

1 **Huxley Review 2020**

2
3 **Selfish genes and sexual selection: the impact of genomic parasites on host reproduction**

4
5
6 Nina Wedell

7 Bioscience, University of Exeter, Penryn Campus, Penryn TR10 9FE, UK

8 N.Wedell@exeter.ac.uk

9
10
11 Running title: Selfish genes and sexual selection

12
13
14
15
16 *Abstract*

17 Selfish genetic elements (SGEs) such as replicating mobile elements, segregation distorters,
18 and maternally inherited endosymbionts, bias their transmission success relative to the rest of
19 the genome to increase in representation in subsequent generations. As such they generate
20 conflict with the rest of the genome. Such intra-genomic conflict is also a hallmark of
21 sexually antagonistic (SA) alleles, which are shared genes between the sexes but that have
22 opposing fitness effects when expressed in males and females. However, while both SGEs
23 and SA alleles are recognised as common and potent sources of genomic conflict, the
24 realisation that SGEs can also generate sexually antagonistic selection and contribute to
25 sexual conflict in addition to generate sexual selection is largely overlooked. Here I show that
26 SGEs frequently generate sex-specific selection and outline how SGEs that are associated
27 with compromised male fertility can shape female mating patterns, play a key role in the
28 dynamics of sex determination systems, and likely be an important source of sexually
29 antagonistic genetic variation. Given the prevalence of SGEs their contribution to sexual
30 conflict is likely to be greatly overlooked.

35 *1. What are Selfish Genetic Elements?*

36

37 Selfish genetic elements (SGEs) are ubiquitous in eukaryotes and prokaryotes (Burt &
38 Trivers, 2006; Lindholm *et al.*, 2016). As the name implies, these are genes that do not play
39 fair but manipulate the rest of the genome in a variety of ways to enjoy a transmission
40 advantage to subsequent generations and therefore increase in frequency. As such they are an
41 important source generating intra-genomic conflict (conflict between different agents within
42 the genome due to biased transmission) in addition to the potential negative impact on gene
43 function of their activity (e.g. increasing/ decreasing gene expression or immobilising genes
44 by translocation/ insertion/ deletions, Table 1). Furthermore, their mode of generating
45 transmission bias can have substantial fitness costs to the host. SGEs frequently target
46 gametogenesis and reproduction to ensure enhanced transmission. There are many different
47 types of SGE that affect the genome in a variety of ways. The different characteristics can be
48 distilled into two types: an over-replication advantage (e.g. mobile genetic elements in
49 genomes) and a transmission distortion advantage (e.g. meiotic drivers in populations), but
50 they all violate the rule of equal inheritance (Table1).

51

52 The most common type of SGE are transposable elements (TEs). TEs increase in frequency
53 by encoding for enzymes that catalyse their copy number within the genome. They are
54 frequent in eukaryotes and prokaryotes and can make up a large part of the genome (e.g.
55 ~45% of the human genome derive from transposable elements (Lander *et al.*, 2001)).
56 Another group of SGE are segregation distorters that include driving chromosomes (meiotic
57 drive), which if associated with the sex chromosomes cause sex ratio distortion (Jaenike,
58 2001). They also include maternally inherited endosymbionts that kill or feminize males as
59 they cannot transmit the endosymbiont, with resources instead diverted to the female function
60 (Werren, 1997). Meiotic drivers are common in insects, mammals and plants (Lindholm *et al.*
61 *et al.*, 2016). Endosymbionts are also ubiquitous (e.g. mitochondria), and bacterial
62 endosymbionts that affect host reproduction by inducing reproductive incompatibility are
63 very common in arthropods (Zeh & Zeh, 1996). There is also a growing recognition that the
64 microbiome of animals shapes many aspects of organismal fitness, but also has the potential
65 to act selfishly, for example by competing over nutrients in the gut at a cost to its host (Bell *et al.*,
66 2019).

67

68 There are several consequences stemming from the intragenomic conflict and direct impact
69 on gene function generated by SGEs. They are a potent force in shaping the structure and
70 function of the genome, can increase the mutation rate, affect the evolution of genes,
71 genomes, cells, gene regulation and gene expression (e.g. Jurica & Stoddard, 1999). In
72 addition, they play a role in the formation of sex chromosomes and sex chromosome
73 turnover, influence effective population size, viability and gene flow and may even aid
74 speciation (Werren, 2011). They can also have dramatic impact on behaviour of individuals,
75 including sexual behaviour (Wedell, 2019). In this review I outline how SGEs can shape
76 sexual selection by affecting mate choice and mating strategies, but also generate sex specific
77 selection, frequently resulting in sexual conflict and sexually antagonistic selection.

78

79

80 *2. How can SGEs affect sexual selection?*

81

82 Seeing that SGEs are ubiquitous and affect most aspects of organismal life it is perhaps not
83 surprising that they also influence sexual selection and sexual conflict. There are several
84 reasons why this is to be expected: individuals should avoid mating with partners carrying

85 genes associated with costs, and many SGEs target sperm production affecting male fertility.
86 Below I outline how these aspects of sexual selection are affected by SGEs.

87

88 *a) SGEs affect mate preferences*

89 We might expect individuals that carry SGEs to be discriminated against during mate choice
90 as they carry genes that result in reduced fertility, reduced offspring production, or offspring
91 of reduced fitness or attractiveness. However, there are remarkably few examples
92 demonstrating that SGEs make their carriers less attractive. There has to be a cue revealing
93 that individuals carry a SGE. Unless there is a change in behaviour, odour or morphology of
94 SGE carriers, it is not clear how individuals could discriminate against them. So, are there
95 cues revealing SGEs? With regards to behavioural changes, unless an individual carrying the
96 SGE suffers a direct cost (i.e. pathogenic effect), it is not always clear whether behavioural
97 changes are to be expected (Wedell, 2019). In insects infected by the endosymbiont
98 *Wolbachia* there is evidence that the bacteria can directly invade brain regions and interfere
99 with the nervous system and affect mate preferences of infected individuals (Strunov *et al.*,
100 2017). In *Drosophila melanogaster* the *wMel* strain is found throughout the insect brain
101 (Albertson *et al.*, 2013). *Wolbachia* has been shown to influence mate preferences in some
102 studies (e.g. Arburthnott *et al.*, 2016), whereas other studies have found no effect (e.g.
103 Champion de Crespigny & Wedell, 2007). It is currently not clear if these different findings
104 indicate that *Wolbachia* has a differential impact depending on the host genotype, or are due
105 to other factors not controlled for. In contrast, in the fly *D. paulistorum* the strain *wPau* is
106 confined to regions in the fly brain that processes olfactory and auditory information (Strunov
107 *et al.*, 2017). *D. paulistorum* is a species complex where different strains of *Wolbachia* cause
108 reproductive incompatibilities between infected and uninfected flies. Remarkably, mate
109 preferences are dependent on the specific strain of *Wolbachia* hosts carry with females
110 preferring to mate with males carrying the same *Wolbachia* strain as their own, ensuring
111 compatible pairings (Miller *et al.*, 2010; Schneider *et al.*, 2019). It is currently not clear what
112 impact *Wolbachia* in the brain has in terms of shaping insect mate preferences. One
113 possibility is that endosymbionts and other SGEs have the potential to modify odour cues
114 used in mate recognition and mate choice by uninfected individuals. For example, *Wolbachia*
115 reduce mate discrimination in *Nasonia jewel* wasps (Chafee, 2011), and in the terrestrial
116 isopod *Armadillium vulgare*, feminizing *Wolbachia* affects mate attraction by altering female
117 cuticular odour cues (Richard, 2017). There is now a growing realisation that endosymbionts,
118 as well as gut microbiota and other bacteria, can directly affect cuticular hydrocarbons, sex
119 pheromone production, and other odour cues used in mate choice (e.g. Engl & Kaltenpoth,
120 2018).

121

122 With non-bacterial SGEs there is more limited evidence of mate preference. For example, in
123 *D. pseudoobscura* harbouring a sex-ratio distorting meiotic driver (SR), females do not
124 discriminate against males despite large fitness cost (Price *et al.*, 2012). By mating with SR-
125 carrying males, females will produce the more common sex (daughters) and may also suffer
126 reduced fertility as SR males transfer smaller ejaculates (Price *et al.*, 2008a, b). In *Teleopsis*
127 *dalmanni* stalk-eyed flies carrying a sex ratio distorter (an X-linked meiotic driver), females
128 prefer to mate with males with long eye-stalks. This signals that they carry a genetic
129 suppressor of sex-ratio drive meaning females will sire both sons and daughters (Cotton *et*
130 *al.*, 2014). On the other hand, in mice carrying an autosomal meiotic driver, the *t*-complex,
131 heterozygous females avoid mating with males carrying the *t*-haplotype. This may be
132 advantageous because homozygous recessives are lethal (Lenington, 1991). Again odour cues
133 are involved, with the *t*-complex being contained in an inversion system that also harbours
134 the MHC alleles used in kin recognition (Lindholm *et al.*, 2013). However, mate choice is not

135 always present and it is suggested that *t*-specific female preferences may not be
136 evolutionarily stable (Sutter & Lindholm, 2016).

137

138 In summary, there is only limited evidence for mate discrimination against carriers of SGEs.
139 For the cases where this has been documented, mate choice appears to be based on cues that
140 are directly linked to the SGE – usually odour cues, although eye-stalk length appears to be a
141 reliable signal of males carrying a genetic suppressor of sex ratio drive in stalk-eyed flies. So
142 why is there such scant evidence of SGEs-based mate choice? One reason may be a lack of
143 genetic linkage between the SGE and the preference allele due to recombination (Nicholls &
144 Butlin, 1998; Lande & Wilkinson, 1999). It is interesting to note, that in the stalk-eyed flies
145 there is evidence of a tight linkage between the preference alleles and sex ratio drive (Johns
146 *et al.*, 2005). A recent theoretical model also shows that preference can only persist in the
147 presence of a cue that reliably indicates a male's distorter genotype (Manser *et al.*, 2017). We
148 may therefore predict that selfish endosymbionts are more likely to have an effect on mate
149 choice than other SGEs, as there is scope for these bacteria to have a direct impact on both
150 odour production and invading the central nervous system of their host where cue processing
151 takes place.

152

153 *b) SGEs affect male fertility and sperm competition*

154 In contrast to the somewhat limited evidence of SGEs shaping mate preferences, there is
155 ample evidence to show that SGE-carriers frequently suffer reduced gamete production
156 (Zanders & Unckless, 2019). Males in particular that carry different types of SGE have
157 reduced sperm production (Price & Wedell, 2008). While female gamete-killers operate by
158 exploiting the asymmetric meiosis in females, where one meiotic product is selected to
159 become the gamete (Chmatal *et al.*, 2014), they are less commonly observed than SGEs that
160 target sperm. This may be because female drive can result in population extinction
161 (Hamilton, 1967), and to a greater impact of gamete reduction on female compared to male
162 fitness. There are two main ways SGEs target male spermatogenesis to increase their
163 transmission success. Segregation distorters do this by eliminating allelic rivals during
164 meiosis by selectively killing sperm that do not carry the distorter. Meiotic drivers achieve
165 their transmission advantage by being the only sperm type remaining in drive-carrying males'
166 ejaculate (Courret *et al.*, 2019). Post-segregation distorters such as maternally inherited
167 endosymbionts achieve their transmission advantage by killing or feminizing males, or by
168 modifying sperm function resulting in zygote death when eggs lacking the endosymbiont are
169 fertilized. This resulting reproductive incompatibility (cytoplasmic incompatibility, CI)
170 means that uninfected females have dramatically reduced offspring production, whereas
171 infected females who are compatible with both infected and non-infected males' sperm
172 produce offspring that carry the endosymbiont. This differential offspring production
173 translates into a large transmission advantage favouring the spread of the endosymbiont
174 through a population (Werren, 1997). However, sperm modification by post-segregation
175 distorters, and sperm immobilisation and killing by segregation distorters, result in reduced
176 sperm production and therefore may result in transfer of less sperm to females at mating
177 compared to non-carrying males. There are exceptions to this rule, for example male *T.*
178 *dalmanni* carrying sex ratio drive (SR) do not suffer reduced sperm production, but instead
179 produce and deliver as many sperm as wild-type males. It is suggested that males have
180 evolved to compensate for sperm loss due to SR by increased sperm production to match wild
181 type male ejaculate production (Meade *et al.*, 2019). Whether this is due to lower overall
182 sperm production and delivery by *T. dalmanni* males per mating compared to other fly
183 species and/or due to unknown trade-offs with other fitness related traits, is currently not
184 clear (Meade *et al.*, 2020).

185
186 The magnitude of the sperm killing/modification of SGE-carrying males can be substantial.
187 The reduction in male fertility ranges from no significant impact on sperm numbers (e.g. *T.*
188 *dalmanni* mentioned above) to a reduction of more than 50% as has been shown in several
189 species carrying sex-ratio drive (Price & Wedell, 2008). In addition, the mechanism whereby
190 the gametes are rendered inviable can have deleterious impacts on the surviving SGE-
191 carrying sperm (Price & Wedell, 2008). For example, in *D. pseudoobscura* SR males only
192 produce X-linked sperm as all the Y-sperm are killed. However, the act of sperm killing
193 appears to have a spill-over effect reducing the vigour of the surviving sperm that carry SR
194 (Price *et al.*, 2008a). It is also possible that female behaviour post mating affects the number
195 of sperm delivered by SGE-carrying males thereby reducing the likelihood of fertilization
196 (i.e. cryptic female choice (Eberhard, 1996)). This requires that the cost of mating is
197 relatively low allowing polyandrous females to discriminate against specific males post-
198 mating. In many animals, females eject sperm following insemination. For example, female
199 feral fowl eject the ejaculate after being inseminated by a subordinate male (Pizzari &
200 Birkhead, 2000), and sperm ejection is common in many other birds, mammals, and insects
201 (e.g. Snook & Hosken, 2004). It is currently not known if females preferentially eject sperm
202 following mating with males carrying SGEs. In *D. simulans*, sperm are preferentially lost
203 from the females' sperm storage following mating to males carrying sex-ratio drive (SR).
204 However, it is not known if the removal of SR males' sperm is due to a specific response by
205 females to sperm carrying the SR driver, or is a response to receiving small overall ejaculates
206 (Angelard *et al.*, 2008). There is little previous evidence that females can detect meiotic
207 drivers in sperm, and it therefore seems likely that *D. simulans* females respond to the
208 significantly smaller ejaculates transferred by SR males (Price *et al.*, 2009). Whether female
209 sperm dumping is a general strategy to guard against ejaculates carrying SGEs is not known,
210 and is predicted to occur only when the cost of mating to females is low.

211
212 Even if females are unable to detect the ejaculate of SGE-carrying males and preferentially
213 eject sperm following insemination, there are additional strategies that they can adopt to
214 reduce the risk of fertilizing their eggs with SGE carrying males' sperm. As SGEs frequently
215 compromise males' sperm production, this often translates into reduced sperm competitive
216 ability (Price & Wedell, 2008). This is because the outcome of sperm competition is often
217 dependent on relative sperm number (Parker, 1970). In addition, the method of sperm
218 killing/modification by SGEs often results in reduced performance in sperm competition over
219 and above the impact of reduced sperm numbers (e.g. Price *et al.*, 2008a). This critically sets
220 up a link between males carrying SGEs and poor sperm competitive ability, which in theory
221 should favour polyandry (female multiple mating) as a strategy to promote sperm
222 competition and reduce the risk of fertilizing their eggs with SGE-carrying males' sperm
223 (Zeh & Zeh, 1996). Again, the cost of polyandry has to be relatively low. In support of this
224 prediction, female *D. pseudoobscura* evolving in the presence of males carrying a sex ratio
225 distorter (SR) rapidly evolved increased mating frequency and rate of remating (Price *et al.*,
226 2008b). Subsequent work has shown that polyandry is a very effective strategy that
227 undermines the transmission advantage of SR (Price *et al.*, 2010). Female mating patterns are
228 influenced by the presence of SGEs that reduce male fertility also in house mice and flies
229 (Lindholm *et al.*, 2016). This indicates the presence of SGEs may in general promote
230 polyandry as a female strategy to reduce the risk of producing offspring sired by SGE-
231 carrying males, and as a consequence also limit the spread of the SGE.

232
233 In summary, there is ample evidence that SGEs have a detrimental impact on the
234 reproductive success of SGE-carrying males by compromising their fertility. Reduced male

235 fertility can affect female mating decisions, often by promoting polyandry and sperm
236 competition as a strategy to reduce the risk of siring their offspring by SGE-carrying males.

237

238

239 3. SGEs affect sex determination

240

241 SGEs have been shown to play a key role in the evolution and turnover of sex chromosomes
242 (Kozielska *et al.*, 2010; Mank *et al.*, 2014). Selfish sex chromosomes cause sex ratio bias
243 (Table 1) that in turn can result in population extinction (Hamilton, 1967; Price *et al.*, 2010),
244 or suppression (Jaenike, 2001). Sex ratio distorters either promote genetic suppression or
245 evolution of new sex determination systems as a way to restore sex ratio to unity. The cost of
246 drive and the strength of selection associated with sex ratio distortion is suggested to affect
247 the outcome, with strong drive favouring a change in the sex determination system, whereas
248 weak drive favours accumulation of suppressors (Lyttle, 1981; Kozielska *et al.*, 2010).
249 Selfish endosymbionts can cause feminization of genetic males, and microbe-induced
250 parthenogenesis regularly occurs in arthropods (Kageyama *et al.*, 2012). It is also suggested
251 that TEs through their influence on the expression of sexual development genes, often with
252 pronounced sex-specific effect, can influence sex determination including sex chromosome
253 evolution (Dechaud *et al.*, 2019). Hence a variety of SGEs have a major influence on the
254 evolutionary dynamics of sex chromosomes.

255

256 *SGEs, sex chromosome evolution and sex chromosome turnover*

257 SGEs that cause sex ratio distortion (Table 1) often target sex determination mechanisms
258 including the sex chromosomes themselves (Ma *et al.*, 2014; Courret *et al.*, 2019). As such
259 sex chromosomes are vulnerable to the invasion of segregation distorters. This may not be
260 surprising seeing that any gene on the X/Z can efficiently drive against the Y/W (and *vice*
261 *versa*) resulting in sex ratio distortion (Hamilton, 1967). This in turn will promote strong
262 selection to restore sex ratio to unity, which can favour the evolution of new sex
263 chromosomes or new ways to determine sex. For example, segregation distorters have
264 promoted the evolution of new mechanisms of sex-determination in rodents (e.g. wood-
265 lemmings, moles and voles), as well as in flies (including the house fly), and scale insects
266 (Beukeboom & Perrin, 2014). A recent model has even suggested that meiotic drive can give
267 rise to sex chromosomes because any new sex determining allele will be favoured when
268 linked to a sex-specific meiotic driver and therefore rapidly spread as a new sex chromosome
269 (Úbeda *et al.*, 2015). In support of this prediction is the recent finding that in a population of
270 the African monarch butterfly *Danaus chrysippus* harbouring male-killing *Spiroplasma*
271 endosymbionts, a neo-W sex chromosome has hitchhiked to high frequency as the male killer
272 has spread through the population. There appears to be a perfect genealogical congruence
273 between the genome of the male-killing *Spiroplasma* and the neo-W sex chromosome
274 (Martin *et al.*, 2020), suggesting that male-killing has favoured the rise of this new sex
275 chromosome. In general sex-chromosome turnover frequently appears to involve autosome-
276 sex chromosome fusion resulting in neo-sex chromosomes in vertebrates (e.g. Kitano &
277 Peichel, 2012), and invertebrates (e.g. Carabel Paladio *et al.*, 2019) and are associated with
278 faster evolution of post-zygotic isolation and diversification (Turelli & Begun, 1997; Lima,
279 2014). In turn neo-sex chromosomes often involve small and repeat-rich chromosomes (e.g.
280 Ahola *et al.*, 2014), suggesting a role for SGEs such as TEs.

281

282 Sex determination and differentiation of arthropods can also be perturbed by endosymbionts
283 and promote evolution of new sex chromosomes. For example, some populations of *A.*
284 *vulgare* pill bugs harbour feminizing *Wolbachia* that turn ZZ males into females (Leclercq *et*

285 *al.*, 2016). As a consequence, the effective population size of the W chromosome is reduced
286 eventually resulting in its elimination (Rigaud, 1997). As a consequence of *Wolbachia*-
287 induced feminization, all individuals are females but ZZ genetic males; those inheriting
288 *Wolbachia* develop as females, whereas uninfected embryos develop as males, meaning there
289 has been a transition from genetic to endosymbiont-determined sex determination. In
290 addition, a new female determining factor that converts genetic males into females has
291 recently been discovered. Females from these lines are thought to be ZZ genetic males
292 converted into females by an unknown feminizing agent termed the “*f* element”. Further
293 work has shown that this genetic element has triggered the evolution of a new W sex
294 chromosome by horizontal transfer of part of the bacterial genome into the pillbug’s nuclear
295 genome (Leclercq *et al.*, 2016). This complicated scenario in *A. vulgare* suggests that
296 *Wolbachia* promoted sex chromosome turnover by first causing the loss of the W sex
297 chromosome, and then by inserting a new sex-determining region into the nuclear genome.
298 This sequence of events suggests that the birth of the new sex chromosome in the pill bug has
299 its origin in the horizontal gene transfer of an initially feminizing endosymbiont (Leclercq *et*
300 *al.*, 2016). Evidence of the wide-spread ongoing tension between SGE-fuelled sex
301 determination and mechanisms to restore sex-ratio to unity, is the frequent occurrence of a
302 variety of aberrations such as gynandromorphs, in addition to sex-specific lethality (e.g. male
303 killing) and conversion of gender (e.g. feminization of genetic males). Such sexual
304 abnormalities can be caused by selfish maternally transmitted endosymbionts such as
305 *Wolbachia*, *Rickettsia*, *Arsenophonus*, *Spiroplasma* and *Cardinium* bacteria, and by
306 microsporidian protists (Kageyama *et al.*, 2012) that interfere with the sex-determining
307 systems (Ma *et al.*, 2014).

308
309 Segregation distorters also have the potential to fuel the turnover of sex chromosomes by
310 invasion and initiating silencing mechanisms to suppress their action (Meiklejohn & Tao,
311 2009). Silencing of sex-linked genes is a common occurrence and involves meiotic sex
312 chromosome inactivation (MSCI), and other inactivation mechanisms such as RNA
313 interference and methylation (Bird, 2019; Vogel *et al.*, 2019). The co-evolution of SGEs and
314 their silencing mechanisms on the sex chromosome can lead to reproductive incompatibilities
315 between populations harbouring different segregation distorters and suppressors and may
316 even contribute to speciation (Meiklejohn & Tao, 2009). Furthermore, in addition to the
317 reduced recombination of sex chromosomes, these silencing mechanisms can promote new
318 sex determination systems that allow SGEs to escape inactivation and sex chromosome
319 degeneration. For example, it is suggested that gene silencing of the Y chromosome in the fly
320 *D. albomicans* may have initiated the process of degeneration (Zhou & Bachtrog, 2012). In
321 addition, new sex-determining mechanisms such as novel sex chromosomes can facilitate a
322 selective sweep of the sex determining region that may also result in hitchhiking of linked
323 genes with large fitness effects (Hall, 2004; Nolte *et al.*, 2013, Miyata *et al.*, 2017). This
324 means there is the potential that SGEs can also increase in spread by being tightly linked to
325 high-fitness alleles under positive selection (Mank *et al.*, 2014).

326
327 In summary, selfish sex chromosomes and maternally inherited endosymbionts that cause sex
328 ratio distortion can favour new ways of determining sex to restore sex ratio to unity. This can
329 involve a variety of mechanisms and we are only now beginning to unravel the complex
330 interaction between SGEs and novel ways to determine sex.

331

332

333 *4. SGEs can generate sexual conflict and sexually antagonistic selection*

334

335 SGEs enjoy a selfish transmission advantage with many showing asymmetrical transmission
336 either through males (e.g. sperm killers), or females (e.g. selfish endosymbionts). While there
337 are some SGEs that are exclusively transmitted in males (e.g. paternal sex ratio in *Nasonia*
338 wasps (Werren, 1991)), many SGEs predominately show a sex-biased transmission in
339 females. For example, mitochondria and other cytoplasmically transmitted agents are
340 (almost) exclusively inherited from mother to offspring (Werren, 1997). Female gametes are
341 usually substantially larger than sperm, in part due to a larger volume of cytoplasm that can
342 harbour selfish endosymbionts that are hitchhiking to the next generation. It is even
343 suggested that one reason that sperm are generally small (over and above the numerical
344 superiority favoured by sperm competition (Parker, 1970)) is because they carry little
345 cytoplasm, which reduces the risk of passing on hitchhiking SGEs to offspring (Randerson &
346 Hurst, 1999). Because of the asymmetrical sexual inheritance of some SGEs, this can
347 translate to differential selection imposed on males and females.

348
349 For SGEs that are equally transmitted through both sexes, the overall cost to the individual
350 carrying the SGEs will shape the transmission success. In some cases, the outcome is a less
351 effective transmission of the SGE if greater transmission is associated with considerable
352 fitness costs to the host. For endosymbionts and females this conflict is less apparent and may
353 even be non-existing as their respective fitnesses are often aligned. For example, in the fly *D.*
354 *simulans*, the Riverside strain of *Wolbachia* has gone from imposing a 15-20% fecundity cost
355 to providing a 10% fecundity benefit to females in less than 20 years of coevolution (Weeks
356 *et al.*, 2007). Moreover, many SGEs while not causing sex ratio distortion, also have sex-
357 specific effects. For example, many TEs show pronounced sex-specific activity (Dechaud *et*
358 *al.*, 2019). The differential expression of SGEs in males and females has the potential to
359 generate sexual conflict through their potentially sexually antagonistic effect. This is because
360 males and females share most of their genome and develop many of the same traits, but each
361 sex frequently has different optimal trait values, creating intra-locus sexual conflict
362 (Bonduriansky & Chenoweth, 2009). This means that SGEs have the potential to fuel such
363 intra-locus sexual conflict by their sex-specific effects (Wedell, 2013; Mank *et al.*, 2014).
364 Below I outline a few examples to illustrate how different SGEs can generate sexual conflict.

365 366 *a) Endosymbionts promote female fitness through feminizing selection*

367 Endosymbionts are almost exclusively maternally inherited and therefore the evolutionary
368 interests of the endosymbiont and female function are often aligned, which frequently
369 translate into feminizing selection to promote female fitness. An extreme example is the
370 situation in the wasp *Asobara tabida*, where female ovary development is entirely dependent
371 on *Wolbachia* infection – if females are cured of *Wolbachia* they become sterile (Dedeine *et*
372 *al.*, 2001). In general, we predict that maternally inherited endosymbionts such as *Wolbachia*
373 in arthropods and mitochondria in animals would enhance female fecundity as this increases
374 their own transmission success and hence the evolutionary interests of females and
375 endosymbionts are frequently aligned. In support of this suggestion, in lab-adapted *D.*
376 *melanogaster* females, *Wolbachia* increases insulin/IGF-like signalling (IIS) resulting in
377 increased fecundity (Tomoatsu *et al.*, 2009). Endosymbiont-enhancing female fitness is also
378 predicted to increase the longer the duration for coevolution, a prediction supported by
379 empirical findings (e.g. Weeks *et al.*, 2007). However, due to the fact that males and females
380 share a genome, genes that are shaped by feminizing selection to maximize female fitness can
381 result in reduced male fitness when expressed in males. For example, in *D. simulans*, the
382 Riverside strain of *Wolbachia* is associated with increased female fecundity (Weeks *et al.*,
383 2007), whereas in males *Wolbachia* reduces sperm production (Snook *et al.*, 2000), and
384 sperm competitive success (Champion de Crespigny & Wedell, 2006). *Wolbachia* therefore

385 generate strong sex-specific fitness differences. Similarly, endosymbionts that cause
386 feminization of genetic males will clearly impose a cost on the male function. For example,
387 feminization of males has the potential to have a detrimental impact on sexually selected
388 traits expressed in males such as odour and visual cues used in mate choice. While there is to
389 date no definitive evidence that feminizers have a detrimental effect on male reproductive
390 success by affecting the expression of sexually selected traits, this is a real possibility. In
391 many feminized systems, some males tend to escape feminization and there are naturally
392 occurring curing agents such as exposure to high temperature and antimicrobial products that
393 remove the endosymbiont resulting in the resurgence of males (Werren, 1997), allowing this
394 prediction to be tested.

395
396 A negative intersexual genetic correlation for fitness is frequently interpreted as evidence for
397 the existence of widespread intra-locus sexual conflict where a high male fitness genotype
398 gives rise to a low fitness female and *vice versa*. However, this negative intersexual
399 correlation may instead be due to endosymbionts causing reproductive incompatibilities
400 between infected males and uninfected females (Duffy *et al.*, 2019). For example, *Wolbachia*
401 (and other endosymbionts) frequently cause reproductive failure in crosses between infected
402 males and uninfected females (CI). *Wolbachia* will therefore reduce the fitness of uninfected
403 females mated to infected males, while uninfected males will not suffer this fitness reduction
404 if they mate with infected females. In fact, uninfected males often have higher fitness than
405 infected males that can have compromised sperm production and sperm competitive ability
406 (e.g. Champion de Crespigny & Wedell, 2006). This asymmetry in fitness between the sexes
407 can generate a strong negative intersexual genetic correlation for fitness, thus mimicking
408 intra-locus sexual conflict. In support of this prediction, experimental findings in *D. simulans*
409 crosses coupled with simulations show that *Wolbachia* can generate signals of intra-locus
410 sexual conflict (Duffy *et al.*, 2019). This possibility is currently largely overlooked as a
411 potentially common source generating sexual conflict in arthropods, but is likely to be of
412 genuine importance seeing the prevalence of CI-inducing endosymbionts.

413
414 The uniparental inheritance of mitochondria, the powerhouse of cells, generates a conflict
415 with the nuclear genome over sex determination and sex ratio, and also creates the
416 opportunity for sexually antagonistic selection as mitochondria can increase maternal fitness
417 but with a potential detrimental side-effect to males – often referred to as ‘mothers curse’
418 (Gemmell *et al.*, 2004; Havird *et al.*, 2019). This occurs because mtDNA cannot evolve for
419 male function as their heritability in males is zero. For example, in dioecious plants there is
420 evidence that mitochondria can induce cytoplasmic sterility and abort pollen production
421 altogether, instead diverting these resources to enhance the female function which will favour
422 mitochondrial transmission (e.g. Loussaert *et al.*, 2017). This generates selection on the
423 nuclear genome to suppress the action of such selfish mitochondria and restore sex ratio to
424 unity (Fujii *et al.*, 2011). Less overt is the situation where mitochondrial genes have a
425 negative effect on male fitness that can include compromised sperm function and fertility
426 without affecting female fitness (Patel *et al.*, 2016; Vaught & Dowling, 2018). Such reduced
427 male reproductive fitness can persist, as low fertility genes are not removed by selection since
428 they are inherited through females where they are never expressed. However, selfish
429 mitochondria can also generate antagonistic selection by favouring the female function at a
430 cost to male fitness. One such example is a mutation in the cytochrome B identified in *D.*
431 *melanogaster* that increase female fitness whilst simultaneously decreasing male fertility
432 (Camus *et al.*, 2018). It is therefore likely that selfish mitochondria also represent a
433 ubiquitous source generating sexually antagonistic selection.

434

435 In summary, because of the asymmetrical inheritance of many SGEs, it is perhaps not
436 surprising they often generate sex-specific fitness impacts. There is extensive evidence that
437 maternally inherited cytoplasmic SGEs can generate sex-specific and sexually antagonistic
438 selection. Future research will reveal the relative importance of endosymbionts such as
439 *Wolbachia* and the mitochondria for generating sex-specific selection, but it is worth noting
440 that the inheritance patterns will promote genetic hitchhiking between these two cytoplasmic
441 agents eventually resulting in linkage. Similarly, the frequently reported nuclear-
442 mitochondrial interactions affecting male fertility may be due to endosymbionts such as
443 *Wolbachia*, *Spiroplasma* and *Cardinium*, rather than a linkage disequilibrium between certain
444 maternal mitochondrial haplotypes and the nuclear genome. Hence, endosymbionts may have
445 an overlooked role to play in generating the reported ‘mitochondrial load’ reducing male
446 fertility reported in several insects. The origin of mitochondria stems from an ancient
447 endosymbiosis, and hence share features with other endosymbionts, albeit subject to billion
448 years of coevolution (Zachar *et al.*, 2018). It is therefore possible there are lessons to be
449 learnt from studying coevolved associations of different ages to explore the importance of the
450 interactions between nuclear and cytoplasmic genes for the pattern of sex-specific and
451 sexually antagonistic effects and the potential for resolution of such SGE-generated sexual
452 conflicts.

453
454 *b) Sex-ratio distorters are sex-specific and can generate conflict*

455 Most sex ratio distorters target males by killing sperm, males, or by feminization of genetic
456 males, and inducing parthenogenesis and therefore by their very nature, generate strong sex-
457 specific effects. There are examples of sex-ratio distorters that bias sex ratio towards males
458 such as *psr* in *Nasonia* wasps that convert diploid eggs into haploid eggs resulting in male
459 offspring. Nevertheless, despite being paternally inherited, this results in complete
460 elimination of the sperm-derived hereditary material (Aldrich *et al.*, 2017). Paternal genome
461 elimination (PGE) also occurs in mealybugs where males are diploid but only transmit the
462 maternally inherited chromosomes with the paternal ones eliminated from their sperm
463 (Normark, 2003). As a consequence, mothers in effect monopolise the parentage of sons at
464 the cost of fathers’ reproductive success generating a conflict between maternal and paternal
465 genomes over gene transmission. PGE is a type of meiotic drive in which the entire maternal
466 chromosomal complement drives, and hence we expect there to be strong selection for
467 suppression of PGE to evolve as is the case in many other meiotic drive systems (Jaenike,
468 2001). Crosses between *Planococcus citri* and *P. ficus* mealybugs have the potential to
469 uncover such an arms-race between maternal and paternal chromosomes. Recent experiments
470 revealed that elimination of paternally derived chromosomes was not completely effective,
471 implying scope for intragenomic conflict, but no evidence for an ongoing arms race was
472 found (de la Filia *et al.*, 2019). As yet, it is not known if the incomplete PGE is associated
473 with any fitness differences between male genotypes, but it would appear that there is almost
474 complete maternal control over inheritance. Less extreme examples of sex ratio distorters
475 exerting sex-specific selection are found in other taxa harbouring sperm and male killers, and
476 feminizers.

477
478 Above, I have provided several examples of SGEs generating sexual selection and sexual
479 conflict and also outlined why we might expect this to be the case, i.e. asymmetrical
480 inheritance and the generation of sex-specific selection. There are several similarities
481 between the conflict generated by segregation distorters such as meiotic drivers and sexually
482 antagonistic alleles (SA, alleles with opposing fitness effects when expressed in males and
483 females) that stem from the reproductive conflict between the two sexes (Trivers & Burt,
484 2006). A recent model has even shown that meiotic drive attracts SA alleles and can increase

485 the opportunity for polymorphism, and similarly that the opportunity for polymorphism at a
486 driving locus also increases when linked to a SA locus (Patten, 2014). The initial model was
487 developed for autosomal drive but the findings also holds true for X-linked drive: the driving
488 sex chromosome becomes enriched for sexually antagonistic effects that benefits the sex in
489 which the drive occurs (Rydzewski *et al.*, 2016). Both processes have the potential to
490 maintain genetic variation within populations, but to date there has been little empirical
491 exploration into the possibility that meiotic drive and sexually antagonistic selection
492 stemming from SA alleles can reinforce each other and contribute to genetic variation of
493 fitness related traits.

494
495 The frequency of drive alleles is predicted to increase when a drive allele is linked to a
496 sexually antagonistic polymorphism. In addition, drivers are predicted to accumulate SA
497 alleles and to favour reduced recombination, analogous to a sex-determining locus (Patten,
498 2014; Rydzewski *et al.*, 2016). Previous models have shown that sexual antagonism should in
499 itself favour reduced recombination (Rice, 1987) hence the combined impact of drive and
500 sexual antagonism should strengthen the speed of evolution of reduced recombination
501 (Patten, 2014; Rydzewski *et al.*, 2016). We therefore predict that there should commonly be
502 haplotypes with driving and sexually antagonistic effects that in theory should promote new
503 sex-determining alleles. This is especially true for meiotic drivers with strong sex-specific
504 fitness effects that may give rise to new sex determining alleles. It is known that sex
505 chromosomes are particularly vulnerable to the invasion of drivers (Jaenike, 2001), but
506 maybe drivers themselves have an unappreciated role to play in the origin of new sex
507 chromosomes (Kozielska *et al.*, 2010; Patten, 2014).

508
509 In summary, sex-linked meiotic drivers and sexual antagonism appear to be intrinsically
510 linked and their joint selective force may exert dramatic impact on sex chromosome
511 evolution and fuel sexual conflict. This is especially likely to be the case when involving X-
512 chromosome drivers (Rydzewski *et al.*, 2016). Drive is more likely to occur on the X
513 chromosome than on the autosome (Jaenike, 2001), and the X chromosome is predicted to
514 accumulate SA alleles (Rice, 1987). Hence, there is a predicted link between sexual
515 antagonism, meiotic drive and sex determination – any one of them will favour the other two
516 in a population (Patten, 2014).

517
518 *c) Other SGEs as sexually antagonistic alleles*

519 Segregation distorters are unequally exposed to selection in males and females, a trait they
520 have in common with SA alleles. While many SGEs such as segregation distorters act
521 through brute force via killing of males and sperm, or through feminization of genetic males
522 resulting in sex-bias, other SGEs are inherited equally through males and females such as
523 TEs and exert a more subtle sex-specific effect. It is worth remembering that the transmission
524 success of TEs is reliant on sex, as sexual reproduction and outcrossing provide TEs with a
525 means of spreading to all individuals in a population (Wright & Finnegan, 2001). This
526 prediction is supported by findings that in yeast asexual reproduction is shown to reduce the
527 load of TEs (Bast *et al.*, 2019). In mammals, it appears that oocytes are more resilient to TE
528 activity than the male germline, and it is suggested that this difference could be due to the
529 ongoing division of sperm cells, in contrast to oocytes, which undergo a long meiotic arrest.
530 Cell division is required for TE transposition, and many more cell divisions occur in the male
531 germline (Dechaud *et al.*, 2019). But there are also sex-differences in expression patterns of
532 TEs that affect reproductive fitness. For example, in *D. melanogaster* insecticide resistance is
533 due to the action of a TE element inserted into the promotor region of a P450 detoxification
534 gene (*Cyp6g1*) that result in upregulation and resistance (ffrench-Constant, 2013).

535 Interestingly there are large sex-differences in the expression pattern of the TE-generated
536 insecticide resistance allele with females showing greater expression and greater resistance to
537 insecticides compared to males (Schmidt *et al.*, 2010). Even without the TE insertion there
538 appear to be sex differences in the expression pattern of *Cyp6g1* (Catalan *et al.*, 2012).
539 Importantly, these sex-differences in expression are associated with sex-specific fitness
540 differences depending of the genetic background. In most genetic backgrounds examined,
541 resistant females enjoy a fecundity advantage compared to their susceptible counterparts
542 implying no cost to resistance (McCart *et al.*, 2005; Rostant *et al.*, 2015; Hawkes *et al.*,
543 2016). In contrast, in males increased expression of *Cyp6g1* conferring resistance can be
544 associated with large fitness costs in terms of reduced mating success and reproductive output
545 (Smith *et al.*, 2011; Hawkes *et al.*, 2016; Rostant *et al.*, 2017). In other words, the resistance
546 allele functions as a SA allele conferring high fitness females and low fitness males and this
547 sex-difference in fitness is sufficient to maintain polymorphism at this locus (Rostant *et al.*,
548 2015). As yet it is not clear if the differential expression of *Cyp6g1* due to the TE activity
549 between the sexes is an outcome to reduce the detrimental SA effects in males, or is an
550 intrinsic effect of TE activity. But it is remarkable what large-scale impact upregulation of
551 one gene has on the behaviour, morphology and fitness of *D. melanogaster* flies indicating
552 substantial pleiotropic effects of this gene (Rostant *et al.*, 2017). Seeing that TEs are present
553 in both bacteria and eukaryotes and can dramatically affect expression of individual genes
554 and gene networks, often in a sex specific manner, it is highly likely there will be many more
555 examples of TEs with sexually antagonistic effects to be discovered.

556

557

558 5. Summary and future prospects

559

560 The selfish nature of SGEs generates conflict with the rest of the genome that will select for
561 suppression and silencing of selfishness. This is especially true for SGEs causing sex ratio
562 distortion, that in turn can promote the evolution of new sex chromosomes. However,
563 changes to sex determination, such as going from male heterogamety to female heterogamety
564 or *vice versa* will alter the opportunity for selection. Heterogamety exposes recessive alleles
565 to selection and therefore generates differential selection on sex-linked genes expressed in
566 males and females (Rice, 1984). In principle, any SGE that is already present on a sex
567 chromosome (or on a former autosome now involved in sex determination) will experience a
568 shift in the strength of sex-specific selection. And as mentioned, segregation distorters such
569 as sex-linked meiotic drivers are themselves magnets for SA alleles and hence are expected
570 to accumulate on the driving sex chromosome (Rydzewski *et al.*, 2016). Many SGEs
571 associated with sex ratio bias may therefore have dramatically different fitness effects when
572 expressed in males or females following a shift in sex determination, depending on the
573 population sex ratio and the degree of sex bias. For example, a genome that has experienced
574 extensive periods of feminizing selection (e.g. by feminizing, male killing, or
575 parthenogenesis-inducing bacteria) may have accumulated female-benefit alleles that lower
576 male fitness when expressed in “rescued” males after the evolution of suppressors of sex-
577 ratio distortion. We may predict that over time the cost of expressing such newly exposed SA
578 alleles in the “rescued” sex should be ameliorated (Bonduriansky & Chenoweth, 2009). The
579 resurgence of SA alleles may therefore be more prominent in populations experiencing a
580 recent spread of a segregation-distorting suppressor allele or a shift in sex determination. In
581 general, the rapid turn-over of sex chromosomes generated by sex ratio distorters will alter
582 the exposure of sex-linked SA alleles to selection and contribute to sexual conflict. Seeing
583 that sex chromosomes are magnets for SGEs and SA alleles, and in turn SGEs promote sex

584 chromosome turnover, there is a direct link between the recurrent intragenomic conflict
585 caused by SGEs and the resurgence and exposure of SA alleles on sex chromosomes.

586
587 SGEs may also represent an overlooked source generating balancing selection. Theory shows
588 that because of the predicted tight linkage that is expected to accumulate between segregation
589 distorters and SA alleles, they will contribute to increased polymorphism at driving and SA
590 loci and thus maintain overall genetic variation (Patten, 2014). However, also non-driving
591 SGEs have the potential to maintain genetic variation in sexually selected traits by generating
592 strong opposing selection. For example, feminizing endosymbionts have the potential to
593 expose male genomes to extensive feminizing selection that could compromise trait
594 expression when males eventually escape feminization through naturally occurring curing
595 events. As yet there is no definitive verification of this suggestion although preliminary
596 findings indicate that male ultra-violet wing colouration – a sexually selected trait in male
597 *Eurema hecabe* butterflies - is eroded when exposed to feminizing selection caused by a
598 maternally-inherited female-biasing agent (Wedell & Kemp, *unpubl.*). Future work will
599 reveal to what extent this reduction in male trait value is directly due to feminizing selection
600 imposed by the endosymbiont, and therefore raises the possibility it may balance the
601 increased trait value favoured by female choice (Kemp, 2008).

602
603 In this review I have outlined several ways in which SGEs can directly shape sexual selection
604 and sexual conflict by promoting sex chromosome evolution (e.g. sex-ratio distorters),
605 affecting gene expression of sex-linked genes with SA effects (e.g. TEs), generating strong
606 sex-specific selection (e.g. maternally transmitted endosymbionts and mitochondria) and
607 acting as a magnet for SA alleles (e.g. segregation distorters). It is likely that there are many
608 more undetected cases of SGEs with the potential to generate sexual selection and sexual
609 conflict, but that have largely gone undetected (Lindholm *et al.*, 2016). Genetic conflict that
610 involves antagonistic coevolution of SGEs and suppressors are often only uncovered in
611 interpopulation crosses. Seeing the prevalence of SGEs in nature, this source of sexual
612 conflict is likely to be greatly overlooked.

613
614
615 *Acknowledgments*

616 I thank Professor Nigel Bennett and the Editorial Board for the honour and opportunity to
617 write this review. I also thank Professor David Hosken for his encouragement and sharing his
618 insights, and Professors Hosken and Pizzari and three anonymous referees for the very
619 helpful comments on the MS.

620
621

622 **References**

623

624 Albertson, R., Tan, V., Leads, R.R. *et al.* (2013). Mapping *Wolbachia* distributions in the
625 adult *Drosophila* brain. *Cell Microbiol.* **15**, 1527–1544.

626

627 Ahola, V., Lehtonen, R., Somervuo, P., Salmela, L., Koskinen, P., Rastas, P., Välimäki, N.,
628 Paulin, L., Kvist, J., Wahlberg, N., Tanskanen, J., Hornett, E.A., Ferguson, L.C., Luo, S.,
629 Cao, Z., de Jong, M.A., Duploy, A., Smolander, O-P., Vogel, H., McCoy, R.C., Qian, K.,
630 Chong, W.S., Zhang, Q., Ahmad, F., Haukka, J.K., Joshi, A., Salojärvi, J., Wheat, C.W.,
631 Grosse-Wilde, E., Hughes, D., Katainen, R., Pitkänen, E., Ylinen, J., Waterhouse, R.M.,
632 Turunen, M., Vähärautio, A., Ojanen, S.P., Schulman, A.H., Taipale, M., Lawson, D.,
633 Ukkonen, E., Mäkinen, V., Goldsmith, M.R., Holm, L., Auvinen, P., Frilander, M.J. &
634 Hanski, I. (2014). The Glanville fritillary genome retains an ancient karyotype and reveals
635 selective chromosomal fusions in Lepidoptera. *Nature Comm.* **5**, 4737.

636

637 Aldrich, J.C., Leibholz, A., Cheema, M.S., Ausio, J. & Ferree, P.M. (2017). A ‘selfish’ B
638 chromosome induces genome elimination by disrupting the histone code in the jewel wasp
639 *Nasonia vitripennis*. *Sci. Rep.* **13**, 42551. doi: 10.1038/srep42551.

640

641 Angelard, C., Montchamp-Moreau, C. & Joly, D. (2008). Female driven mechanisms,
642 ejaculate size and quality contribute to the lower fertility of *sex-ratio* distorter males in
643 *Drosophila simulans*. *BMC Evol. Biol.* **8**, 326. doi: 10.1186/1471-2148-8-326.

644

645 Arburthnott, D., Levin, T.C., & Promislow, D.E. (2016). The impacts of *Wolbachia* and the
646 microbiome on mate choice in *Drosophila melanogaster*. *J. Evol. Biol.* **29**, 461–468.

647

648 Bast, J., Jaron, K.S., Schiseil, D., Roze, D. & Schwander, T. (2019). Asexual reproduction
649 reduces transposable element load in experimental yeast populations. *eLife* **8**, e48548. doi:
650 10.7554/eLife.48548.

651

652 Beeman, R., Friesen, K. & Denell, R. (1992). Maternal-effect selfish genes in flour beetles.
653 *Science* **256**, 89–92.

654

655 Bell, A., Brunt, J., Crost, E. *et al.* (2019). Elucidation of a sialic acid metabolism pathway in
656 mucus-foraging *Ruminococcus gnavus* unravels mechanisms of bacterial adaptation to the
657 gut. *Nature Microbiol.* **4**, 2393–2404.

658

659 Beukeboom, L.W. & Perrin, N. (2014). *The Evolution of Sex Determination*. Oxford
660 University Press.

661

662 Bird, A. (2020). The selfishness of law-abiding genes. *Trends Genet.* **36**, 1 doi.org/10.1016/
663 j.tig.2019.10.002

664

665 Burt, A. & Trivers, R. (2006). *Genes in Conflict*. Belknap Press of Harvard University Press,
666 Cambridge, MA.

667

668 Bonduriansky, R. & Chenoweth, S.F. (2009). Intralocus sexual conflict. *Trends Ecol. Evol.*
669 **24**, 280–288.

670

671 Camus, M.F. & Dowling, D.K. (2018). Mitochondrial genetic effects on reproductive
672 success: signatures of positive intrasexual, but negative intersexual pleiotropy. *Proc. R. Soc.*
673 *Lond. B* **285**, 20180187.

674
675 Chafee, M.E. (2011). Decoupling of host–phage coadaptations following transfer between
676 insect species. *Genetics* **187**, 203–215.

677
678 Champion de Crespigny, F.E. & Wedell, N. (2006). *Wolbachia* infection reduces sperm
679 competitive ability in an insect. *Proc. R. Soc. Lond. B* **273**, 1455-1458.

680
681 Champion de Crespigny, F.E. & Wedell, N. (2007). Mate preferences in *Wolbachia* infected
682 *Drosophila*. *Behav. Ecol. Sociobiol.* **61**, 1229-1235.

683
684 Carabel Paladio, L.Z., Provaznikova, I., Befrger, M., Bass, C., Aratchige, N.S., Lopez, S.N.,
685 Marec, F. & Nguyen, P. (2019). Sex chromosome turnover in moths of the diverse
686 superfamily *Gelechioidea*. *Genome Biol. Evol.* **11**, 1307-1319.

687
688 Catalan, A., Hutter, S. & Parch, J. (2012). Population and sex differences in *Drosophila*
689 *melanogaster* brain gene expression. *BMC Genomics* **13**, 654.

690
691 Chmatal, L., Gabriel, S.I., Mitsainas, G.P., Martinez-Vargas, J., Ventura, J., Searle, J.B.,
692 Schultz, R.M., & Lampson, M.A. (2014). Centromere strength provides the cell biological
693 basis for meiotic drive and karyotype evolution in mice. *Curr. Biol.* **24**, 2295–2300.

694
695 Cotton, A.J., Foldvari, M., Cotton, S. & Pomiankowski, A. (2014). Male eye span size is
696 associated with meiotic drive in wild stalk-eyed flies (*Teleopsis dalmanni*). *Heredity* **112**,
697 363-369.

698
699 Courret, C., Chang, C.-H., Wei, K.H.-C., Montchamp-Moreau, C. & Larracuent, A.M.
700 (2019). Meiotic drive mechanisms: lessons from *Drosophila*. *Proc. R. Soc. Lond. B* **286**,
701 20191430.

702
703 Dechaud, C., Volff, J.-N., Schartl, M. & Naville, M. (2019). Sex and the TEs: transposable
704 elements in sexual development and function in animals. *Mobile DNA* **10**, 42
705 doi.org/10.1186/s13100-019-0185-0.

706
707 Dedeine, F., Vavre, F., Fleury, F., Loppin, B., Hochberg, M.E. & Boulétreau, M. (2001).
708 Removing symbiotic *Wolbachia* bacteria specifically inhibits oogenesis in a parasitic wasp.
709 *Proc. Natl. Acad. Sci.* **98**, 6247–6252.

710
711 De la Folia, A.G., Fenn-Moltu, G. & Ross, L. (2019). No evidence for an intragenomic arms
712 race under paternal genome elimination in *Planococcus* mealybugs. *J. Evol. Biol.* **32**, 491-
713 504.

714
715 Duffy, E., Archer, C.R., Sharma, M.D., Prus, M., Joag, R., Radwan, J., Wedell, N. & Hosken,
716 D. 2019. *Wolbachia* infection can bias estimates of intralocus sexual conflict. *Ecol. Evol.* **9**,
717 328-338.

718
719 Dunn, A.M., Terry, R.S. & Smith, J.E. (2001). Transovarial transmission in the
720 *Microsporidia*. *Adv. Parasitol.* **48**, 57-100.

721
722 Eberhard, W.G. (1996). *Female Control: Sexual Selection by Cryptic Female Choice*.
723 Princeton University Press.
724
725 Engels, W.R. (1997). Invasions of P elements. *Genetics* **145**, 11–5.
726
727 Engl, T. & Kaltenpoth, M. (2018). Influence of microbial symbionts on insect pheromones.
728 *Nat. Prod. Rep.* **35**, 386–397
729
730 ffrench-Constant, R.H. (2013). The molecular genetics of insecticide resistance. *Genetics*
731 **194**, 807–815.
732
733 Fujii, S., Bond, C.S., & Small, I.D. (2011). Selection patterns on restorer-like genes reveal a
734 conflict between nuclear and mitochondrial genomes throughout angiosperm evolution. *Proc.*
735 *Natl. Acad. Sci.* **108**, 1723–1728.
736
737 Gemmell, N.J., Metcalf, V.J., & Allendorf, F.W. (2004). Mother’s curse: the effect of
738 mtDNA on individual fitness and population viability. *Trends Ecol. Evol.* **19**, 238–244.
739
740 Hall, D.W. (2004). Meiotic drive and sex chromosome cycling. *Evolution* **58**, 925–931.
741
742 Hamilton, W.D. (1967). Extraordinary sex ratios. *Science* **156**, 477–488.
743
744 Hammerberg, C. & Klein, J.A.N. (1975). Evidence for postmeiotic effect of *t* factors causing
745 segregation distortion in mouse. *Nature* **253**, 137–138.
746
747 Havecker, E.R., Gao, X. & Voytas, D.F. (2004). The diversity of LTR retrotransposons.
748 *Genome Biol.* **5**, 225.
749
750 Havird, J. C., Forsythe, E. S., Williams, A. M., Werren, J. H., Dowling, D. K. & Sloan, D. B.
751 (2019). Selfish mitonuclear conflict. *Curr. Biol.* **29**, R496-R511.
752
753 Hawkes, M.F., Gamble, C.E., Turner, E.C.R., Carey, M.R., Wedell, N. & Hosken, D.J.
754 (2016). Intralocus sexual conflict and insecticide resistance. *Proc R Soc Lond B* **283**,
755 20161429
756
757 Jaenike, J. (2001). Sex chromosome meiotic drive. *Annu. Rev. Ecol. Syst.* **32**, 25–49.
758
759 Johns, P.M., Wolfenbarger, L.L. & Wilkinson, G.S. (2005). Genetic linkage between a
760 sexually selected trait and X chromosome meiotic drive. *Proc. R. Soc. Lond. B* **272**, 2097–
761 2103.
762
763 Jurica, M.S. & Stoddard, B.L. (1999). Homing endonucleases: structure, function and
764 evolution. *Cell. Mol. Life Sci.* **55**, 1304–1326.
765
766 Kageyama, D., Narita, S. & Watanabe, M. (2012). Insect sex determination manipulated by
767 their endosymbionts: incidences, mechanisms and implications. *Insects* **3**, 161-199.
768
769 Keller, L. & Ross, K.G. (1998). Selfish genes: a green beard in the red fire ant. *Nature* **394**,
770 573-575.

771
772 Kemp, D.J. (2008). Female mating biased for bright ultraviolet iridescence in the butterfly
773 *Eurema hecabe* (Pieridae). *Behav. Ecol.* **19**, 1-8.
774
775 Kitano, J. & Peichel, C.L. (2012). Turnover of sex chromosomes and speciation in fishes.
776 *Env. Biol. Fishes* **94**, 549-558.
777
778 Koonin, E.V. & Dolja, V.V. (2014). Virus world as an evolutionary network of viruses and
779 capsidless selfish elements. *Microbiol. Mol. Biol. Rev.* **78**, 278-303.
780
781 Kozielska, M., Weissing, F.J., Beukeboom, L.W. & Pen, I. (2010). Segregation distortion and
782 the evolution of sex-determining mechanisms. *Heredity* **104**, 100–112.
783
784 Lande, R. & Wilkinson, G. S. (1999). Models of sex-ratio meiotic drive and sexual selection
785 in stalk-eyed flies. *Genet. Res.* **74**, 245–253.
786
787 Lander, E.S., Linton, L.M., Birren, B., Nusbaum, C., Zody, M.C., Baldwin, J., Devon, K.,
788 Dewar, K., Doyle, M., FitzHugh, W., Funke, R., Gage, D., Harris, K., Heaford, A., Howland,
789 J., Kann, L., Lehoczy, J., LeVine, R., McEwan, P., McKernan, K., Meldrim, J., Mesirov,
790 J.P., Miranda, C., Morris, W., Naylor, J., Raymond, C., Rosetti, M., Santos, R., Sheridan, A.,
791 Sougnez, C., Stange-Thomann, Y., Stojanovic, N., Subramanian, A., Wyman, D., Rogers, J.,
792 Sulston, J., Ainscough, R., Beck, S., Bentley, D., Burton, J., Clee, C., Carter, N., Coulson, A.,
793 Deadman, R., Deloukas, P., Dunham, A., Dunham, I., Durbin, R., French, L., Grafham, D.,
794 Gregory, S., Hubbard, T., Humphray, S., Hunt, A., Jones, M., Lloyd, C., McMurray, A.,
795 Matthews, L., Mercer, S., Milne, S., Mullikin, J.C., Mungall, A., Plumb, R., Ross, M.,
796 Shownkeen, R., Sims, S., Waterston, R.H., Wilson, R.K., Hillier, L.W., McPherson, J.D.,
797 Marra, M.A., Mardis, E.R., Fulton, L.A., Chinwalla, A.T., Pepin, K.H., Gish, W.R., Chisoe,
798 S.L., Wendl, M.C., Delehaunty, K.D., Miner, T.L, Delehaunty, A., Kramer, J.B., Cook, L.L.,
799 Fulton, R.S., Johnson, D.L., Minx, P.J., Clifton, S.W., Hawkins, T., Branscomb, E., Predki,
800 P., Richardson, P., Wenning, S., Slezak, T., Doggett, N., Cheng, J.F., Olsen, A., Lucas, S.,
801 Elkin, C., Uberbacher, E., Frazier, M., Gibbs, R.A., Muzny, D.M., Scherer, S.E., Bouck, J.B.,
802 Sodergren, E.J., Worley, K.C., Rives, C.M., Gorrell, J.H., Metzker, M.L., Naylor, S.L.,
803 Kucherlapati, R.S., Nelson, D.L., Weinstock, G.M., Sakaki, Y., Fujiyama, A., Hattori, M.,
804 Yada, T., Toyoda, A., Itoh, T., Kawagoe, C., Watanabe, H., Totoki, Y., Taylor, T.,
805 Weissenbach, J., Heilig, R., Saurin, W., Artiguenave, F., Brottier, P., Bruls, T., Pelletier, E.,
806 Robert, C., Wincker, P., Smith, D.R., Doucette-Stamm, L., Rubenfield, M., Weinstock, K.,
807 Lee, H.M., Dubois, J., Rosenthal, A., Platzer, M., Nyakatura, G., Taudien, S., Rump, A.,
808 Yang, H., Yu, J., Wang, J., Huang, G., Gu, J., Hood, L., Rowen, L., Madan, A., Qin, S.,
809 Davis, R.W., Federspiel, N.A., Abola, A.P., Proctor, M.J., Myers, R.M., Schmutz, J.,
810 Dickson, M., Grimwood, J., Cox, D.R., Olson, M.V., Kaul, R., Raymond, C., Shimizu, N.,
811 Kawasaki, K., Minoshima, S., Evans, G.A., Athanasiou, M., Schultz, R., Roe, B.A., Chen, F.,
812 Pan, H., Ramser, J., Lehrach, H., Reinhardt, R., McCombie, W.R., de la Bastide, M., Dedhia,
813 N., Blöcker, H., Hornischer, K., Nordsiek, G., Agarwala, R., Aravind, L., Bailey, J.A.,
814 Bateman, A., Batzoglou, S., Birney, E., Bork, P., Brown, D.G., Burge, C.B., Cerutti, L.,
815 Chen, H.C., Church, D., Clamp, M., Copley, R.R., Doerks, T., Eddy, S.R., Eichler, E.E.,
816 Furey, T.S., Galagan, J., Gilbert, J.G., Harmon, C., Hayashizaki, Y., Haussler, D.,
817 Hermjakob, H., Hokamp, K., Jang, W., Johnson, L.S., Jones, T.A., Kasif, S., Kasprzyk, A.,
818 Kennedy, S., Kent, W.J., Kitts, P., Koonin, E.V., Korf, I., Kulp, D., Lancet, D., Lowe, T.M.,
819 McLysaght, A., Mikkelsen, T., Moran, J.V., Mulder, N., Pollara, V.J., Ponting, C.P., Schuler,
820 G., Schultz, J., Slater, G., Smit, A.F., Stupka, E., Szustakowki, J., Thierry-Mieg, D., Thierry-

- 821 Mieg, J., Wagner, L., Wallis, J., Wheeler, R., Williams, A., Wolf, Y.I., Wolfe, K.H., Yang,
822 S.P., Yeh, R.F., Collins, F., Guyer, M.S., Peterson, J., Felsenfeld, A., Wetterstrand, K.A.,
823 Patrinos, A., Morgan, M.J., de Jong, P., Catanese, J.J., Osoegawa, K., Shizuya, H., Choi, S.,
824 Chen, Y.J., Szustakowki, J. & International Human Genome Sequencing Consortium. (2001).
825 Initial sequencing and analysis of the human genome. *Nature* **409**, 860-921.
- 826 Leclercq, S., Thézé, J., Chebbi, M.A., Giraud, I., Moumen, B., Ernenwein, L., Grève, P.,
827 Gilbert, C. & Cordaux, R. (2016). Birth of a W sex chromosome by horizontal transfer of
828 *Wolbachia* bacterial symbiont genome. *Proc. Natl. Acad. Sci.* **113**, 15036-15041.
- 829 Lenington, S. (1991). The *t*-complex: a story of genes, behavior, and populations. *Adv. Study*
830 *Behav.* **20**, 51–86.
- 831 Lima, T.G. (2014). Higher level of sex chromosome heteromorphism are associated with
832 markedly stronger reproductive isolation. *Nature Comm.* **5**, 4743.
- 833 Lindholm, A.K., Musolf, K., Weidt, A. & König, B. (2013). Mate choice for genetic
834 compatibility in the house mouse. *Ecol. Evol.* **3**, 1231–1247.
- 835
836 Lindholm A.K. Dyer K.A., Firman R.C., Fishman L., Forstmeier W., Holman L.,
837 Johannesson H., Knief U., Kokko H., Larracuenta A.M., Manser A., Montchamp-Moreau C.,
838 Petrosyan V.G., Pomiankowski A., Presgraves D.C., Safronova L.D., Sutter A., Unckless
839 R.L., Verspoor R.L., Wedell N., Wilkinson G.S., and Price T.A.R. (2016). The ecology and
840 evolutionary dynamics of meiotic drive. *Trends Ecol. Evol.* **31**, 315-326.
- 841
842 Loussaert, D., DeBruin, J., San Martin, J.P., Schussler, J., Pape, R., Clapp, J., Mongar, N.,
843 Fox, T., Albertsen, M., Trimnell, M., Collinson, S & Shen, B. (2017). Genetic male sterility
844 (Ms44) increases maize grain yield. *Crop. Sci.* **57**, 2718–2728.
- 845
846 Lyttle, T.W. (1981). Experimental population genetics of meiotic drive systems III.
847 Neutralization of sex ratio distortion in *Drosophila* through sex chromosome aneuploidy.
848 *Genetics* **98**, 317-334.
- 849
850 Ma, W.-J., Vavre, F. & Beukeboom, L.W. (2014). Endosymbiotic manipulation of arthropod
851 sex determination: diversity and molecular mechanisms. *Sex. Dev.* **8**, 59–73.
- 852
853 Mank, J., Hosken, D.J. & Wedell, N. (2014). Conflict on the sex chromosomes: cause, effect,
854 and complexity. In: *Sexual Conflict*. 341-354. Rice, W.R. & Gavrillets, S. (Eds.) Cold Spring
855 Harbor Press.
- 856
857 Manser, A., Lindholm, A.K. & Weissing, F.J. (2017). The evolution of costly mate choice
858 against segregation distorters. *Evolution* **71**, 2817-2828.
- 859
860 Martin, S.M., Singh, K.S., Gordon, I.J., Omufwoko, K.S., Collins, S., Warren, I.A., Munby,
861 H., Brattstrom, O., Traut, W., Martins, D.J., Smith, D.A.S., Jiggins, C.D., Bass, C. & ffrench-
862 Constant, R.H. (2020). Whole-chromosome hitchhiking driven by a male-killing
863 endosymbiont. *PLoS Biol.* **18**, e3000610.
- 864
865 McCart, C., Buckling, A. & ffrench-Constant, R.H. (2005). DDT resistance in flies carries no
866 cost. *Curr. Biol.* **15**, R587-589.

867
868 Meade, L.C., Dinneen, D., Kad, R., Lynch, D.M., Fowler, K. & Pomiankowski, A. (2019).
869 Ejaculate sperm number compensation in stalked-eyed flies carrying a selfish meiotic drive
870 element. *Heredity* **122**, 916–926.
871
872 Meade, L.C., Finnegan, S.R., D.M., Kad, R., Fowler, K. & Pomiankowski, A. 2020.
873 Maintenance of fertility in the face of meiotic drive. *Am. Nat.* doi.org/10.1086/707372.
874
875 Meiklejohn, C.D. & Tao, Y. (2009). Genetic conflict and sex chromosome evolution. *Trends*
876 *Ecol. Evol.* **25**, 215–223.
877
878 Miller, W.J., Ehrman, L. & Schneider, D. 2010. Infectious speciation revisited: impact of
879 symbiont-depletion on female fitness and mating behavior of *Drosophila paulistorum*. *PLoS*
880 *Path.* **6**, e1001214.
881
882 Miyata, M., Konagaya, T., Yukuhiro, K., Nomura, M. & Kageyama, D. (2017). *Wolbachia*-
883 induced meiotic drive and feminization is associated with an independent occurrence of
884 selective mitochondrial sweep in a butterfly. *Biol. Lett.* **13**, 20170153.
885
886 Nichols, R.A. & Butlin, R.K. (1998). Does runaway sexual selection work in finite
887 populations. *J. Evol. Biol.* **2**, 299–313.
888
889 Nolte, V., Pandey, R.M., Kofler, R. & Schlotterer, C. (2013). Genome-wide patterns of
890 natural variation reveal strong selective sweeps and ongoing genomic conflict in *Drosophila*
891 *mauritiana*. *Genome Res.* **23**, 99–110.
892
893 Normark, B. B. (2003). The evolution of alternative genetic systems in insects. *Ann. Rev.*
894 *Entomol.* **48**, 397–423.
895
896 Patel, M.R., Miriyala, G.K., Littleton, A.J., Yang, H.K., Trinh, K., Young, J.M., Kennedy,
897 S.R., Yamashita, Y.M., Pallanck, L.J., & Malik, H.S. (2016). A mitochondrial DNA
898 hypomorph of cytochrome oxidase specifically impairs male fertility in *Drosophila*
899 *melanogaster*. *eLife* **5**, e16923.
900
901 Parker, G.A. (1970). Sperm competition and its evolutionary consequences in insects. *Biol.*
902 *Rev.* **45**, 525-567.
903
904 Patten, M.M. (2014). Meiotic drive influences the outcome of sexually antagonistic selection
905 at a linked locus. *J. Evol. Biol.* **27**, 2360–2370.
906
907 Pizzari, T. & Birkhead, T.R. (2000). Female fowl eject sperm of subdominant males. *Nature*
908 **405**, 787-789.
909
910 Price, T.A.R. & Wedell, N. (2008). Selfish genetic elements and sexual selection: their
911 impact on male fertility. *Genetica* **132**, 295–307.
912
913 Price, T.A.R., Bretman, A.J., Avent, T., Snook, R.R., Hurst, G.D.D. & Wedell, N. (2008a).
914 Sex ratio distorter reduces sperm competitive ability in an insect. *Evolution* **62**, 1644-1652.
915

916 Price, T.A.R., Hodgson, D.J., Lewis, Z., Hurst, G.D.D. & Wedell, N. (2008b). Selfish genetic
917 elements promote polyandry in a fly. *Science* **322**, 1241-1243.

918

919 Price, T.A.R., Lewis, Z. & Wedell, N. (2009). Sperm dumping as a defense against meiotic
920 drive. *J. Biol.* **8**, 6 doi:10.1186/jbiol1109.

921

922 Price, T.A.R., Hurst, G.D.D. & Wedell, N. (2010). Polyandry prevents extinction. *Curr. Biol.*
923 **20**, 471–475.

924

925 Price, T.A.R., Lewis, Z., Smith, D.T., Hurst, G.D.D. & Wedell, N. (2012). No evidence of
926 mate discrimination against males carrying a sex ratio distorter in *Drosophila pseudoobscura*.
927 *Behav. Ecol. Sociobiol.* **66**, 561-568.

928

929 Randerson, J.P. & Hurst, L.D. (1999). Small sperm, uniparental inheritance and selfish
930 cytoplasmic elements: a comparison of two models. *J. Evol. Biol.* **12**, 1110-1124.

931

932 Rice, W.R. (1984). Sex chromosomes and the evolution of sexual dimorphism. *Evolution* **38**,
933 735–742.

934

935 Rice, W.R. (1987). The accumulation of sexually antagonistic genes as a selective agent
936 promoting the evolution of reduced recombination between primitive sex chromosomes.
937 *Evolution* **41**, 911–914.

938

939 Richard, F.-J. (2017). Symbiotic bacteria influence the odor and mating preference of their
940 host. *Front. Ecol. Evol.* **5**, 143.

941

942 Rigaud, T. (1997) Inherited microorganisms and sex determination of arthropod hosts. In:
943 *Influential Passengers: Inherited Microorganisms and Arthropod Reproduction*: 81-101.
944 O'Neill, S.L., Hoffmann, A.A. & Werren, J.H. (Eds). New York: Oxford Univ. Press

945

946 Rostant, W.G., Kay, C., Wedell, N. & Hosken, D.J. (2015). Sexual conflict maintains
947 variation at an insecticide resistance locus. *BMC Biol.* **13**, 34.

948

949 Rostant, W.G., Bowyer, J., Coupland, J., Facey, J., Hosken, D.J. & Wedell, N. (2017).
950 Pleiotropic fitness effects of DDT resistance of male size and behavior. *Behav. Genet.* **47**,
951 449-458.

952

953 Rydzewski, E.T., Carioscia, S.A., Lievano, G., Lynch, V.D. & Patten, M.M. (2016). Sexual
954 antagonism and meiotic drive cause stable linkage disequilibrium and favour reduced
955 recombination on the X chromosome. *J. Evol. Biol.* **29**, 1247–1256.

956

957 Schmidt, J.M., Good, R.T., Appleton, B., Sherrard, J., Raymant, G.C., Bogwitz, M.R.,
958 Martin, J., Daborn, P.J., Goddard, M.E., Batterham, P., & Robin, C. (2010). Copy number
959 variation and transposable elements feature in recent, ongoing adaptation at the *Cyp6g1*
960 locus. *PLoS Genet.* **6**, e1000998.

961

962 Smillie, C., Garcillán-Barcia, M.P., Francia, M.V., Rocha, E.P.C. & de la Cruz, F. (2010).
Mobility of plasmids. *Microbiol. Mol. Biol. Rev.* **74**, 434–452.

- 963 Smith, D.T., Hosken, D.J., Rostant, W.G., Yeo, M., Griffin, R.M., Bretman, A., Price,
 964 T.A.R., ffrench-Constant, R.H. & Wedell, N. (2011). DDT resistance, epistasis and male
 965 fitness in flies. *J. Evol. Biol.* **24**, 1351-1362.
- 966 Snook, R.R. & Hosken, D.J. (2004). Sperm death and dumping in *Drosophila*. *Nature* **428**,
 967 939-941.
- 968 Snook, R.R., Cleland, S.Y., Wolfner, M.F. & Karr, T.L. (2000). Offsetting effects of
 969 *Wolbachia* infection and heat shock on sperm production in *Drosophila simulans*: analyses of
 970 fecundity, fertility and accessory gland proteins. *Genetics* **155**, 167-178.
 971
- 972 Strunov, A., Schneider, D.I., Albertson, R., and Miller, W.J. (2017). Restricted distribution
 973 and lateralization of mutualistic *Wolbachia* in the *Drosophila* brain. *Cell. Microbiol.* **19**,
 974 e12639.
 975
- 976 Sutter, A. & Lindholm, A.K. (2016). No evidence for female discrimination against male
 977 house mice carrying a selfish genetic element. *Curr. Zool.* **62**, 675–685.
 978
- 979 Tomoatsu, I., Broughton, S., Nazif, Al., Grandison, R. & Partridge, L. (2009). The
 980 endosymbiont *Wolbachia* increases insulin/IGF-like signalling in *Drosophila*. *Proc. R. Soc.*
 981 *Lond. B.* **276**, <http://doi.org/10.1098/rspb.2009.0778>
 982
- 983 Turelli, M. & Begun, D.J. (1997). Haldanes' rule and X-chromosome size in *Drosophila*.
 984 *Genetics* **147**, 1799-1815.
 985
- 986 Úbeda, F., Patten, M.M. & Wild, G. (2015). On the origin of sex chromosomes from meiotic
 987 drive. *Proc. R. Soc. Lond. B* **282**, 20141932.
 988
- 989 Vaught, R.C., & Dowling, D.K. (2018). Maternal inheritance of mitochondria: Implications
 990 for male fertility? *Reproduction* **155**, R159-R168.
- 991 Vogan, A.A., Ament-Velásquez, S.L., Granger-Farbos, A., Svedberg, J., Bastiaans, E.,
 992 Debets, A.J.M., Coustou, V., Yvanne, H., Clavé, C., Saupe, S.J. & Johannesson, H. (2019).
 993 Combinations of *Spok* genes create multiple meiotic drivers in *Podospora*. *eLife* **8**, e46454.
- 994 Vogel, E., Santos, D., Mingels, L., Verdonckt, T.-W. & Broeck, J.V. (2019). RNA
 995 interference in insects: protecting beneficials and controlling pests. *Front. Physiol.* **9**, 1912.
 996 doi: 10.3389/fphys.2018.01912.
 997
- 998 Wedell, N. (2013). The dynamic relationship between polyandry and selfish genetic
 999 elements. *Phil. Trans. R. Soc. B* **368**, 20120049.
 1000
- 1001 Wedell, N. (2019). The effect of non-self genes on the behaviour of hosts. In *Genes and*
 1002 *Behaviour: Beyond Nature-Nurture*: 157-180. Hosken, D.J., Hunt, J. & Wedell, N. (Eds.)
 1003 John Wiley & Sons.
 1004
- 1005 Weeks, A.R., Turelli, M., Harcombe, W.R., Reynolds, K.T. & Hoffmann, A.A. (2007). From
 1006 parasite to mutualist: rapid evolution of *Wolbachia* in natural populations of *Drosophila*.
 1007 *PLoS Biol.* **5**, e177.
 1008

1009 Werren, J.H. (1991). The paternal sex-ratio chromosome of *Nasonia*. *Am. Nat.* **137**, 392-402.
1010
1011 Werren, J.H. (1997). The biology of *Wolbachia*. *Ann. Rev. Entomol.* **42**, 587–609.
1012
1013 Werren, J.H. (2011). Selfish genetic elements, genetic conflict, and evolutionary innovation.
1014 *Proc. Natl. Acad. Sci.* **108**, 10863–10870.
1015
1016 Wright, S. & Finnegan, D. (2001). Genome evolution: sex and the transposable element.
1017 *Curr. Biol.* **11**, R296-299.
1018
1019 Zachar, I., Szilagyi, A., Szamado, S. & Szathmary, E. (2018). Farming the mitochondrial
1020 ancestor as a model of endosymbiotic establishment by natural selection. *Proc. Natl. Acad.*
1021 *Sci.* **115**, E1504-E1510.
1022
1023 Zanders, S.E. & Unckless, R.L. (2019). Fertility costs of meiotic drivers. *Curr. Biol.* **29**,
1024 R512–R520.
1025
1026 Zeh, J.A. & Zeh, D.W. (1996). The evolution of polyandry I: intragenomic conflict and
1027 genetic incompatibility. *Proc. R. Soc. Lond. B* **263**, 1711-1717.
1028
1029 Zhou, Q. & Bachtrog, D. (2012). Chromosome-wide gene silencing initiate Y-degeneration
1030 in *Drosophila*. *Curr. Biol.* **22**, 522–525.
1031