Review

The Ecology and Evolutionary Dynamics of Meiotic Drive

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Meiotic drivers are genetic variants that selfishly manipulate the production of gametes to increase their own rate of transmission, often to the detriment of the rest of the genome and the individual that carries them. This genomic conflict potentially occurs whenever a diploid organism produces a haploid stage, and can have profound evolutionary impacts on gametogenesis, fertility, individual behaviour, mating system, population survival, and reproductive isolation. Multiple research teams are developing artificial drive systems for pest control, utilising the transmission advantage of drive to alter or exterminate target species. Here, we review current knowledge of how natural drive systems function, how drivers spread through natural populations, and the factors that limit their invasion.

The Battle for Transmission

One of the few rules in biology is Mendel’s law of equal segregation: the two copies of each gene and/or chromosome in a diploid organism are transmitted with equal probability to its offspring. Although often taken for granted, it is increasingly clear that equal segregation is a fragile détente in a world of constant intragenomic competition (see Glossary) for passage to the next generation. Such conflict plays out in the arenas of meiosis and gametogenesis, and results in meiotic drive [1], the biased transmission of a gene or chromosome against its alternative (Box 1). Because selection on meiotic drive elements operates at a level below that of the individual, drivers can spread through populations even if they reduce organism fitness [2]. By the same process, recently developed synthetic drive elements, which are currently still confined to laboratories, have the potential to rapidly modify genomes in wild populations [3]. Both natural and synthetic drive systems can have profound ecological, evolutionary, and genomic consequences.

Meiotic Drive Systems in Nature

In this review, we explore the ecological and evolutionary dynamics of natural meiotic drive systems. We focus on three types of drive: female meiotic drive, male meiotic drive (sperm killers), and drive in haploid spores (spore killers, Box 1). However, meiotic drive can

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to compete and killing them will not increase the killer’s fitness. However, disabling noncarrier sperm often reduces fertility [8].

Male meiotic drive takes multiple forms – some at least partially meiotic, some entirely post-meiotic – but all involve a driving element that prevents maturation or function of sperm that do not contain it. Because haploid sperm within a single ejaculate compete to fertilise the same pool of eggs, disabling noncarrier sperm results in transmission of the driving element to more than half of the functional gametes and resulting offspring ([5], Box 1). However, disabling noncarrier sperm can compete for inclusion in the gamete and hence transmission to subsequent generations, with failing chromosomes discarded into the polar bodies. Examples of drive through female meiosis have been observed in mice [22,36], maize [80], and many Drosophila species [17]. Finally, in fungi a heterozygous cross between strains carrying a spore killer allele and a sensitive allele results in elimination of haploid ascospores that lack the spore killer allele ([7], Figure 1B). These gametic drivers typically involve a drive locus and a target locus. They can occur on autosomes – as in the mouse t haplotype [56] and the fruit fly Segregation Distorter [31] – or on sex chromosomes, causing distorted sex ratios among progeny – as in Silene flowering plants [81], stalk-eyed flies [82], mosquitoes [17], and many Drosophila species [17].

Spore drive in fungi, in which the products of meiosis are packaged together in an ascus, operates via similar mechanisms. Spores with one haploid genotype will kill or disable spores of the alternative haplotype ([7], Box 1). If spores disperse long distances sibling spores are unlikely to compete and killing them will not increase the killer’s fitness. However, spore killing can be beneficial if there is local resource competition.

Exciting progress has been made in dissecting the genetic and cellular mechanisms of multiple drive systems that span eukaryotic diversity (Box 1). However, we are still in the early stages of understanding how these genetic systems interact with ecology to shape the dynamics of drivers in natural populations. The fate of a meiotic driver depends on the costs of transmission bias, the mating system, environmental factors, and population and geographic structure that affect the fitness of its carriers. These interactions might then affect how drivers contribute to genetic and phenotypic variation within and among populations, potentially contributing to speciation [8]. On a larger timescale, coevolution between drive elements and suppressors encompass a broad range of systems we do not discuss, including supernumerary B chromosomes, zygote killers, and paternal genome eliminators.

Female meiotic drive occurs when homologous chromosomes are differentially transmitted to the egg during meiosis. In plants and animals, female meiosis is asymmetric, with only one of the four meiotic products becoming an egg or, in plants, a megagametophyte ([4], Box 1). Any chromosomal variant that biases its own segregation (e.g., by preferentially associating with and moving toward the egg pole at Meiosis I) will be transmitted to more than half of the maturing eggs. Although this bias does not necessarily reduce the production of eggs (as only one egg matures per meiosis), the fitness of other alleles at the same locus, that do not bias transmission, and alleles linked to them, is reduced. Such meiotic drivers could reduce the fitness of individuals that carry them, if the driving variant is genetically linked to deleterious mutations or has deleterious pleiotropic effects.

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Figure I. Meiotic Drive. The first column shows schematics of three types of meiotic drive, with the second column showing a species that carries that drive system. (A) Female gametogenesis: driving chromosomes relegate rival chromosomes to the polar bodies. The polar bodies are lost, while the drive chromosome enters the egg. (B) Female drive occurs in monkeyflowers. (C) Male gametogenesis: driving chromosomes (‘D’) cause sperm that carry the rival chromosome (‘d’) to die. (D) Sperm killing segregation distortion occurs in stalk-eyed flies. (E) Fungal spore production. Similar to male drivers, spore killers cause the death of spores that carry rival chromosomes. (F) A spore killing system found in Neurospora fungi. Images: (B) Lila Fishman, (D) Gerald Wilkinson, and (F) Hanna Johannesson.
might also shape fundamental aspects of eukaryotic biology, including meiosis, gametogenesis, and genome structure [9–11]. Finally, understanding how ecology influences the population dynamics of meiotic drivers is important for predicting the success of synthetic drive elements, which are currently being engineered and applied to the management of vector populations of important human diseases ([12], Boxes 2 and 3). In this review, we consider the impacts drivers
can have on the genomes, individuals, and populations that harbour them, and then discuss the factors that influence the dynamics of drivers in natural populations.

**Consequences of Drive**

**Genomic Conflict**

Meiotic drivers can pose a significant cost to the rest of the genome, which is then under selection for unlinked alleles that suppress drive and restore equal segregation. Consider a driving allele that resides on an X chromosome in a species with heterogametic (XY) males. The driving X causes Y-bearing sperm to die, such that the driving X is transmitted to all offspring, who become daughters. The spread of the driving X makes the population sex ratio increasingly female-biased, until lack of males causes population collapse and extinction [13]. It is easy to imagine that any Y chromosome that resists drive will be favoured by selection [13,14], even if the driver is rare. Once the population sex ratio has become female-biased, classical Fisherian sex ratio selection will favour any autosomal mutation that suppresses drive [13,15]. Interestingly, a recent comparative study on tetrapods suggests that sex chromosome drive could account for the evolutionary pattern of species with male heterogamy exhibiting more female-biased adult sex ratios than species with female heterogamy [16].

Many drive systems consist of multiple drivers and suppressors, with several loci being involved with drive expression [17]. These systems suggest that the conflict does not end once a drive suppressor has evolved. Instead, enhancers linked to the original drive locus could evolve to restore drive, resuming the conflict. In this way, a drive system can cycle through periods of apparent drive and lack of drive resembling a coevolutionary arms race [18], resulting in a
complex genetic drive system. Recurrent coevolution between drivers and suppressors can contribute to the rapid evolution of genes, satellite DNA, and pathways whose functions might otherwise be expected to be conserved.

**Rapid Divergence in Sequences, Genome Organisation, and Populations**

Drive can contribute to DNA sequence evolution via selfish, driving nucleotide substitutions. For example, the meiotic drive gene *Overdrive* (GenBank: GA19777) of the fruit fly, *Drosophila pseudoobscura bogotana*, differs from the nondriving wild-type allele of its close relative, *Drosophila pseudoobscura pseudoobscura*, by seven nucleotide changes [19]. More often, drive seems to involve copy number variants: the *Segregation Distorter* system of *Drosophila melanogaster* involves a partial duplication of a protein-coding gene [20]; the *t haplotype distorter* system of the house mouse (*Mus musculus*) involves four tandemly duplicated genes [21]; copy number gain of the R2d distorter locus in house mice is associated with drive [22]; and the tandemly repeated, rapidly evolving, testis-expressed ampliconic genes of mammalian sex chromosomes are thought to result from recurrent arms races over gene dosage [23]. Such arms races do not necessarily occur between a driver and suppressors: different allelic variants of a meiotic driver can also compete against one another [24,25]. The rapid evolution of centromeres and centromeric proteins is particularly striking because these essential proteins are otherwise expected to be highly conserved [26]. Early speculation that female meiotic drive might be responsible for this rapid centromeric change is now supported by evidence in *Mimulus* monkeyflowers [27]. Finally, testis-expressed *de novo* genes often arise and spread to fixation but then, once fixed, degenerate into nonfunctional pseudogenes—a pattern suggestive of drive [28]. The recent identification of a young, rapidly evolving heterochromatin protein gene involved in a case of X chromosome drive in *Drosophila simulans* strongly supports this idea [29].

Drive can also have large-scale impacts on genome organisation and chromosome structure. Sperm killing meiotic drive elements often begin with just two loci—a driver and a target sequence, with the driver tightly linked to a resistant target to prevent the production of a suicide chromosome—subsequently driver and target may become elaborated via the recruitment of genetically linked enhancers. Such linked, coadapted gene complexes are expected to evolve in regions of low recombination and can become further protected from recombination by chromosomal inversions [30]. Reduced recombination associated with male drive has been found in *Segregation Distorter* [31], the *t haplotype* [32], *Spore killer* [33], and *Drosophila recens Sex-Ratio* [34]. Female drive can involve dramatic changes in the quantity and sequence content of centromeric satellite DNA and proteins, as centromeres evolve to compete for access to primary oocytes and avoid relegation to the polar bodies, losing their chance for transmission (Box 1, [11,35,36]). Female drive can also favour the evolution of chromosome fusions or fissions, in which two fused centromeres experience a transmission rate different from that of nonfused ones, thus fueling karyotype evolution [4]. As drive is usually exclusive to one sex, it accentuates intralocus sexual conflict [37]. Hence, a drive locus is expected to acquire genetically linked sexually antagonistic loci [38], potentially explaining the origin of sex chromosomes [39].

The combined effects of drive on DNA, genome, and karyotype evolution can lead to rapid divergence between populations and ultimately to speciation. For example, the fixation of alternative chromosome fusions in different populations can result in incompatible karyotypes that cause meiotic segregation problems in heterozygous individuals [36,40]. Recurrent drive and suppression can lead to cryptic drive systems, where fair meiosis has been restored within a species, but in a hybrid individual the dormant or suppressed drive elements can then spring into action [5,41,42]. Owing to reduced recombination and lack of homology, well-differentiated sex chromosomes are more susceptible to the invasion of drive elements. The recurrent fixation of cryptic drive systems on sex chromosomes might explain the prominent role of the X chromosome in the evolution of hybrid sterility in a wide range of species [42–44]. Cryptic drive systems
appear to contribute to reproductive isolation between populations and species of Drosophila [19,45], stalk-eyed flies [46], and yeasts [47].

**Growth and Persistence of Populations**
Drive can also have ecological consequences. Female-biased populations are expected to have higher per capita growth rates [13,48]. Although individuals carrying X-linked drivers might leave fewer descendants than other members of their subpopulation that lack drivers, subpopulations containing an intermediate frequency of drivers might have faster population growth relative to driver-free subpopulations [48] and competing species [49]. Finally, a significant consequence of distorted sex ratios is the potential for population extinction attributable to the lack of one sex [13,50,51], although definite evidence for such extinctions is almost entirely limited to lab populations [52–54].

**Dynamics of Drive**

**Stability of Driver Frequencies in Natural Populations**
All else being equal, drivers are predicted to increase in frequency as a result of biased transmission, and go to fixation. However, the spread of a driver can be limited by genetic suppressors, as well as fitness costs to carriers such as decreased fertility or viability [50]. Most of the known drive elements impose fitness costs on their carriers [6,31,55], either as a result of direct pleiotropic effects of the driver on survival or reproductive success, production of a biased sex ratio (in the case of sex-linked drivers), or via deleterious mutations linked to the driver. The latter are expected to build up in drive systems located in genomic regions with reduced recombination (e.g., inversions). Genetic studies suggest that some well-studied drive systems apparently have persisted for considerable time (estimated ages: t haplotype in mice ca. 2 Mya [56], Drosophila pseudoobscura Sex-Ratio ca. 1 Mya [57]). This long-term stability is surprising: a drive polymorphism is characterised by powerful selection on drivers and suppressors, and simple models suggest even a small change in drive or suppression strength can potentially lead rapidly to extinction or fixation. However, well-studied drivers in stable polymorphisms may represent a biased sample, if most drivers rapidly reach fixation or extinction, thereby becoming almost impossible to detect.

Fitness costs to individuals homozygous for the drive allele might help explain the persistence of some polymorphisms [51,58,59]. As autosomal drivers only benefit from transmission bias when in heterozygotes, they are most likely to be able to drive when rare. At higher frequencies, driver homozygotes become common, unmasking any recessive deleterious mutations linked to the drive allele. Processes that increase homozygote frequency, such as inbreeding, are predicted to reduce autosomal driver frequency [58]. The general prediction of an intermediate equilibrium for drivers with homozygous costs is borne out in some cases; for example, in yellow monkeyflowers, male and female fitness costs measured in the field together predict the observed frequency of a centromere-associated driver [59]. However, driver frequency in natural populations is often substantially lower than predicted by simple models based on homozygote fitness effects [17,60].

Field studies of driver dynamics are rare, as few wild populations harbouring meiotic drivers have been repeatedly sampled [24,54,61,62]. Long-term studies of driver frequencies within populations are even rarer [60]. Several species show apparently stable clines in driver frequency [54,62], for example, the frequencies in Drosophila pseudoobscura populations across North America have remained unchanged for 70 years. By contrast, a strong decline of the house mouse t haplotype frequency within one population was seen over 6 years [60]. There are also examples of rapidly spreading drivers. In D. simulans, a young X driver originating in Africa has spread in the Middle East within the past 2 decades [62] while simultaneously decreasing in East Africa because of genetic suppression. The reasons for the stability of some drive systems, and
the rapid spread and decline of others, are poorly understood and a major focus of drive research.

**Sexual Selection against Driver-Carrying Individuals**

Male and female mating behaviour are predicted to influence driver dynamics. The costs associated with drive create a benefit to avoiding mating with individuals carrying a driver, and thus preferences against driver carriers are expected to evolve [63]. In stalk-eyed flies (*Teleopsis dalmanni*), females prefer to mate with males with males with larger eyespans, and driver-carrying males tend to have smaller eyespans [64,65]. In some house mouse populations, females carrying the haplotype discriminate against driver males in choice tests, although wild-type females show no preference [66,67]. However, as recombination is expected to break linkage between drive elements and traits that allow mate choice [63], with undetectable drivers predicted to rapidly outcompete detectable forms, premating discrimination against driver males might be uncommon [6]. Alternatively, as many sperm killers significantly reduce sperm numbers, females could potentially avoid drivers by preferentially discarding sperm from males transferring small ejaculates, as hinted by a study in *D. simulans* [68]. The production of driver-carrying progeny can also be avoided through sperm competition when females mate with multiple males, assuming driver-carrying males are poor sperm competitors [69]. Both theoretical models [51,60,70] and empirical studies [54,55,61,71–73] support the idea that gamete competition can reduce driver frequencies and limit the spread of male drivers under some conditions (see [51]). Indeed, the presence of drive elements can select for and lead to an increase in female mating frequency. If female mating rates are density-dependent [73], this could make drivers rare in denser populations.

**Spatial Heterogeneity**

Driver distribution varies across space and between habitats, and this aspect of natural drive systems might be important for the successful application of artificial drivers (Boxes 2 and 3). Drivers in mice and monkeyflowers vary in abundance between populations [59,74]. Segregation Distorter is typically found at very low frequencies in *D. melanogaster* [31], while two other *Drosophila* species show latitudinal clines in driver frequency across North America [54,61]. Driver frequency correlates negatively with the frequency of polyandry in these populations, supporting the hypothesis that polyandry impacts the success of drivers in nature. However, in *Drosophila neodtestacea*, the environmental factor that best predicts the frequency of drivers is winter temperature [75], implying that drivers might be limited by elevated susceptibility to cold in driver carriers. Frequency of drivers in *D. pseudoobscura* can cycle yearly [76], suggesting more complex ecological interactions control driver abundance. Sperm killers can interact with other environmental factors that affect male fertility, such as high temperature [77]. It seems that variation in driver fitness between populations can result from interactions between environmental factors and the characteristics of populations harbouring drivers, potentially including differences between populations in deleterious genes linked to drive elements.

**Fixation and Extinction of Drivers**

Stable drive systems might be the exception, not the rule, with most drivers rapidly reaching fixation or extinction and becoming undetectable [50]. Population extinction is frequently predicted by simple models of sex chromosome drive [13,50,51]. It is difficult to measure the frequency of drive-mediated extinction because extinct populations leave no trace: while sampling wild *D. neodtestacea*, Pinzone and Dyer [54] collected 175 flies from an isolated population, 91% of which were female; the following year only three flies were found at the same site, all driver-carrying females, and only one was inseminated. Laboratory experiments suggest that local extinctions are likely [52,53]. Local extinctions might allow drive to persist in a spatial mosaic where drive-related local extinctions are followed by rapid recolonisation from nearby sites [78]. Finding definitive evidence for such processes is very difficult, and the frequency at which such extinctions occur cannot typically be gauged.
Molecular mechanisms of drive

We understand the genetic basis of very few drive systems. Are there general themes in the mechanisms? Do all gametic drive systems target similar pathways, or is each unique? Is the preponderance of drive systems in the Diptera (flies) attributable to some shared weakness in spermatogenesis that drive can exploit? Why is genetic suppression apparently absent in some ancient drive systems? Do these drive systems target something fundamental that cannot be defended, or are these drivers simply evolving faster than their targets?

Contrasting synthetic and natural drive

How similar are the mechanisms of natural drive to synthetic drive systems? As the survivors of generations of counter selection, are natural drivers more robust than synthetic ones? Or are they limited by mutations where the designers of synthetic drivers are not?

Outstanding Questions

Despite involving key processes of life, our understanding of meiotic drive remains rudimentary. Here, we outline some key unresolved questions.

How common is drive?

Drive is the result of a fundamental conflict and potentially occurs in any diploid organism. Yet known drivers come from a limited range of species. Is it simply that drivers are rare? If so, why? Or do drivers usually persist for a very short time before reaching fixation or going extinct? Alternatively, are some taxa particularly susceptible to drive? Indeed, we have little understanding of how often novel mutations create drive. Why are so many of the detected drivers so strong, when theory suggests weak drive should be common? Is it simply that weak drive is difficult to detect?

Drivers across space and time

Despite decades of research, we lack data on how drive varies across time and space. Consequently, we do not know if drive is stable or cycles. We also do not know if drivers require a metapopulation for survival, nor what limits the spread of drivers between populations. Moreover, do drivers spread between hybridising species?

Poorly Understood Dynamics in Many Systems

The ecological dynamics of spore killers in fungi are little known. Although the system is increasingly understood at the genetic level [32,77], the rarity of local resource competition makes the advantage they gain from drive obscure [78]. Ecological understanding of the dynamics of female drivers is also poor, with the exception of Mimulus monkeyflowers [59]. Finally, some documented sperm killer systems are more complex than any existing theoretical models. For example, Drosophila paramelanica has two driving X chromosomes, a Y that is susceptible to both, another Y that is resistant to one of the drivers, and latitudinal differences between populations in the co-occurrence of drivers and Y chromosomes [79]. Currently, little is known about how multiple drivers and resistance chromosomes coexist. Understanding factors that influence natural drive system dynamics is likely to be important to ensure the successful application of synthetic drive systems (Boxes 2 and 3).

Summary and Concluding Remarks

The potential for meiotic drive is probably high in all sexual organisms with a diploid phase, because the conflict over the transmission of homologous chromosomes in haploid gametes is nearly universal. Our understanding of the ecological and evolutionary dynamics of drive is surprisingly poor, even in well-studied systems. Nevertheless, some consistent themes stand out. Genetic suppression can evolve to neutralise drivers to the extent that the driver becomes undetectable, and this suppression can evolve and spread extremely rapidly [62]. Yet
suppression is not universal, and some ancient systems seem to have never evolved resistance or suppression. All well-studied extant drivers have costs, either intrinsic to the mechanism they use to gain their transmission advantage, or resulting from the reduced recombination that commonly associates with drive. Repeated discoveries of such associations suggest that extant drive systems are often complex, using multiple genes, perhaps indicating that successful drivers need modifiers that help them avoid suppression. Active drive systems vary in frequency between populations, and sometimes over seasons and years, suggesting that the fitness of drivers depends on their local environmental conditions, in ways that are currently not well understood.

Novel synthetic drive techniques (Box 2) have the potential to fundamentally alter natural populations in ways analogous to meiotic drive. These synthetic drive systems have enormous potential for bioccontrol, but if they are used without understanding how drive behaves in natural systems, there are serious risks of synthetic drive both failing to achieve its aims and having unintended negative consequences. Work on natural drive systems shows that the consequences of drive are manifold, from speciation to genome organisation, gametogenesis, competition among species, mate choice, and mating systems. Once synthetic drivers are released into nature, the potential for long-term evolutionary changes in the target species and its community are profound.

New natural drive systems will be discovered in coming years (Box 4), for example, by the discovery of non-Mendelian patterns of inheritance in sequence data. Detecting new drivers should help answer many of the outstanding questions in the field (see Outstanding Questions), and without doubt will uncover new mechanisms of drive, as well as unexpected genomic consequences of drive.

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