The dynamic relationship between polyandry and selfish genetic elements

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Selfish genetic elements (SGEs) are ubiquitous in eukaryotes and bacteria, and make up a large part of the genome. They frequently target sperm to increase their transmission success, but these manipulations are often associated with reduced male fertility. Low fertility of SGE-carrying males is suggested to promote polyandry as a female strategy to bias paternity against male carriers. Support for this hypothesis is found in several taxa, where SGE-carrying males have reduced sperm competitive ability. In contrast, when SGEs give rise to reproductive incompatibilities between SGE-carrying males and females, polyandry is not necessarily favoured, irrespective of the detrimental impact on male fertility. This is due to the frequency-dependent nature of these incompatibilities, because they will decrease in the population as the frequency of SGEs increases. However, reduced fertility of SGE-carrying males can prevent the successful population invasion of SGEs. In addition, SGEs can directly influence male and female mating behaviour, mating rates and reproductive traits (e.g. female reproductive tract length and male sperm). This reveals a potent and dynamic interaction between SGEs and polyandry highlighting the potential to generate sexual selection and conflict, but also indicates that polyandry can promote harmony within the genome by undermining the spread of SGEs.

1. Introduction

The number of times a female mates during her lifetime is highly variable both between- and within species. With the use of molecular techniques, we now know that monogamy is the exception rather than the rule, with females of most species mating with more than one male in their lifetime—polyandry [1,2]. Mating can be costly in terms of time and energy expenditure [3], physical damage [4], receipt of seminal proteins [5] and exposure to sexually transmitted infections [6]. Multiple mating, therefore, has to confer benefits to outweigh these costs. Females may derive direct benefits such as paternal care [7], nutrient provisioning [8], sperm replenishment [9,10] and strengthened social cohesion [11] by mating multiply. In addition, they may gain genetic benefits such as increased offspring diversity [12], viability [13] and attractiveness of sons [14]. A recent meta-analysis indicated there is only weak evidence for the genetic benefits of polyandry, largely due to insufficient data from well-designed studies [15]. Females may also mate multiply to acquire compatible genotypes [16,17]. Genetic compatibility is affected by male and female relatedness [18,19], and selfish genetic elements (SGEs) [20,21]. Incompatibility stemming from relatedness is due to the exposure of deleterious recessive alleles [22]. SGEs are a frequent source of intra-genomic conflict that can cause reproductive incompatibilities between male and female genomes [23]. It has also been suggested that females may mate multiply to reduce the cost of male harassment. Such female harm minimization is predicted to be widespread owing to the potential conflict between the sexes over female mating rate [24], or as a means to obscure paternity to reduce the risk of male infanticide [25]. It is likely that more than one benefit can be obtained by polyandry to individual females.

The frequency at which females remate is of key importance to many areas in ecology and evolution. The frequency of polyandry can affect the level of gene flow and effective population size [26], and, therefore, population viability [27,28]. Polyandry also determines the level of post-copulatory sexual selection...
and sexual conflict, giving rise to a range of adaptations and counter-adaptations involving reproductive manipulation [29,30]. In addition to sexual conflict, female remating frequency determines the within family relatedness, thus affecting the intensity of parent–offspring conflict [31] and the potential evolution of cooperation and sociality [32,33]. While inhibiting cooperation at the family level, recent studies also indicate that polyandry can promote harmony within the genome, through undermining the spread of SGEs that distort transmission ratios [34]. It is not clear to what extent polyandry is generally favoured as a strategy by females to reduce the risks and costs associated with SGEs, although it is suggested to favour evolution of female multiple mating [20]. To date, there have been remarkably few experimental examinations of this possibility.

The existence of genetic incompatibility is predicted to favour polyandry as a female strategy to reduce the risk of fertilization of their eggs with sperm from genetically incompatible males [23]. The idea initially put forward is that by mating more than once, females will reduce the risk of only mating to males who carry incompatible genes. However, this bet-hedging strategy only works under very restricted circumstances [35]. Potentially of wider importance is that, whenever females mate multiply, sperm from different males will compete for fertilization of the female’s ovum [36]. If females mate several times, they may potentially swamp the sperm of incompatible males with that of genetically superior males. Of particular importance in this context is the finding that males that carry SGEs frequently have reduced fertility [37,38] and, therefore, often poor sperm competitive ability [39,40].

SGEs are genes that are present in the genome or cell of an organism that ensure they are passed on at a higher frequency than the rest of the genome to subsequent generations. This ‘selfish’ nature of SGEs ensures they will accumulate within the genome [41]. However, the subversion of the normal pattern of inheritance by SGEs generates a conflict with the rest of the genome [41,42]. SGEs are ubiquitous in living organisms, can make up a large proportion of the genome [42,43], and the intra-genomic conflicts they create are thought to have had major impacts on the evolution of sex and reproductive systems [41–44]. For example, the scarcity of males caused by sex-ratio distorting SGEs can have dramatic effects on mating systems [43]. There is strong selection for suppression of SGEs to ensure a more equitable inheritance and to restore the sex ratio to unity. Such genetic suppression of SGEs can have far reaching impacts. For example, it is suggested that methylation arose to silence SGEs, and was key to the evolution of genomic imprinting and the placenta in mammals [45]. There is also evidence that SGEs can directly affect sexual selection. Many SGEs, including meiotic drive elements, B chromosomes, endosymbionts and some transposons, target male gametes in order to increase their transmission rate, and have been shown to impair male fertility through manipulation of spermatogenesis. This often also reduces the success of male carriers in sperm competition [37]. Polyandry is, therefore, hypothesized to decrease the frequency of any SGEs that reduce the sperm competitive ability of males [34] by undermining their transmission advantage [20]. Hence, there is a dynamic link between the presence of SGEs and mating systems, with female multiple mating that promote sperm competition being favoured when they are at risk of mating with SGE-carrying males, and conversely that polyandry can also regulate the frequency of SGEs. Here, I explore the evidence for this.

2. Selfish genetic elements and sexual selection

Because SGEs are associated with a variety of costs such as reduced fertility and production of the more common sex in the case of sex-ratio distorters, non-carrying individuals are expected to avoid mating with SGE-carrying mates. There is evidence that both males and females discriminate against SGE-carriers prior to mating in some species. For example, in populations of stalk-eyed flies that harbour a sex-ratio distorter, females prefer to mate with males with long eye-stalks, as this is an indicator of a genetic suppressor of drive ensuring females will produce both sons and daughters [46]. Similarly, some populations of house mice carry a recessive lethal autosomal driving chromosome, the t-complex [47]. Heterozygous individuals prefer to mate with homozygous wild-types, and avoid other heterozygote individuals using odour cues [47]. A recent fascinating example comes from Drosophila paulistorum flies, where it is demonstrated that both males and females show assortative mating based on the strain of the endosymbiont (Wolbachia) they carry [48]. This is favoured as crosses between males and females carrying different strains of Wolbachia result in reproductive incompatibilities and embryo death (cytoplasmic incompatibility—(CI)). It is as yet not clear what the mechanism is that promotes Wolbachia-assortative mate preferences in this fly species complex, although it is possible the different strains of Wolbachia may be reflected in different cuticular hydrocarbons potentially used in mate choice. In other Drosophila species, there is evidence that different diets cause differences in the commensal gut microbiota that can directly affect mate preferences by changing the cuticular hydrocarbon sex pheromones [49,50]. Females of the two-spotted spider Tetranychus urticae also show assortative mating and oviposition preferences with respect to CI-inducing Wolbachia; uninfected females prefer uninfected males, whereas infected females aggregate their offspring, thereby promoting mating with their infected sibs [51]. In the isopod, Armadillidium vulgare harbouring feminizing Wolbachia, males prefer real females to feminized males [52]. It is possible that feminized (i.e. genotypic) males lack the ability to produce female sexual pheromones and, therefore, appear less ‘attractive’, and hence mate discrimination is not due to the presence of Wolbachia itself. There is also evidence that males may modify their mate preferences in response to SGEs. For example, in Gammarus duebeni shrimp populations infected by feminizing microsporidia, males prefer to mate with real females and provide them with more sperm. This is advantageous as feminized males are less fecund, and males are severely sperm limited [53]. However, Wolbachia-based mate preferences are not present in Drosophila melanogaster or Drosophila simulans flies, indicating that this is far from a universal pattern [54] (but see [55]).

There is also evidence for ‘maladaptive’ mate choice based on SGEs. In Xiphophorus hellerii male populations carrying a cancerous melanoma, females prefer to mate with cancerous males [56]. These males have a spotted caudal melanin pattern (Sc), which is associated with the Xmrk oncogene (melanoma receptor kinase), that has been maintained for several million years. High expression of Xmrk leads to the
development of a large black spot on the fish’s side—a cancer that destroys muscle, reduces swimming speed and causes early death—but is also associated with bigger size and increased aggressiveness. Females prefer black, cancerous and bigger males. Frequency-dependent selection maintains this gene as individuals with two copies of \( Xmrk \) have reduced viability. However, the majority of cases examining the potential for SGE-based mate choice have failed to find any conclusive evidence, despite severe fitness consequences of not discriminating against SGE-carrying individuals [54,57]. The reason for the general lack of avoidance of SGE-carriers may be the problem of recombination breaking up any association between an ornament and the SGE, and a potential preference allele allowing for SGE-based mate choice to evolve [58]. Support for this suggestion is that the only known examples that are not associated with either behavioural or phenominal differences of SGE-carriers do indeed show reduced recombination. There is little recombination between the driving and non-driving X-chromosome in stalk-eye flies [59]. The \( I \)-complex in mices is contained within a large inversion system that also contains the major histocompatibility complex and odour preference gene(s) [60]. In the swordtail mollies, it is suggested a pre-existing sensory bias in females is responsible for the preference of black cancerous males, as preference for dark males is present in populations without the oncogene, in combination with black males being more visible [56].

The limited evidence for the importance of SGEs in promoting pre-copulatory mate choice suggests that post-copulatory effects may be more important, thereby favouring polyandry. Many SGEs target male spermatogenesis in order to get transmitted to the next generation. This can be achieved by modifying sperm during development (e.g. \textit{Wolbachia} [61,62]). Other SGEs destroy sperm that do not pass on the selfish gene to subsequent generations by killing non-carrying sperm during spermatogenesis (e.g. meiotic drive [63]). However, the method of sperm killing can also have a detrimental side effect on the surviving carrier sperm that will compromise sperm performance (e.g. sex-ratio drive [40]). As a consequence of such sperm manipulation to enhance transmission of SGEs, male carriers frequently suffer reduced fertility and sperm competitive ability compared with non-carrier males [37,38]. This provides an important link between SGE-carrying and reduced fertility, and sperm competitive ability in males, which is predicted to promote polyandry as a female strategy to avoid passing on SGEs to offspring [20,37].

3. Do selfish genetic elements promote polyandry?

Is there any evidence that the presence of SGEs favours polyandry? In \textit{D. simulans} carrying the Riverside strain of \textit{Wolbachia} (\( uRi \)), crosses between uninfected females and infected males result in greater than 90 per cent hatching failure owing to CI. This enhances the transmission of \textit{Wolbachia} as all other crosses involving infected individuals are fertile and result in \textit{Wolbachia}-infected offspring. Uninfected females do not discriminate against infected males as mates [54]. However, infected males are poor sperm competitors relative to uninfected males [39]. Uninfected females mating multiply with at least one uninfected male are able to recuperate their fitness loss caused by CI owing to the poor sperm performance of infected males, and have the same reproductive output as infected females (figure 1a,b). As a consequence, polyandry should be favoured by infected females. There is some evidence this is indeed the case. Uninfected females (that are at risk of reproductive incompatibilities) remate sooner than infected females [64]. However, despite the poor sperm competitive ability of infected males, this benefit cannot promote polyandry in itself. The reason is the relatively low probability of uninfected females mating with both an infected and uninfected male, which will become increasing less likely as the frequency of \textit{Wolbachia} increases and most individuals are infected (see §4).

The situation is different when the benefit of polyandry is not dependent on the frequency of male and female genotypes (e.g. uninfected females and infected males), but solely linked to the magnitude of the fertility reduction of male carriers. In \textit{Hypolimnas bolina} butterflies for example, many South Pacific populations harbour a male-killing strain of \textit{Wolbachia} [65]. Intriguingly, different populations have different frequency of male killers that is associated with differences in degree of female-biased sex ratio, with a higher frequency of male killers associated with a more severely female-biased population sex ratio [66]. As a consequence, the mating system differs in relation to the frequency of male killers. In high prevalence populations, males provide smaller sperm packets than in low prevalence populations, which is likely due to their higher mating frequency in these high prevalence populations as a consequence of the pronounced female sex-ratio bias (figure 2a). This in turn promotes female multiple mating, likely due to the increased severity of sperm limitation experienced in high prevalence populations (figure 2b). This creates a cycle

![](http://rstb.royalsocietypublishing.org/)
poor sperm competitors [40]. Female multiple mating by promoting sperm competition may thus be an effective strategy to undermine the transmission of SR by biasing paternity towards non-carrying males. We therefore predict that females that are at risk of SR should evolve to remate more frequently and at a higher rate. This possibility was examined using experimental evolution in replicate populations, demonstrating that females do indeed rapidly evolve higher remating rates in the presence of SR (figure 3; [69]). This effect could be attributed to the presence of SR and not due to the associated female-biased population sex ratio, as SR alone did not result in evolution of higher female remating rates. The reason for the rapid evolution of higher female remating rates in SR populations may be partially due to sperm and/or ejaculate depletion, which will be exacerbated in sex-ratio males under a female sex-ratio bias.

In mice populations harbouring an autosomal meiotic drive system, the $t$-complex, it has recently been shown that polyandry is favoured, because female multiple mating undermines the frequency of the $t$-allele by promoting sperm competition that disfavours heterozygous $t/+ \, \text{males}$ owing to the production of reduced sperm numbers [7]. Recent theoretical work together with extensive laboratory and field experiments also found that the dynamics of $t$ alleles in a natural population of house mice could only be explained by the transmission disadvantage owing to poor sperm competitive ability [70], which will favour polyandrous females. Similarly, in *Solenopsis invicta* fire ants, there is evidence that a selfish gene ($\text{Gp-9b}$) that determines the social organization of the ant colony, also directly influences the level of polyandry in queens. Workers carrying the $\text{Gp-9b}$ allele only tolerate sexuals that also have the $\text{Gp-9b}$ allele, and always reject and kill $\text{Gp-9b}^{\text{e}}$ queens. However, males carrying the $\text{Gp-9b}$ allele have low sperm counts and as a consequence suffer reduced paternity in mixed broods compared with $\text{Gp-9b}^{\text{e}}$ males. In addition, the low sperm counts of $\text{Gp-9b}$ males result in increased female remating rates, presumably as a direct consequence of receiving limited amount of sperm. Reduced paternity in sperm competition together with an inability to suppress female receptivity represents a dramatic reduction to the reproductive success of $\text{Gp-9b}$ males [71]. Polyandry in this ant species is, therefore, largely

of male fatigue with a concomitant female sperm shortage that will further promote polyandry. At extreme levels of male killing, female mating becomes directly limited by access to males [66]. Polyandry is favoured in this species because of the limited supply of sperm owing to the shortage, and exhaustion of the few males in high prevalence populations because of the female-biased sex ratio that in some populations can be as high as 100 females to one male [65].

Some populations of *Drosophila pseudoobscura* harbour an X-linked meiotic drive element, ‘sex ratio’ (SR) that results in female only broods owing to the elimination of all Y-chromosone sperm during spermatogenesis [63]. In females, SR has no consistent effect. Despite being an ancient drive system of approximately 70,000 years, there is no genetic resistance or suppression of SR drive in *D. pseudoobscura* [67], and females cannot distinguish male carriers from normal males [57]. Moreover, there is considerable variation in the frequency of SR, which has remained remarkably stable for more than 50 years [68]. The loss of sperm by SR-carrying males makes them

**Figure 2.** (a) Spermatophore size as a function of male mating rate in the butterfly *H. bolina*. Negative correlation is highly significant (Spearman’s rank correlation test, $\rho = -0.66, S = 2206, p = 0.0002$). (b) Female mating rate as a function of population sex ratio. Model comparison showed that a regression containing both a linear and a quadratic term (dashed curve) fits the data significantly better than a linear one ($F_{1,17} = 12.10, p = 0.002$) or a purely quadratic one ($F_{1,17} = 11.34, p = 0.004$). Female mating rate was estimated from the mean number of spermatophores per female. Sex ratio is given as log of number of females per male. Adapted from Charlat et al. [66].

**Figure 3.** The proportion of experimental evolution line *Drosophila pseudoobscura* females remating at their first opportunity, for each selection regime ($n = 11$ lines), when offered standardized stock males, showing median, interquartile range, and range ($*p = 0.0383; \text{n.s., not significant}$). Adapted from Price et al. [69].

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determined by the reduced sperm production of Gp-9^b males, again corroborating the existence of a link between carrying a SGE and reduced sperm numbers [37].

4. When do selfish genetic elements promote polyandry?

It was initially suggested by Zeh & Zeh [20,23] that genetic incompatibilities have the potential to promote polyandry as a female strategy to bias paternity against incompatible males. While there is evidence that SGEs are a frequent source generating genetic incompatibilities between male and female genomes, there is remarkably little experimental examination of this hypothesis. Despite this, there has been an explosion of reviews examining the potency of genetic incompatibilities to promote polyandry [15,16,21,29,72]. While there is some evidence that polyandry confers genetic benefit to females by enabling post-copulatory female choice (see reviews [15,16,21,29,72]), there is little experimental support regarding the potency of SGE causing reproductive incompatibilities to favour polyandry. The one study explicitly examining the potential for CI-inducing Wolbachia to promote polyandry found that, although polyandrous females have higher fitness than monogamous females, in itself this cannot promote polyandry [64]. This is because the benefit to females depends on the frequency with which they encounter incompatible males. In this study, females were equally likely to mate with an incompatible and a compatible male, as is often the case for studies examining genetic incompatibility avoidance, where the paternity achieved by related and non-related males is compared [18]. However, the rate at which incompatible matings occur has crucial implications for the reproductive benefits obtained by polyandrous females. As the frequency of infected females in the population increases, the probability of an uninfluenced female mating with both an infected (incompatible) and an uninfected (compatible) male is reduced. Furthermore, despite the increased reproductive success obtained by polyandrous females, the spread of polyandry is constrained by associated costs and the low frequency of incompatible matings when Wolbachia has reached a stable equilibrium. The probability of mating with an incompatible mate in conjunction with the potential costs associated with polyandry imposes severe restrictions on the conditions in which incompatibilities caused by Wolbachia generate a selective advantage for polyandry. Therefore, although incompatibility avoidance may be a benefit of polyandry, models based on laboratory data do not support the hypothesis that genetic incompatibilities caused by Wolbachia promote the evolution of polyandry [64]. However, the models show that the disadvantage in sperm competition can inhibit or prevent the invasion of Wolbachia [64]. This is an important finding as it shows the sperm competitive disadvantage of Wolbachia-infected males relative to uninfected males can undermine the successful invasion and establishment of CI-inducing Wolbachia in polyandrous populations. If the sperm of uninfected males win in competition against sperm from infected males, polyandry may well act to inhibit the invasion and spread of Wolbachia, and although in principle sperm competition may promote polyandry when Wolbachia is established, the advantage is typically too weak to resist even very minor costs of polyandry.

In contrast, polyandry can rapidly evolve when SGEs are associated with reduced male fertility and does not result in genetic incompatibilities based on the combination of male and female genotypes. This situation is common as many SGEs compromise both male fertility and sperm competitive ability [37,38]. The impact of SGEs can either be direct or indirect. In the fire ant, for example, males carrying the Gp-9^b allele produce few sperm, resulting in reduced capacity to suppress remating in the queens that together with poor sperm competitive ability undermines male reproductive success [71]. In H. bolina butterflies, male-killing Wolbachia result in a female-biased population sex ratio, which causes females to experience sperm limitation owing to lack of males, in turn promoting female multiple mating to obtain more sperm [66]. This exacerbates the issue of sperm depletion for males (and, therefore, also for females), as males become increasingly sperm limited, generating a cycle of increasing female mating demand and male fatigue. In this situation male-killing Wolbachia favours polyandry in response to male (i.e. sperm) limitation. In principle, any sex-ratio distorter has the potential to promote polyandry as a female response to sperm limitation owing to the lack of mating males. This possibility needs to be more widely examined in species harbouring sex-ratio distorters that cause female-biased population sex ratios. There is also additional direct evidence that the presence of a sex-ratio distorter with associated poor sperm competitive ability of male carriers can promote rapid evolution of polyandry. In experimental populations of D. pseudoobscura harbouring SR drive, females rapidly evolve increased remating rate when at risk of sex-ratio distorting males [69]. Moreover in laboratory populations, polyandry is effective at biasing paternity against SR males by promoting sperm competition [28], thereby undermining the transmission advantage of SR and suppressing its frequency [28]. In two species of stalk-eyed flies, females are found to mate more frequently in populations with higher frequency of meiotic drive. In addition, mating rate increased as the frequency of drive increases across species [73]. In at least one species, males carrying the drive allele suffer reduced sperm competitive ability [74], which is likely to be the case also in the other species. In general, polyandry is likely to be favoured whenever SGE-carrying males suffer reduced fertility and sperm competitive ability. This situation is fulfilled for many SGEs [37]. To date, there are few experimental examinations of this possibility, particularly in the wild, although field data suggest this may be the case in mice [70].

5. Impact of selfish genetic elements on reproductive behaviours

Endosymbionts can dramatically alter the behaviour and reproductive morphology of their host [75]. There is plenty of evidence to suggest that the presence of SGEs more generally can have a dramatic effect on female mating patterns that can influence the mating system, including the degree of multiple mating. For example, it can alter the reproductive behaviour of female butterflies in populations harbouring male-killing bacteria, resulting in sex role reversal. In Acraea encedon butterflies, lekking is normally carried out by males in non-female-biased populations, whereas females form leks in populations with high frequency of male killers and female-biased sex ratio. Female lekking is suggested to
increase the likelihood of mating [76], and may also increase the likelihood of multiple mating. Changes in female mating patterns in response to the presence of SGEs will also influence male mating behaviours and strategies. In D. pseudoobscura, for example, non-carrier males that coevolved with females that evolved increased remating rates in the presence of SR males were found to evolve increased sperm transfer at mating. This is likely a response to the higher risk of sperm competition experienced by males in these high polyandry populations compared with populations evolving without the SR allele, where females did not evolve to mate at a higher rate [77]. In addition, males evolving in the high polyandry populations were also better at suppressing female remating, and there was a tight relationship between the level of female multiple mating and males’ ability to suppress female remating across populations, with males from populations where females evolved the highest remating rates being best able to suppress female remating. Despite this, females were able to maintain high remating rates, indicating that it is variation in female remating rate that drives male ejaculate evolution. This provides evidence for strong male–female coevolution driven by the presence of the SR allele, even when present at a low frequency (less than 5%). Similarly, in isopods infected with the presence of the SR allele, even when present at a high frequency (less than 5%), males from populations where females evolved the highest remating rates being best able suppress female remating. Despite this, females were able to maintain high remating rates, indicating that it is variation in female remating rate that drives male ejaculate evolution. This provides evidence for strong male–female coevolution driven by the presence of the SR allele, even when present at a low frequency (less than 5%).

SGEs can also affect morphological traits. In the Allonemobius socius cricket species complex, for example, Wolbachia modify the length of the spermathecal duct of females. There is a causal link between Wolbachia infection status and the length of the spermathecal duct, as curing reduces the length to resemble uninfected female tract morphology [79]. This may have important implications for male fertilization success, as in D. melanogaster longer reproductive tract length is associated with greater female influence over the outcome of sperm competition and therefore exerts selection on sperm length [80]. Inspired by the finding in crickets, a recent model suggests that endosymbiont-induced changes to female reproductive traits can directly impact on the dynamics of sexual conflict by directly affecting the mating rates of females. This, in turn, can maintain the infection within the host population, even in the absence of any reproductive manipulation such as sex-ratio distortion or reproductive incompatibilities [81]. It is unclear to what extent endosymbionts and other SGEs can manipulate morphological traits important for reproduction in other species, or indeed by what mechanism, but they clearly have the potential to directly affect female mating rate and, therefore, polyandry levels by manipulating reproductive traits.

There is also evidence that the presence of SGEs can directly affect male mating strategies. In D. melanogaster and D. simulans harbouring CI-inducing Wolbachia, infected males have significantly higher mating rates than uninfected males [82]. Moreover, the difference in male mating rate covaries with the level of CI. It is more pronounced in D. simulans where wki induces a more severe CI, with more than 95 per cent of crosses between uninfected females and infected males failing to hatch compared with less than 30 per cent in similar crosses in D. melanogaster. There is evidence that the level of CI declines with both male age and male mating frequency [83]. It is therefore possible that the higher level of mating by infected male Drosophila may be a strategy to regain reproductive compatibility with all the females in the population [82]. An alternative interpretation is that the infection is beneficial to males by enabling them to maintain a higher mating rate than uninfected males. Although there are benefits of Wolbachia infection in terms of conferring protection against RNA viruses in both fly species [84,85], this benefit is enjoyed by both sexes and cannot explain why infected males should mate at a higher rate than uninfected males (and endosymbiont protection against viruses is not universal [86]). In addition, infected D. simulans males suffer reduced sperm production [61] and sperm competitive ability [39] relative to uninfected males, further suggesting that Wolbachia infection is not associated with a reproductive advantage to males. This result taken together with the finding that uninfected D. simulans females remate sooner (the only ones at risk of CI), suggests that Wolbachia infection can favour both male and female multiple mating as a strategy to avoid associated reproductive incompatibilities, but that the impact differs depending on the infection status. The net result is an increased level of multiple mating and polyandry in populations harbouring Wolbachia.

6. Consequences of polyandry for the dynamics of selfish genetic elements

The presence of SGEs can promote polyandry, but conversely polyandry can also directly regulate the frequency of SGEs by undermining their transmission advantage. An indication that this may be the case comes from laboratory studies in D. pseudoobscura showing that polyandry is very effective at suppressing the frequency of SR. While the frequency of SR was high in populations evolving under monogamy, just one remating opportunity was sufficient to dramatically suppress the frequency of SR, with no further benefits from additional mating in terms of increased SR suppression [28]. In support for the hypothesis that sperm competition per se is sufficient to effectively reduce the frequency of sex-ratio distorsers, both theory and empirical studies have shown that less than two sires per brood are required to allow invasion and persistence of sex-ratio drive [28,87]. Polyandry may generally play a major role in controlling the abundance of many SGEs in nature, although to date only one study has examined this possibility. The population dynamics of autosomal meiotic driving t alleles in mice have been investigated for decades [88]. Models commonly predict t allele frequencies much higher than those found in natural populations [89]. Recent work using models parameterized with laboratory and field experiments shows that the dynamics of t alleles in a natural population of house mice could best be explained by the transmission disadvantage owing to sperm competition [70].

The potency of polyandry to regulate the dynamics of SGEs can have important implications for population viability. Laboratory studies in D. pseudoobscura suggest that polyandry may directly enhance survival of populations harbouring SR drivers. Populations evolving under monogamy were found to have a significantly higher probability of going extinct [28]. Each extinction event was preceded by the production of very few males, indicating the reason that populations went extinct was due to the lack of males stemming from a high frequency of SR. This finding indicates that monogamy is not a viable option in
populations harbouring sex-ratio distorters, further promoting polyandry. If in nature, monogamous populations harbouring sex-ratio distorters are generally at a higher risk of going extinct, this would further add to an association between polyandry and the presence of SGEs. This possibility may be difficult to quantify in the wild, because documenting extinction events of entire populations is inherently difficult to observe [90]. The recent discovery of a cline in polyandry that underlies the cline in SR in D. pseudoobscura across North America may hint at such a relationship between degree of polyandry and the frequency of SR (T. A. R. Price, G. D. D. Hurst & N. Weddell 2012, unpublished data).

As yet, we have no idea how important variation in female multiple mating is for determining the frequency of SGEs, although models predict polyandry can allow the persistence of SGEs at a stable equilibrium in the population [87]. We need more systematic surveys of populations harbouring SGEs with clear differences in degree of polyandry to allow us to examine if there is a corresponding variation in the frequency of SGEs. One possible example could be the fly Drosophila neotestacea that shows a clinal distribution in the frequency of a meiotic driver across the USA, implicating temperature as the regulating factor [91], although it is not known whether there is corresponding variation also in degree of polyandry that may regulate the frequency of meiotic drive. Another potential example may be the weevil Curculio sikkimensis that also shows a clinal distribution in the frequency of Wolbachia [92], but to date, there is no information regarding possible underlying variation in polyandry along this cline. The difficulty with any association between the frequency of an SGE and degree of polyandry is that it may not be possible to determine the causal relationship: is the frequency of SGEs determined by the level of polyandry, or is the level of polyandry determined by the frequency of the SGEs? Ideally, additional experimental manipulations are needed to disentangle the link between the two, although it is possible, or even likely, that both processes are operating simultaneously, ultimately determining a stable equilibrium of both the frequency of SGEs and the level of polyandry.

7. Summary and future prospects

SGEs clearly have the potential to generate sexual selection, sexual conflict and to promote polyandry. Sexual selection and conflict can occur as a side effect of selection on females’ mating behaviour to avoid mating with SGE-carrying males, which in turn can generate bouts of selection and counter-adaptation by males. This possibility has been demonstrated (e.g. increased female mating frequency in butterflies carrying male-killing bacteria [66], SR drive promoting evolution of polyandry in flies [69]). The main cause of the impact of SGEs on female mating patterns is driven by the association between low fertility and reduced paternity in sperm competition of males carrying SGEs. This favours female multiple mating, which can increase sexual conflict over mating frequency [37,39]. A less well-studied situation is the realization that the SGE itself can have sex-specific effects when present in males and females, potentially causing sexual conflict [93]. A recent example of this is DDT-resistance alleles (DDT-R) in D. melanogaster flies. DDT-R is caused by a SGE—a retrotransposon inserted into the promoter region of a detoxification gene (Cyp6g1) that upregulates this gene in both sexes. Intriguingly, DDT-resistant females are more fecund and have offspring of higher fitness than susceptible females [94]. Despite this fitness advantage to females, DDT-R did not spread before the use of pesticides [95]. This implies a cost to males in order to balance the benefits to females—the hallmark of a sexually antagonistic allele. Remarkably, this seems to be the case. In males, DDT-R makes them less likely to obtain matings when competing for females with susceptible males [93]. This cost (relative fitness reduction to DDT-R males –0.28) almost perfectly balances the fitness benefit to females (+0.25 [94]) when DDT-R is expressed in the same genetic background. However, there is also evidence of extensive epistasis between the DDT-R allele and the genetic background in which it is expressed [93], but it shows that the sexual conflict it can generate clearly has the potential to affect the spread of resistance alleles. It is possible that other SGEs may function as sexually antagonistic alleles with opposite fitness effects when expressed in males and females. If this is the case, it indicates that SGEs may not only generate sexual selection and sexual conflict by favouring polyandry, as this will result in increased risk of sperm competition with potential loss of paternity for males, but may also generate conflict by acting as a sexually antagonistic allele with large sex-specific fitness effects. To date, the extent to which SGEs may have such sex-specific fitness effects is not known, but considering their prevalence throughout the genome and their direct involvement in gene regulation, it is likely to be an overlooked possibility.

So how important are SGEs in general for variation in female mating frequency between species and populations? The simple answer is that we do not know. However, seeing the ubiquitous nature of SGEs in animals and plants, it is likely to be a more common reason for polyandry than previously recognized, although the pervasiveness of polyandry across taxa is likely due to more than one reason. Future studies should focus on exploring the potential that high polyandry populations may have arisen as a female response to the existence of SGEs, although the feasibility of determining this possibility may be hampered by the difficulty of identifying the SGE within the genome of most species. One fruitful approach may be to examine the impact of various endosymbionts known to manipulate host reproduction (there are diagnostic tools (PCR) available for several arthropod endosymbionts—e.g. Wolbachia, Spiroplasma, Cardinium) on the level of polyandry between females and populations with or without endosymbionts. A caveat is that some endosymbionts also have a beneficial role in many insects [84,85].

In nature, genetic suppressors of SGEs frequently evolve, thereby obscuring their presence. It is therefore likely that SGEs are more common in populations than we can appreciate at first glance. Such genetic suppressors are often population specific; hence divergent populations may be at risk when coming into secondary contact as they may be exposed to a SGE without the protection of a corresponding suppressor [90]. The impact can be pronounced by generating severe reproductive incompatibilities and hybrid dysfunction [96] that can contribute to species divergence and potentially even speciation [97]. This situation is likely to occur on secondary contact of previously isolated populations, or when a species colonizes a new habitat. The emergence of sex-ratio bias when populations interbreed is a strong contender for the re-surfacing of an unsuppressed distorter [98]. It is
possible that polyandry under such circumstances is favoured if it provides protection to females by reducing the risk of siring offspring by males with a novel and unexpressed SGE. This may provide an additional explanation for the observation that colonizing females appear to be less choosy [99,100]. Theory shows that polyandry is favoured during colonization events by reducing the risk of inbreeding [101]. A similar argument could be made for dispersing females at risk of encountering males with novel SGEs without the protection of corresponding suppressors.

The wide range of SGEs can provide the key combination of low sperm competitive ability and low fitness that in many circumstances will favour polyandry. As a corollary that SGEs can promote polyandry, theory and laboratory experimental evolution studies indicate that high levels of polyandry can aid genomic cohesion by protecting a population against the invasion of SGEs: for example, natural populations of D. pseudoobscura where females are more polyandrous do indeed suffer a lower burden of deleterious sex-ratio distorters [102]. Hence, in this species, variation in polyandry could potentially determine the frequency of an SGE and therefore the population sex ratio in natural populations. Because SGEs are ubiquitous in living organisms and frequently compromise male fertility and sperm competitive ability, they may provide a generally overlooked explanation for why polyandry is widespread. Conversely, although polyandry can be a potent determinant of the intensity of sexual selection, sexual conflict, parent–offspring conflict, and the evolution of cooperation and sociality, there is growing evidence that polyandry may also protect a population against the invasion and spread of SGEs.

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