression of IASC can be illustrated by differentiating four distinct phases (Figure I), as follows.

Phase 1. Before IASC

A shared trait *z* with no history of IASC is expected to be under weak, stabilising selection in both sexes (females: solid black curve; males: dashed black curve), with the mean of the common phenotypic distribution (solid blue curve) corresponding to the optimum (**1a**). For such a 'typical' trait, allelic effects on fitness (females: dashed red line; males: solid blue line) are similar in both sexes (**1b**). Additive genetic variance for fitness is expected to be negligible (blue dot), probably rendering r_{mf} for fitness unmeasurable in practice. However, any additive genetic variance maintained by mutation–selection balance should generate $r_{mf} = 1$ (**1c**). Intersexual heritability (i.e. heritability based on mother–son or father–daughter regression) for *z* is high (**1d**).

Phase 2. Acute IASC

IASC might often originate when a change in physical or social conditions generates a novel vector of sexual selection on a trait in males. Lande [4] showed that sexual selection on males will initially displace both sexes from their common optimum, but evolution toward the male optimum will stop once net costs for females exactly balance net benefits for males. During this phase, IASC is intense, with strong sexually antagonistic selection (**2a**) and sexually antagonistic allele effects on fitness (**2b**), negative r_{mf} and intersexual heritability for fitness (**2c**), whereas r_{mf} and intersexual heritability for *z* remain high and positive (**2d**). The displacement of both sexes from their sex-specific phenotypic optima generates a gender load [19].

Phase 3. Attenuated IASC

Lande [4] reasoned that selection will favour sex-linked genetic modifiers that erode r_{mf}, permitting the phenotypes of females (solid blue curve) and males (dotted red curve) to diverge toward their sex-specific optima. During this phase, sexually antagonistic selection and sexually antagonistic fitness variation will abate as sexual dimorphism evolves (**3a**, **3b**, **3c**), and intersexual heritability of *z* will diminish as r_{mf} declines (**3d**).

Phase 4. Resolved IASC

Finally, if evolution of the trait genetic architecture permits full resolution of the conflict, an optimal sexual dimorphism will evolve and both sexes will again be under stabilising selection (**4a**). Once this phase is reached, sexually antagonistic fitness variation will vanish (**4b**) and r_{mf} for fitness will return to unity (**4c**). However, theory [36,76,77] suggests that r_{mf} and intersexual heritability for *z* could vary, depending on the strength of selection, mutation rate and the genetic architecture that has evolved (**4d**).

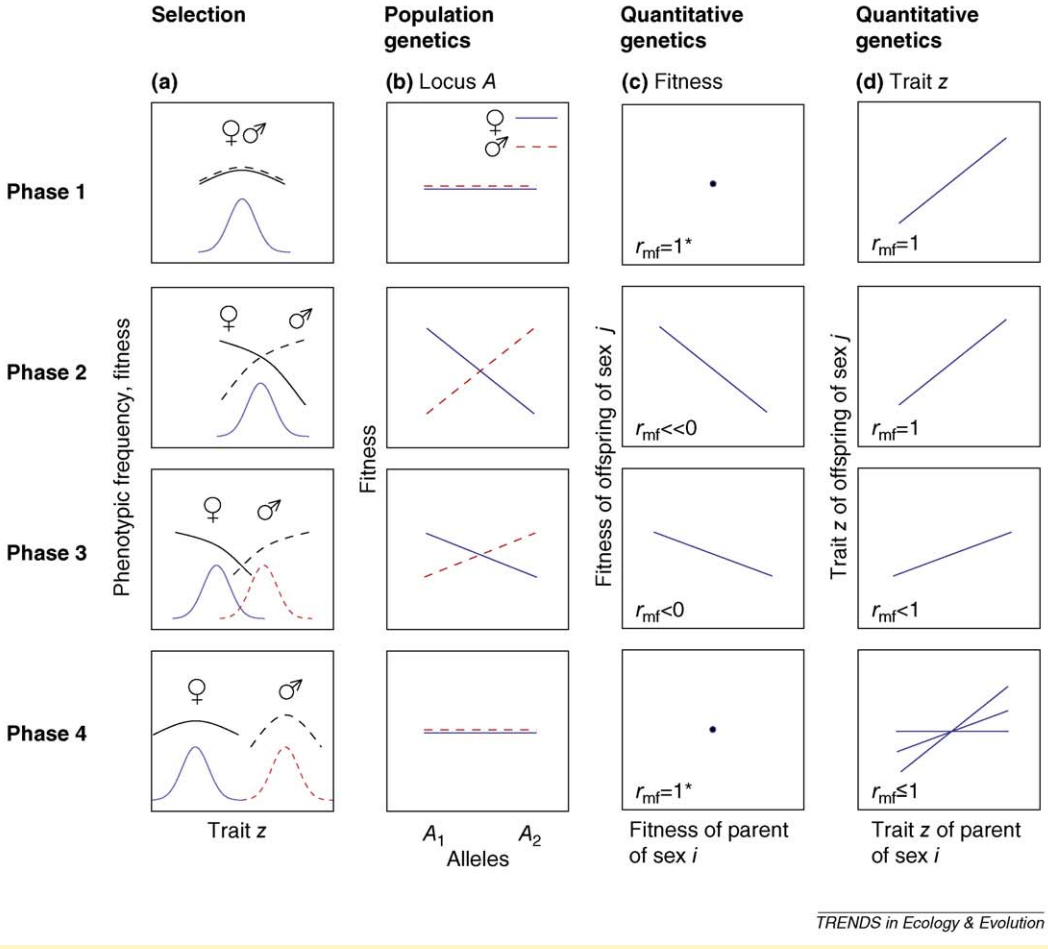

Figure I. Phases of intralocus sexual conflict. The hypothesised progression of IASC for a trait *z* affected by autosomal, X-linked or Z-linked loci, with **columns 1–4** representing the phases of the conflict and **rows a–d** representing distinct perspectives on the conflict (see text for explanation).

traits. Third, existing dimorphism can reflect fully resolved IASC (Box 2). Thus, although sexual dimorphism points to the possibility of past or ongoing IASC, estimates of selection and r_{mf} are required to confirm IASC.

Sex-biased experimental evolution

Under sexual conflict, removing the opportunity for selection in one sex should result in increased fitness in the other sex [13–15]. However, to attribute this result to

Table 1. Studies demonstrating intralocus sexual conflict

Type of evidence	Taxon	Details	Refs
(i) Trait-focussed correlational	*Drosophila melanogaster*	Sexually antagonistic selection on locomotory activity in a laboratory-adapted population; positive r_{mf} for locomotory activity	[11]
	Collared flycatcher (*Ficedula albicollis*)	Sexually antagonistic natural selection on body size; $r_{mf} = 1$ for body size	[78,79]
	Zebra finch (*Taeniopygia gutta*)	Sexually antagonistic selection on bill colour; strong r_{mf} for bill colour	[80,81]
(ii) Fitness-focussed correlational	*Drosophila melanogaster*	Negative r_{mf} for adult but not juvenile fitness in a laboratory-adapted population	[6]
		Sex-specific inheritance pattern for fitness consistent with a net-negative r_{mf} for total fitness	[64]
		Sexually antagonistic fitness effects of mitochondrial genotype	[62]
	Ground cricket (*Allonemobius socius*)	Sex-specific inheritance of fitness resulting in negative r_{mf} for fitness	[82]
	Red deer (*Cervus elaphus*)	Negative r_{mf} for fitness in a natural population	[13]
	Collared flycatcher (*Ficedula albicollis*)	Negative r_{mf} for lifetime reproductive success in a natural population	[83]
(iii) Fitness-focussed experimental	*Drosophila melanogaster*	Increased fitness in males following male-limited transmission of whole genomes; decrease in one female fitness component	[14,15]
		Increased male and decreased female fitness following male-limited evolution; also evolution of dimorphic trait to become more 'male like'	[16]
		Applied both male- and female-limited evolution for 26 generations using a 'middle-class neighborhood' design; observed decreased fitness in the unselected sex	[84]

release from IASC rather than IRSC requires demonstrating that genotypes from selected lines produce low-fitness phenotypes when expressed in the unselected sex. For example, Prasad and colleagues [16] prevented females from responding to selection over 25 generations in replicate *D. melanogaster* populations: a suite of sexually dimorphic traits became more 'male like,' while male performance increased and female performance declined.

Figure 1. Key empirical evidence of IASC is provided by four animals. Top left: in wild red deer *Cervus elaphus* (male above and female; photo: A. Morris), high-fitness males sire low-fitness daughters. Top right: in wild collared flycatchers *Ficedula albicollis* (female above and male; photo: T. Veen), r_{mf} for fitness is strongly negative. Bottom left: captive flies *Drosophila melanogaster* (male left and female; photo: R.B.) provided some of the first compelling evidence of IASC. Bottom right: high-fitness male crickets *Allonemobius socius* (female left and male; photo: K. Fedorka) captured in the wild sired low-fitness daughters in the laboratory.

Mechanisms that resolve intralocus sexual conflict

In theory, the genetic architecture of a trait under persistent sexually antagonistic selection will ultimately evolve to reduce genetic constraints on the independent evolution of the sexes, allowing a more optimal sexual dimorphism to evolve [4,17–19]. We refer to such modifications to trait genetic architecture as mechanisms for the resolution of IASC. We outline mechanisms that have substantial empirical support, and discuss other potential mechanisms that are either predicted by theory or suggested by recent empirical studies.

Many interesting questions remain. For example, do the different mechanisms outlined below resolve IASC to different degrees, and do some mechanisms evolve more readily or rapidly than others? Do some mechanisms serve as evolutionarily unstable, transitional solutions? Are different sex-determination systems (e.g. XX-XY, ZZ-ZW, environmental sex determination) amenable to different mechanisms of IASC resolution? Such questions can be addressed by comparing the genetic architectures that evolve in response to IASC in different taxonomic groups, and by tracking changes in genetic architecture through the phases of IASC (Box 2). As yet, no empirical or theoretical studies have tackled these questions.

Mechanisms with empirical support

Widely observed autosomal quantitative trait loci (QTLs) with sex-specific effects [20–22] suggest that sex-specific expression of autosomal loci, via sex-linked modifiers or alternative splicing mechanisms [23], can often contribute to IASC resolution (Figure 2a).

IASC is one key hypothesis for the evolution of sex-biased gene expression [24,25]. Autosomal and sex-linked

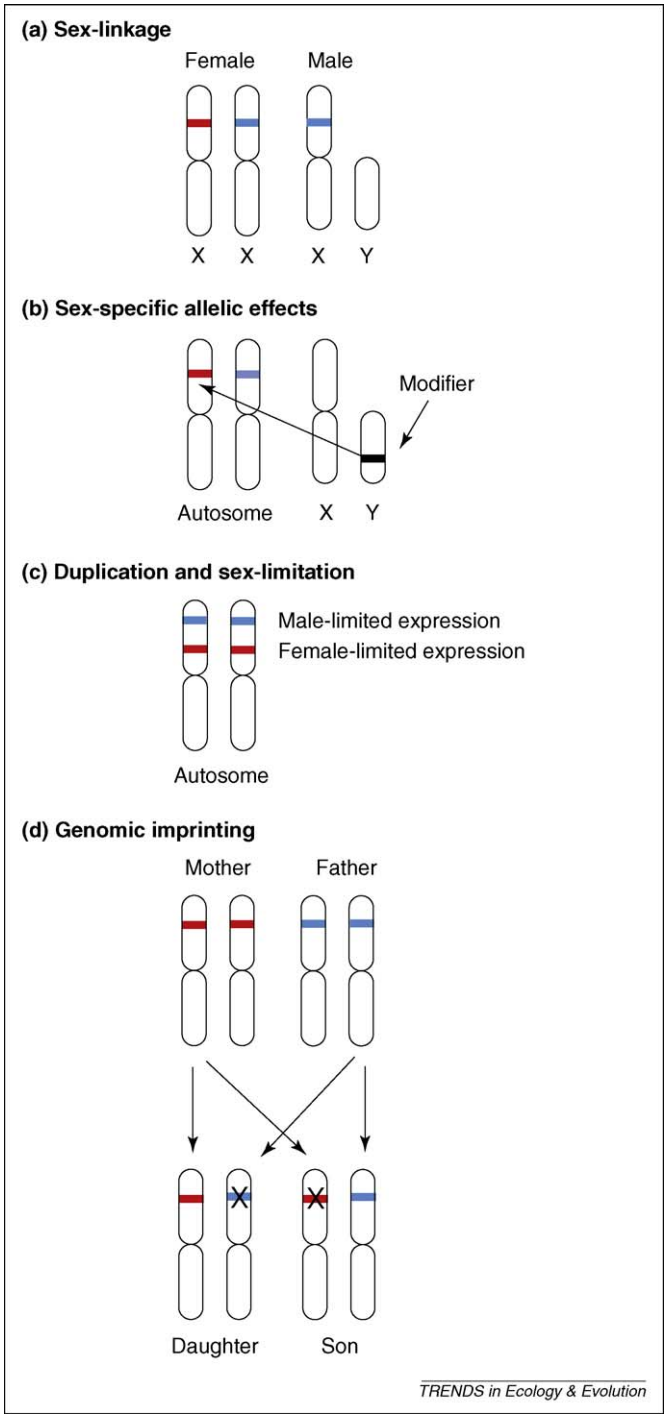

Figure 2. Genomic mechanisms of IASC resolution. Consider an ancestral autosomal locus segregating two sexually antagonistic alleles: one that benefits males but is detrimental to females (blue) and another with the opposite effects (red). Genetic mechanisms capable of mitigating such conflict include: **(a)** sex linkage where male-benefiting recessive and female-benefiting dominant alleles are favoured on the X chromosome (opposite allelic effects work for ZZ-ZW systems) [5]; **(b)** the evolution of sex-specific allelic effects via modifiers that are associated with a sex-determining locus [18]; **(c)** gene duplication and sex limitation of paralogous loci; and **(d)** genomic imprinting whereby alleles are silenced in a sex-specific manner depending on parent of origin [49].

loci might interact directly [26]. Alternatively, a sexually antagonistic locus might be modified by a locus with sex-specific expression that is further down the sex-determination cascade [27]. Sexually dimorphic abdominal pigmentation in *D. melanogaster* is modulated by an interaction between two autosomal loci, *bric-a-brac* and *doublesex*, the latter being alternatively spliced for male- and female-specific variants, and activated via the X-linked sex-determination gene *sex-lethal* [28]. Recent work [29] has revealed that male-specific pigmentation evolved from a sexually monomorphic ancestor via distinct forms of gene expression in each sex, involving modification of existing sex-specific expression pathways. These results suggest that once the genetic machinery for sex-specific regulation of expression has evolved, new dimorphisms can arise via co-option of existing sex-specific expression networks [29].

Gene duplication, regarded as a major source of genomic novelty [30], might also mitigate IASC (Figure 2b) [19,31,32]. Male-biased genes in *Drosophila* are more likely to have paralogues than unbiased genes, suggesting more frequent duplication [33]. Further, sex limitation appears to have evolved in novel duplicates of autosomal loci that are expressed in both sexes [34]. An intriguing example is the visual system of the butterfly *Lycaena rubidus* [35]: males possess two colour pigments in the dorsal eye that facilitate detection of conspecific males, whereas females possess two additional pigments (resulting from a duplicated gene encoding opsin) that enhance ability to detect food plants.

Sex linkage has long been thought to contribute to IASC resolution (Figure 2c) [5,19], but the evidence remains equivocal [36,37]. Sex chromosomes can either amplify or reduce IASC, depending on dominance, sex-specific allelic effects and pleiotropy [5,19]. For example, the X chromosome accounted for 97% of genome-wide sexually antagonistic fitness variation in a laboratory-adapted *D. melanogaster* population [38]. By contrast, in *D. serrata*, although dimorphic cuticular hydrocarbons undergo sexually antagonistic sexual selection [39], X-chromosome effects appear to weaken genome-wide r_{mf} to the extent that each sex could evolve independently during experimental evolution [40,41]. Adaptation to IASC could also account for the striking 'demasculinisation' of the X chromosome in flies [42–44] and worms [45], although alternative explanations exist [46–48].

Other potential mechanisms

Theory suggests that genomic imprinting (Figure 2d) could mitigate IASC by reducing the expression of alleles inherited from the opposite-sex parent because, on average, such alleles confer lower fitness than alleles inherited from the same-sex parent [49]. Consistent with this model, a recent study shows imprinting in genes affecting sexual performance in mice [50]. Sexually dimorphic imprinting (a pattern uniquely predicted by the model) has also been reported [51].

The evolution of condition dependence can, in principle, also contribute to the resolution of IASC [52–54]. The genic capture model [55] postulates the evolution of sex-limited pleiotropy that causes the expression of a phenotypic trait (such as a secondary sexual trait in males) to reflect variation in genetic and environmental factors influencing condition. A male-limited modifier that activates genic capture could shift breeding values for the secondary sexual trait toward higher levels of

expression in high-condition males, relative to low-condition males and females, potentially yielding an optimal sexual dimorphism. However, the efficacy of this mechanism depends on the degree of sex limitation of genic capture—an open question [52,54].

Recent examples also point to facultative mechanisms. In red deer, a positive maternal effect appears to weaken the negative mother–son phenotypic covariance for fitness, thus partially resolving IASC without altering the negative mother–son genetic covariance for fitness [13]. In lizards, females seem to produce sons and daughters using sperm from different males [56,57], thus altering the mother–offspring genetic covariance by preventing the production of low-fitness offspring genotypes.

Although we have focussed on the more typical case of organisms with sex chromosomes, much of the above discussion is relevant to organisms with environmental sex determination (ESD). In such species, IASC is expected to occur, and mechanisms of IASC resolution are expected to be associated genetically with the sex-determining developmental cascade (see Ref. [37]).

Can intralocus sexual conflict be fully resolved?

It is likely that IASC is fully resolved at some loci affecting primary sex traits because such loci can experience a consistent pattern of sex-specific selection over a vast number of generations, allowing ample time for mechanisms of IASC resolution to evolve. However, we argue that IASC is unlikely to be resolved fully throughout the genome (also see Ref. [58]).

Sexual coevolution models predict rapid, ongoing coevolutionary cycles and 'chases' that give rise to new secondary sexual traits [59,60]. A male trait that exploits a female sensory bias will eventually be rendered ineffective by the evolution of female resistance, selecting for new, more effective male traits [59,60]. Sexual coevolution can thus frequently generate novel patterns of sex-specific selection at shared loci affecting the expression of sexually antagonistic traits, resulting in novel IASC. Mechanisms that resolve IASC are likely to evolve slowly relative to these coevolutionary cycles [19].

Moreover, a weakened form of IASC is likely to persist even after mechanisms of IASC resolution have evolved. For example, following the evolution of locus duplication and sex limitation (i.e. silencing in one sex), deleterious alleles entering the population via mutation or gene flow will be sheltered from selection when present in the non-expressing sex [49]. Thus, even though sex-limited loci do not contribute to r_{mf} for the traits they affect, such loci can contribute to IASC [49]. Likewise, loci on the sex-determining chromosome could evolve to function as modifiers that resolve IASC at loci on other chromosomes. However, sex-determining chromosomes are often small and degenerate, and therefore unlikely to accommodate many such modifiers [36]. Moreover, the evolution of novel pleiotropic modifier effects at existing sex-limited loci (such as Y-linked loci affecting sperm production) could disrupt those genes' primary functions, spreading rather than eliminating IASC [19]. Finally, exclusively maternal transmission of cytoplasmic genes (e.g. in mitochondria) can result in suboptimal mitochondrial function in males [31,61,62]—a form of IASC that apparently cannot be resolved, because selection on mitochondria in males cannot produce a response. Thus, sexual coevolution might often generate new IASC whereas conflict resolution is likely to be slow and incomplete, resulting in an accumulating gender load (reduction in population mean fitness as a consequence of sexual conflict).

Impediment or driver of adaptive evolution?

Current thinking stresses the potential for IASC to impede sexual coevolution, but theory suggests that IASC could also drive adaptive evolution and speciation. We consider both perspectives below.

Intralocus sexual conflict as a brake on evolution

IASC is defined as an impediment to the evolution of optimal sexual dimorphism. However, because the bearer

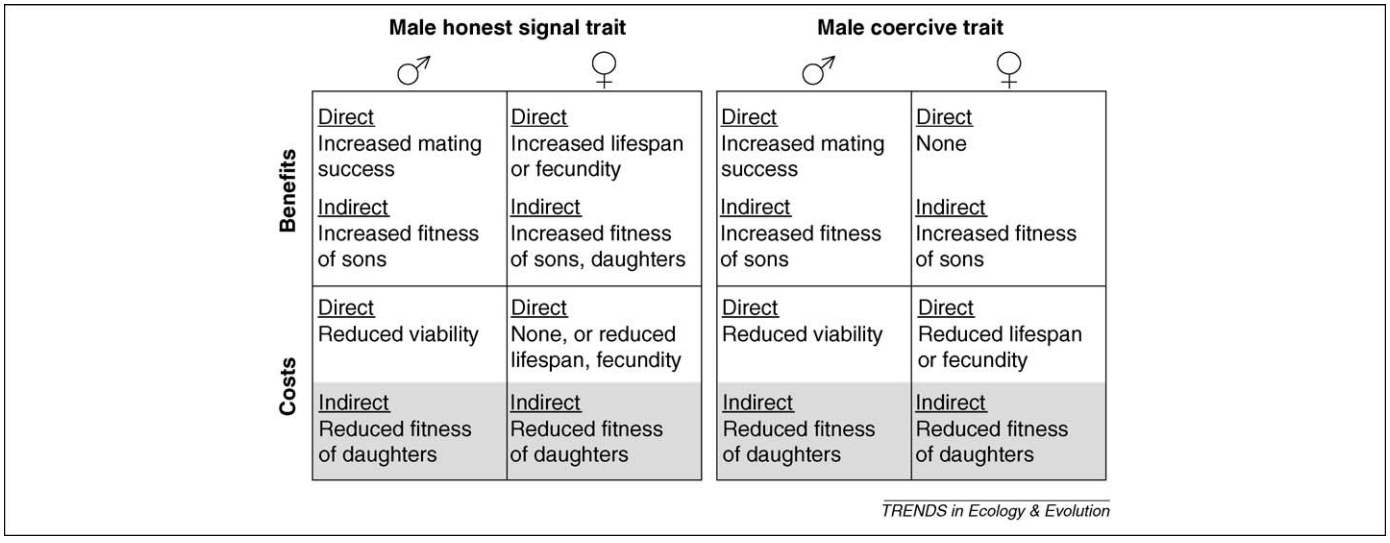

Figure 3. Direct and indirect benefits and costs to males and females of a male honest signal of 'good genes' (i.e. a trait whose expression conveys information about heritable genetic quality) (left) or coercive trait involved in IRSC (right) that could occur, according to theory [3,64]. The shaded area represents some of the indirect costs associated with IASC (additional indirect costs will reflect suboptimal trait expression in males as a result of opposing selection in females). Such costs will be reduced if the trait is male limited when it first appears, or becomes male limited through modifications to the genetic architecture. Analogous benefits and costs pertain to a female-benefit trait.

of any novel secondary sexual trait suffers indirect costs that offset its benefits (Figure 3), we argue that IASC can impede any form of sexual coevolution. It has been noted that IASC challenges 'good genes' models based on the evolution of female mating preferences for honest indicators of male genetic quality [6,63,64]. Under IASC, the most successful males sire low-quality daughters, and even their sons might be of only average quality if many male-benefit loci are X linked and, thus, never transmitted from father to sons [64]. IASC can thus reduce or eliminate the potential indirect benefits to females of preference for male signals of high genetic quality. However, for analogous reasons, IASC could impede the evolution of male traits that are harmful to females (Figure 3), and thus curb the escalation of sexual arms races.

We note that, by impeding sexual coevolution, IASC could have varying consequences for the evolution of population mean fitness. Theory [65] and some empirical evidence (e.g. [66]) suggest that sexual conflict can reduce population mean fitness and increase extinction risk. IASC could contribute to such effects [4]. However, because IASC could also impede the escalation of IRSC, the net effect of IASC on population mean fitness probably depends on the dominant form of sexual coevolution occurring in the population.

Many arguments about the consequences of IASC trace back to the fundamental question concerning the processes that maintain additive genetic variance (V_A) for fitness in natural populations. We know very little of the relative contributions of mutation, fluctuating and sexually antagonistic selection and migration to observed levels of V_A [63]. If V_A for fitness is largely mutational, IASC could be relatively inconsequential because most mutations are likely to be deleterious for both sexes. By contrast, IASC is likely to be of critical importance if sexually antagonistic selection contributes substantively to V_A, as often suggested [13,16,32]. Indeed, laboratory experiments with flies suggest that a large fraction of standing V_A for adult fitness could be sexually antagonistic [6,38].

Interestingly, whereas single-locus models show that sexually antagonistic selection can maintain stable polymorphism [5,67], multilocus analysis shows that V_A is not maintained at more than two loci [68]. Thus, for each trait subject to persistent IASC, sexually antagonistic fitness variation could be dominated by the effects of just one or two loci.

Intralocus sexual conflict as an engine of evolution

Paradoxically, theory also suggests that IASC could promote adaptive evolution and speciation. Two decades ago, Lande and Kirkpatrick [69] showed that selection on one sex can drag both sexes to the bottom of a fitness valley and, ultimately, allow the population to ascend a new fitness peak. Thus, trait evolution is initially driven by selection on one sex, and imposes viability costs (i.e. IASC occurs). However, once trait evolution reaches a certain phenotypic threshold, the trait permits the exploitation of a new niche, and further evolution can be driven by concordant selection in both sexes. The long-standing problem of peak shift on the fitness landscape [70] is thus potentially solved by IASC because, whereas viability selection cannot move a population to a point of lower mean fitness, sexual conflict can readily do so (see Ref. [65]). Even if the trait under sex-specific selection evolves sex-limited expression, once the genetic machinery exists, the evolutionary modification of a simple genetic switch can bring about the trait's expression in the other sex (e.g. see Ref. [71]). Low viability of hybrids between the new and ancestral populations can then generate strong selection favouring reproductive isolation mechanisms and promoting speciation [69].

Evidence for a primary role for sexual selection and conflict in speciation is equivocal [72], but the potential role of IASC in niche shift and the evolution of novel adaptations merits further investigation. If IASC promotes adaptive evolution, we can expect cases where an ancestral state of trait elaboration in one sex only gives rise to increased trait elaboration in both sexes. In extant species, we can expect to see similar traits having sexual functions in one sex in some groups, but viability-related functions in both sexes in related groups. There are many possible examples of adaptations that could evolve in this way. For example, colour patterns in fish, birds and butterflies that enhance viability through crypsis, mimicry or aposematic signaling could have evolved from sexual signals. Similarly, horns and antlers that serve as antipredator defences in mammals could have evolved from weapons used in male–male competition. Comparative approaches could be used to test such hypotheses.

Prospects

The study of IASC is in its infancy, and many important questions remain to be answered (Box 3). Theoretical analyses have addressed the efficacy of sex linkage [5], genomic imprinting [49] and sex-specific allelic effects [18] as solutions to IASC, but other mechanisms, such as gene duplication, sex limitation, condition dependence and sex-ratio manipulation await formal analysis. Ultimately, we require a general model of the evolution of genetic architectures under different patterns of selection, alongside detailed empirical dissections of the genetic architecture of sexual dimorphism. It is likely that next-generation sequencing technologies will provide new empirical insights here, deepening our understanding of the sex specificity of expression in model species and broadening the study of genetic architecture to a wider range of taxonomic groups. The most ambitious questions concern

Box 3. Outstanding questions

- How much sexually antagonistic fitness variation do natural populations support?
- What phenotypic traits mediate IASC, and what is the form of natural and sexual selection on such traits?
- How often do novel conflicts arise, and how much do changes in sex-specific optima versus sex-biased mutational effects contribute to this process?
- What genetic architectures evolve to resolve intralocus sexual conflict, and what are the intermediate stages in this process?
- How rapidly and fully are intralocus sexual conflicts resolved?
- Does intralocus sexual conflict impede the evolution of secondary sexual traits and the rate of sexual coevolution? Can intralocus sexual conflict drive adaptive evolution and speciation?

the rate and extent of conflict resolution, the frequency with which new conflicts arise and the consequences of IASC for sexual coevolution, adaptation and speciation.

Because most empirical studies focus on existing sexually dimorphic traits, a consistent challenge is to determine whether the observed genetic architecture is in fact a direct consequence of sexually antagonistic selection (e.g. [73]). Badly needed are studies that trace the steps leading to the evolution of sexual dimorphism by comparing patterns of gene expression as well as selection in conspecific populations and in related species. Within a phylogenetic context, such an approach would yield a historical picture of parallel changes in genetic architectures and patterns of sex-specific selection. Moreover, much of our understanding of IASC still rests on laboratory studies of *D. melanogaster*. Studies on a broad range of species, especially under natural conditions, are needed to confirm the generality of these findings, provide comparative data and identify traits mediating IASC.

A word of caution: in laboratory studies, artefactual IASC can arise (or become intensified) when the transfer of study organisms from the wild to the laboratory alters selection in a sex-specific manner. Consider a trait that functions in predator avoidance or dispersal in both sexes in the wild, but also plays a role in male sexual competition. Under laboratory conditions, the predator-avoidance and dispersal functions are eliminated, whereas the sexual functions are maintained. Transfer to the laboratory would thus effect a much larger shift in the female optimum than in the male optimum for this trait, and generate sexually antagonistic fitness variation (although this could be overwhelmed initially by sexually concordant fitness variation generated by the novel environment). Great care is needed to avoid such artefacts.

However, artificial IASC can also be a powerful research tool. Sexually antagonistic selection can be applied to sexually monomorphic traits, with follow-up assays tracking the evolutionary response at the genetic and phenotypic levels. Such experiments, combined with QTL, microarray and expression studies that dissect the genetic architecture in detail [29], are likely to yield the most valuable insights.

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