Asymmetric inheritance

Asymmetric inheritance: the diversity and evolution of non-Mendelian reproductive strategies

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Abstract

The ability to reproduce is the key trait that distinguishes living organisms from inorganic matter, and the strategies used to achieve successful reproduction are almost as diverse as the organisms themselves. In animals, the most widespread form of reproduction involves separate male and female sexes: each sex produces haploid gametes via meiosis, and two gametes fuse to form a new diploid organism. In some cases, both parents contribute equally to the nuclear and cytoplasmic genomes of their offspring. However, such fully symmetric reproduction of both parents represents the extreme end of a continuum towards complete asymmetry where offspring inherit their nuclear and cytoplasmic genomes from only one of the two parents. Asymmetries also occur with respect to the fate of maternally and paternally inherited genomes or which sex is affected by non-Mendelian inheritance. In this review we describe the diversity of animal reproductive systems along different axes with a symmetry-asymmetry continuum, and suggest evolutionary routes that may have led to increased levels of asymmetry.

1. Introduction

Reproduction is a key trait distinguishing living organisms from inorganic matter and it is a central part of every species' life cycle. Among animals, the most widespread form of reproduction involves separate male and female sexes: each sex produces haploid gametes via meiosis, and two gametes fuse to form a new diploid organism (Bachtrog et al. 2014; Jarne & Auld 2006; Normark 2006). There is an inherent asymmetry between gametes in animals. Namely, males produce smaller, highly motile gametes (sperm) and females produce larger, immoble gametes (eggs). How this asymmetry evolved is not yet clear, but two explanations currently exist. First, gamete asymmetry may be the result of disruptive selection favoring either small, numerous, gametes (i.e. sperm) or few, resource-rich gametes (i.e. eggs) (Parker et al. 1972). Alternatively, it may result from the specialization of gametes to solve the problem of finding each other in a complex environment. One type would specialize in searching and be chemotactic (i.e.,

sperm), whereas the other would remain immobile (i.e., the egg) and produce chemical signals used by the chemotactic gamete (Hadjivasiliou & Pomiankowski 2016).

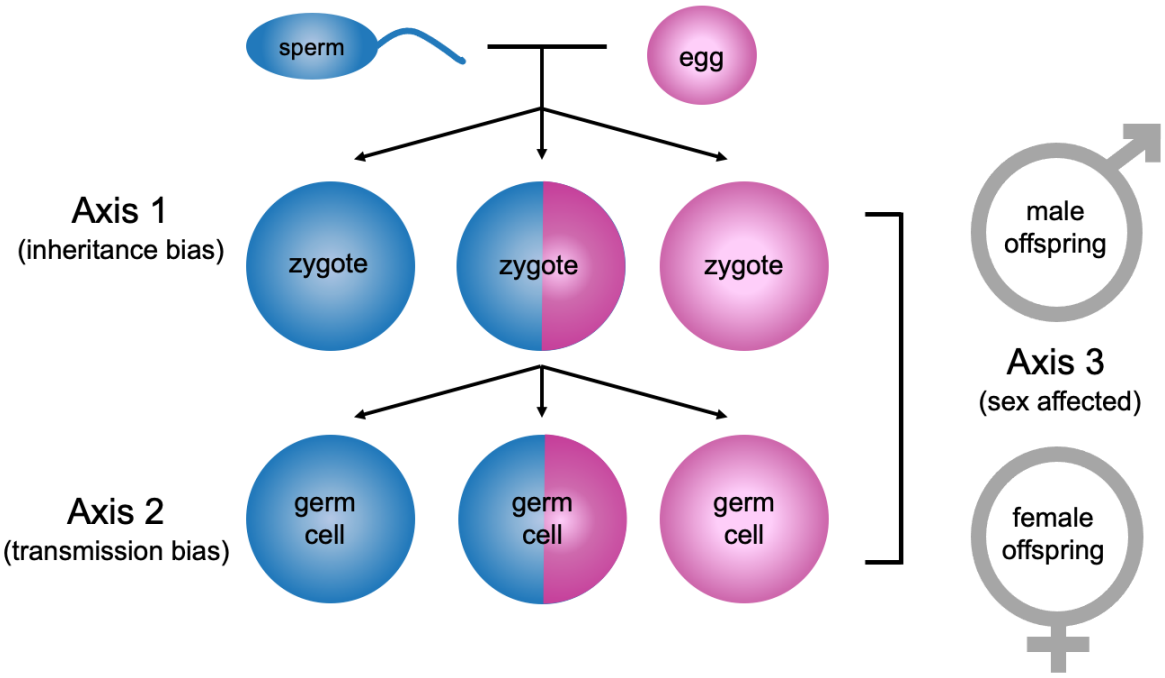
The size asymmetry between male and female gametes is associated with another asymmetry. Cytoplasmic elements, most prominently mitochondria, chloroplasts and vertically transmitted bacterial endosymbionts, are generally transmitted solely by the female parent. Although exceptions likely exist, such as uniparental inheritance of mitochondria from either the female or male parent, or even rare events of biparental inheritance (Hoeh et al. 1991; Skibinski et al. 1994a,b; Zouros et al. 1992, 1994), we are not considering asymmetric cytoplasmic inheritance here, as there are several reviews on this topic (Birky 2001; Greiner et al. 2015; Ladoukakis & Zouros 2017; Xu 2005). Instead, we focus solely on asymmetry in nuclear inheritance.

In contrast to the asymmetry in gamete size and cytoplasmic inheritance, nuclear inheritance is generally symmetrical, with the male and female parents contributing equally to the nuclear genome of each offspring. Yet deviations from this classical system exist at different steps of the sexual reproduction pathway along a continuum from relatively symmetric to strongly asymmetric inheritance patterns. We discuss three axes of asymmetry in inheritance patterns (Fig 1). The first is **asymmetric inheritance from male and female parents**. The center of this axis represents a completely symmetric system with equal contribution of both parents to the offspring's nuclear genome. On the two ends of the continuum are fully asymmetric systems where the offspring's genome derives from only the female or only the male parent. These include female-producing parthenogenesis (thelytoky) and male asexuality (androgenesis).

The second axis of asymmetric inheritance represents **asymmetry in the fate of the two nuclear genomes (maternal and paternal) in a diploid individual**. There are two striking examples of this. First, species with paternal genome elimination, in which males are diploid but only include maternally-derived chromosomes in their sperm. And second, hybridogenetic species, in which individuals (males or females) are diploid and systematically transmit either solely their maternal, or solely their paternal genome to their gametes.

The final axis of asymmetry is determined by **the sex of offspring affected by asymmetric inheritance**, for in haplodiploid and paternal genome elimination (PGE) systems, only males are affected while reproduction through females is fully symmetrical. Distinguishing between these different axes of asymmetry is useful to classify the different reproductive systems found in animals. What will become clear by doing so is that reproductive systems are often asymmetrical along multiple axes, so that in order to understand the evolution of asymmetric inheritance, we often have to consider multiple axes simultaneously.

a)


sperm
egg
Axis 1
(inheritance bias)
zygote
zygote
zygote
germ cell
germ cell
germ cell
Axis 2
(transmission bias)
male offspring
Axis 3
(sex affected)
female offspring

b)

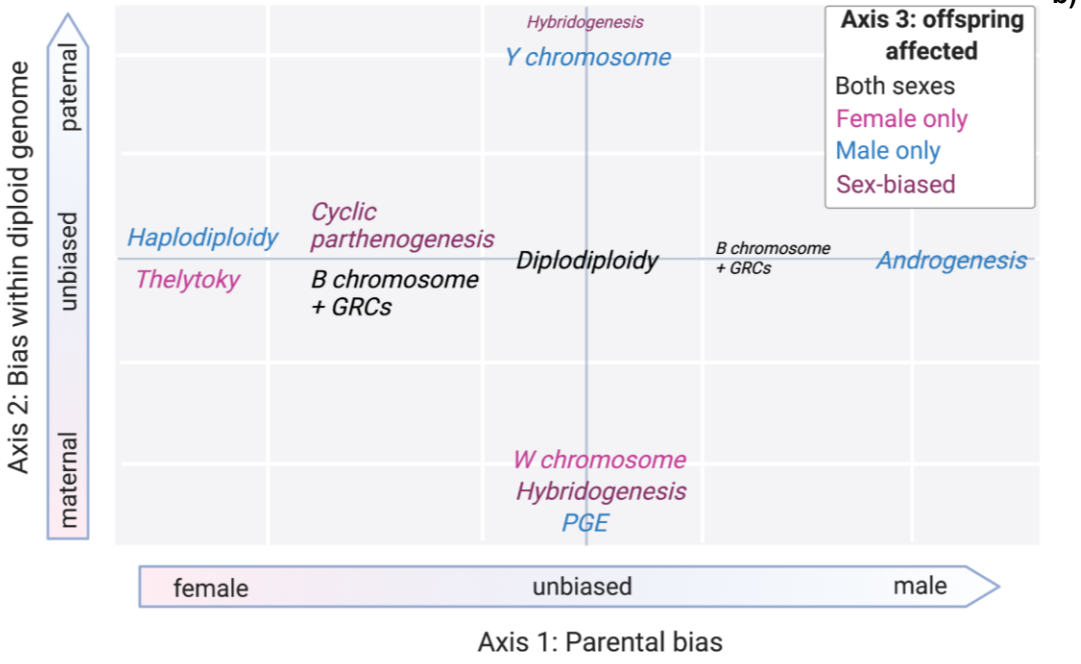

Axis 2: Bias within diploid genome
paternal
unbiased
maternal
Hybridogenesis
Y chromosome
Axis 3: offspring affected
Both sexes
Female only
Male only
Sex-biased
Haplodiploidy
Thelytoky
Cyclic parthenogenesis
B chromosome + GRCs
Diplodiploidy
B chromosome + GRCs
Androgenesis
W chromosome
Hybridogenesis
PGE
female
unbiased
male
Axis 1: Parental bias

Figure 1

a) Schematic illustration of the three axes of asymmetry we describe: Asymmetric inheritance from parents, Asymmetric behavior parental genotypes within offspring, Asymmetry in which offspring sex is affected and **b)** how each of the described reproductive systems fit on this asymmetry continuum from relatively balanced to fully asymmetric across the three axes. Note that the axes are not orthogonal and in fact are correlated for many systems. Also note that some systems can occur in multiple places as the direction of asymmetry can vary among species, we have indicated the less common direction in a smaller font. PGE: Paternal Genome Elimination; GRCs: Germline Restricted Chromosomes. Figure created with biorender.com

2. Characterization and description of reproductive systems

In this section we will describe the diversity of reproductive systems among animals and for each system highlight along which axes of asymmetry they fall.

2.1 Asymmetric inheritance from male and female parents

Chromosome transmission to gametes (i.e., nuclear inheritance patterns) is most symmetric under **sexual reproduction without sex chromosomes** (or with homomorphic ones) as well as in species that lack separate sexes (hermaphrodites). Asymmetric transmission of individual chromosomes can arise from **differentiated sex chromosomes** in species with separate sexes**, driving sex chromosomes, B chromosomes and germline restricted chromosomes.** At the extreme are systems that are asymmetric with respect to inheritance of all chromosomes.

Systems with asymmetric inheritance of single chromosomes

In many animals with genetic sex determination, the sex chromosome pair has diverged in size, sequence, and gene content (Bachtrog et al. 2014). **Differentiated sex chromosomes** create an asymmetry in inheritance by their role in sex determination. Consider the familiar XX/XY chromosome system of mammals. Females have two X chromosomes, so all eggs receive one X. Males, however, are heterogametic (having one X and one Y). Even if transmission is unbiased (e.g. 50% of sperm receive the X chromosome and 50% receive the Y), the end result is still asymmetric because the Y chromosome is only transmitted by males and the X chromosome is transmitted twice as often by females as males. Systems with female heterogametic sex determination (ZZ/ZW systems) have the same type of asymmetric inheritance, but here the sex-specific chromosome is in females. Furthermore, in some cases, **X-chromosome drive** in males can increase asymmetry by generating female-biased sex ratios (meaning that the X spends even more time in females than males than under 50:50 sex ratios (Burt & Trivers 2006)). **Y-chromosome drive** can have similar consequences.

In a few taxa unusual sex chromosome asymmetries have evolved, which often include obligate sex chromosome drive. One such example is found in the nematode *Auanema rhodensis*, in which males and females co-occur with hermaphrodites (referred to as trioecy, Tandonnet et al. 2018). Females and hermaphrodites both have two X chromosomes, and males have only one. X transmission is as expected in females, but is highly asymmetric in hermaphrodites. In hermaphrodites, oocytes never receive an X (they are nullo-X) whereas all sperm cells carry two X chromosomes. In sperm cells produced by males, X chromosome inheritance is also unusual. Instead of the expected 50% of cells carrying a single X, and 50% nullo-X cells, the latter category is never produced, and all cells carry a single X (Tandonnet et al. 2018). A similar case of obligate X-chromosome drive in males is found in aphids. Aphid males are XO,

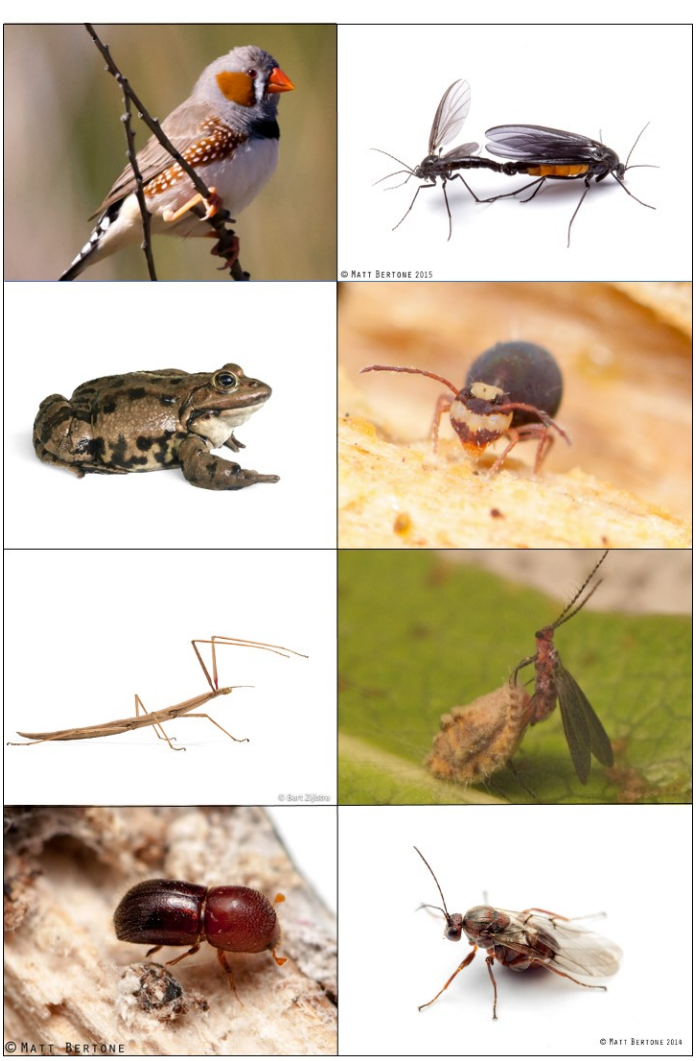
but only produce X-bearing sperm and therefore only father daughters (Blackman 1985; Wilson et al. 1997). There are also a number of unusual sex chromosome systems in vertebrates that lead to enhanced (and often extreme) sex chromosome asymmetry. In several species of rodents these include feminizing X chromosomes which lead to the production of XY females. Sex chromosome transmission in these XY females varies between species, with in some cases a transmission advantage for the X chromosome, while in others only the Y-chromosome is transmitted (Saunders et al. 2022). In response to this asymmetric sex chromosome transmission by XY females, a conditional form of sex chromosome drive evolved in males: they preferentially transmit the Y in crosses with XX females, but transmit the X in crosses with XY females (Romanenko et al. 2020; Saunders et al. 2022). Similar examples of sex-change associated with sex-chromosome drive exist, such as a mascunilizing mutation in the Tongue sole with ZW sex determination (Cui et al. 2018) or the asymmetric sex chromosome inheritance in the creeping vole (*Microtus oregoni*). In the latter case the Y chromosome fused with the X. This new fused chromosome comes in two flavours, the X^P only found in males, and the X^M found in both sexes. Females are somatically X^MO, but carry two identical copies of X^M in their germline so that all oocytes transmit the X^M (X-drive). In males on the other hand the X^M is eliminated during spermatogenesis so that males produce either X^P or nullo-X sperm (Couger et al. 2021).

Transmission asymmetry is also frequently observed for supernumerary **B chromosomes,** estimated to be present in at least 10% of eukaryotic species (Ahmad & Martins 2019; Camacho 2005; Camacho et al. 2000). B chromosomes are genomic parasites that exist within cells of some individuals, sometimes as multiple copies, in addition to the core genome. B chromosomes are maintained in populations often in spite of neutral or even deleterious fitness effects, through drive mechanisms that are generally poorly understood (Houben 2017; Jones 1991). These mechanisms typically lead to asymmetric inheritance, with transmission exclusive to or at least more pronounced in one sex. Many Bs appear to exploit existing asymmetries in reproduction: for example, securing their inclusion into the oocyte rather than polar bodies during oogenesis (e.g. in a locust, Pardo et al. 1994). In other cases, the drive mechanism exploits asymmetries in transmission of chromosomes from parents to offspring: In the haplodiploid wasp *Nasonia vitripennis*, haploid males normally do not pass genetic material on to male offspring. However a sperm-derived B chromosome in this species drives by eliminating all other paternally-inherited autosomes from female embryos during embryogenesis. This turns them into haploidised males, which in turn will incorporate the B chromosome into their sperm (Benetta et al. 2020; Nur et al. 1988). Another example of B chromosomes exploiting asymmetric inheritance is in a mealybug species that reproduces through paternal genome elimination (see below); there the B chromosome escapes elimination with the other paternal chromosomes during spermatogenesis. Finally, asymmetric inheritance of B chromosomes may interact with sex determination (Nokkala et al. 2003). A clear example is a B chromosome in the gibel carp (*Carassius gibelio*) that is associated with male development in an otherwise all-female parthenogenetic species (Li et al. 2016). More speculative is the suggestion that some sex chromosomes such as the Y chromosome in *Drosophila melanogaster* and the W chromosome in butterflies and moths may derive from a B chromosome that gained a sex determining function (Carvalho 2002; Fraïsse et al. 2017; Vicoso & Bachtrog 2015).

Another class of chromosomes that can display sex-specific inheritance are **germline restricted chromosomes** (GRCs). They are eliminated from somatic cells early in development such that they are restricted to the germline in adults. GRCs are found in several animal clades including songbirds (Fig 2), hagfish, and three families of flies: the gall midges (Cecidomyiidae), the non-biting midges (Chironomidae) and the fungus gnats (Sciaridae) (Fig 2) (Hodson & Ross 2021; Wang & Davis 2014). They can range from a single chromosome to up to 80 in some of the flies and can make up a large proportion of the genome. Much remains unclear about the evolution and function of GRCs, although many have been found to contain a large number of genes, some of which are important for germline

function (Hodson et al. 2021; Kinsella et al. 2019). GRCs are not inherently asymmetric: they are present in the germline of both sexes in all clades, but their transmission is often biased. In songbirds (at least in those species that have been studied), GRCs are primarily maternally transmitted (Pigozzi & Solari 2005). The GRC duplicates prior to female meiosis and segregates during meiosis so that one GRC copy is present in the egg. In males GRCs are eliminated during spermatogenesis, although there is some paternal leakage (Pei et al. 2022). In gall midges GRCs are also maternally transmitted and eliminated during male meiosis together with the paternally derived genome (this clade reproduces through paternal genome elimination (PGE) see below) (White 1973). In fungus gnats, another PGE clade, GRCs are transmitted through both males and females. However in *B. coprophila*, the best-studied fungus gnat, GRC transmission is male biased as two GRCs are transmitted through sperm but only one is transmitted through eggs (Crouse et al. 1971). It is currently unclear why GRCs are often biased in their transmission and why the direction of the bias seems to differ between clades.

a) **b)**



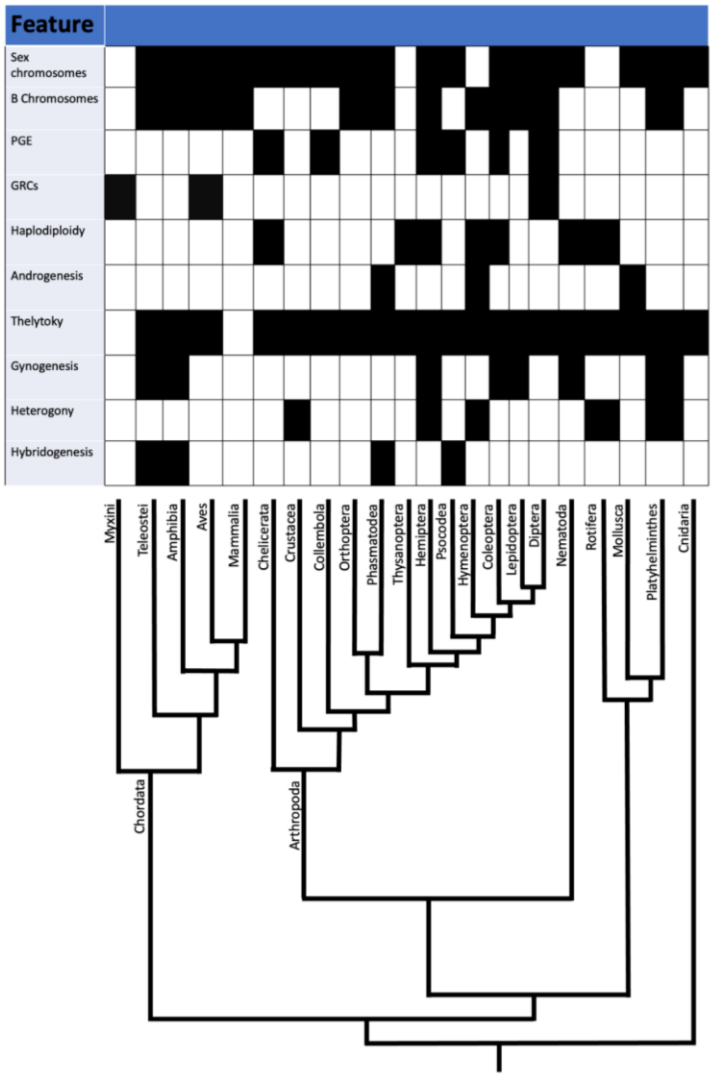


Figure 2: Examples of species with asymmetric inheritance, **a)** from top left: Zebra finch, maternally-transmitted germline restricted chromosomes. Mating male and female fungus gnats, paternal genome elimination and germline restricted chromosomes. Female edible frog *Pelophylax esculentus,* hybridogenesis; Globular springtail, paternal genome elimination. Female *Bacillus* stick insect, hybridogenesis, photo copyright Bart Zijlstra https://www.bartzijlstra.com/, species in the same genus are capable of obligate and facultative parthenogenesis as well as androgenesis. Mating hermaphrodite and male *Icerya purchasi* scale insects, androdioecy and haplodiploidy, photo copyright Enric Frago. Ambrosia beetle, haplodiploidy. Gall wasp, cyclic parthenogenesis. Photos b, d, g, and h are copyright Matt Bertone. **b)** the taxonomic distribution of asymmetric reproduction with black squares indicating presence in a clade. Tree topology based on (Irie et al. 2018; Marlétaz et al. 2019; Misof et al. 2014).

Systems with asymmetric inheritance of entire genomes

Haplodiploidy or arrhenotoky is a sex determination system in which females develop from fertilized, diploid eggs and males from haploid, unfertililzed ones. As inheritance is symmetric for daughters and only asymmetric for sons (only the female contributes), this system lies somewhere in between the symmetric nuclear inheritance in sexual species without heteromorphic sex chromosomes and the completely asymmetric inheritance found under the different forms of asexuality described below (see also Fig 1). Most instances of haplodiploidy are found in arthropods including the entire orders Hymenoptera (wasps and allies), and Thysanoptera (thrips), as well as several smaller clades such as whiteflies, some groups of ambrosia and bark beetles (Fig 2), and several families of mites (Blackmon et al. 2015; de la Filia et al. 2015; Normark 2004). It is also found in other invertebrate clades including a family of nematodes (Adamson 1989) and the monogonont rotifers (Nogrady 1993). Haplodiploidy has evolved repeatedly with an estimated 20 origins, and includes approximately 12% of all animal species. As males are haploid, spermatogenesis involves a highly modified meiosis with only a single equational division (the reductional division is aborted) (Ferree et al. 2019).

Haplodiploidy occasionally occurs within the context of other asymmetric or otherwise unusual inheritance systems. One example is the evolution of haplodiploidy in the scale insect tribe the Icerynii, in which diploid, female-like hermaphrodites co-exist with haploid males (Hughes-Schrader 1925; Royer 1975). Hermaphrodites have an ovotestis which is a chimera of two tissue types, diploid cells in which female gametes develop and haploid tissue in which spermatogenesis takes place. Reproduction is primarily through self fertilization, although hermaphrodites and males can mate and outcross (Mongue et al. 2021). It is currently not clear if haplodiploidy co-evolved with this mating system, or preceded its evolution. Another example of haplodiploidy co-evolving with asymmetric inheritance is the evolution of an unusual type of reproduction called **paedogenesis** in both gall midges (Diptera) and a species of beetle (see more details below).

An extreme kind of asymmetric inheritance is parthenogenesis, where a single parent contributes the full nuclear genome of offspring. **Female-producing parthenogenesis (thelytoky**) occurs in different forms - it can be obligate, sperm-dependent, facultative, spontaneous or cyclical (reviewed in Jalvingh et al. 2016). Obligate parthenogenetic animal species are rare compared to sexual species, but widespread across most major taxa (Bell 1982). The vast majority of the taxonomically described parthenogenetic species represent independent transitions to parthenogenesis as most of these species are “tips on the tree of life”, meaning there are few genera or higher taxa consisting solely of parthenogenetic species (Bell 1982). Oribatid mites (Oribatida), darwinulid ostracods (Darwinulidae), and bdelloid rotifers are the best known exceptions to this general pattern. Several animal groups appear to be particularly rich in transitions to obligate parthenogenesis. This is the case for certain hymenopteran genera (van der Kooi et al. 2017) as well as certain insect orders in general (Normark 2003), especially stick insects (Phasmatodea), in the latter perhaps because sexual species are generally capable of spontaneous or facultative parthenogenesis (see below).

Some obligately parthenogenetic species are characterized by **sperm-dependent parthenogenesis (gynogenesis)** (reviewed in Beukeboom & Vrijenhoek 1998). Here, paternal components, often the centrioles, are still required for triggering embryo development. Thus, parthenogenetic females need to mate with males of related sexual strains to produce offspring, but the paternal genome does not contribute to the zygote’s genome. Sperm-dependent parthenogenesis is found in many taxa, including vertebrates, molluscs, flatworms, nematodes, and different arthropod orders (Beukeboom & Vrijenhoek 1998). **Facultative parthenogenesis** characterizes lineages in which each female can use both biparental sex and female-producing parthenogenesis to generate offspring. Facultatively parthenogenetic females can flexibly shift between the two reproductive modes and parthenogenesis is generally meiotic (Suomalainen et al. 1987; White 1973). The efficiency of parthenogenesis and sexual

reproduction (number of offspring produced) is comparable under facultative parthenogenesis, distinguishing it from spontaneous parthenogenesis in sexual species where the hatching success of unfertilized eggs is very low. However, survival rates are typically higher for sexually than parthenogenetically produced offspring such that, given the option, females will prefer to produce sexual rather than parthenogenetic offspring. Facultative parthenogenesis occurs and may be widespread in some insect groups such as phasmids, mayflies or termites, but is most likely rare in other animal groups. More frequent is mixed reproduction (species with sexual and parthenogenetic strains) however females in each strain are obligately sexual or obligately parthenogenetic.

Cyclical parthenogenesis (also called **heterogony**) is a type of life cycle in which a sexual generation (bisexual or hermaphroditic) alternates with one or more generations of parthenogenetic reproduction. Six large animal groups are characterized by this life cycle (Bell 1982): trematodes (a parasitic class of flatworms), rotifers, cladocerans (water fleas such as Daphnia), aphids (including adelgids, and phylloxerids), and cynipids (gall wasps)(Fig 2). Parthenogenesis typically predominates under favorable conditions; deteriorating or stressful conditions (for example linked to seasonality, resource depletion or crowding) trigger the production of males and sexual females. **Paedogenesis,** an unusual type of cyclical parthenogenesis that also involves haplodiploidy, is found in cecidomyiids (gall midges) (White 1973) and in a single species of beetle, the telephone pole beetle *Micromalthus debilis* (Pollock & Normark 2002). The parthenogenetic part of the life cycle involves reproduction by immature females that reproduce either as larvae or as pupae. The offspring are mostly female and develop within their mothers body and often consume their mother before emerging. Some of the offspring of female larvae develop into adults caple of sexual reproduction. Sexual females are able to produce males by laying unfertilized eggs which develop into haploid males (haplodiploidy, see below). Males and adult females mate and produce diploid female offspring, which again reproduce parthenogenetically during the immature stages.

Each form of parthenogenesis is characterized by variable cellular mechanisms underlying the production of offspring (Suomalainen et al. 1987; White 1973), but these mechanisms are poorly studied, or not at all, in the vast majority of species. Egg production is tightly linked to meiosis, hence parthenogenesis involves a modified meiosis that allows for the maintenance of ploidy levels between generations. The meiosis modifications can include a duplication of all chromosomes prior to meiosis, or a secondary fusion/ suppression of cytokinesis. In some cases, meiosis is fully maintained and females lay haploid eggs that undergo diploidization secondarily during early mitotic divisions in embryos.

Completely asymmetric inheritance of the nuclear genome is also the case for male asexualiy, generally referred to as androgenesis (reviewed in Schwander & Oldroyd 2016). **Androgenesis** is a form of reproduction in which a male's gamete develops into a new male. Two forms of androgenesis are recognized. Under the first form, known in *Corbicula* clams, a sperm fuses with an egg, but the maternal nuclear genome is eliminated and does not contribute to the developing offspring (Ishibashi et al. 2003; Komaru et al. 1998). Under the second form, known in different organisms including stick insects (Mantovani & Scali 1992; Tinti & Scali 1995) and ants (Fournier et al. 2005, Ohkawara et al. 2006, Kobayashi et al. 2008, Pearcy et al. 2011), an embryo develops from a male gamete in an egg that lacks a functional maternal nucleus. Thus, when such 'non-nucleate' eggs are fertilized they develop into an offspring whose entire nuclear genome is of paternal origin. Androgenesis in haplodiploid organisms does not require additional mechanisms for the maintenance of male ploidy, since eggs fertilized by a single sperm can develop into normal, haploid males. In diploid organisms, however, androgenesis depends on the production of diploid sperm (in clams; Ishibashi et al. 2003; Komaru et al. 1998), or on the fusion of two haploid sperm cells in the egg (in stick insects; Mantovani & Scali 1992; Tinti & Scali 1995).

2.2. Asymmetry in the fate of the two nuclear genomes (maternal and paternal) in a diploid individual.

Under the second axis of asymmetry we discuss reproductive strategies where the fate of the maternal and paternal genomes *within offspring* differs strikingly, with only one of the two genomes being transmitted to future generations. Asymmetric maternal or paternal genome transmission is known in species with separate male and female sexes, in which transmission asymmetry can occur in only one or both sexes, but it is also known in unisexual, generally female-only species.

Hybridogenesis is a form of reproduction that occurs in hybrids between two related species or lineages and involves the selective transmission of only the maternal or only the paternal nuclear genome. It is also referred to as "hemiclonal reproduction", to reflect that half of the genome is clonally transmitted. Hybridogenesis has been reported in phylogenetically diverse animals, including different fish taxa (e.g., Kimura-Kawaguchi et al. 2014, Schultz 1969), frogs and salamanders (Heppich 1978), stick insects (Mantovani & Scali 1992) and most likely booklice (Hamilton et al. 2018).

There is little knowledge of the proximate mechanisms underlying the specific transmission of only one of the two available genomes, but there appear to be two common patterns across different hybridogens. The first is that genome elimination occurs typically in juveniles, early during the differentiation of the germline. Such early elimination is supported by research in different taxa, including *Hypseleotris* carp gudgeons (Majtánová et al. 2021), *Bacillus* stick insects (Tinti & Scali 1992, Marescalchi & Scali 2001) and *Pelophylax* water frogs (Fig 2) (Chmielewska et al. 2018). In the water frogs, this elimination appears to occur via the formation of micronuclei which contain the eliminated parental genome in a heterochromatized form (Chmielewska et al. 2018). The second common pattern is that hemiclonal transmission involves a premeiotic duplication of the parental genome to be transmitted. As a consequence, meiosis starts with a diploid cell comprising two identical copies of one parental genome.

Two types of reproductive systems in ants have been referred to as “social hybridogenesis” because of their conceptual similarity with hybridogenesis. In the first system, two co-occurring lineages hybridize with each other at each generation, but all offspring produced from within-lineage matings develop into new queens while inter-lineage hybrid offspring develop into workers (Cahan & Vinson 2003; Cahan et al. 2002; Julian et al. 2002; Norman et al. 2016; Romiguier et al. 2017; Volny & Gordon 2002). Because workers are sterile but required for colony functioning, they can be compared to the hybrid soma in hybridogenetic species, while queens, reflecting the germline, are non-hybrid. However, this system does not feature asymmetry along the axes we focus on here. In the second system of social hybridogenesis, queens are produced via parthenogenesis, while workers are produced from fertilized eggs (Kuhn et al. 2020; Leniaud et al. 2012; Pearcy et al. 2004). In some cases, queens mate with males of a diverged lineage to produce workers, which also reproduce asexually via androgenesis (Fournier et al. 2005). These systems thus combine two cases (parthenogenesis and androgenesis) of the most extreme asymmetric inheritance from male and female parents described above.

Paternal genome elimination (PGE) is a type of reproduction that in many respects is similar to haplodiploidy with the important exception that males develop from fertilized eggs, and therefore PGE is symmetric in terms of parental inheritance (axis 1). However, while males develop from diploid eggs, they only ever pass on their mothers genome to their offspring because they fail to include their father’s genome in their sperm. PGE systems have independently evolved seven times, in diverse groups of arthropods: the globular springtails (Symphypleona) (Fig 2) (Dallai et al. 1999; Jaron et al. 2021b), parasitic lice (de la Filia et al. 2017; McMeniman & Barker 2005) and *Liposcelis* booklice (Hodson et al. 2017), scale insects (Coccoidea) (Nur 1990; Ross et al. 2010), the gall midges (Cecidomyiidae) (White

1973), the fungus gnats (Sciaridae) (Metz 1938), *Hypothenemus* bark beetles (Brun et al. 1995; Normark 2003) and several families of parasitiformes mites (Norton et al. 1993). While the patterns of inheritance are similar for both haplodiploidy and PGE, there are several important differences. First of all, under haplodiploidy males are always haploid, while under PGE males are often partially or even completely diploid (de la Filia et al. 2015, 2021). This is because in most species the paternal genome is only eliminated during spermatogenesis and therefore present in all somatic and germline cells. There are some exceptions to this: in mites and some scale insects the entire paternal genome is eliminated in early embryos, so in those systems males are haploid during most of their development (embryonic PGE) (Norton et al. 1993; Nur 1990; Ross et al. 2010). In the other PGE clades, males remain genetically diploid throughout development (germline PGE) but can be partially haploid either because they suppress the expression of paternal alleles (scale insects, coffee borer beetle, *Liposcelis* booklouse)(Brun et al. 1995; de la Filia et al. 2021; Hodson et al. 2017), or because they eliminate paternally-derived sex chromosomes in early development (fungus gnats, gall midges and globular springtails)(Gerbi 1986; Jaron et al. 2021b; White 1973). Finally, male parasitic lice appear to be fully diploid until spermatogenesis (De la Filia et al. in prep). Another unusual type of reproduction reminiscent of PGE has evolved in a species of the otherwise haplodiploid *Encarsia* parasitoid wasps. Here, diploid female offspring parasitize the primary hemipteran hosts, while males are hyperparasites of female conspecifics. However, some initially diploid eggs can develop into males within primary hosts after losing their paternal genome in early development (Hunter et al. 1993). It is not clear what triggers this occasional haploidization through genome loss.

Most PGE species remain poorly studied and therefore we know very little about the molecular mechanisms that govern different aspects of this unusual reproductive strategy. It involves the evolution of novel mechanisms of meiosis and spermatogenesis, recognition of the parental origin of the haploid genomes, sex determination and ploidy. Here we will briefly outline what is known. Male meiosis in species with germline PGE is highly aberrant. The exact details differ between taxonomic groups but there are a few common themes: in all clades there is no recombination in males and chromosomes segregate based on parent of origin, with half of the meiotic products receiving only maternal and the other half only receiving paternal chromosomes (as reviewed in Burt & Trivers 2006). In mealybugs this unusual meiotic sequence is accompanied by inverted meiosis, where the reductional division is preceded by the equational division (Bongiorni et al. 2004). At the end of meiosis II, only those nuclei containing maternal chromosomes develop into mature sperm, while the others degrade. In fungus gnats that have sex chromosomes and XO males, meiosis II involves an unequal division where both X chromatids segrate into one nucleus. Only the nucleus that inherits both the maternal autosomes as well as the two X chromosomes develops as a sperm cell; the remaining ¾ of nuclei degrade (Gerbi 1986). This latter system is thereby somewhat equivalent to oogenesis where ¾ of nuclei form polar bodies rather than eggs. It is unclear exactly why the maternal and paternal chromosomes segregate into different nuclei. In both mealybugs (a group of scale insects) and fungus gnats, maternal and paternal chromosomes appear to be enriched for different histone modifications which could play a role. In the mealybugs the paternal chromosomes also appear more condensed throughout meiosis. In both systems there is a suggestion that the reductional division of meiosis involves the formation of a uni-spindle that only attaches to maternally-derived chromosomes, however the data supporting this observation in both lineages is limited.

2.3. Asymmetry in which offspring is affected by asymmetric inheritance

In the previous two sections we have described reproductive systems where there is either asymmetry in which of the parents contributes genetic material to the next generation (axis 1) or in the fate of maternally/paternally derived genomes within their offspring (axis 2). However in several of these

systems there is a third axis of asymmetry: the offspring sex that is affected. This is true for both haplodiploidy (asymmetric inheritance) and PGE (fate asymmetry of parental genomes) in which the asymmetry is present only in male offspring, while inheritance through females is fully symmetrical. It also holds for species with heteromorphic sex chromosomes, where one of the sex chromosomes (the Y or the W) is only inherited through a single sex. However, there are also systems in which both offspring sexes are affected by different types of asymmetry: in hymenopterans with facultative (female-producing) parthenogenesis, males are always produced parthenogenetically, developing from unfertilized haploid eggs, while females are diploid and can either be produced through asymmetric inheritance (parthenogenesis) or symmetric inheritance (outcrossing). Other examples come from several unusual reproductive systems in nematodes: in *Mesorhabditis belari* nematodes, females are produced through sperm-dependent parthenogenesis (asymmetric inheritance, Grosmaire et al. 2019). Contrary to other known sperm-dependent parthenogens, *Mesorhabditis* females do not mate with males from other species to trigger egg development, but instead produce a few sons from fertilized eggs. Curiously, fertilized eggs always develop into males because only Y-carrying sperm succeed in fertilizing eggs (genome-specific fate) (Grosmaire et al. 2019). Another example are the *Auanema* nematodes with three sexes (hermaphrodites, males and females) described above, where there is asymmetric X chromosome behaviour (genome-specific fate) in hermaphrodites and males, but not in females (Tandonnet et al. 2018).

3. Original spread and maintenance of asymmetric systems

What evolutionary forces are responsible for the evolution of these three different axes of asymmetric inheritance? The answer is complex and in part will depend on the particular type of asymmetric reproduction. However it appears that three features are key to governing all systems of asymmetric reproduction.

First, **a transmission advantage**. The spread of all asymmetric inheritance systems is linked to part or all of the genome gaining a transmission advantage relative to more symmetric systems (Brown 1964; Burt & Trivers 2006; Hurst & Werren 2001; Maynard-Smith 1978). What varies among systems is where and how increased transmission occurs. Under haplodiploidy and PGE, males only pass on a haploid copy of their mothers genome. This is a way for mothers to monopolize the parentage of sons. Females able to do so pass on their genes at a higher rate through their male offspring, giving them an overall fitness advantage (Brown 1964; Bull 1979; Gardner & Ross 2014; Hartl & Brown 1970; Ross et al. 2019; Smith 2000) . Males able to generate at least some offspring via androgenesis transmit an extra copy of their genome to every androgenetic descendant relative to normal sexually produced offspring, which results in the spread of the genetic basis for androgenesis (Lehtonen et al. 2013; McKone & Halpern 2003). In hybridogenetic species, every allele in the genome that is hemiclonally transmitted between generations sees its transmission rate increased to 100%, rather than the 50% expected under canonical meiosis (Lehtonen et al. 2013; Vrijenhoek 1989). Under parthenogenesis, because asexual females do not produce any males which cannot themselves produce offspring, the rate of population growth is faster in asexual than sexual species, again resulting in the spread of the asymmetric system within the sexual population with symmetric inheritance (Maynard-Smith 1978; Williams 1975). Finally there are several types of **asymmetrically-transmitted selfish elements** that could act as drivers for the extension of asymmetric inheritance (Hurst 1995; Hurst & Werren 2001). Selfish elements are able to enhance their transmission compared to the rest of the genome, and therefore often rapidly spread through populations (Burt & Trivers 2006). They are common, and include chromosomes that enhance transmission through meiotic drive (e.g sex chromosome meiotic drive and many B chromosomes) or endosymbiotic bacteria (such as *Wolbachia*) which have evolved varied ways to increase their transmission by manipulating their host's reproduction. The spread of these elements can at least temporarily lead to asymmetric inheritance

of single chromosomes (e.g. for B chromosomes), but can also drive the evolution of more long term or genome-wide asymmetries. Perhaps the clearest example of a selfish element causing transitions to asymmetric inheritance is *Wolbachia* inducing parthenogenesis in their host (Werren et al. 2008). Other examples include the possible role of driving X chromosomes in the origin of PGE (Anderson et al. 2020; Haig 1993) and the suggestion that the germline restricted chromosome found in songbirds has originated from a B chromosome (Hansson 2019).

Second, **sex and recombination**. Asymmetric inheritance patterns involve modifications of the meiosis pathway, which almost always leads to a decrease in the rate of recombination. For example, there is no recombination in haploid males and recombination is typically reduced or may be completely lacking under parthenogenesis, hybridogenesis and androgenesis. Sex and recombination are believed to be generally favored because they increase the efficacy of natural selection by reducing different forms of selective interference between loci (recently reviewed in Otto, 2021).There is evidence in natural populations of parthenogenetic species that beneficial mutations fix more slowly and deleterious mutations are purged less effectively (Bast et al. 2018; Jaron et al. 2021a,c). Other predictions however, such as the accumulation of transposable elements (TEs) could not be corroborated. This is most likely because lineage-level selection will tend to eliminate parthenogenetic species with high TE loads, a mechanism that does not generally apply for non-recombining genome portions of sexual species sex chromosomes (Jaron et al. 2021c,a). While similar consequences of reduced recombination can be expected for other asymmetric inheritance systems, empirical tests remain scarce.

A third is the effect of **ploidy**. In many asymmetric reproductive systems either part (e.g. sex chromosomes) or all of the genome (e.g. male haploidy) become haploid. Haploidy directly exposes recessive alleles to selection and can therefore also increase the efficiency of selection (Gerstein et al. 2011; Hedrick & Parker 1997; Rice 1984; Werren 1993). On the other hand, male haploidy means a reduction in effective population size, so that the effects of drift will also be stronger, which will tend to reduce population polymorphism and the fixation rate for advantageous mutations but increase it for deleterious mutations (Wright, 1931; Vicoso & Charlesworth, 2009).

Ploidy variation associated with asymmetric inheritance of genome portions can also affect the fixation probability of sexually antagonistic alleles (Rice, 1984; Gibson *et al.*, 2002; Mullon *et al.*, 2012) In diploid male heterogametic species, the X chromosome spends two-thirds of its time in females, giving an advantage to dominant female-beneficial alleles on the X. By extension, we could predict that genomes in haplodiploid species are more optimized for female than male phenotype (relative to diplo-diploid species), given that the genome is more often inherited through females than males (Hitchcock et al. 2021; Klein et al. 2021). In the most extreme cases, genomes experience selection in only one sex, i.e., females in asexual species (Parker et al. 2019), males in androgenetic species, and males or females only (depending on the species) in hybridogens. Empirical tests of these predictions are clearly needed to evaluate similarities and differences between sex chromosome evolution and evolution under asymmetric inheritance systems. Some of the more unusual asymmetric systems might be particularly useful for such an approach. For example species with paternal genome elimination, where males are often at least partially diploid, would allow us to disentangle the effects of hemizygosity and asymmetric inheritance, which are usually confounded in studies on sex chromosome evolution.

Selection acting on these three features is central to the evolution and fate of all forms of asymmetric inheritance systems. However, many additional important factors that depend on species-specific ecologies, such as the amount of sexual conflict or sex-specific selection, or the amount of inbreeding, can interact with one or more of these features. These interactions can either favor or hamper the spread of asymmetric inheritance systems. Inbreeding, for example, can reduce offspring fitness by exposing

recessive deleterious alleles (Charlesworth & Willis 2009). In this context, it was suggested that some asymmetric inheritance systems may have spread because they reduce costs of inbreeding (Pearcy et al. 2011). This is a possibility in several ant genera, in which the production of workers (correcponding to the soma of the colony) relies on the hybridization between lineages, which avoids the costs of inbreeding, while males and queens (the "germline") are produced asexually. On other hand, inbreeding allows purging of recessive deleterious alleles and therefore might facilitate the evolution of reproductive systems that involve an extended haploid phase (Bull & Cummings 1983; de la Filia et al. 2015; Otto & Jarne 2001). More generally, how frequently we observe particular asymmetric inheritance systems will thus be determined by the relative importance of factors hampering or favoring their spread as well as how likely asymmetric systems are to go extinct. Systems that are primarily asexual (parthenogenesis, hybridogenesis, androgenesis) are generally seen as evolutionary dead ends because they are prone to extinction, with their frequency driven by the rates of emergence and extinction (Schwander & Crespi 2009). Few primarily asexual taxa have diversified into ecologically diversified "species", both because most groups are too recent for diversification to have occurred and because "speciation" processes under asexuality are fundamentally different from those in sexual species (Barraclough et al. 2003; Birky & Barraclough 2009). On the other hand, haplodiploidy and paternal genome elimination are conserved traits in old and species-rich clades, suggesting that their distribution is primarily constrained by infrequent origins, not high extinction. In fact, haplodiploid and PGE clades appear to have increased diversity compared to their diploid sister clades (Lohse & Ross 2015). This has led several authors to suggest that haplodiploidy and PGE could increase diversification rates. For example, male haploidy can reduce the rate of gene flow between hybridizing species, because females mated to heterospecific males only produce hybrid daughters, because sons only carry maternal genes (Patten et al. 2015), and because haploidy can enhance hybrid breakdown caused by Bateson–Dobzhansky–Muller incompatibilities (Bendall et al. 2021). This effect is likely to be more pronounced in haplodiploids than in PGE taxa as males in these systems are at least partially diploid.

4. <u>Origin and taxonomic distribution</u>

While there are often good explanations for how asymmetric transmission genetics can spread, these explanations fail to capture how asymmetric transmission originates in the first place and why it has done so in some taxonomic groups but not others. One of the challenges is the assumption that the key components of newly emerged systems are equivalent to what we currently observe. For example, when considering the spread of a new parthenogenetic lineage arising within a sexual population, it is generally assumed that incipent parthenogens are as fit as the lineages we observe today. This may be the case for cyclical or facultative parthenogens, where the capacity for parthenogenesis is already quite old and where obligate parthenogenesis can appear as a consequence of simple loss of function mutations (Molinier et al. 2021; Neiman et al. 2014) . However, this is quite unlikely for *de novo* origins of parthenogenesis, as many forms of parthenogenesis require the combination of multiple adaptations, which are unlikely to emerge all at once, meaning the efficiency of parthenogenesis likely increases over time (Schwander et al. 2010; Templeton 1982). Furthermore, many forms of parthenogenesis involve meiosis and a secondary restitution of diploidy, which results in the loss of heterozygosity similar to selfing (Suomalainen et al. 1987; Pearcy et al. 2004). In this case the fitness of incipient parthenogens is likely reduced as a consequence of expression of recessive deleterious alleles (Archetti 2004; Pearcy et al. 2004). Similarly, the spread of haplodiploidy or PGE in modeling approaches generally requires males to be at least half as fit compared to regular diploid males (Bull 1979; Hartl & Brown 1970). This may be unlikely since haploid males would suddenly expose recessive deleterious mutations, while PGE males are eliminating between ½ to ¾ of their sperm and are therefore likely to be less fertile (de la Filia et al. 2015). So it is possible that, similar to female fitness limiting the spread of parthenogenesis, male fitness is a key factor in limiting the spread of haplodiploidy/PGE. Reduced male or female fitness may also limit

the spread of other asymmetric systems such as hybridogenesis and androgenesis, although much less work has focused on the *de novo* emergence of these systems.

More generally, the emergence of asymmetric systems is likely hampered by various developmental and genetic constraints (Engelstädter 2008). These constraints can be reduced by taxon-specific traits, such as particular developmental systems, specific life history traits or sets of ecological conditions. For example, PGE may evolve more readily in species with a monogamous mating system where selection on male fertility is weak, and reducing sperm number does not strongly impact male fitness. We would also expect parthenogenesis and male haploidy to spread more readily in taxa with reduced genetic loads, as could be expected in species with a history of inbreeding, which allows for the purging of the deleterious load (Archetti 2004; Engelstädter 2008). Low ancestral recombination rates, including the complete lack of recombination and absence of chromosome pairing (achiasmy) in one of the two sexes, may also alleviate constraints. Achiasmy or low recombination for example might make it easier to modify meiosis in the non-recombining sex and reduce ferlility effects of doing so. Finally in some cases the evolution of asymmetric inheritance might have knock-on effects on sex determination resulting in biased sex ratios, which could either hinder or promote its spread (Gardner & Ross 2014; Ross et al. 2019).

The idea that constraints hampering the spread of asymmetric systems can be reduced by taxon-specific traits is supported by the striking observation of phylogenetic clustering of many of the systems we describe. A key example is the co-occurence of haplodiploidy and PGE in mites, scale insects and beetles. Parthenogenesis, hybridogenesis and androgenesis are all known in the stick insect genus *Bacillus* (Fig. 2). *Liposcelis* booklice, a genus with PGE and parthenogenesis, also has some females with only maternal genome transmission, most likely a type of hybridogenesis (Hodson & Perlman 2019; Hodson et al. 2017; Mockford 1971) In many cases, new patterns of asymmetric inheritance emerge within clades that are already asymmetric. Examples include the co-occurence of PGE and germline restricted chromosomes in the gall midges and fungus gnats, or the B chromosomes in mealybugs which exploit the transmission asymmetry of PGE to enhance their own transmission. *Micromalthus debilis* and several genera of gall midges combine haplodiploidy with paedogenetic cyclic parthenogenesis. Finally several ant species employ both female parthenogenesis (for queen production) and androgenesis (for male production). So understanding why asymmetric systems evolve more readily in some groups than others will require a better understanding of the diversity of developmental systems, and the key pathway, meiosis, that has to be modified for the emergence of inheritance asymmetry. While meiosis is often thought of as a very conserved molecular mechanism, recent work across a larger range of organisms shows that there is substantial variability (reviewed in Lenormand et al. 2016). It is likely that a better understanding of the molecular mechanisms of taxa with asymmetric inheritance, as well as their closest sister clades will provide further insight into the factors that enable modified meiosis to evolve.

5. Conclusions and outstanding questions

In this review we have outlined three axes of asymmetric inheritance that together can help classify the diverse reproductive strategies found among animals. We have also discussed the possible mechanisms and selection pressures that have led to their origin, maintenance, and evolutionary consequences. Asymmetric inheritance from parents (axis 1) and asymmetric fates of parental genomes within individuals (axis 2) are two alternative ways through which genomes, or genome fractions, can increase their transmission to future generations, and they often affect offspring of only one sex (asymmetry along axis 3). Because asymmetric inheritance generates or enhances intra- and intergenomic conflicts (Burt &

Trivers 2006; Normark 2006), asymmetry along one of the three axes can increase selection for asymmetry along the other two axes and drive further reproductive system diversification.

Increased transmission along the first two axes appears to be generally in favor of females or the maternal genome in offspring (Figure 1b). While for completely asymmetric transmission by parents (i.e., either androgenesis or parthenogenesis), this bias can be explained by androgenetic males always depending on eggs for offspring production, there is no *a priori* reason for this bias in the remaining systems. For example, although paternal genome transmission in hybridogens exists, maternal genome transmission is much more widespread, and GRCs are more often biased towards female- than male transmission. The most extreme situation is illustrated by the many independent transitions to (functionally) haploid males in haplodiploids and species with PGE: **Why are there no species with haploid or PGE females?**

A possible explanation for generally female-biased asymmetry is that male and female meiosis are very different, such that modifications to achieve asymmetric transmission will also vary. An example of this are the different mechanisms underlying genetic drive in males versus females: in males, drive typically occurs via sperm killing, while in females it occurs via preferential segregation into the oocyte instead of the polar bodies (Kruger & Mueller 2021). In the case of preferential segregation, a key role may be played by cytoplasmic elements: Because cytoplasmic elements are generally maternally inherited, their evolutionary interests are aligned with the maternal genome, which can lead to positive co-evolution between the cytoplasm and the maternal nuclear genome (Normark 2004). Such co-evolution could explain why interactions between signals in the cytoplasm and the centromeres cause meiotic drive favoring maternal chromosomes in hybrid mice (Akera et al. 2019) and eliminate paternal chromosomes in hybrid *Arabidopsis* (Marimuthu et al. 2021). They could also help explain the high prevalence of mitochondrial rearrangements in taxa with uniparental males (Cameron 2014), including the evolution of several mitochondrial “chromosomes” in parasitic lice (Shao et al. 2009), extensive rearrangements in thrips and multiple smaller rearrangements in several hymenopteran clades, scale insects, white flies (all hemipterans) and gall midges (Diptera). Nevertheless, male-biased asymmetry systems do evolve, and certain modifications of male meiosis for asymmetric transmission may be mechanistically easier, or less often associated with fertility costs in males than in females. This could be one possible explanation for why PGE which involves whole-genome meiotic drive tends to occur during male meiosis.

As these examples illustrate, **research directed towards understanding the molecular mechanisms that govern the different types of asymmetric inheritance is clearly needed.** This understanding is key in order to understand the drivers and constraints that govern the evolution of different systems. Knowledge of these mechanisms may reveal unexpected convergence across different asymmetric inheritance systems. For example, heterochromatinization of genetic material destined for elimination is described for almost all known cases of asymmetric reproduction involving programmed DNA elimination. During elimination of germline-restricted chromosomes in *Sciara* flies (Escriba & Goday 2013) and zebra finches (Goday & Pigozzi 2010), as well as during PGE in *Planococcus citri* mealybugs (Bongiorni et al. 2009) and hybridogenesis in *Pelophylax* water frogs (Chmielewska et al. 2018), strong heterochromatinization and abnormal H3S10 phosphorylation are believed to hamper proper chromatin compaction, which prevents the attachment of spindle microtubules to chromosomes. Another common theme among different systems is the degradation of spermatocytes (e.g. those not carrying a drive X chromosome, or those carrying the paternal genome in PGE species (Bressa et al. 2015; Brown & Nur 1964; Goday & Esteban 2001; Herbette et al. 2021). It could be that this type of asymmetric inheritance exploits a pathway for apoptosis of damaged sperm.

Another important focus for future work should be taxa where asymmetric inheritance has recently originated, as well as as the closest outgroups, as outgroups are key to distinguishing consequences of asymmetric inheritance from taxon-specific idiosyncrasies (Jaron et al. 2021c). They are also important for pinpointing modifications to the meiosis pathways in asymmetric systems, given that these pathways are very diverse even among species with classical sexual reproduction (Lenormand et al. 2016). While identifying appropriate outgroup species should be possible at least for recently derived systems, this may be difficult for ancient systems, such as most PGE and haplodiploid clades, with the coffee borer beetle that reproduces through PGE being a notable exception that may be useful focus for such studies.

Most of the examples we discuss in this review are drawn from insects, which is perhaps to be expected given that they make up the vast majority of animal diversity. However it is likely that asymmetric inheritance systems are far more frequent and widespread. Most, if not all, were discovered serendipitously and it therefore seems likely that asymmetric inheritance is overlooked in many species. Asymmetric inheritance is difficult to detect, unless it changes the sex ratio at population level or affects another highly visible phenotype. Thus, many systems were discovered when people did extensive surveys of karyotypes, or more recently, during population genetics surveys designed to study patterns not related to asymmetric inheritance. The ongoing efforts of large scale taxonomic sampling for genome sequencing are likely to uncover many more examples.

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