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# Sex and Conflict

**Linda Partridge and Laurence D. Hurst**

REVIEW

Evolutionary conflict occurs when the deterministic spread of an allele lowers the fitness either of its bearer or of other individuals in the population, leading to selection for suppressors. Sex promotes conflict because associations between alleles are temporary. Differing selection on males and females, sexual selection, and differences in transmission patterns between classes of nuclear and cytoplasmic genes can all give rise to conflict. Inert Y chromosomes, uniparental inheritance of cytoplasmic genes, mating strains and sexes, and many features of sexual behavior may have evolved in part as a result of evolutionary conflict. Estimates of its quantitative importance, however, are still needed.

Why do around 5 percent of species of flowering plant have a significant proportion of individuals that are male sterile (*1*)? Why does the Y chromosome of the fruit fly *Drosophila melanogaster* contain multiple copies of a gene whose sole function appears to be suppression of the effects of another multicopy gene on the X chromosome (*2*)? And why does mating sometimes kill female fruit flies (*3, 4*)? These failures in individual adaptation can be understood through the theory of evolutionary conflict. Conflict occurs when the spread of an allele at one locus in a population lowers the fitness either of the individuals in which it resides or of other members of the same population. The spread of this "harmful" allele therefore results in natural selection for suppressors at other gene loci, which reduce the phenotypic effects of the original allele (*5*).

One of the first people to document this situation was Östergren (*6*) [see also (*7, 8*)] who argued that the small B chromosomes in many plants could be "parasitic." B chromosomes can be costly to their host (*9*), and they themselves will be subject to the fitness reduction that they cause. However, as Östergren noticed, some B chromosomes have mechanisms by which they are transmitted at a greater than Mendelian rate. This "overrepresentation" can be sufficient to ensure their spread in the population, even if they are bad for the plant, or as Östergren concluded, "They need not be useful for the plants. They need only be useful to themselves" (*6*, p. 163) (*10*).

Let us assume that an organism with a B chromosome has fitness $1 - s$, whereas an organism with none has fitness 1. If an organism with the B chromosome transmits this to a proportion, $k$, of its progeny, the spread of the element is possible if $2k(1 - s) > 1$. If the element can gain over-representation ($0.5 < k \leq 1$) then $s > 0$ can hold, that is, the chromosome can both be deleterious and spread. Related calculations can apply to other genetic elements such as transposable elements (*11*) and meiotic drive genes (*12–14*). The spread of a parasitic chromosome that reduces the fitness of its host creates the conditions for the spread of suppressors (*15*). There is then a potential conflict between the B chromosome and the genes of the host genome (*16*). For didactic purposes, we shall here present verbal evolutionary arguments. However, purely verbal arguments can mislead, and those we present have, in the main, been subject to extensive theoretical analysis. Where the arguments are on a less secure footing, we shall point it out.

Conflict can also be instigated as a result of interactions between individual organisms. Consider, for example, an allele that when present in a male bird causes him to prevent the female from mating with other males, to the point where he interferes with her feeding success (*17*). The reduced feeding success of the female may reduce her fertility or survival. However, the allele in the male may increase its own rate of transmission, despite reducing the fertility of his mate, because it will increase the proportion of her offspring that are sired by this particular male (*18–20*). There may now be selection on other gene loci to suppress the fertility reduction caused by the mate-guarding behavior. This outcome would increase the fitness not only of alleles present in females, but also of alleles in males, provided that it did not also reduce the effectiveness with which males other than the mate were denied access to the female. This type of evolutionary conflict has received less theoretical attention than has intragenomic conflict.

Sexual reproduction greatly increases the likelihood of evolutionary conflict. In an asexual, clonal species all the genes present in an individual are in permanent association and share their evolutionary fate. The fitness effects of one allele on the individual therefore affects its own transmission in the same way as that of all the other genes in the organism. In contrast, in a sexual population, associations among genes at different loci are temporary and are broken up through sex and recombination. Intragenomic conflict is therefore more likely with sex (*21*). Situations in which conflicts may occur can be deduced from Price's notion of fitness covariance (*22*). When two genes are in permanent association, a positive increment in the fitness of one is a positive increment in the fitness of the other: their fitness covariance is positive. The conditions permissive for negative fitness covariance are those permissive for

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conflicts. Sex and recombination generate the potential for negative fitness covariance.

Different transmission patterns of genes to offspring create the context for negative fitness covariance. Because B chromosomes can reduce the fitness of their host organism but can be transmitted to other individuals at a greater than Mendelian rate, the B chromosome's fitness and that of the other genes in the host genome can negatively covary. Cytoplasmic genes, such as those in chloroplasts and mitochondria, are present in both sexes but usually transmitted by females alone; Hence, a gene increasing cytoplasmic genomic fitness, while decreasing male fitness, can spread. These differences in transmission patterns can result in conflicts over the sex ratio (*12, 23*) and in the evolution of novel forms of sex determination (*24, 25*). When a gene in one sex can increase its own transmission by means that reduce the fitness of the mate, the fitness covariance between it and the genes in the mate is negative. Sexual selection (*26*) is therefore a potential source of evolutionary conflict.

Conflicts have some unusual features. They can, for example, lead to the evolution of genetic redundancy (*27*) and to antagonistic coevolution involving sequential episodes of gene substitution (*28*), with the potential to lead to rapid population divergence, reproductive isolation (*29*) and even major evolutionary novelties (*16, 30*) [for example, transposable elements may play a role in karyotype evolution (*30*)]). They can even, in theory, lead to species extinction (*12, 31*). Not all conflicts have these unusual properties; some can be resolved and are temporary.

## Forms of Conflict

The spread of a harmful allele starts all conflicts, but sometimes the harm is incidental to the spread (*32*). Suppose that in the competition for females, male deer with larger antlers are more successful. An allele that increases antler size will then be favored in males but may be disadvantageous in females, leading to sexually antagonistic selection if the allele is expressed in both sexes. If there is a net advantage, the mutant will spread through the population, to the detriment of females. Here the harm is incidental, and if either the allele itself, or any other gene, can confine expression of the allele to males (sex-limited expression), then it too will spread, the conflict will be resolved, and that will be the end of it (*33, 34*). In other instances the harm caused by the allele is necessary to its spread. When a male forces a female to mate with him, any cost to the female in not being able to have further mates (if her mate is subfertile or an otherwise less fit mate than alternative males) is necessary for the allele to increase the fitness of its male bearers.

The stability of the evolutionary outcome of conflict will therefore depend in part on whether conflicting selection can be prevented. Stability will depend also on the availability of new mutations to continue the response to conflicting selection on different genes.

*The evolution of sex differences and genetically inert Y chromosomes.* Are the sexes held back in their separate adaptive evolution by their common gene pool, or is sexually antagonistic selection merely a brief passing phase in the evolution of sex differences? The evolution of sex-limitation is likely to be slow (*34*), in part because it requires de novo evolution of sex-specific control sequences (*35*). Detailed studies of selection in natural populations [see for example (*36*)] have demonstrated conflicting selection on particular genomic segments in the two sexes. An artificial selection experiment with *Drosophila* showed that when the three main chromosomes (99% of the haploid set of genes) were experimentally confined to males for 32 generations and then transferred into females, the result was a reduction in net female fitness, mainly as a result of delayed development. This reduction in female fitness was a consequence of an increase in frequency of sexually antagonistic (male-benefit and female-detriment) alleles, because male mating speed, mating success, and success in sperm displacement were all higher in the experimental than in the control males (*37*). The indications are that their common gene pool does both result in sexually antagonistic selection and restrict the rate of adaptive evolution of males and females, but quantitative assessment of these effects is a task for the future.

Sex-specific selection has been important in the evolution of sex chromosomes. Heteromorphic sex chromosomes have evolved independently many times, from different precursors, and probably by somewhat different routes in animals and plants, but similar forces are thought to have been at work in each case (*38–40*). A dominant male-determining region on a proto-Y chromosome is expected to accumulate male-advantage, female-disadvantage alleles in its vicinity [compare with (*41*)]. There will be selection for suppression of recombination between the sex-determination region and the male-advantage/female-disadvantage genes linked to it, because then they will not be transmitted to daughters. Suppression of recombination would also increase the fitness of other genes in the genome (*42, 43*). Modifiers of recombination in particular genomic locations are known to be present in natural populations (*44*).

Selection for restriction to males may explain (*39*) why in guppies a high proportion of loci known to code for sexual ornaments appear closely linked to the male-determining locus (*45*). These ornaments increase vulnerability to predators but are of advantage to males in obtaining mates. Suppression of recombination on the Y chromosome resolves sexual antagonism, but it may carry the seeds of destruction for the affected genes on the Y. Several theoretical approaches have shown how the nonrecombining Y chromosome would be expected to deteriorate from the effects of mutation (*46*), an effect experimentally demonstrated (*47*). As the Y chromosome genes deteriorate, there is selection for some form of dosage compensation on the X chromosome (*38*). The evolutionary stage of homology between the X and Y chromosome, but with deterioration of genes on the Y, has been detected in *Drosophila miranda* (*48*) and a dioecious plant (*49*).

If the expression of a phenotype that reduces female fitness but increases male fitness becomes confined to males, the fitness of all the alleles with which it is associated when in females is restored. The spread of an allele that produces this sex-limited expression will therefore resolve the conflict; it does not induce selection for further suppressors, and the equilibrium is stable. In other cases, where there is not a phenotypic solution that removes conflicting selection, any equilibrium will be unstable, and the potential exists for continuing antagonistic coevolution at different gene loci.

*Meiotic drive and the iteration of conflicts.* Meiotic drive occurs when a nuclear gene obtains, during meiosis, a transmission advantage (*50*). Reduced fitness of the host can arise, for instance, through the destruction of half of the parent's gametes or through biasing the sex ratio in offspring (if the driving allele is on a sex chromosome). Consider an X-linked meiotic drive gene that kills Y-bearing sperm from the same father. There is then selection for Y-linked (*14*) and autosomal suppressors of drive (*12, 51*), and these have been found, for instance, in natural populations of *Drosophila simulans* (*52*). Suppose that such a suppressor arises on the Y chromosome and spreads, and the usual sex ratio is restored. If the suppression were dose-sensitive, increased expression of the X-linked drive gene could result in drive once more and provide the conditions for the evolution of suppression, and the cycle could be repeated many times.

Such a history may explain the evolution of multicopy gene families on both the X and the Y chromosome of *D. melanogaster* (*25, 53*). *Suppressor of Stellate* is a Y-linked multicopy gene

whose sole activity appears to be suppression of the transcription and translation of the multicopy *Stellate* gene on the X chromosome. The Stellate protein is homologous to the β-subunit of casein kinase II and hypothesized to be involved in the packing of the Y chromosome (*53*). Support for the proposal that the two coevolved in cycles of drive-suppression has come from the finding that, in the absence of the Y-linked suppressor, there is meiotic drive (*54*), and the intensity of this drive goes up as *Stellate* copy number goes up (*27*). Further evidence is required on the effects of deletion of *Stellate* on transmission rates of the X chromosome.

*Conflict between nucleus and cytoplasm.* The gene *Suppressor of Stellate* acts simply to inhibit the putative distorter. Other suppressors alter the form of the conflict (*16*). Consider, for example, the interaction between the nuclear and cytoplasmic genomes.

Deletion of a small part of a mitochondrial genome will often interfere with the normal functioning. Despite being deleterious to the mitochondrion and to the cell in which it resides, small mitochondrial genomes, like those of yeast petite mutants (*55*), replicate faster and can potentially spread in a population if reproduction is sexual and there is biparental inheritance (otherwise they would be stuck in a clone, and their transmission rate would be the same as that of the host genome). Could this be the reason that inheritance of organelles is predominately uniparental? Theory supports such a proposition (*56, 57*).

The spread of a deleterious fast-replicating organelle genome (Fig. 1) creates the conditions for spread of a nuclear allele that produces uniparental inheritance of organelles. If the nuclear allele kills the organelles from its partner cell, then if that allele is initially associated with the advantageous, slower replicating organelle genome, it stays associated with it. Unlike competing theories (*58*) this one is consistent with observed patterns of uni- and biparental inheritance (*59*). The theory can also be extended (*57, 60–62*) to explain the evolution of mating-type alleles (mating strains) in organisms where gametes are all the same size (*63*) and the small size of sperm in species with male and female gametes (*64*). Genetic candidates for a nuclear gene that digests DNA of the partner's chloroplast in *Chlamydomonas* have been identified, but none has been characterized mechanistically (*65*).

Once uniparental inheritance evolved, the potential conflicts between cytoplasmic and nuclear genes were not over, but they altered their form. Consider a cytoplasmic factor that converts males into females. It would spread, because males do not transmit cytoplasmic genes (*43, 66*). More generally, selection can act on cytoplasmic genes to distort the sex ratio toward females (*23, 66–68*). In some isopod crustaceans cytoplasmic bacteria, *Wolbachia*, turn males into females (*24*), and in ladybirds other bacteria kill sons (*68*) (Fig. 2).

The best described cytoplasmic sex-ratio distorters are the mitochondrial genes associated with male sterility in monoecious plants (*69, 70*). The theoretical conditions for the spread of cytoplasmic male sterility are very much broader than those for nuclear-induced male sterility (*71*). The importance of the transmission dynamics for the spread of the trait is indicated by the finding that most spontaneous mutations inducing male sterility are nuclear, but most recovered in the field are cytoplasmic (*72*). The mutations producing male sterility, at least in domesticated plants, are rearrangements of the mitochondrial genome that result in the formation of new chimeric genes with novel products (*69*). The distortion of the sex ratio sets up selection for nuclear genes that restore the sex ratio, and these are present in many natural populations with cytoplasmic sex-ratio distorters (*73*). In

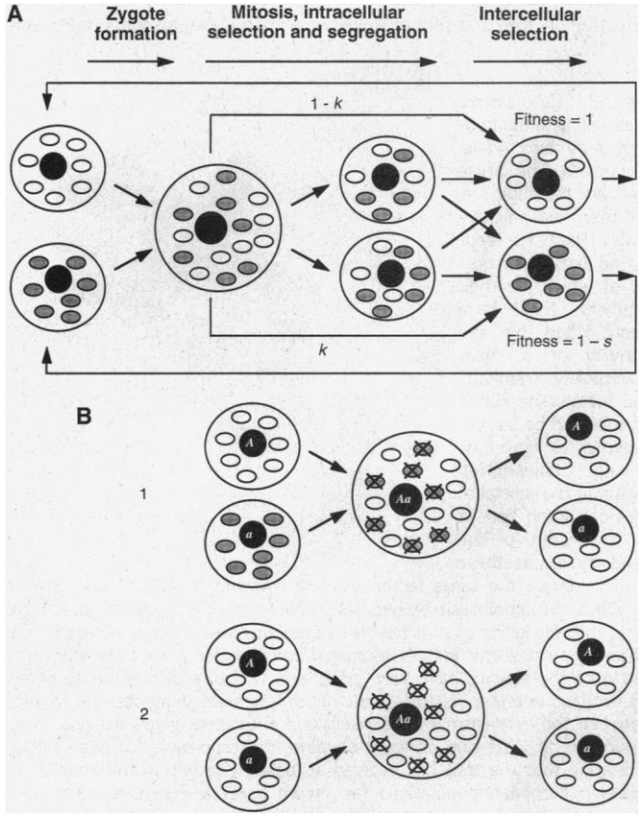


**Fig. 1.** A possible route to the evolution of uniparental inheritance of cytoplasmic organelles. **(A)** In a population with biparental inheritance, an organelle containing a fast-replicating but deleterious genome (gray) can arise by mutation (wild-type organelles shown as unfilled ovals, nuclei as black circles). The deleterious genome can spread if the population is sexual, because, after zygote formation, the fast-replicating genomes out-compete the slower ones in intracellular competition. A short time after zygote formation, cells may still be heteroplasmic, but the faster dividing mitochondrial genome is expected to be more frequent. Organelle replication rate differences and organelle segregation, through random sampling during cell division, tend to lead to cells being homoplasmic. The population genetics of deleterious organelles can be approximated by considering the step between zygotic heteroplasmy and postzygotic homoplasmy as effectively instantaneous; hence, we can reduce the problem to two parameters: (i) the proportion ($k$) of progeny of heteroplasmic zygotes that are homoplasmic for the deleterious genomes; if the deleterious organelle is fast replicating, $k$ is greater than 1/2; (ii) the selective coefficient ($s$) acting on cells homoplasmic for the deleterious organelle; because these genomes are deleterious, they are at a disadvantage in intercell competition (cells with the disadvantageous genome have fitness $1 - s$, $1 > s > 0$). Spread is possible if the intracellular competitive advantage outweighs the intercell competitive disadvantage, that is, when $2k(1 - s) > 1$. **(B)** After the invasion of the deleterious organelle genome, a nuclear modifier that forces uniparental inheritance can spread. Invasion conditions are most broad when the modifier (the *A* allele at the nuclear and Mendelian *Aa* locus) is initially found with the organelle type associated with high cellular fitness. If the *A* allele excludes the organelles from the partner cell, it will always be associated with the "fitter" organelle genome. Initially the allele will be rare, so only two matings are possible: (i) a mating between *A* and a cell with *a* and the deleterious organelle genome; *A* eliminates the deleterious genome and so prevents intracellular competition; and (ii) a mating between *A* and a cell with *a* and the advantageous genome. In this case the action of the modifier is neutral. The alternative allele, *a*, may be associated with the deleterious organellar genome. As a consequence, the relative fitness of the *A* allele will be higher than that of the *a* allele and can spread. Note that, if the *A* allele were initially associated with the deleterious organellar genome, then it would stay associated with it and would be lost from the population. Note too that, were fast-replicating, advantageous organellar genomes to arise by mutation, uniparental inheritance would slow, but not prevent their fixation.

the sunflower, restoration is achieved by regulation of the activity of the new, chimeric mitochondrial gene responsible for the male sterility (*74*).

*Conflicts between mates: Molecules to behavior.* Thus far we have concentrated on intragenomic conflicts expressed at the molecular level and interindividual conflicts expressed at the individual level (for example, male control of female activity). There is, however, a possibility of cross-talk between the levels. There are claims, for example, that females might prefer males without sex ratio or other distorters (*75*). Others speculate that the costs associated with distorter-related incompatibilities may have provided the conditions for the evolution of polyandry (*76*). Conflicts between individuals may have many potential outlets, some behavioral and overt, others molecular and subtle. Consider conflict between mates. If monogamy is not absolute, and if success in the current reproductive attempt can be increased by traits that lower the fitness of the mate, then alleles for the traits will spread (*19, 20, 77*). There will be selection at other loci to counter the fitness reduction (*77*).

As a consequence of their higher input of time and energy into each offspring, females in general spend less of their time in the pool of available mates than do males (*78–80*). Mating is not a neutral event for females; it can attract the attentions of predators (*81*), it can injure the female (*82*), it can infect her (*83*), and it can adversely affect her interactions with other males (*84*). Given an approximately equal sex ratio, encounters between males and females will often be asymmetrical, with males selected to mate and females not. Sexual conflict over mating is therefore common.

Female resistance to mating can produce reproductive costs for males. *Drosophila* males perform elaborate courtship, and sexual activity shortens the life-span (*85*), mainly as a cost of courtship (*86*). Male courtship may in turn affect the sensory systems of females so as to cause them to mate more often than is necessary for full fertility, selecting in turn for female resistance to male courtship; more evidence on this point is needed (*87*). Males can also induce females to mate by persistence (*78*); by mimicry of preferred-male mating status (*88*); and by physical force, intimidation, and punishment of persistent refusal to mate (*19, 20*). Females therefore often avoid areas where males are abundant and associate with males capable of fending off other males (*20*).

**Fig. 2.** Cytoplasmic male killing in the two-spot lady bird *Adalia bipunctata*. The larvae that are hatching are, for the most part, females. There are feeding on eggs the majority of which are their brothers that have been killed by the activity of a cytoplasmically transmitted factor. The killing of sons can be advantageous to cytoplasmically transmitted factors if the mortality provides some benefit to the sisters of the dead males, as these sisters contain the same factor and will transmit it (*68*). In the case of ladybirds, the cannibalism by females probably provides a growth advantage. Note that the killing of sons has no deleterious consequences as regards the fitness of cytoplasmic factors because these were not going to be transmitted were the sons to have survived. It is likely to be deleterious to genes transmitted in a Mendelian fashion; hence, there exists a potential conflict between the cytoplasmic male killers and autosomal genes in sons. In *A. bipunctata* at least two different bacteria are responsible for male killing, indicating that the trait has evolved at least twice within this species. In Western European populations (as shown here) a rickettsial bacteria is responsible, whereas in Eastern European populations a *Sprioplasma* is the causative agent. Both are strictly maternally inherited. For further examples of male killing see (*68*). [Picture courtesy of G. D. D. Hurst and M. E. N. Majerus.]

Sexual conflict can continue after mating. The seminal fluid of male *D. melanogaster* contains proteins that increase the female's egg-laying rate (*89, 90*), that reduce her sexual attractiveness (*91*) and her receptivity to further matings (*89, 92*), and that attack and defend against sperm of other mates of the female (*3*). These activities increase the reproductive success of the male. However, proteins in the seminal fluid can also kill females that mate at a high rate (*3, 4*), possibly as an unselected side effect (*93*). Females normally avoid the killing effect, at least in part by a low mating rate (*94*). In an artificial selection experiment, females were prevented from evolving in response to males—while male adaptation to the females continued for 41 generations and suffered high death rates when reexposed to the adapting males, suggesting that there may be continuous antagonistic coevolution between the sexes. The adapting males mated more often with the stalled females than did control males, and their matings had a greater female-killing effect, possibly as a consequence of more toxic seminal fluid (*35*), although this was not directly established. Some of the molecules in the seminal fluid responsible for its biological activity in the female have been identified (*89, 92*) and at least one shows an unusually high evolutionary rate and patterns of variation within and between species that suggest that natural selection is acting (*95*).

Conflicts may also underlie some features of egg-sperm interactions at fertilization, with genes in sperm selected to produce rapid penetration of the egg and genes in the egg to resist polyspermy (*96*). Conflict need not end with fertilization. When females produce offspring by more than one male, paternally derived genes will have increased fitness if they cause the offspring to gain more than an equitable share of maternal resources, whereas maternally derived genes are more likely to have maximal fitness with a more equitable distribution (*77*). Similar logic provides a hypothesis (*97*) for the evolution of genomic imprinting, the differential activity of some genes dependent on the sex of parent from which they were inherited. Although the hypothesis provides a parsimonious interpretation of the interaction of insulin-like growth factor 2 and its antagonists (*98*), a significant number of details are not obviously consistent with the hypothesis (*99, 100*) and its current status is unclear. The angiosperm endosperm, like the placenta in mammals, is the tissue that mediates transfer of resources from mother to seed. The double maternal component of the triploid endosperm (*101*) and its imprinting pattern (*102*) may ensure greater maternal control of resource allocation to different seeds, although whether this was the selective force for its evolution is hard to determine.

## The Bigger Picture

How much evolutionary change is the result of conflict, how much conflict is there at any one time, and how would we know? When we look at the biological world, are we seeing the smiling faces of children furiously kicking one another under the table, or is the appearance of harmonious organismal adaptation real as well as apparent? Is conflict a pervasive, gradualistic force likely to shape genomes and organisms, or rather a rare and exceptional one?

As long as different genetic entities within a population can increase their fitness by producing different trait values from each other, the potential for evolutionary conflict will exist (*30*). However, natural selection is not the only force at work here, and the incidence of conflict can be understood only by also including details of biology and the possible range of phenotypic effects of new mutations that these allow. For instance, *Wolbachia* can convert males into females

but this appears to be prevalent only in isopod crustaceans (*24, 67*). In these animals, female is the default state, and induction of maleness is hormonal. Abolition of the tissue inducing masculinity is hence adequate to induce feminization. Organisms with cell-specific sex determination, or where male is the default state, are therefore less likely to be affected. Comparable vulnerability can explain in part the phylogenetic distribution of *Wolbachia*-induced asexuality (*103*). However, it is not understood why *Wolbachia*, rather than other bacteria or organelles, is so often responsible. Driving sex-ratio distorters are present in *Drosophila pseudoobscura* (*104*), and there is therefore likely to be selection for suppressors, but none has been found. Possibly details of the molecular biology of the case, as opposed to other cases where suppressors are present (*52*), may provide an explanation.

Beyond the details of biology of individual cases, can we make any general statements about which entities will win conflicts where conflicting selection is persistent? Where individual organisms, such as males and females, are in conflict, theoretical analysis has suggested that asymmetry between them, both in the strength of selection on the value of winning the conflict, and in the costs to them of the contest, will determine the outcome. But again, details of biology, such as the ability to force mating or inflict punishment, are important (*19, 20, 79*). Even less is understood about when conflicts will persist, and indeed empirical evidence for persistence is largely lacking. In part this may be because conflict can be cryptic. Natural populations of *Drosophila simulans* have a ~1:1 sex ratio, suggesting that sex-ratio drive is absent. However, males from interpopulation crosses can sire female-biased broods, suggesting that the 1:1 ratio is the result of suppression of drive (*105*). Remarkably, a geographic survey based on crossing with standard strains found driving X chromosomes in all populations (*52*). The lack of phenotypic drive was no evidence for the absence of driving genes. Genes involved in persistent conflicts may, like host immune genes (*106*) and parasite antigens (*107*), evolve rapidly at the molecular level. Such a possibility may explain the rapid evolution of genes involved in maternal-fetal interactions (*99, 108*), of molecules in the seminal fluid of fruit flies (*95*), and of bacterial colicins (*28*). However, it is not clear when we should expect to see ongoing antagonistic coevolution, and when instead we might expect equilibrium. If imprinted genes are the outcome of a conflict (*97*), they might be expected to show rapid evolution, but they do not (*108*). Does this mean the idea is wrong, or that only some genes involved in conflict evolve quickly? Perhaps antagonistic coevolution did occur in the past, but stasis has now been reached. Detailed phylogenetic analysis could help resolve the issue.

In theory every sexual organism has multiple potential conflicts. In consequence, genetic systems, like social insect colonies (*109*), may have evolved to prevent the spread of harmful alleles. Methylation may be a means of suppression of transposable elements (*110, 111*), and the case of repeat induced methylation or mutation in fungi is a striking example (*110*). If conflict is responsible for species extinction (and there is no empirical evidence on this point), then traits producing a low mutation rate toward conflict could evolve (*16, 112*). We remain far from an understanding of which potential conflicts become real ones and of the quantitative extent of their evolutionary effect.

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5. We exclude those cases where an allele spreads due to a net beneficial effect on an individual, but that has deleterious pleiotropic effects in the same individual. For example, an allele that increases net fitness due to some effect in the kidney might also have a small deleterious effect on the liver. This provides the conditions for compensatory mutations (for example, those restricting expression to the kidney). This is different from sexual antagonism, where the deleterious effects occur in an individual separate from the one that has the fitness advantage. The important point is that liver and kidney cannot independently reproduce, but males and females can. We also restrict discussion of conflict to those cases where the spread of the harmful allele is deterministic.
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