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REVIEW

Patterns and mechanisms in instances of endosymbiont-induced parthenogenesis

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Abstract

Female-producing parthenogenesis can be induced by endosymbionts that increase their transmission by manipulating host reproduction. Our literature survey indicates that such endosymbiont-induced parthenogenesis is known or suspected in 124 host species from seven different arthropod taxa, with *Wolbachia* as the most frequent endosymbiont (in 56–75% of host species). Most host species (81%, 100 out of 124) are characterized by haplo-diploid sex determination, but a strong ascertainment bias likely underestimates the frequency of endosymbiont-induced parthenogenesis in hosts with other sex determination systems. In at least one taxon, hymenopterans, endosymbionts are a significant driver of transitions from sexual to parthenogenetic reproduction, with one-third of lineages being parthenogenetic as a consequence of endosymbiont infection. Endosymbiont-induced parthenogenesis appears to facilitate the maintenance of reproductive polymorphism: at least 50% of species comprise both sexual (uninfected) and parthenogenetic (infected) strains. These strains feature distribution differences similar to the ones documented for lineages with genetically determined parthenogenesis, with endosymbiont-induced parthenogens occurring at higher latitudes than their sexual relatives. Finally, although gamete duplication is often considered as the main mechanism for endosymbiont-induced parthenogenesis, it underlies parthenogenesis in only half of the host species studied thus far. We point out caveats in the methods used to test for endosymbiont-induced parthenogenesis and suggest specific approaches that allow for firm conclusions about the involvement of endosymbionts in the origin of parthenogenesis.

Introduction

Female-producing parthenogenesis or 'thelytoky' (hereafter: parthenogenesis) refers to the production of female offspring without genetic contributions from males. Parthenogenetic animals derive from sexual ancestors whereby the transition from sexual reproduction to parthenogenesis occurs via three main evolutionary routes. First, the transition from sexual reproduction to parthenogenesis can be caused by spontaneous mutations. This is especially likely in cyclical parthenogens such as *Daphnia* water fleas (Beaton & Hebert, 1988) or monogonont rotifers (King & Snell, 1977), where parthenogenetic and sexual generations alternate in the life cycle (reviewed in Neiman *et al.*, 2014). Indeed, a single loss-of-function mutation can be enough to suppress the sexual cycle in these organisms and thus generate new obligately parthenogenetic lines (e.g. Stelzer, 2008; Stelzer *et al.*, 2010).

Second, parthenogenetic lineages can arise as a consequence of hybridization between two sexually reproducing species, a mechanism that appears to be frequent among vertebrates. Direct evidence for hybrid origins of parthenogenesis stems from fish. Choleva *et al.* (2012) experimentally generated parthenogenetic F1 hybrid females of *Cobitis* fish by crossing the sexual species *C. taenia* and *C. elongatoides*. Hybridization

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between the same two sexual species also generated different parthenogenetic lineages in natural populations (Janko *et al.*, 2003). The mechanisms through which F1 hybrid ancestry causes parthenogenetic reproduction remain unknown. Recombination can be suppressed in F1 hybrids because genetic divergence between genomic regions greatly reduces the local rate of recombination (Waldman, 2008). However, recombination suppression alone is generally not sufficient to generate new parthenogenetic lineages as parthenogenetic females need to produce unreduced gametes that develop spontaneously without any contribution from sperm (reviewed in Neiman *et al.*, 2014).

Finally, the third main route from sexual reproduction to parthenogenesis and focus of this review is parthenogenesis induced by endosymbiont infection (e.g. Stouthamer *et al.*, 1990). The ultimate reasons why endosymbionts induce parthenogenesis in their hosts are well understood (e.g. Stouthamer *et al.*, 1993; Werren, 1997). Endosymbionts are vertically transmitted from mothers to their offspring via the egg cytoplasm; they are not transmitted by males. By inducing female-producing parthenogenesis in their hosts (or by inducing increased investment into daughters via other types of reproductive manipulation not discussed here), endosymbionts increase their rate of transmission (Werren, 1997; Werren *et al.*, 2008).

In this review, we first conducted a careful literature survey to establish a database of all known species in which parthenogenesis is caused by endosymbiont infection. This database currently comprises 124 species from seven different arthropod taxa (state February 2017), including 54 confirmed (Table 1) and 70 speculative cases (Tables 2 and S1). The 124 species are characterized by different sex determination systems. In most species, sex is determined by haplo-diploidy, where males develop from haploid, unfertilized eggs and females from diploid, fertilized ones, but systems in which sexual differentiation depends on sex chromosomes in male or female heterogametic species (hereafter referred to diplo-diploidy) are also represented. We use this database to discuss similarities and differences across independent, endosymbiont-mediated transitions to parthenogenesis. We also highlight the different approaches used to identify endosymbionts as the cause of parthenogenesis in their hosts, and to infer the types of parthenogenesis induced by endosymbionts. Finally, we point to caveats that have led to unsupported conclusions in some systems.

Instances of endosymbiont-induced parthenogenesis

Frequency and reproductive polymorphism

How frequent endosymbiont-induced parthenogenesis is relative to other causes of parthenogenesis (i.e. spontaneous mutation or hybridization) is difficult to estimate. Indeed, for the vast majority of parthenogenetic species, the mechanisms that caused the transition from sexual reproduction to parthenogenesis are not known. Given our database of species with endosymbiont-induced parthenogenesis, we can provide an estimate of the minimum frequency of endosymbiont-induced parthenogenesis in taxa with available estimates for the frequency of parthenogenetic species. Such estimates are available for hymenoptera (wasps, bees, ants and sawflies), where parthenogenesis and endosymbiont-induced parthenogenesis have been studied extensively. Stouthamer (1997) indicates that there are at least 270 hymenopteran species with female-producing parthenogenesis. From our database, we identified 46 hymenopterans in which parthenogenesis is endosymbiont-induced (Table 1) and an additional 39 hymenopterans for which endosymbiont-induced parthenogenesis is speculative (Table S1). In combination, these estimates suggest that the frequency of endosymbiont-induced parthenogenesis in hymenopterans is at least 15–31%. Although this frequency is based on only a small fraction of the total hymenopteran diversity and will likely change with future studies, it indicates that endosymbionts are an important driver for transitions from sexual to parthenogenetic reproduction.

From our database, we further infer that species with endosymbiont-induced parthenogenesis generally display reproductive polymorphism. That is, at least 50% of species comprise both parthenogenetic (infected) and sexual (noninfected) females. Specifically, reproductive polymorphism characterizes at least 27 of 54 species with confirmed, endosymbiont-induced parthenogenesis whereas parthenogenesis and infection are fixed in only seven species (Table 1). For the remaining 20 species, parthenogenesis may be fixed but reproductive strategies have only been investigated in one or few populations such that sexual populations may have been missed (Table 1). Reproductive polymorphism is likely to be even more frequent. Many species considered to be obligately sexual may have currently unknown parthenogenetic populations, perhaps because of endosymbiont infection. These species would all display reproductive polymorphism, but are not included in the database.

Although all species with confirmed endosymbiont-induced parthenogenesis are haplo-diploid (see section below), reproductive polymorphism also appears widespread among diplo-diploid species in which endosymbiont-induced parthenogenesis is suspected but speculative. Indeed, reproductive polymorphism characterizes at least 32% and up to 96% (8–24 of 25) of these species; only 4% (1 of 25) are fixed for parthenogenesis (Table 2).

The frequent maintenance of reproductive polymorphism in species with endosymbiont-induced

Table 1 Species with a firm demonstration of endosymbiont-induced parthenogenesis (see text for details). Sex determination is haplo-haploidy in *Brevipalpus* and haplo-diploidy in all remaining species.

Host order	Host species	Common name	Approaches to verify	Reproductive polymorphism*	References
Parthenogenesis induced by *Wolbachia*					
Trombidiformes	*Bryobia praetiosa*	Mite	1) Antibiotic treatment; 2) *Wolbachia* PCR assay (*ftsZ* primers)	No?	Weeks & Breeuwer (2001)
Trombidiformes	*Bryobia sp.*	Mite	1) Antibiotic treatment; 2) *Wolbachia* PCR assay (*ftsZ* primers)	No?	Weeks & Breeuwer (2001)
Thysanoptera	*Franklinothrips vespiformis*	Thrips	1) Antibiotic and heat treatments; 2) *Wolbachia* PCR assay (*ftsZ* primers)	Yes	Arakaki *et al.* (2001)
Thysanoptera	*Hercinothrips femoralis*	Thrips	1) Antibiotic and heat treatments: 2) *Wolbachia* PCR assay (16S rDNA and *ftsZ* primers)	No	Kumm & Moritz (2008)
Hymenoptera	*Aphytis diaspidis*	Wasp	1) Antibiotic treatment; 2) *Wolbachia* PCR assay (*ftsZ* primers)	No	Gottlieb *et al.* (1998); Zchori-Fein *et al.* (1995)
Hymenoptera	*Aphytis lingnanensis*	Wasp	1) Antibiotic treatment; 2) *Wolbachia* PCR assay (*ftsZ* primers); 3) electron microscopy	Yes	Zchori-Fein *et al.* (1994, 1995, 1998); Gottlieb *et al.* (1998)
Hymenoptera	*Apoanagyrus diversicornis*	Wasp	1) Antibiotic and heat treatments; 2) *Wolbachia* PCR assay (*wsp* primers)	Yes	Pijls *et al.* (1996); Van Meer *et al.* (1999)
Hymenoptera	*Asobara japonica*	Wasp	1) Antibiotic treatment; 2) *Wolbachia* PCR assay (*wsp* primers) and general bacterial diagnostic PCR	Yes	Kremer *et al.* (2009); Reumer *et al.* (2012)
Hymenoptera	*Diaphorencyrtus aligarhensis*	Wasp	1) Antibiotic treatment 2) *Wolbachia* PCR assay (*wsp* primers)	No	Meyer & Hoy (2007)
Hymenoptera	*Encarsia formosa*	Wasp	1) Antibiotic treatment; 2) *Wolbachia* PCR assay (*wsp* primers)	No	Zchori-Fein *et al.* (1991); Hunter (1999); Giorgini *et al.* (2007)
Hymenoptera	*Eretmocerus mundus*	Wasp	1) Antibiotic treatments; 2) *Wolbachia* PCR assay (*ftsZ* primers)	Yes	De Barro & Hart (2001)
Hymenoptera	*Gronotoma micromorpha*	Wasp	1) Antibiotic treatment; 2) *Wolbachia* PCR assay (*ftsZ* primers)	No?	Arakaki *et al.* (2001)
Hymenoptera	*Leptopilina clavipes*	Wasp	1) Antibiotic treatment; 2) *Wolbachia* PCR assay (*ftsZ* primers)	Yes	Werren *et al.* (1995); Pannebakker *et al.* (2004a,b, 2005)
Hymenoptera	*Megastigmus pinsapinis*	Wasp	1) Antibiotic treatments; 2) multiple endosymbiont PCR assay	No?	Boivin *et al.* (2014)
Hymenoptera	*Muscidifurax uniraptor*	Wasp	1) Antibiotic treatment; 2) *Wolbachia* PCR assay (*wsp* primers)	No?	Stouthamer *et al.* (1993); Gottlieb & Zchori-Fein (2001), Gottlieb *et al.* (2002)
Hymenoptera	*Telenomus nawai*	Wasp	1) Antibiotic and heat treatments; 2) *Wolbachia* PCR assay (*ftsZ* primers, 16S rRNA)	Yes	Arakaki *et al.* (2000)
Hymenoptera	*Trichogramma atopovirilla*	Wasp	1) Antibiotic treatment 2) *Wolbachia* PCR assay (*wsp* primers)	Yes	Ciociola *et al.* (2001), de Almeida *et al.* (2010)
Hymenoptera	*Trichogramma brassicae*	Wasp	1) Heat treatment 2) *Wolbachia* PCR assay	Yes	Nazeri *et al.* (2015)
Hymenoptera	*Trichogramma brevicapillum*	Wasp	1) Antibiotic treatment 2) *Wolbachia* PCR assay (*wsp* primers)	Yes	Stouthamer & Werren (1993)
Hymenoptera	*Trichogramma chilonis*	Wasp	1) Antibiotic and heat treatments; 2) *Wolbachia* PCR assay (*ftsZ* primers)	Yes	Stouthamer *et al.* (1990); Stouthamer & Werren (1993); Schilthuizen & Stouthamer (1997)

Table 1 (*Continued*)

Host order	Host species	Common name	Approaches to verify	Reproductive polymorphism*	References
Hymenoptera	*Trichogramma cordubensis*	Wasp	1) Antibiotic and heat treatment; 2) *Wolbachia* PCR assay (*wsp* primers); 3) microscopy	No?	Cabello-García & Vargas-Piqueras (1985); Stouthamer & Werren (1993); Pintureau *et al.* (1999, 2002)
Hymenoptera	*Trichogramma deion*	Wasp	1) Antibiotic and heat treatment; 2) *Wolbachia* PCR assay (*wsp* primers)	Yes	Stouthamer *et al.* (1990); Stouthamer (1997); Stouthamer & Werren (1993); Stouthamer & Kazmer (1994); Van Meer *et al.* (1999); Pintureau *et al.* (2000)
Hymenoptera	*Trichogramma embryophagum*	Wasp	1) Antibiotic and heat treatments; 2) *Wolbachia* PCR assay (*wsp* primers); 3) microscopy	Yes	Stouthamer *et al.* (1990); Stouthamer & Werren (1993); Pintureau *et al.* (2000)
Hymenoptera	*Trichogramma evanescens*	Wasp	1) Antibiotic treatment; 2) *Wolbachia* PCR assay (*wsp* primers)	Yes	Grenier *et al.* (2002)
Hymenoptera	*Trichogramma kaykai*	Wasp	1) Antibiotic and heat treatments; 2) *Wolbachia* PCR assay (*wsp* primers)	Yes	Schilthuizen *et al.* (1998); Van Meer *et al.* (1999); Tulgetske (2010); Tulgetske & Stouthamer (2012)
Hymenoptera	*Trichogramma nr. deion*	Wasp	1) Antibiotic and heat treatment; 2) *Wolbachia* PCR assay (*wsp* primers)	Yes	Stouthamer *et al.* 1990; Stouthamer, 1997; Stouthamer & Werren (1993); Stouthamer & Kazmer (1994); Van Meer *et al.* (1999); Pintureau *et al.* (2000)
Hymenoptera	*Trichogramma oleae*	Wasp	1) Heat treatments; 2) *Wolbachia* PCR assay (*wsp* primers); 3) microscopy	No?	Stouthamer *et al.* (1993); Stouthamer & Werren (1993); Pintureau *et al.* (2000, 2002)
Hymenoptera	*Trichogramma platneri*	Wasp	1) Antibiotic and heat treatments; 2) *Wolbachia* PCR assay (*ftsZ* primers); 3) microscopy	Yes	Stouthamer *et al.* (1990); Stouthamer & Werren (1993); Schilthuizen & Stouthamer (1997)
Hymenoptera	*Trichogramma pretiosum*	Wasp	1) Antibiotic and heat treatments; 2) *Wolbachia* PCR assay (*wsp* primers)	Yes	Stouthamer *et al.* 1990; Stouthamer, 1997; Stouthamer & Werren (1993); Stouthamer & Kazmer (1994); Van Meer *et al.* (1999); Pintureau *et al.* (2000);Grenier *et al.* (2002)
Hymenoptera	*Trichogramma rhenana*	Wasp	1) Antibiotic and heat treatments; 2) *Wolbachia* PCR assay (*wsp* primers); 3) microscopy	Yes	Stouthamer *et al.* (1990); Stouthamer & Werren (1993); Pintureau *et al.* (2000)
Parthenogenesis induced by *Cardinium*					
Trombidiformes	*Brevipalpus californicus*	Mite	1) Antibiotic treatments; 2) *Cardinium* PCR assay (*gyrB* sequences) and bacterial community screening (16S rRNA)	No?	Groot & Breeuwer (2006)
Trombidiformes	*Brevipalpus obovatus*	Mite	1) Antibiotic treatments; 2) *Cardinium* PCR assay (*gyrB* sequences) and bacterial community screening (16S rRNA)	No?	Groot & Breeuwer (2006)
Trombidiformes	*Brevipalpus phoenicis*	Mite	1) Antibiotic treatments; 2) *Cardinium* PCR assay (*gyrB* sequences) and bacterial community screening (16S rRNA)	No?	Groot & Breeuwer (2006)
Hymenoptera	*Encarsia guadeloupae*	Wasp	1) Antibiotic treatment; 2) bacterial community screening (16S rDNA)	No	Giorgini *et al.* (2007)
Hymenoptera	*Encarsia hispida*	Wasp	1) Antibiotic treatment; 2) bacterial community screening 16S rDNA; 3) microscopy	No	Zchori-Fein *et al.* (2004); Giorgini *et al.* (2007, 2009)
Hymenoptera	*Encarsia pergandiella*	Wasp	1) Antibiotic treatment; 2) *Cardinium* PCR assay; 3) electron microscopy	No	Zchori-Fein *et al.* (2004); Giorgini *et al.* (2007); Gebiola *et al.* (2016)
Hymenoptera	*Plagiomerus diaspidis*	Wasp	1) Antibiotic treatment; 2) endosymbiont community screening; 3) Fluorescent *in situ* hybridization	No?	Gordh & Lacey (1976); Matalon *et al.* (2007)

Table 1 (*Continued*)

Host order	Host species	Common name	Approaches to verify	Reproductive polymorphism*	References
Parthenogenesis induced by *Rickettsia*					
Hymenoptera	*Neochrysocharis formosa*	Wasp	1) Antibiotic treatment; 2) endosymbiont community screening (16S rRNA)	Yes	Arakaki & Kinjo (1998); Hagimori *et al.* (2006); Adachi-Hagimori *et al.* (2008b), Adachi-Hagimori & Miura (2011)
Hymenoptera	*Pnigalio soemius*	Wasp	1) Antibiotic treatment; 2) endosymbiont community screening (16S rRNA); 3) Fluorescent *in situ* hybridization	Yes	Giorgini *et al.* (2010)
Parthenogenesis induced by undetermined/unknown endosymbiont					
Thysanoptera	*Aptinothrips rufus*	Thrips	1) Antibiotic treatment; 2) bacterial community screening (16S rRNA); *Wolbachia* PCR assay	Yes	Van der Kooi & Schwander (2014b); Fontcuberta García-Cuenca *et al.* (2016)
Hymenoptera	*Anagrus atomus*	Wasp	Antibiotic and heat treatments	Yes	Choudhury & Copland (2003)
Hymenoptera	*Aphelinus semiflavus*	Wasp	Heat treatment	Yes	Schlinger & Hall (1959)
Hymenoptera	*Aphytis mytilaspidis*	Wasp	Cytogenetics	Yes	Rössler & Debach (1972, 1973)
Hymenoptera	*Gilpinia hercyniae*	Wasp	Heat treatment	No?	Smith (1955)
Hymenoptera	*Encarsia meritoria*	Wasp	Antibiotic and heat treatment	Yes	Giorgini (2001)
Hymenoptera	*Galeopsomyia fausta*	Wasp	1) Antibiotic treatment; 2) *Wolbachia* PCR assay (*ftsZ* primers)	No?	Argov & Gottlieb (2000)
Hymenoptera	*Habrolepis rouxi*	Wasp	Antibiotic treatment	No?	Flanders (1945)
Hymenoptera	*Hexacola sp.* near *websteri*	Wasp	Heat treatment	No?	Eskafi & Legner (1974)
Hymenoptera	*Ooencyrtus fecundus*	Wasp	Heat treatment	No?	Laraichi (1978)
Hymenoptera	*Ooencyrtus submetallicus*	Wasp	Heat treatment	No?	Wilson & Woolcock (1960)
Hymenoptera	*Coccidoxenoides perminutus*	Wasp	Heat treatment	No?	Flanders (1965)
Hymenoptera	*Signiphora borinquensis*	Wasp	Antibiotic treatment	No?	Quezada *et al.* (1973)
Hymenoptera	*Trechnites insidiosus*	Wasp	Antibiotic treatment	No?	Braig *et al.* (2002)
Hymenoptera	*Trichogramma semifumatum*	Wasp	Heat treatment	Yes	Bowen & Stern (1966)

*Presence of sexual (uninfected) and parthenogenetic (infected) females in natural populations.

Table 2 Parthenogenesis in diplo-diploid species believed to be endosymbiont induced (no formally confirmed examples).

Host order	Host species	Common name	Sex determination	Endosymbiont identity	Approaches to verify	Reproductive polymorphism*	Reference
Psocoptera	*Liposcelis bostrychophila*	Booklouse	XX/XO	*Rickettsia*	Electron microscopy	No	Yusuf *et al.* (2000), Perotti *et al.* (2006)
Coleoptera	*Naupactus purpureoviolaceus*	Weevil	XX/XY	*Wolbachia* ?	*Wolbachia* PCR assay (multilocus sequence typing: *cosX*, *fbpA*, *ftsZ*, *gatB*, *hcpA*)	Yes?	Rodriguero *et al.* (2010a)
Coleoptera	*Naupactus tremolerasi*	Weevil	XX/XY	*Wolbachia* ?	*Wolbachia* PCR assay (multilocus sequence typing: *cosX*, *fbpA*, *ftsZ*, *gatB*, *hcpA*)	Yes?	Rodriguero *et al.* (2010a)
Coleoptera	*Pantomorus auripes*	Weevil	XX/XY	*Wolbachia* ?	*Wolbachia* PCR assay (multilocus sequence typing: *cosX*, *fbpA*, *ftsZ*, *gatB*, *hcpA*)	Yes?	Rodriguero *et al.* (2010a)
Psocoptera	*Cerobasis guestfalica*	Booklouse	XX/XO	*Wolbachia*	1) antibiotic and heat treatments; 2) endosymbiont community screening (16S rDNA); 3) microscopy	Yes?	Yusuf & Turner (2004)
Coleoptera	*Aramigus conirostris*	Weevil	XX/XY	*Wolbachia*	*Wolbachia* PCR assay (multilocus sequence typing: *cosX*, *fbpA*, *ftsZ*, *gatB*, *hcpA*)	Yes?	Rodriguero *et al.* (2010a)
Coleoptera	*Atrichonotus taeniatulus*	Weevil	XX/XY	*Wolbachia*	*Wolbachia* PCR assay (multilocus sequence typing: *cosX*, *fbpA*, *ftsZ*, *gatB*, *hcpA*)	Yes?	Rodriguero *et al.* (2010a)
Coleoptera	*Eurymetopus fallax*	Weevil	XX/XY	*Wolbachia*	*Wolbachia* PCR assay (multilocus sequence typing: *cosX*, *fbpA*, *ftsZ*, *gatB*, *hcpA*)	Yes?	Rodriguero *et al.* (2010a)
Coleoptera	*Eurymetopus globosus*	Weevil	XX/XY	*Wolbachia*	*Wolbachia* PCR assay (multilocus sequence typing: *cosX*, *fbpA*, *ftsZ*, *gatB*, *hcpA*)	Yes?	Rodriguero *et al.* (2010a)
Coleoptera	*Mimographus ocellatus*	Weevil	XX/XY	*Wolbachia*	*Wolbachia* PCR assay (multilocus sequence typing: *cosX*, *fbpA*, *ftsZ*, *gatB*, *hcpA*)	Yes?	Rodriguero *et al.* (2010a)
Coleoptera	*Naupactus condecoratus*	Weevil	XX/XY	*Wolbachia*	*Wolbachia* PCR assay (multilocus sequence typing: *cosX*, *fbpA*, *ftsZ*, *gatB*, *hcpA*)	Yes?	Rodriguero *et al.* (2010a)
Coleoptera	*Naupactus dissimilis*	Weevil	XX/XY	*Wolbachia*	*Wolbachia* PCR assay (multilocus sequence typing: *cosX*, *fbpA*, *ftsZ*, *gatB*, *hcpA*)	Yes?	Rodriguero *et al.* (2010a)
Coleoptera	*Naupactus leucoloma*	Weevil	XX/XY	*Wolbachia*	*Wolbachia* PCR assay (multilocus sequence typing: *cosX*, *fbpA*, *ftsZ*, *gatB*, *hcpA*)	Yes?	Rodriguero *et al.* (2010a)
Coleoptera	*Naupactus minor*	Weevil	XX/XY	*Wolbachia*	*Wolbachia* PCR assay (multilocus sequence typing: *cosX*, *fbpA*, *ftsZ*, *gatB*, *hcpA*)	Yes?	Rodriguero *et al.* (2010a)
Coleoptera	*Naupactus peregrinus*	Weevil	XX/XY	*Wolbachia*	*Wolbachia* PCR assay (multilocus sequence typing: *cosX*, *fbpA*, *ftsZ*, *gatB*, *hcpA*)	Yes?	Rodriguero *et al.* (2010a)
Coleoptera	*Naupactus verecundus*	Weevil	XX/XY	*Wolbachia*	*Wolbachia* PCR assay (multilocus sequence typing: *cosX*, *fbpA*, *ftsZ*, *gatB*, *hcpA*)	Yes?	Rodriguero *et al.* (2010a)
Coleoptera	*Pantomorus cinerosus*	Weevil	XX/XY	*Wolbachia*	*Wolbachia* PCR assay (multilocus sequence typing: *cosX*, *fbpA*, *ftsZ*, *gatB*, *hcpA*)	Yes?	Rodriguero *et al.* (2010a,b)
Coleoptera	*Pantomorus viridisquamosus*	Weevil	XX/XY	*Wolbachia*	*Wolbachia* PCR assay (multilocus sequence typing: *cosX*, *fbpA*, *ftsZ*, *gatB*, *hcpA*)	Yes?	Rodriguero *et al.* (2010a)
Hemiptera	*Aspidiotus nerii*	Scale insect	XX/XO ?	*Cardinium*	Endosymbiont community screening (16S rDNA)	Yes	Provencher *et al.* (2005)
Hemiptera	*Eucalymnatus tessellatus*	Scale insect	XX/XO	Unknown	Microscopy	Yes	Nur (1972)
Hemiptera	*Parthenolecanium cerasifex*	Scale insect	XX/XO	Unknown	Microscopy	Yes	Nur (1972)
Isotomidae	*Folsomia candida*	Springtail	XX/XO	*Wolbachia*	1) antibiotic treatments; 2) *Wolbachia* PCR assay (16S rDNA); 3) electron microscopy	Yes	Palévody (1973); Pike & Kingcombe (2009)

Table 2 (*Continued*)

Host order	Host species	Common name	Sex determination	Endosymbiont identity	Approaches to verify	Reproductive polymorphism*	Reference
Coleoptera	*Aramigus tessellatus*	Weevil	*XX/XY*	*Wolbachia*	*Wolbachia* PCR assay (multilocus sequence typing: *cosX*, *fbpA*, *ftsZ*, *gatB*, *hcpA*)	Yes	Normark (1996); Rodriguero *et al.* (2010a)
Coleoptera	*Asynonychus cervinus*	Weevil	*XX/XY*	*Wolbachia*	*Wolbachia* PCR assay (16S rDNA, *ftsZ*)	Yes	Rodriguero *et al.* (2010a,b)

*Presence of sexual (uninfected) and parthenogenetic (infected) females in natural populations.

parthenogenesis is interesting as it appears to be much rarer in species with other causes of parthenogenesis. Although there are currently no estimates available for reproductive polymorphism in this case, data on mostly diplo-diploid species from Suomalainen *et al.* (1987) suggest that only 11% of species without endosymbiont-induced parthenogenesis maintain reproductive polymorphism. However, as our database only comprises species with endosymbiont-induced parthenogenesis and we did not generate a list of species with genetic causes of parthenogenesis, the estimates extracted from Suomalainen *et al.* (1987) should be interpreted with caution. Nevertheless, given that 50% of species with endosymbiont-induced parthenogenesis also feature sexual strains, it is clear that there must be mechanisms facilitating the maintenance of polymorphism. Many studies have addressed mechanisms that can contribute to the maintenance of sex in general (e.g. reviewed by Bell, 1982; Agrawal, 2006; Otto, 2009; Lively & Morran, 2014), but we speculate that three hypotheses may explain reproductive polymorphism specifically in endosymbiont-induced parthenogenesis.

First, most cases of confirmed endosymbiont-induced parthenogenesis could be very recent, such that the infection has not invaded all populations yet. In this case, reproductive polymorphism would be transient, and either sexual or parthenogenetic populations may eventually disappear. Unfortunately, this hypothesis cannot be evaluated, as there are no age estimates available for lineages with endosymbiont-induced parthenogenesis.

Second, reproductive polymorphism in endosymbiont-induced parthenogenesis could be maintained, in rare cases, because of resistance mechanisms evolving in the host population during the spread of the initial infection (Stouthamer *et al.*, 2010). The evolution of such mechanisms requires that parthenogenetic and sexual strains are not fully reproductively isolated and that 'parthenogenetic' females are able to facultatively produce offspring sexually upon mating with males. Indeed, endosymbiont transmission to eggs is typically $< 100\%$ (Haine *et al.*, 2005) and can be as low as 10% (e.g. in the genus *Trichogramma*, Stouthamer *et al.*, 2010). Eggs that remain endosymbiont free could thus be fertilized and develop into sexual females. During the spread of a parthenogenesis-inducing endosymbiont in a population, the sex ratio becomes increasingly female-biased. Females with resistance mechanisms against endosymbionts would continue to produce sons, which would have many opportunities to father offspring in a female-biased population. As a consequence, resistance mechanisms would be favoured and polymorphism maintained, at least transiently (Stouthamer *et al.*, 2001, 2010; Jeong & Stouthamer, 2005). Among species with endosymbiont-induced parthenogenesis, facultative sex has only been documented in the

parasitoid wasp genus *Trichogramma* (Stouthamer *et al.*, 2001; Huigens, 2003; Van Vugt *et al.*, 2003), but the finding of gene flow between parthenogenetic and sexual strains in some species (e.g. *Tetrastichus*, Reumer *et al.*, 2013) suggests it could be more widespread. The evolution of resistance against infection is also only known in *Trichogramma*, where two resistance mechanisms have been described (Stouthamer *et al.*, 2001; Huigens, 2003; Van Vugt *et al.*, 2003). In *T. kaykai*, a B chromosome solely transmitted by males eliminates the maternal genome from fertilized (i.e. female) eggs and thereby converts female eggs into male eggs (Stouthamer *et al.*, 2001; Van Vugt *et al.*, 2003). In *T. deion*, a nuclear parthenogenesis suppressor gene inducing male production in infected females is hypothesized to maintain male production, but the exact mechanism currently remains unknown (Huigens, 2003). In both cases, reproductive polymorphism of sexual and facultatively parthenogenetic females can thus be maintained via negative frequency-dependent selection, given the disproportionate reproductive success of males in female-biased populations.

Finally, a third possible explanation for the maintenance of reproductive polymorphism is that under endosymbiont-induced parthenogenesis, reversals from parthenogenetic to sexual reproduction may occur upon losses of the infection, for example because of incomplete endosymbiont transmission. One or several losses of infection can thus regenerate sexual lineages (and reproductive polymorphism) in fully asexual populations. By contrast, parthenogenesis to sex reversals is less likely (or perhaps impossible) under spontaneous mutations and hybrid origins (Van der Kooi & Schwander, 2014a).

Another factor that can contribute to the maintenance of reproductive polymorphism and that is widespread among species with genetically determined parthenogenesis is geographic parthenogenesis. Species with genetically determined parthenogenesis typically feature distributions at higher latitudes in Northern Hemisphere and higher altitudes than their sexual relatives (e.g. Glesener & Tilman, 1978; Beaton & Hebert, 1988; Haag & Ebert, 2004; Hörandl, 2009; Verhoeven & Biere, 2013). Geographic parthenogenesis can be explained by several, mutually nonexclusive mechanisms that have been the focus of previous reviews (e.g. Glesener & Tilman, 1978; Lynch, 1984; Hörandl, 2009; Tilquin & Kokko, 2016). These mechanisms include, for example, reduced biotic interactions and selection for reproductive insurance at high latitudes and altitudes, avoidance of outbreeding depression in sink populations and frequent transitions to parthenogenesis in marginal habitats. However, whether similar distribution differences also exist in the case of endosymbiont-induced parthenogenesis is not known (Tilquin & Kokko, 2016). Tilquin & Kokko (2016) speculated that distributions of sexual and parthenogenetic strains may be more similar in the case of endosymbiont-induced parthenogenesis than under genetically determined parthenogenesis where parthenogens are often polyploid and/or of hybrid origin. We searched for information on the geographic distributions of sexual and parthenogenetic strains for the 27 haplo-diploid species with reproductive polymorphism and confirmed endosymbiont-induced parthenogenesis (Table S3). We found that the distribution differences between sexual and parthenogenetic strains for endosymbiont-induced cases match those for genetically determined cases, with parthenogens featuring ranges shifted towards higher latitudes (Fig. S1; we did not obtain sufficient information on altitudinal distributions). This suggests that geographic parthenogenesis is strongly driven by sexual vs. asexual reproduction *per se* and not by hybrid ancestry or polyploidy of parthenogens.

Taxonomic diversity of parthenogenesis-inducing endosymbionts and their hosts

All currently known cases of endosymbiont-induced parthenogenesis are caused by bacteria. However, there is no *a priori* reason for such a taxonomic restriction. Vertically transmitted viral endosymbionts, such as sigma viruses, are suggested to be common in insects (Longdon *et al.*, 2010, 2011; Longdon & Jiggins, 2012). Similarly, vertically transmitted protozoans are described for a range of species, including termites and wood-feeding cockroaches (e.g. Ohkuma, 2008). It is thus possible (or perhaps even likely) that the sole reason for why all known cases of endosymbiont-induced parthenogenesis involve bacteria is that these cases are easy to detect (e.g. by removal of the endosymbionts via antibiotic treatment, see below).

Similar to the taxonomic restriction for the endosymbionts, endosymbiont-induced parthenogenesis is only confirmed in host taxa with haplo-diploid sex determination; all 54 confirmed examples of endosymbiont-induced parthenogenesis are in haplo-diploid hosts, as well as 46 of the 70 speculative cases (Table S1). Currently, only a small number of species with other sex determination mechanisms (19%, 24 of 124) are believed to have endosymbiont-induced parthenogenesis (Tables 2 and S1). However, the strong overrepresentation of haplo-diploid species among species with known or suspected endosymbiont-induced parthenogenesis is most likely due to an ascertainment bias and may not necessarily represent a biological pattern.

Indeed, endosymbiont-induced parthenogenesis is typically easy to reveal in haplo-diploid taxa. By contrast, it is difficult or often impossible to formally demonstrate endosymbiont-induced parthenogenesis in taxa with diplo-diploid sex determination systems. In haplo-diploid species, curing parthenogenetic females of their endosymbionts by antibiotics or heat treatment usually leads to the production of haploid sons instead

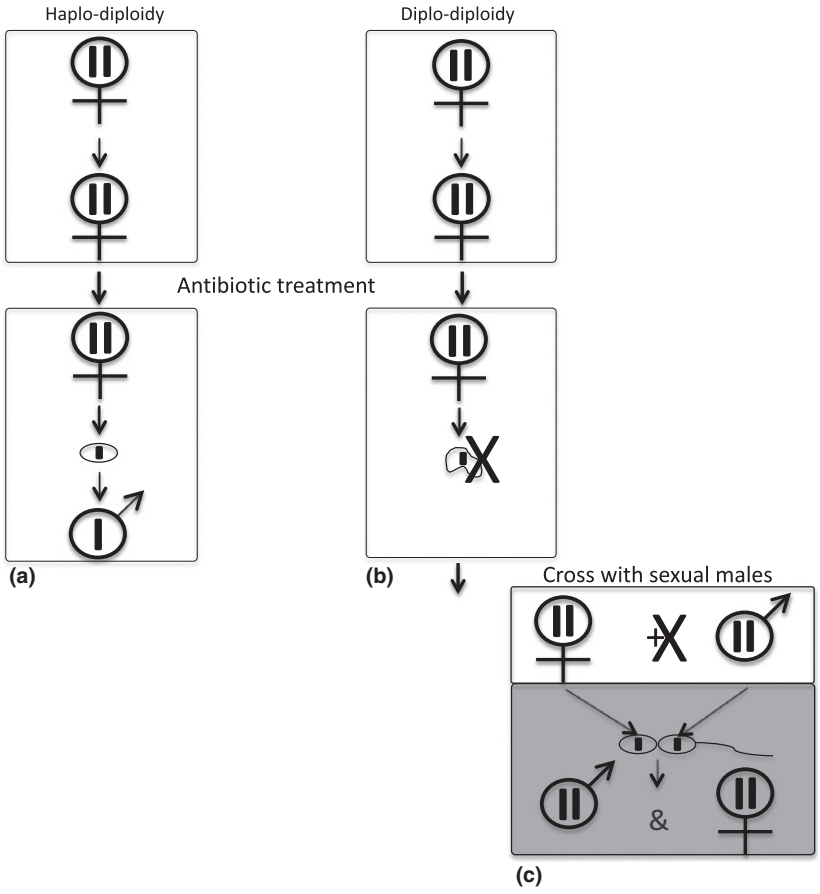

Fig. 1 Consequences of endosymbiont removal under (a) haplo-diploidy and (b) diplo-diploidy in species with bacteria-induced parthenogenesis. Under haplo-diploid sex determination, endosymbiont removal leads to females producing haploid males, similar to virgin sexual females (a). Following the same treatment, no offspring are produced by diplo-diploid females, because haploid eggs do not develop (b). An important step in diplo-diploid species is to allow cured parthenogenetic females to mate with sexual males. If they produce both female and male offspring upon mating, direct evidence for endosymbiont-induced parthenogenesis in a diplo-diploid species could be obtained (c, in grey).

of diploid daughters (Fig. 1a), providing direct evidence for the role of endosymbionts in causing parthenogenesis in their hosts. This approach was used for the first discovery of endosymbiont-induced parthenogenesis (Stouthamer *et al.*, 1990), and has since then been applied to reveal endosymbiont-induced parthenogenesis in haplo-diploid species from diverse taxonomic groups including thrips, mites and many different species of wasps (see Tables 1 and S1).

Removal of endosymbionts via heat or antibiotic treatment in parthenogenetic females of diplo-diploid species can also provide useful information on the cause of parthenogenesis. If parthenogenesis has genetic causes and is not endosymbiont induced, one would expect treated females to continue producing diploid eggs that may or may not be viable (as a consequence of the treatment, see below). Alternatively, parthenogenetic females in diplo-diploid species may lay haploid eggs upon treatment, suggesting endosymbionts as the cause of parthenogenesis. In diplo-diploid species, haploid eggs however do not develop further as they have to be fertilized to generate sons or daughters. Endosymbiont-cured females should therefore be mated to males of a sexual strain (Fig. 1c). However, key sexual traits, and especially the ability to fertilize eggs, are typically decayed in parthenogenetic females (Van der Kooi & Schwander, 2014a), such that crosses between cured parthenogenetic females and males of sexual strains may often not yield any offspring from fertilized eggs. Removing endosymbionts from females in diplo-diploid species with endosymbiont-induced parthenogenesis can therefore result in the production of nonviable eggs or no eggs at all (Fig. 1b). Such a phenotype alone cannot be interpreted as endosymbiont-induced parthenogenesis because it is equally likely that endosymbionts are required for successful oogenesis in the absence of parthenogenesis. For example, the requirement for endosymbiont infection for successful oogenesis is well documented in the sexual wasp species *Asobara tabida*. Females of this species cannot produce viable oocytes if cured of their endosymbionts (Dedeine *et al.*, 2001). A similar case of dependence on endosymbionts for the production of viable oocytes is known in parthenogenetic *Folsomia candida*, a diplo-diploid species (Pike & Kingcombe, 2009; Timmermans & Ellers, 2009). *F. candida* females cured of their endosymbionts solely produce nonviable eggs; thus, it is impossible to distinguish whether the lack of egg viability is because of endosymbiont-induced parthenogenesis or because oogenesis in this species is dependent on endosymbiont infection as

in *A. tabida*. Matings of cured *F. candida* females with sexual males have thus far not been documented. Nevertheless, endosymbionts primarily occupy the cytoplasmic asters in eggs from parthenogenetic *F. candida* females, probably as a replacement for paternally provided centrioles needed for centrosome formation in sexual species (Riparbelli *et al.*, 2006). These findings strongly support the hypothesis that parthenogenesis in *F. candida* is endosymbiont-induced, making *F. candida* the currently strongest case for endosymbiont-induced parthenogenesis in a diplo-diploid species.

It is worth noting that endosymbiont-induced parthenogenesis in haplo-diploid species is very similar to another host phenotype induced by some endosymbionts, feminization. In this case, endosymbionts feminize genotypic males (for example with a XY karyotype) to develop as females, thereby increasing their transmission to future generations (Werren, 1997; Werren *et al.*, 2008). Endosymbiont-induced feminization is well described in woodlice, but is also known in mites, shrimp, butterflies and moths (reviewed by Kageyama *et al.*, 2012). The difference between parthenogenesis induction and feminization is that parthenogenesis generates daughters from unfertilized eggs whereas feminization generates daughters from fertilized eggs and therefore requires mating. In haplo-diploid species, genetic males are haploid and are produced without mating such that feminization and parthenogenesis induction are essentially indistinguishable. For example, in parthenogenetic spider mites (*Brevipalpus phoenicis, B. californicus, B. obovatus*), endosymbionts feminize unfertilized haploid eggs that consequently develop into females instead of males (Groot & Breeuwer, 2006). In other haplo-diploid parthenogenetic species, endosymbionts both diploidize and feminize unfertilized haploid eggs (Table 1, see also Ma *et al.*, 2015). These examples illustrate that although the distinction between parthenogenesis induction and feminization is clear in diplo-diploid species, the two phenotypes often overlap in haplo-diploid species.

Parthenogenesis-inducing endosymbionts

Bacteria from three different genera, namely *Wolbachia*, *Cardinium* and *Rickettsia,* are currently known to induce parthenogenesis in a subset of the host species they infect. Bacteria of a fourth, recently described genus (*Xiphinematobacter*), also occur in parthenogenetic hosts and it was suggested they may be involved in reproductive manipulation (Coomans & Cleys, 1998; Vandekerckhove *et al.*, 2000). However, it was later discovered that this symbiont is probably a nutritional mutualist, as it is mostly found in the gut and also in occasionally produced males (Brown *et al.*, 2015; Palomares-Rius *et al.*, 2016). Among the three bacterial genera *Wolbachia*, *Cardinium* and *Rickettsia*, *Wolbachia* appears to be the most frequent cause of parthenogenesis (but see discussion below on why *Wolbachia*-induced parthenogenesis is overestimated). For the 54 confirmed host species with endosymbiont-induced parthenogenesis, 56% (30 cases) are caused by *Wolbachia*, 13% (seven cases) by *Cardinium*, 4% (two cases) by *Rickettsia* and the remaining 28% (15 cases) by different, currently unknown/undescribed, bacterial endosymbionts (Table 1). Among the 70 host species where endosymbiont-induced parthenogenesis is speculative, *Wolbachia* is also the most widespread (86–90%; 60–63 of 70 cases), with only one case for *Cardinium*, three cases for *Rickettsia* and three cases for unknown endosymbiont species (Tables 2 and S1). In combination, these data suggest that in 56–75% of hosts with endosymbiont-induced parthenogenesis, the endosymbiont is *Wolbachia*, *Cardinium* in 6–13% of hosts, *Ricksettsia* in 4% and other (unidentified) bacteria in 15–28%. Unfortunately, these estimates remain speculative as the data available from the literature are most likely strongly biased. Many studies solely test for *Wolbachia* infection, without considering other endosymbionts as the cause of parthenogenesis. The implications are that even the 56% *Wolbachia*-induced parthenogenesis among confirmed examples of endosymbiont-induced parthenogenesis are most likely an overestimation. Indeed, over the past decade the estimated frequency of *Wolbachia*-induced parthenogenesis has already decreased considerably; in 2003, as many as 85% cases of endosymbiont-induced parthenogenesis were believed to be caused by *Wolbachia* (Huigens & Stouthamer, 2003) and the estimates will likely change with further studies.

Methods to reveal parthenogenesis induced by bacterial endosymbionts

Three different approaches have been used to study bacteria-induced parthenogenesis. First, as discussed above, bacteria-induced parthenogenesis is revealed via antibiotic and/or heat treatments that remove endosymbionts and cause parthenogenetic females to produce sons from haploid eggs (in haplo-diploid species; see Table 1 for examples) or from fertilized eggs (in diplo-diploid species; currently no examples).

Second, a characterization of the endosymbiont community can help identify the endosymbiont causing parthenogenesis. Typically, tests for endosymbionts only cover bacteria known to induce parthenogenesis, usually *Wolbachia*, *Cardinium* and *Ricksettia*. Restricting screens to these specific bacteria impedes the discovery of other endosymbionts capable of host reproductive manipulation. It may further lead to false conclusions under multiple infections with different endosymbionts. For example, an undescribed bacteria may induce parthenogenesis in a host that is also infected by a widespread strain of *Wolbachia*, with *Wolbachia* causing

a different, or no, phenotype in the host. More generally, the challenge for endosymbiont community screens is to distinguish between endosymbionts involved in reproductive manipulation and those involved in other processes, for example protecting hosts from parasites and parasitoids (e.g. Vorburger *et al.*, 2009; Hamilton & Perlman, 2013) or providing nutritional benefits (e.g. Hosokawa *et al.*, 2010). Useful information can be obtained from the physical location of the endosymbionts via microscopy – reproductive manipulators are associated with host ovaries, whereas endosymbionts providing nutritional benefits are associated with the digestive system (e.g. reviewed by Dillon & Dillon, 2004). Notably, the original description of *Wolbachia* as a manipulator of host reproduction was linked to the bacteria being clustered around the ovaries (Stouthamer & Werren, 1993). A complementary approach could involve a comparison of bacterial communities from multiple, geographically separate parthenogenetic strains with bacterial communities from different sexual strains. This can allow identifying bacteria specific to parthenogenetic strains independently of geography.

Finally, a direct approach to identifying the endosymbiont(s) causing parthenogenesis would be to cure hosts of their endosymbionts and re-infect them with individual endosymbiont strains. Unfortunately, isolation and *in vitro* culture of endosymbionts are often very laborious (Fenollar *et al.*, 2003; Dale *et al.*, 2006; Perotti *et al.*, 2006; Rasgon *et al.*, 2006) or even impossible. Given the practical constraints that impede controlled infections, an alternative approach is to modify the relative titres of different endosymbionts with specific narrow-spectrum antibiotics or specific antibiotic concentrations. For example, the sexual wasp species *Asobara tabida* is infected by three different strains of *Wolbachia*. One strain is required for host oogenesis and the other two strains induce cytoplasmic incompatibility. It was possible to reveal these different phenotypes by removing individual *Wolbachia* strains with different antibiotic concentrations (Dedeine *et al.*, 2004; Mouton *et al.*, 2004).

Many unconfirmed cases of endosymbiont-induced parthenogenesis

Unfortunately, the conclusion of endosymbiont-induced parthenogenesis in a host is often based on little evidence. For example, *Wolbachia*-induced parthenogenesis was suggested in at least 63 parthenogenetic species solely because they are infected with *Wolbachia* (listed as speculative cases in Tables 2 and S1). However, *Wolbachia* infection is very widespread among arthropods, with 40–66% of described species infected (Hilgenboecker *et al.*, 2008; Zug & Hammerstein, 2012). As mentioned above, *Wolbachia* does not always have detectable phenotypic effects on its host (e.g. Vavre *et al.*, 2002) and even if it does, reproductive manipulation is only one of many possible effects. As a consequence, the presence of *Wolbachia* in a parthenogenetic host does not indicate its involvement in parthenogenesis.

Another frequent shortcut is the screening of a single parthenogenetic host strain instead of multiple strains. In many species that feature *Wolbachia* infection, only a subset of individuals is actually infected (typically $< 10\%$ or $> 90\%$ of individuals; Hilgenboecker *et al.*, 2008; Zug & Hammerstein, 2012). In the case of endosymbiont-induced parthenogenesis, this may mean that not *Wolbachia*, but a different endosymbiont is causing parthenogenesis in its host. For example, in the grass thrips *Aptinothrips rufus*, endosymbiont-induced parthenogenesis was revealed by antibiotic treatment (Van der Kooi & Schwander, 2014b). Subsequent genetic screening of parthenogenetic females from different populations revealed that only 69% of them were infected by *Wolbachia*. This indicates that parthenogenesis is either induced by another (non-*Wolbachia*) bacteria or that *Wolbachia* was lost after parthenogenesis was genetically fixed in some host lineages.

Mechanisms of parthenogenesis and mechanisms of endosymbiont-induced parthenogenesis

In sexual species, females produce haploid oocytes which are fertilized by sperm and develop as diploid individuals. In parthenogenetic species, females produce oocytes that develop as diploid (or in some cases polyploid) daughters without fertilization via different cellular mechanisms (Suomalainen *et al.*, 1987). Importantly, there is no apparent phylogenetic pattern with respect to which species uses which cellular mechanism of parthenogenesis and closely related species may employ different mechanisms of parthenogenesis (Suomalainen *et al.*, 1987). The cellular mechanisms underlying parthenogenesis have variable consequences on heterozygosity, as well described by Suomalainen *et al.* (1987). Notably, barring effects of gene conversion (e.g. Tucker *et al.*, 2013), heterozygosity is fully maintained under mitotic and certain forms of meiotic parthenogenesis. Under mitotic parthenogenesis, there are no meiotic divisions and offspring are genetically identical to their mother (Fig. 2a, also referred to as clonal reproduction or 'apomixis'; Suomalainen *et al.*, 1987). Under meiotic parthenogenesis, meiosis is maintained partly or entirely (see Fig. 2b through f). Three types of meiotic parthenogenesis result in offspring being identical to their mother, that is, parthenogenesis is 'functionally' mitotic even though meiosis is present. First, under 'endoduplication', a duplication of the entire chromosome set precedes a normal meiosis. During meiosis, ploidy is then reduced again, leading to the ploidy level present prior to chromosome duplication (Fig. 2c). Offspring are genetically identical to their

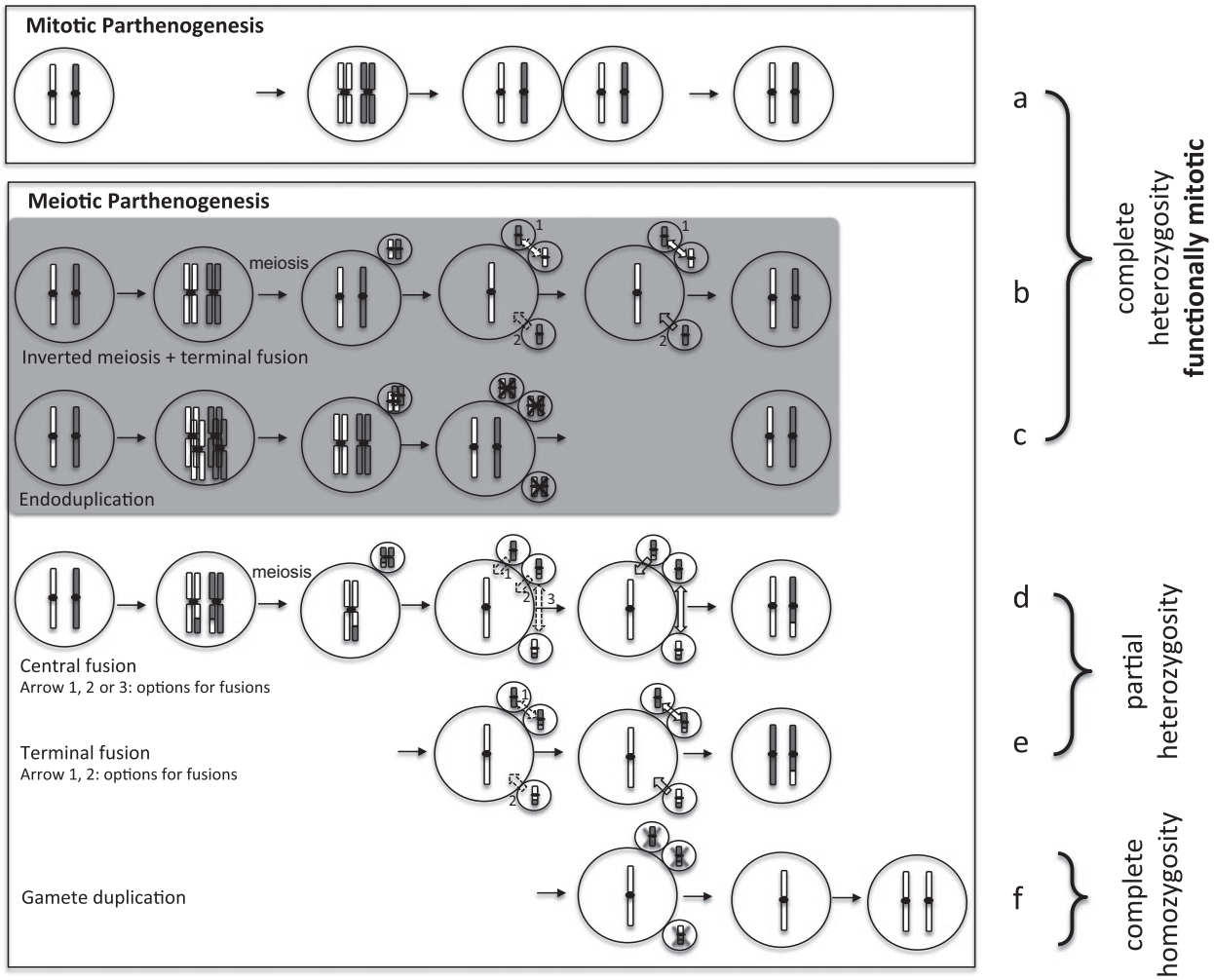

Fig. 2 Different cellular mechanisms of parthenogenesis. Cellular mechanisms currently not known for endosymbiont-induced parthenogenesis are highlighted in dark grey. Homologous chromosomes are represented by grey and white bars; oocytes are represented by large chromosome-containing circles and polar bodies by small chromosome-containing circles. 'X' indicates that a polar body degenerates. Under mitotic parthenogenesis, offspring are genetically identical to their mother and thus retain heterozygosity (a). The same is true for inverted meiosis with terminal fusion or endoduplication (b, c). For inverted meiosis with terminal fusion (b), central fusion (d), terminal fusion (e) and gamete duplication (f), recombination can generate new allele associations (small white and grey chromosome segments). Figure is modified from Neiman *et al.*, 2014. Drawings are not to scale.

mothers because meiotic recombination occurs between two identical chromosome copies (Suomalainen *et al.*, 1987). Second, heterozygosity is maintained under inverted meiosis and terminal fusion (sometimes referred to as 'gonoid thelytoky' in the literature). Inverted meiosis means that during the first meiotic division, instead of the separation of chromosome homologues in normal meiosis, the sister chromatids of each chromosome are separated; the second meiotic step is then the separation of chromosomes (Feiertag-Koppen, 1980) (Fig. 2b). Interestingly, this form of meiosis seems to occur solely in species with holocentric chromosomes (e.g. in sedges, Cabral *et al.*, 2014 or in oribatid mites, Taberly, 1987, 1988). Finally, if recombination is suppressed, 'central fusion' also generates offspring that are clones of their mother (Fig. 2d). However, if recombination occurs, central fusion (and terminal fusion under inverted meiosis) results in a loss of heterozygosity between generations. This is because ploidy restoration occurs via fusion of meiotic products which are derived from a single mother cell. The extent of heterozygosity loss depends on which meiotic products fuse (terminal and central fusions; Suomalainen *et al.*, 1987; see Fig. 2d,e for details). Finally, complete homozygosity is attained within one generation in the case where an egg with a haploid genome undergoes genome duplication ('gamete duplication', Fig. 2f).

Endosymbiont-induced parthenogenesis could in principle occur via any of the above-described cellular mechanisms of parthenogenesis. However, currently only a subset of cellular mechanisms are known for endosymbiont-induced parthenogenesis. The cellular mechanisms of endosymbiont-induced parthenogenesis have been studied in 19 species (Table 3). Among these, endosymbionts induce central fusion in three (*Encarsia pergandiella*, *E. hispida*, *E. guadeloupe*), and terminal fusion in one species (*Aphytis mytilaspidis*). In 11 of the 19 species (57.8%), parthenogenesis occurs via gamete duplication. In the remaining four species where cellular mechanisms were studied, gamete duplication could be excluded as the mechanism of parthenogenesis because of heterozygosity patterns, but the exact cellular mechanism is not known. In one of these species (*Aptinothrips rufus*, Table 3), gamete duplication can be excluded because individuals are heterozygous at several loci but nothing further can be deduced about the cellular mechanisms underlying parthenogenesis. In the three remaining species (*Bryobia praetiosa, Neochrysocharis formosa* and *Folsomia candida*), parthenogenesis is functionally mitotic (heterozygosity is maintained between generations), but the detailed mechanism has not been studied (Table 3).

The high frequency of gamete duplication in endosymbiont-induced parthenogenesis is interesting as it is rare among species with genetically determined parthenogenesis. For example, among the parthenogenetic species discussed by Suomalainen *et al.* (1987), only 2 to 4% of (diplo-diploid) species with genetically caused parthenogenesis are characterized by gamete duplication (4 to 10 of 242 species, Table S2). Gamete duplication in general is probably rare because it is associated with low fitness in females of incipient parthenogenetic lineages, given it causes genomewide homozygosity and therefore results in the expression of the full genetic load. The genetic load is usually reduced in haplo-diploids because recessive deleterious alleles can be purged if expressed in haploid males (Blackmon *et al.*, 2015; Tien *et al.*, 2015). However, considering only species with haplo-diploid sex determination (given all confirmed cases of endosymbiont-induced parthenogenesis are in haplo-diploids), there is not a single species (of 17) known that features genetically caused parthenogenesis via gamete duplication (Table S4). Gamete duplication might then be frequent in endosymbiont-induced parthenogenesis because gamete duplication would be mechanistically easy to induce by endosymbionts. In other words, parthenogens with gamete duplication would generally be disfavoured relative to parthenogens with other cellular mechanisms, but the proportion of incipient (novel) parthenogens with gamete duplication would be much higher in endosymbiont-induced cases than in species where parthenogenesis evolved via spontaneous mutations or hybridization.

It is also interesting to compare cellular mechanisms of parthenogenesis induced by different endosymbionts. Notably, *Wolbachia* infection causes parthenogenesis via gamete duplication in nine cases, via functional mitosis in two cases, and in one case via an unknown mechanism that is not gamete duplication (Table 3). In the single known case of *Rickettsia,* parthenogenesis occurs via mitotic parthenogenesis. *Cardinium* causes central fusion in all three studied host species (but note that these all belong to the genus *Encarsia*). Finally, for the three hosts with unknown/undescribed endosymbionts, two feature gamete duplication (*Eucalymantus tessellatus, Parthenolecanium cerasifex*) and one terminal fusion (*Aphytis mytilaspidis*, Table 3).

It would be interesting to know the cellular mechanisms of parthenogenesis in additional species with endosymbiont-induced parthenogenesis as such knowledge may help develop insights into the mechanisms through which endosymbionts interfere with host reproduction. Given the sparse data, it is currently impossible to conclude that certain endosymbionts cause certain types of parthenogenesis or that there is an interaction between host taxon and endosymbiont. Indeed for the majority of species with suspected or confirmed endosymbiont-induced parthenogenesis (105 cases of 124 species, 85%), the cellular mechanisms of parthenogenesis are completely unknown (Table S1).

The cellular mechanism of parthenogenesis can be investigated using cytological studies and/or genetic markers. Cytological studies are by far the most precise way to identify parthenogenesis mechanisms. However, it is often time-consuming and technically difficult to perform such studies. Genotyping mothers and their offspring at multiple loci is a much simpler approach and can provide insights into the mechanisms of parthenogenesis depending on the maintenance or loss of heterozygosity between generations. Unfortunately, it typically does not allow for identification of the exact mechanism because often too few loci are used and their genomic locations are unknown. Recently, Svendsen *et al.* (2015) developed a new method by mapping a high density of markers on individual chromosomes. Because the rate of transition from heterozygosity to homozygosity between generations should vary among loci depending on the type of parthenogenesis but also depending on how close they are to the centromere, mapping heterozygosity along chromosome arms allows identifying the exact mechanism of parthenogenesis. This method is especially powerful as it also allows for the identification of mixed parthenogenesis (e.g., some offspring being produced by central and others by terminal fusion; Svendsen *et al.*, 2015).

Table 3 Mechanisms of endosymbiont-induced parthenogenesis (species in which endosymbiont-induced parthenogenesis is suspected but not confirmed are highlighted in grey).

Host order	Host species	Common name	Sex determination	Endosymbiont	Approaches to verify	Mechanism	Method for mechanism	References
Trombidiformes	*Bryobia praetiosa*	Mite	Haplo-diploidy	*Wolbachia*	1) Antibiotic treatment; 2) *Wolbachia* PCR assay (*ftsZ* primers)	Functional apomixis	Genetic markers (microsatellite loci)	Weeks & Breeuwer (2001)
Thysanoptera	*Aptinothrips rufus*	Thrips	Haplo-diploidy	Undescribed bacterium or *Wolbachia*	1) Antibiotic treatments; 2) bacterial community screening (16S rRNA); *Wolbachia* PCR assay	? (Not gamete duplication)	Genetic markers (microsatellite loci)	Van der Kooi & Schwander (2014b); Fontcuberta García-Cuenca *et al.* (2016)
Hymenoptera	*Leptopilina clavipes*	Wasp	Haplo-diploidy	*Wolbachia*	1) Antibiotic treatment; 2) *Wolbachia* PCR assay (*ftsZ* primers)	Gamete duplication	Cytogenetic approach	Werren *et al.* (1995); Pannebakker *et al.* (2004a,b, 2005)
Hymenoptera	*Muscidifurax uniraptor*	Wasp	Haplo-diploidy	*Wolbachia*	1) Antibiotic treatment; 2) *Wolbachia* PCR assay (*wsp* primers)	Gamete duplication	Cytogenetic approach	Stouthamer *et al.* (1993); Gottlieb & Zchori-Fein (2001), Gottlieb *et al.* (2002)
Hymenoptera	*Encarsia formosa*	Wasp	Haplo-diploidy	*Wolbachia*	1) Antibiotic treatment; 2) *Wolbachia* PCR assay (*wsp* primers)	Gamete duplication	Cytogenetic approach	Zchori-Fein *et al.* (1991); Hunter (1999); Giorgini *et al.* (2007)
Hymenoptera	*Trichogramma deion*	Wasp	Haplo-diploidy	*Wolbachia*	1) Antibiotic and heat treatment; 2) *Wolbachia* PCR assay (*wsp* primers)	Gamete duplication	Cytogenetic approach and genetic markers	Stouthamer *et al.* 1990; Stouthamer, 1997; Stouthamer & Werren (1993); Stouthamer & Kazmer (1994); Van Meer *et al.* (1999); Pintureau *et al.* (2000)
Hymenoptera	*Trichogramma nr. deion*	Wasp	Haplo-diploidy	*Wolbachia*	1) Antibiotic and heat treatment; 2) *Wolbachia* PCR assay (*wsp* primers)	Gamete duplication	Cytogenetic approach and genetic markers	Stouthamer *et al.* 1990; Stouthamer, 1997; Stouthamer & Werren (1993); Stouthamer & Kazmer (1994); Van Meer *et al.* (1999); Pintureau *et al.* (2000)
Hymenoptera	*Trichogramma pretiosum*	Wasp	Haplo-diploidy	*Wolbachia*	1) Antibiotic and heat treatments; 2) *Wolbachia* PCR assay (*wsp* primers)	Gamete duplication	Cytogenetic approach and genetic markers	Stouthamer *et al.* (1990); Stouthamer & Werren (1993); Stouthamer & Kazmer (1994); Van Meer *et al.* (1999); Pintureau *et al.* (2000)
Hymenoptera	*Encarsia pergandiella*	Wasp	Haplo-diploidy	*Cardinium*	1) Antibiotic treatment; 2) *Cardinium* PCR assay; 3) electron microscopy	Central fusion	Cytogenetic approach	Zchori-Fein *et al.* (2004); Giorgini *et al.* (2007)

Table 3 (*Continued*)

Host order	Host species	Common name	Sex determination	Endosymbiont	Approaches to verify	Mechanism	Method for mechanism	References
Hymenoptera	*Encarsia hispida*	Wasp	Haplo-diploidy	*Cardinium*	1) Antibiotic treatment; 2) bacterial community screening 16S rDNA; 3) microscopy	Central fusion	Cytogenetic approach	Zchori-Fein *et al.* (2004); Giorgini *et al.* (2007, 2009)
Hymenoptera	*Encarsia guadeloupae*	Wasp	Haplo-diploidy	*Cardinium*	1) Antibiotic treatment; 2) bacterial community screening (16S rDNA)	Central fusion	Cytogenetic approach	Giorgini *et al.* (2007)
Hymenoptera	*Neochrysocharis formosa*	Wasp	Haplo-diploidy	*Rickettsia*	1) Antibiotic treatment; 2) endosymbiont community screening (16S rRNA)	Apomixis	Cytogenetic approach and genetic markers	Arakaki & Kinjo (1998); Hagimori *et al.* (2006); Adachi-Hagimori *et al.* (2008a)
Hymenoptera	*Aphytis mytilaspidis*	Wasp	Haplo-diploidy	Unknown	Cytogenetics	Terminal fusion	Cytogenetic approach, genetic markers	Rössler & Debach (1972, 1973)
Hymenoptera	*Diplolepis rosae*	Wasp	Haplo-diploidy	*Wolbachia*	*Wolbachia* PCR assay (*ftsZ* primers)	Gamete duplication	Cytogenetic approach	Stille and Därvring (1980); van Meer *et al.* (1999)
Hymenoptera	*Tetrastichus coeruleus*	Wasp	Haplo-diploidy	*Wolbachia*	*Wolbachia* PCR assay (*gatB, coxA, hcpA, ftsZ* and *fbpA* primers)	Gamete duplication	None	Reumer *et al.* (2010, 2013)
Hymenoptera	*Diplolepis spinosissimae*	Wasp	Haplo-diploidy	*Wolbachia*	*Wolbachia* PCR assay (*ftsZ* primers)	Gamete duplication	Genetic markers	Plantard *et al.* (1998)
Isotomidae	*Folsomia candida*	Springtail	XX/XO	*Wolbachia*	1) antibiotic treatments; 2) *Wolbachia* PCR assay (16S rDNA); 3) electron microscopy	Terminal fusion	Cytogenetic approach	Palévody (1973); Vandekerckhove *et al.* (1999); Riparbelli *et al.* (2006); Pike & Kingcombe (2009)
Hemiptera	*Eucalymnatus tessellatus*	Scale insect	XX/XO	Unknown	Microscopy	Gamete duplication	Cytogenetic approach	Nur (1972)
Hemiptera	*Parthenolecanium cerasifex*	Scale insect	XX/XO	Unknown	Microscopy	Gamete duplication	Cytogenetic approach	Nur (1972)

Proximate (molecular) mechanisms of parthenogenesis induction

Despite considerable research effort into the proximate mechanisms through which endosymbionts induce parthenogenesis, these mechanisms remain elusive. Even in cases where the cellular mechanism underlying parthenogenesis is known, it is difficult to disentangle which molecular phenotypes are generated by the bacteria or by the host, given that parthenogenesis results from an interaction between hosts and endosymbionts. For example, in haplo-diploid species, the endosymbiont could diploidize haploid, unfertilized eggs, which might then develop into females as a consequence of the host sex determination system (Beukeboom, 1995; Heimpel & de Boer, 2008). Alternatively, endosymbionts could feminize haploid eggs, which would be rendered diploid by the host, perhaps to match ploidy with gender. This idea is consistent with data from parthenogenetic *Encarsia hispida* wasps; if females are cured of *Cardinuim*, they produce diploid sons (Giorgini *et al.*, 2009). Finally, it is possible that the endosymbionts regulate both, diploidization and feminization of originally haploid eggs, which appears to be the case in at least two wasp species (Tulgetske, 2010; Ma *et al.*, 2015).

A major constraint to understanding the molecular basis of endosymbiont-induced parthenogenesis is the lack of knowledge of the molecular basis of sex determination in the hosts. Although the sex determination pathway is rather conserved across major insect orders, the upstream cues for 'male' vs. 'female' fate remain unknown in the vast majority of species (recently reviewed in Beukeboom, 2012; Ma *et al.*, 2014). Once species with different upstream signals are identified, a very elegant way to investigate proximate mechanisms of parthenogenesis induction would be injection of identical endosymbiont strains into sexual females with different sex determination signals and characterize the induced phenotypes. However, as discussed above, it is currently a technical challenge to culture/transfect any of the known parthenogenesis-inducing endosymbionts.

Conclusions

Endosymbiont-induced parthenogenesis occurs in at least 54–124 different host species from three to seven different arthropod orders. At least in hymenopterans, endosymbionts are a significant driver of transitions from sexual to parthenogenetic reproduction, with one-third of lineages being parthenogenetic as a consequence of endosymbiont infection. Endosymbiont-induced parthenogenesis has been intensively studied in haplo-diploid species, but most likely also occurs in species with other sex determination systems. Indeed, endosymbiont-induced parthenogenesis has been suggested in 24 such species, but has thus far been difficult to confirm due to technical and biological constraints.

In many species featuring endosymbiont-induced parthenogenesis, endosymbiont infection is not fixed (i.e. each species comprises uninfected sexual and infected parthenogenetic strains), but the mechanisms maintaining reproductive polymorphisms remain speculative. Geographically distinct distributions of sexual and parthenogenetic strains could help explain the maintenance of reproductive polymorphism. Indeed, similar to parthenogens with genetic causes underlying parthenogenesis, endosymbiont-induced parthenogens feature distributions towards higher latitudes than their sexual relatives. *Wolbachia* seems to be the most frequent agent of endosymbiont-induced parthenogenesis (in 56–75% of host species) although a lack of screens for other known or unknown endosymbionts causing parthenogenesis may result in an overestimation of the true frequencies of *Wolbachia*-induced parthenogenesis. Gamete duplication is often considered as the main mechanism for endosymbiont-induced parthenogenesis, despite very little empirical data and despite gamete duplication being the mechanism in only half of the known examples of endosymbiont-induced parthenogenesis. More species need to be investigated regarding the cellular mechanisms before a proper conclusion can be made regarding general patterns and a better understanding of the molecular basis of host sex determination is required to develop insights into proximate processes underlying endosymbiont-induced parthenogenesis. Finally, many suggested cases of endosymbiont-induced parthenogenesis should be verified using appropriate methods and samples.

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Conflict of interest

We declare no conflict of interest.

References

Adachi-Hagimori, T. & Miura, K. 2011. Development of a multiplex method to discriminate between *Neochrysocharis formosa* (Hymenoptera: Eulophidae) reproductive modes. *J. Econ. Entomol.* **201**: 1510–1514.

Adachi-Hagimori, T., Miura, K. & Stouthamer, R. 2008a. A new cytogenetic mechanism for bacterial endosymbiont-induced parthenogenesis in Hymenoptera. *Proc. R. Soc. B* **275**: 2667–2673.

Adachi-Hagimori, T., Miura, K. & Abe, Y. 2008b. Gene flow between sexual and asexual strains of parasitic wasps: a possible case of sympatric speciation caused by a parthenogenesis-inducing bacterium. *J. Evol. Biol.* **24**: 1254–1262.

Agrawal, A.F. 2006. Evolution of sex: why do organisms shuffle their genotypes? *Curr. Biol.* **16**: R696–R704.

de Almeida, R.P., van Lenteren, J.C. & Stouthamer, R. 2010. Does *Wolbachia* infection affect *Trichogramma atopovirilia* behaviour? *Braz. J. Biol.* **70**: 435–442.

Arakaki, N. & Kinjo, K. 1998. Notes on the parasitoid fauna of the serpentine leafminer *Liriomyza trifolii* (Burgess) (Diptera: Agromyzidae) in Okinawa, southern Japan. *Appl. Entomol. Zool.* **33**: 577–581.

Arakaki, N., Noda, H. & Yamagishi, K. 2000. *Wolbachia*-induced parthenogenesis in the egg parasitoid *Telenomus nawai*. *Entomol. Exp. Appl.* **96**: 177–184.

Arakaki, N., Miyoshi, T. & Noda, H. 2001. *Wolbachia*-mediated parthenogenesis in the predatory thrips *Franklinothrips vespiformis* (Thysanoptera: Insecta). *Proc. R. Soc. B* **268**: 1011–1016.

Argov, Y. & Gottlieb, Y. 2000. Possible symbiont-induced thelytoky in *Galeopsomyia fausta*, a parasitoid of the citrus leafminer *Phyllocnistis citrella*. *Phytoparasitica* **28**: 212–218.

Beaton, M.J. & Hebert, P.D.N. 1988. Geographical parthenogenesis and polyploidy in *Daphnia pulex*. *Am. Nat.* **132**: 837–845.

Bell, G. 1982. *The Masterpiece of Nature: The Evolution and Genetics of Sexuality*. University of California Press, Berkeley, Croom Helm, London.

Beukeboom, L.W. 1995. Sex determination in Hymenoptera: a need for genetic and molecular studies. *BioEssays* **17**: 813–817.

Beukeboom, L.W. 2012. Microbial manipulation of host sex determination. *BioEssays* **34**: 484–488.

Blackmon, H., Hardy, N.B. & Ross, L. 2015. The evolutionary dynamics of haplodiploidy: genome architecture and haploid viability. *Evolution* **69**: 2971–2978.

Boivin, T., Henri, H., Vavre, F., Gidoin, C., Veber, P., Candau, J.N. *et al.* 2014. Epidemiology of asexuality induced by the endosymbiotic *Wolbachia* across phytophagous wasp species: host plant specialization matters. *Mol. Ecol.* **23**: 2362–2375.

Bowen, W.R. & Stern, V.M. 1966. Effect of temperature on the production of males and sexual mosaics in a uniparental race of *Trichogramma semifumatum* (Hymenoptera: Trichogrammatidae). *Ann. Entomol. Soc. Am.* **59**: 823–834.

Braig, H.R., Turner, B.D. & Normark, B.B. 2002. Microorganism-induced parthenogenesis. In: *Reproductive Biology of Invertebrates* (R.N. Hughes, ed.), pp. 1–62. John Wiley & Sons, Chichester. [Adiyodi, R.G. (Series Editor): Progress in Asexual Reproduction, vol XI]

Brown, A.M.V., Howe, D.K., Wasala, S.K., Peetz, A.B., Inga, A.Z. & Denver, D.R. 2015. Comparative genomics of a plant-parasitic nematode endosymbiont suggest a role in nutritional symbiosis. *Genome Biol. Evol.* **7**: 2727–2746.

Cabello-García, T. & Vargas-Piqueras, P. 1985. Temperature as a factor influencing the form of reproduction of *Trichogramma cordubensis* Vargas and Cabello (Hym., Trichogrammatidae). *J. Appl. Entomol.* **100**: 434–441.

Cabral, G., Marques, A., Schubert, V., Pedrose-Harand, A. & Schlögelhofer, P. 2014. Chiasmatic and achiasmatic inverted meiosis of plants with holocentric chromosomes. *Nat. Commun.* **5**: 5070.

Choleva, L., Janko, K., De Gelas, L., Bohlen, J. & Šlechtová, V. 2012. Synthesis of clonality and polyploidy in vertebrate animals by hybridization between two sexual species. *Evolution* **66**: 2191–2203.

Choudhury, D.A.M. & Copland, M.J.W. 2003. A new record of thelytoky in the egg parasitoid *Anagrus atomus* (Linnaeus) (Hymenoptera: Mymaridae). *Pak. J. Biol. Sci.* **6**: 500–504.

Ciociola, A.I. Jr, de Almeida, R.P., Zucchi, R.A. & Stouthamer, R. 2001. Detecção de *Wolbachia* emu ma População Telítocq de *Trichogramma atopovirilia* Oatman & Platner (Hymenoptera: Trichogrammatidae) via PCR com o Primer Específico wsp. *Neotrop. Entomol.* **30**: 489–491.

Coomans, A. & Cleys, M. 1998. Structure of the female reproductive system of *Xiphinema americanum* (Nematoda: Longidoridae). *Fundam. Appl. Nematol.* **21**: 569–580.

Dale, C., Beeton, M., Harbison, C., Jones, T. & Pontes, M. 2006. Isolation, pure culture, and characterization of "*Candidatus* Arsenophonus arthropodicus", an intracellular secondary endosymbiont from the Hippoboscid Louse fly *Pseudolynchia canariensis*. *Appl. Environ. Microbiol.* **72**: 2997–3004.

De Barro, P. & Hart, P. 2001. Antibiotic curing of parthenogenesis in *Eretmocerus mundus* (Australian parthenogenic form). *Entomol. Exp. Appl.* **99**: 225–230.

Dedeine, F., Vavre, F., Fleury, F., Benjamin, L., Michael, E.H. & Boulétreau, M. 2001. Removing symbiotic *Wolbachia* bacteria specifically inhibits oogenesis in a parasitic wasp. *Proc. Natl. Acad. Sci. USA* **98**: 6247–6252.

Dedeine, F., Vavre, F., Shoemaker, D.D. & Boulétreau, M. 2004. Intra-individual coexistence of a *Wolbachia* strain required for host oogenesis with two strains inducing cytoplasmic incompatibility in the wasp *Asobara tabida*. *Evolution* **58**: 2167–2174.

Dillon, R.J. & Dillon, V.M. 2004. The gut bacteria of insects: nonpathogenic interactions. *Annu. Rev. Entomol.* **49**: 71–92.

Eskafi, F.M. & Legner, E.F. 1974. Parthenogenetic reproduction in *Hexacola* sp. near websteri, a parasite of *Hippelates* eye gnats. *Ann. Entomol. Soc. Am.* **67**: 767–768.

Feiertag-Koppen, C.C.M. 1980. Cytological studies of the two-spotted spider mite *Tetranychus urticase* Koch (Tetranychidae, Trombidiformes) II. Meiosis in growing oocytes. *Genetica* **54**: 173–180.

Fenollar, F., La Scola, B., Inokuma, H., Dumler, J.S., Taylor, M.J. & Raoult, D. 2003. Culture and phenotypic characterization of a *Wolbachia pipientis* isolate. *J. Clin. Microbiol.* **41**: 5434–5441.

Flanders, S.E. 1945. The bisexuality of uniparental Hymenoptera, a function of the environment. *Am. Nat.* **79**: 122–141.

Flanders, S.E. 1965. On the sexuality and sex ratios of Hymenopterous populations. *Am. Nat.* **99**: 489–494.

Fontcuberta García-Cuenca, A., Dumas, Z. & Schwander, T. 2016. Extreme genetic diversity in asexual grass thrips populations. *J. Evol. Biol.* **29**: 887–899.

Gebiola, M., Monti, M.M., Roishene, C.J., Woolley, J.B., Hunter, M.S., Giorgini, M. *et al.* 2016. A revision of the *Encarsia pergandiella* species complex (Hymenoptera: Aphelinidae) shows cryptic diversity in parasitoids of whitefly pests. *Syst. Entomol.* **42**: 31–59.

Giorgini, M. 2001. Induction of males in thelytokous populations of *Encarsia meritoria* and *Encarsia protransvena*: A systematic tool. *Biocontrol* **46**: 427–438.

Giorgini, M., Hunter, M.S., Mancini, D. & Pedata, P.A. 2007. Cytological evidence for two different mechanisms of

thelytokous parthenogenesis in *Encarsia* parasitoids harbouring *Wolbachia* or *Cardinium* bacteria. Poster. In: *Abstr. X Eur. Workshop Insect Parasit*. Erice (TP), pp. 17–21.

Giorgini, M., Monti, M., Caprio, E., Stouthamer, R. & Hunter, M.S. 2009. Feminization and the collapse of haplodiploidy in an asexual parasitoid wasp harboring the bacterial symbiont *Cardinium*. *Heredity* **102**: 365–371.

Giorgini, M., Bernardo, U., Monti, M.M., Nappo, A.G. & Gebiola, M. 2010. *Rickettsia* symbionts cause parthenogenetic reproduction in the parasitoid wasp *Pnigalio soemius* (Hymenoptera: Eulophidae). *Appl. Environ. Microbiol.* **76**: 2589.

Glesener, R.R. & Tilman, D. 1978. Sexuality and the components of environmental uncertainty: clues from geographic parthenogenesis in terrestrial animals. *Am. Nat.* **112**: 659–673.

Gordh, G. & Lacey, L. 1976. Biological studies of *Plagiomerus diaspidis* Crawford, a primary internal parasite of diaspidid scale insects (Hymenoptera: Encyrtidae; Homoptera: Diaspididae). *Proc. Entomol. Soc. Wash.* **78**: 132–144.

Gottlieb, Y. & Zchori-Fein, E. 2001. Irreversible thelytokous reproduction in *Muscidifurax uniraptor*. *Entomol. Exp. Appl.* **100**: 271–278.

Gottlieb, Y., Zchori-Fein, E., Faktor, O. & Rosen, D. 1998. Phylogenetic analysis of parthenogenesis-inducing *Wolbachia* in the genus *Aphytis* (Hymenoptera: Aphelinidae). *Insect Mol. Biol.* **7**: 393–396.

Gottlieb, Y., Zchori-Fein, E., Werren, J.H. & Karr, T.L. 2002. Diploidy restoration in *Wolbachia*-infected *Muscidifurax uniraptor* (Hymenoptera: Pteromalidae). *J. Invertebr. Pathol.* **81**: 166–174.

Grenier, S., Gomes, S.M., Pintureau, B., Lassablière, F. & Bolland, P. 2002. Use of tetracycline in larval diet to study the effect of *Wolbachia* on host fecundity and clarify taxonomic status of *Trichogramma* species in cured bisexual lines. *J. Invertebr. Pathol.* **80**: 13–21.

Groot, T.V.M. & Breeuwer, J.A.J. 2006. *Cardinium* symbionts induce haploid thelytoky in most clones of three closely related *Brevipalpus* species. *Exp. Appl. Acarol.* **39**: 257–271.

Haag, C.R. & Ebert, D. 2004. A new hypothesis to explain geographic parthenogenesis. *Ann. Zool. Fennici.* **41**: 539–544.

Hagimori, T., Abe, Y., Date, S. & Miura, K. 2006. The first finding of a *Rickettsia* bacterium associated with parthenogenesis induction among insects. *Curr. Microbiol.* **52**: 97–101.

Haine, E.R., Pickup, N.J. & Cook, J.M. 2005. Horizontal transmission of *Wolbachia* in a *Drosophila* community. *Ecol. Entomol.* **30**: 464–472.

Hamilton, P. & Perlman, S.J. 2013. Host defense via symbiosis in *Drosophila*. *PLoS Pathog.* **9**: 31003808.

Heimpel, G.E. & de Boer, J.G. 2008. Sex determination in the Hymenoptera. *Annu. Rev. Entomol.* **53**: 209–230.

Hilgenboecker, K., Hammerstein, P., Schlattmann, P., Telschow, A. & Werren, J.H. 2008. How many species are infected with *Wolbachia*? – a statistical analysis of current data. *FEMS Microbiol. Lett.* **281**: 215–220.

Hörandl, E. 2009. Geographical parthenogenesis: opportunities for asexuality. In: *Lost Sex* (I. Schön, K. Martens & P. van Dijk, eds), pp. 161–186. Springer, Amsterdam, The Netherlands.

Hosokawa, T., Koga, R., Kikuchi, Y., Meng, X.Y. & Fukatsu, T. 2010. *Wolbachia* as a bacteriocyte-associated nutritional mutualist. *Proc. Natl. Acad. Sci. USA* **107**: 769–774.

Huigens, M.E. 2003. On the evolution of *Wolbachia*-induced parthenogenesis in *Trichogramma* wasps. PhD thesis. University of Wageningen, The Netherlands.

Huigens, M.E. & Stouthamer, R. 2003. Parthenogenesis associated with *Wolbachia*. *Insect. Symbiosis* **1**: 247–266.

Hunter, M.S. 1999. The influence of parthenogenesis-inducing *Wolbachia* on the oviposition behavior and sex-specific developmental requirements of autoparasitoid wasps. *J. Evol. Biol.* **12**: 735–741.

Janko, K., Kotlík, P. & Ráb, P. 2003. Evolutionary history of asexual hybrid loaches (Cobitis: Teleostei) inferred from phylogenetic analysis of mitochondrial DNA variation. *J. Evol. Biol.* **16**: 1280–1287.

Jeong, G. & Stouthamer, R. 2005. Genetics of female functional virginity in the parthenogenesis-*Wolbachia* infected parasitoid wasp *Telenomus nawai* (Hymenoptera: Scelionidae). *Heredity* **94**: 402–407.

Kageyama, D., Narita, S. & Watanabe, M. 2012. Insect sex determination manipulated by their endosymbionts: incidences, mechanisms and implications. *Insects* **2**: 161–199.

King, C.E. & Snell, T.W. 1977. Sexual recombination in rotifers. *Heredity* **39**: 357–360.

Kremer, N., Charif, D., Henri, H., Bataille, M., Prévost, G., Kraaijeveld, K. *et al.* 2009. A new case of *Wolbachia* dependence in the genus *Asobara*: evidence for parthenogenesis induction in *Asobara japonica*. *Heredity* **103**: 248–256.

Kumm, S. & Moritz, G. 2008. First detection of *Wolbachia* in arrhenotokous populations of thrips species (Thysanoptera: Thripidae and Phlaeothripidae) and its role in reproduction. *Environ. Entomol.* **37**: 1422–1428.

Laraichi, M. 1978. L'effect de hautes temperatures sur le taux sexuel de *Ooencyrtus fecundus* Hymenoptera: Encyrtidae. *Entomol. Exp. Appl.* **23**: 237–242.

Lively, C.M. & Morran, L.T. 2014. Sex uncovered special issue: the ecology of sexual reproduction. *J. Evol. Biol.* **27**: 1292–1303.

Longdon, B. & Jiggins, F.M. 2012. Vertically transmitted viral endosymbionts of insects: do sigma viruses walk alone? *Proc. R. Soc. B* **279**: 3889–3898.

Longdon, B., Obbard, D.J. & Jiggins, F.M. 2010. Sigma viruses from three species of *Drosophila* form a major new clade in the rhabdovirus phylogeny. *Proc. R. Soc. B* **177**: 35–44.

Longdon, B., Wilfert, L., Obbard, D.J. & Jiggins, F.M. 2011. Rhabdoviruses in two species of *Drosophila*: vertical transmission and a recent sweep. *Genetics* **188**: 141–150.

Lynch, M. 1984. Destabilizing hybridization, general-purpose genotypes and geographic parthenogenesis. *Q. Rev. Biol.* **59**: 257–290.

Ma, W.-J., Vavre, F. & Beukeboom, L.W. 2014. Manipulation of arthropod sex determination by endosymbionts: diversity and molecular mechanisms. *Sex. Dev.* **8**: 59–73.

Ma, W.-J., Pannebakker, B.A., van de Zande, L., Schwander, T., Wertheim, B. & Beukeboom, L.W. 2015. Diploid males support a two-step mechanism of endosymbiont-induced thelytoky in a parasitoid wasp. *BMC Evol. Biol.* **15**: 84.

Matalon, Y., Katzir, N., Gottlieb, Y., Portnoy, V. & Zchori-Fein, E. 2007. Cardinium in *Plagiomerus diaspidis* (Hymenoptera: Encyrtidae). *J. Invertebr. Pathol.* **96**: 106–108.

Meyer, J.M. & Hoy, M.A. 2007. *Wolbachia*-associated thelytoky in *Diaphorencyrtus aligarhensis* (Hymenoptera: Encyrtidae), a parasitoid of the Asian citrus psyllid. *Fla. Entomol.* **90**: 776–779.

Mouton, L., Dedeine, F., Henri, H., Boulétreau, M., Profizi, N. & Vavre, F. 2004. Virulence, multiple infections and regulation of symbiotic population in the *Wolbachia-Asobara tabida* symbiosis. *Genetics* **168**: 181–189.

Nazeri, M., Ashouri, A. & Hosseini, M. 2015. Can *Wolbachia* infection improve qualitative characteristics of *Trichogramma brassicae* reared on cold stored eggs of the host? *Int. J. Pest Manag.* **61**: 243–249.

Neiman, M., Sharbel, T.F. & Schwander, T. 2014. Genetic causes of transitions from sexual reproduction to asexuality in plants and animals. *J. Evol. Biol.* **27**: 346–359.

Normark, B.B. 1996. Phylogeny and evolution of parthenogenetic weevils of the *Aramigus tessellatus* species complex (Coleoptera: Curculionidae: Naupactini): evidence from mitochondrial DNA sequences. *Evolution* **50**: 734–735.

Nur, U. 1972. Diploid arrhenotoky and automictic thelytoky in soft scale insects. *Chromosoma* **39**: 381–401.

Ohkuma, M. 2008. Symbioses of flagellates and prokaryotes in the gut of lower termites. *Trends Microbiol.* **16**: 345–352.

Otto, S.P. 2009. The evolutionary enigma of sex. *Am. Nat.* **174**: S1–S14.

Palévody, C. 1973. Etude cytologique de la parthénogenèse chez Folsomia candida (Collembole, Isotomide). *C. R. Acad. Sci.* **277**: 2501–2504.

Palomares-Rius, J.E., Archidona-Yuste, A., Cantalapiedra-navarrete, C., Prieto, P. & Castillo, P. 2016. Molecular diversity of bacterial endosymbionts associated with dagger nematodes of the genus *Xiphiema* (Nematoda: Longidoridae) reveals a high degree of phylogenetic congruence with their host. *Mol. Ecol.* **25**: 6225–6247.

Pannebakker, B.A., Pijnacker, L.P., Zwaan, B.J. & Beukeboom, L.W. 2004a. Cytology of *Wolbachia*-induced parthenogenesis in *Leptopilina clavipes* (Hymenoptera: Figitidae). *Genome* **303**: 299–303.

Pannebakker, B.A., Beukeboom, L.W., van Alphen, J.J.M., Brakefield, P.M. & Zwaan, B.J. 2004b. The genetic basis of male fertility in relation to haplodiploid reproduction in *Leptopilina clavipes* (Hymenoptera: Figitidae). *Genetics* **168**: 341–349.

Pannebakker, B.A., Schidlo, N.S., Boskamp, G.J.F., Dekker, L., van Dooren, T.J.M., Beukeboom, L.W. *et al.* 2005. Sexual functionality of *Leptopilina clavipes* (Hymenoptera: Figitidae) after reversing *Wolbachia*-induced parthenogenesis. *J. Evol. Biol.* **18**: 1019–1028.

Perotti, M.A., Clarke, H.K., Turner, B.D. & Braig, H.R. 2006. *Rickettsia* as obligate and mycetomic bacteria. *FASEB J.* **20**: 2372–2374.

Pijls, J.W.A.M., van Steenbergen, H.J. & van Alphen, J.J.M. 1996. Asexuality cured: the relations and differences between sexual and asexual *Apoanagyrus diversicornis*. *Heredity* **76**: 506–513.

Pike, N. & Kingcombe, R. 2009. Antibiotic treatment leads to the elimination of *Wolbachia* endosymbionts and sterility in the diplodiploid collembolan *Folsomia candida*. *BMC Biol.* **7**: 54.

Pintureau, B., Lassablière, F., Khatchadourian, C. & Daumal, J. 1999. Parasitoides oophages et symbiotes de deux thrips européens. *Ann. Soc. Entomol. Fr.* **35**: 416–420.

Pintureau, B., Chaudier, S., Lassablière, F., Charles, H. & Grenier, S. 2000. Addition of *wsp* sequences to the *Wolbachia* phylogenetic tree and stability of the classification. *J. Mol. Evol.* **51**: 374–377.

Pintureau, B., Grenier, S., Heddi, A. & Charles, H. 2002. Biodiversity of *Wolbachia* and of their effects in *Trichogramma* (Hymenoptera: Trichogrammatidae). *Ann. Soc. Entomol.* **38**: 333–338.

Plantard, O., Rasplus, J.-Y., Mondor, G., Le Clainche, I. & Solignac, M. 1998. Wolbachia-induced thelytoky in the rose gallwasp *Diplolepis spinosissimae* (Giraud) (Hymenoptera: Cynipidae), and its consequences on the genetic structure of its host. *Proc. R. Soc. B* **265**: 1075–1080.

Provencher, L.M., Morse, G.E., Weeks, A.R. & Normark, B.B. 2005. Parthenogenesis in the *Aspidiotus nerii* Complex (Hemiptera: Diaspididae): a single origin of a worldwide, polyphagous lineage associated with *Cardinium* bacteria. *Ann. Entomol. Soc. Am.* **98**: 629–635.

Quezada, J.R., DeBach, P. & Rosen, D. 1973. Biological and taxonomic studies of *Signophora borinquensis*, new species (Hymenoptera: Signiphoridae), a primary parasite of diaspine scales. *Hilgardia* **41**: 543–604.

Rasgon, J.L., Ren, X.X. & Petridis, M. 2006. Can *Anopheles gambiae* be infected with *Wolbachia pipientis*? Insights from an *in vitro* system. *Appl. Environ. Microbiol.* **72**: 7718–7722.

Reumer, B.M., van Alphen, J.J.M. & Kraaijeveld, K. 2010. Ecology, *Wolbachia* infection frequency and mode of reproduction in the parasitoid wasp *Tetrastichus coeruleus* (Hymenoptera: Eulophidae). *Mol. Ecol.* **19**: 1733–1744.

Reumer, B.M., van Alphen, J.J.M. & Kraaijeveld, K. 2012. Occasional males in parthenogenetic populations of *Asobara japonica* (Hymenoptera: Braconidae): low *Wolbachia* titer or incomplete coadaptation? *Heredity* **108**: 341–346.

Reumer, B.M., van Alphen, J.J.M. & Kraaijeveld, K. 2013. Population genetics of *Wolbachia*-infected, parthenogenetic and uninfected, sexual populations of *Tetrastichus coeruleus* (Hymenoptera: Eulophidae). *Mol. Ecol.* **22**: 4433–4444.

Riparbelli, M.G., Giordano, R. & Callaini, G. 2006. Centrosome inheritance in the parthenogenetic egg of the collembolan *Folsomia candida*. *Cell Tissue Res.* **326**: 861–872.

Rodriguero, M.S., Confalonieri, V.A., Guedes, J.V. & Lanteri, A.A. 2010a. *Wolbachia* infection in the tribe *Naupactini* (Coleoptera, Curculionidae): association between thelytokous parthenogenesis and infection status. *Insect Mol. Biol.* **19**: 631–640.

Rodriguero, M.S., Lanteri, A.A. & Confalonieri, V.A. 2010b. Mito-nuclear genetic comparison in a *Wolbachia* infected weevil: insights on reproductive mode, infection age and evolutionary forces shaping genetic variation. *BMC Evol. Biol.* **10**: 340.

Rössler, Y. & Debach, P. 1972. The biosystematics relations between a thelytokous and an arrhenotokous form of *Aphytis mytilaspidis* (Le Baron) [Hymenoptera: Aphelinidae]. *Entomophaga* **17**: 425–435.

Rössler, Y. & Debach, P. 1973. Genetic variability in a thelytokous form of *Aphytis mytilaspidis* (Le Baron) (Hymenoptera: Aphelinidae). *Hilgardia* **42**: 149–176.

Schilthuizen, M. & Stouthamer, R. 1997. Horizontal transmission of parthenogenesis-inducing microbes in *Trichogramma* wasps. *Proc. R. Soc. Lond. B Biol. Sci.* **264**: 361–366.

Schilthuizen, M., Honda, J. & Stouthamer, R. 1998. Parthenogenesis inducing *Wolbachia* in *Trichogramma kaykai*. *Ann. Entomol. Soc. Am.* **91**: 410–414.

Schlinger, E.I. & Hall, J.C. 1959. A synopsis of the biologies of three imported parasites of the spotted Alfalfa aphid. *J. Econ. Entomol.* **52**: 154–157.

Smith, S.G. 1955. Cytogenetics of obligatory parthenogenesis. *Can. Entomol.* **87**: 131–135.

Stelzer, C.P. 2008. Obligate asex in a rotifer and the role of sexual signals. *J. Evol. Biol.* **21**: 287–293.

Stelzer, C.P., Schmidt, J., Wiedlroither, A. & Riss, S. 2010. Loss of sexual reproduction and dwarfing in a small metazoan. *PLoS ONE* **5**: e12854.

Stille, B. & Därvring, L. 1980. Meiosis and reproductive strategy in the parthenogenetic gall wasp *Diplolepis rosae* (L.) (Hymenoptera, Cynipidae). *Hereditas* **92**: 353–362.

Stouthamer, R. 1997. *Wolbachia*-induced parthenogenesis. In: *Influential Passengers: Inherited Microorganisms and Arthropod Reproduction* (S.L. O'Neill, A.A. Hoffmann & J.H. Werren, eds), pp. 102–122. Oxford University Press, Oxford, UK.

Stouthamer, R. & Kazmer, D.J. 1994. Cytogenetics of microbe-associated parthenogenesis and its consequences for gene flow in *Trichogramma* wasps. *Heredity* **73**: 317–327.

Stouthamer, R. & Werren, J.H. 1993. Microbes associated with parthenogenesis in wasps of the genus *Trichogramma*. *J. Invertebr. Pathol.* **61**: 6–9.

Stouthamer, R., Luck, R.F. & Hamilton, W.D. 1990. Antibiotics cause parthenogenetic *Trichogramma* Hymenoptera/Trichogrammatidae) to revert to sex. *Proc. Natl. Acad. Sci. USA* **87**: 2424–2427.

Stouthamer, R., Breeuwert, J.A. & Werren, J.H. 1993. Molecular identification of microorganisms associated with parthenogenesis. *Nature* **361**: 66–68.

Stouthamer, R., van Tilborg, M., de Jong, J.H., Nunney, L. & Luck, R.F. 2001. Selfish element maintains sex in natural populations of a parasitoid wasp. *Proc. R. Soc. Lond. B* **268**: 617–622.

Stouthamer, R., Russell, J.E., Vavre, F. & Nunney, L. 2010. Intragenomic conflict in populations infected by parthenogenesis inducing *Wolbachia* ends with irreversible loss of sexual reproduction. *BMC Evol. Biol.* **10**: 229.

Suomalainen, E., Saura, A. & Lokki, J. 1987. *Cytology and Evolution in Parthenogenesis*. CRC Press, Boca Raton, FL.

Svendsen, N., Reisser, C.M., Dukić, M., Thuillier, V., Ségard, A., Liautard-Haag, C. *et al.* 2015. Uncovering cryptic asexuality in *Daphnia magna* by RAD sequencing. *Genetics* **201**: 1143–1155.

Taberly, G. 1987. Recherches sur la pathénogenèse thélytoque de deux espèces d'acariens oribatides: *Trhypochthonius tectorum* (Berlese) et *Platynothrus peltifer* (Koch). III. Observations sur les males atavique. *Acarologia* **29**: 95–107.

Taberly, G. 1988. Recherches sur la pathénogenèse thélytoque de deux espèces d'acariens oribatides: *Trhypochthonius tectorum* (Berlese) et *Platynothrus peltifer* (Koch). IV. Etude anatomique, histologique et cytologique des femelles parthenogenetiques. *Acarologia* **28**: 389–403.

Tien, N.S.H., Sabelis, M.W. & Egas, M. 2015. Inbreeding depression and purging in a haplodiploid: gender-related effects. *Heredity* **114**: 327–332.

Tilquin, A. & Kokko, H. 2016. What does the geography of parthenogenesis teach us about sex? *Phil. Trans. R. Soc. B* **371**: 20150538.

Timmermans, M.J.T.N. & Ellers, J. 2009. *Wolbachia* endosymbiont is essential for egg hatching in a parthenogenetic arthropod. *Evol. Ecol.* **23**: 931–942.

Tucker, A.E., Ackerman, M.S., Eads, B.D., Xu, S. & Lynch, M. 2013. Population-genomic insights into the evolutionary origin and fate of obligatory asexual *Daphnia pulex*. *Proc. Natl. Acad. Sci. USA* **110**: 15740–15745.

Tulgetske, G.M. 2010. Investigations into the mechanisms of *Wolbachia*-induced parthenogenesis and sex determination in the parasitoid wasp, *Trichogramma*, Riverside, California: PhD thesis.

Tulgetske, G.M. & Stouthamer, R. 2012. Characterization of intersex production in *Trichogramma kaykai* infected with parthenogenesis-inducing *Wolbachia*. *Naturwissenschaften* **99**: 143–152.

Van der Kooi, C.J. & Schwander, T. 2014a. Evolution of asexuality via different mechanisms in grass thrips (Thysanoptera: *Aptinothrips*). *Evolution* **68**: 1883–1893.

Van der Kooi, C.J. & Schwander, T. 2014b. On the fate of sexual traits under asexuality. *Biol. Rev. Camb. Philos. Soc.* **89**: 805–819.

Van Meer, M.M., Witteveldt, J. & Stouthamer, R. 1999. Phylogeny of the arthropod endosymbiont *Wolbachia* based on the *wsp* gene. *Insect Mol. Biol.* **8**: 399–408.

Van Vugt, J.F.A., Salverda, M., de Jong, J.H. & Stouthamer, R. 2003. The paternal sex ratio chromosome in the parasitic wasp *Trichogramma kaykai* condenses the paternal chromosomes into a dense chromatin mass. *Genome* **46**: 580–587.

Vandekerckhove, T.T.M., Watteyne, S., Willems, A., Swings, J.G., Mertens, J. & Gillis, M. 1999. Phylogenetic analysis of the 16S rDNA of the cytoplasmic bacterium *Wolbachia* from the novel host *Folsomia candida* (Hexapoda, collembolan) and its implications for wolbachial taxonomy. *FEMS Microbiol. Lett.* **180**: 279–286.

Vandekerckhove, T.T.M., Willems, A., Gillis, M. & Coomans, A. 2000. Occurrence of novel verrucomicrobial species, endosymbiotic and associated with parthenogenesis in *Xiphinema americanum*-group species (Nematoda, Longidoridae). *Int. J. Syst. Evol. Microbiol.* **50**: 2197–2205.

Vavre, F., Fleury, F., Varaldi, J., Fouillet, P. & Boulétreau, M. 2002. Infection polymorphism and cytoplasmic incompatibility in Hymenoptera-*Wolbachia* associations. *Heredity* **88**: 361–365.

Verhoeven, K.J. & Biere, A. 2013. Geographic parthenogenesis and plant-enemy interactions in the common dandelion. *BMC Evol. Biol.* **13**: 1.

Vorburger, C., Gehrer, L. & Rodriguez, P. 2009. A strain of the bacterial symbiont *Regiella insecticola* protects aphids against parasitoids. *Biol. Lett.* **6**: 109–111.

Waldman, A.S. 2008. Ensuring the fidelity of recombination in mammalian chromosomes. *BioEssays* **30**: 1163–1171.

Weeks, A.R. & Breeuwer, J.A.J. 2001. *Wolbachia*-induced parthenogenesis in a genus of phytophagous mites. *Proc. R. Soc. Lond. B* **268**: 2245–2251.

Werren, J.H. 1997. Biology of *Wolbachia*. *Annu. Rev. Entomol.* **42**: 587–609.

Werren, J.H., Zhang, W. & Guo, L.R. 1995. Evolution and phylogeny of *Wolbachia:* reproductive parasites of arthropods. *Proc. R. Soc. Lond. B Biol. Sci.* **261**: 55–63.

Werren, J., Baldo, L. & Clark, M.E. 2008. *Wolbachia*: master manipulators of invertebrate biology. *Nat. Rev. Microbiol.* **6**: 741–751.

Wilson, F. & Woolcock, L. 1960. Temperature determination of sex in a parthenogenetic parasite. *Ooencyrtus submetallicus* (Howard) (Hymnopera: Encyrtidae). *Austr. J. Zool.* **8**: 158–169.

Yusuf, M. & Turner, B. 2004. Characterisation of *Wolbachia*-like bacteria isolated from the parthenogenetic stored-

product pest psocid *Liposcelis bostrychophila* (Badonnel) (Psocoptera). *J. Stored Prod. Res.* **40**: 207–225.
Yusuf, M., Turner, B.D., Miles, R.J., Whitfield, P.J. & Pacey, J. 2000. Electron microscopical evidence of a vertically transmitted *Wolbachia*-like parasite in the parthenogenetic, stored-product pest *Liposcelis bostrychophila* (Psocoptera: Insecta). *J. Stored Prod. Res.* **36**: 169–175.
Zchori-Fein, E., Roush, R.T. & Hunter, M.S. 1991. Male production induced by antibiotic treatment in *Encarsia formosa* (Hymenoptera: Aphelinidae). *Experientia* **48**: 102–105.
Zchori-Fein, E., Rosen, D. & Roush, R.T. 1994. Microorganisms associated with thelytoky *Aphytis lingnansensis* compere (Hymenoptera aphelinidae). *Int. J. Insect Morphol. Embryol.* **23**: 169–172.
Zchori-Fein, E., Faktor, O., Zeidan, M., Gottlieb, Y., Czosnek, H. & Rosen, D. 1995. Parthenogenesis-inducing microorganisms in *Aphytis* (Hymenoptera: Aphelinidae). *Insect Mol. Biol.* **4**: 173–178.
Zchori-Fein, E., Roush, R.T. & Rosen, D. 1998. Distribution of parthenogenesis-inducing symbionts in ovaries and eggs of *Aphytis* (Hymenoptera: Aphelinidae). *Curr. Microbiol.* **36**: 1–8.
Zchori-Fein, E., Perlman, S.J., Kelly, S.E., Katzir, N. & Hunter, M.S. 2004. Characterization of a '*Bacteroidetes*' symbiont in *Encarsia* wasps (Hymenoptera: Aphelinidae). *Int. J. Syst. Evol. Microbiol.* **54**: 961–968.
Zug, R. & Hammerstein, P. 2012. Still a host of hosts for *Wolbachia*: analysis of recent data suggests that 40% of terrestrial arthropod species are infected. *PLoS ONE* **7**: e38544.

Supporting information

Additional Supporting Information may be found online in the supporting information tab for this article:
Figure S1 Geographical distribution of 27 endosymbiont-induced parthenogens with both sexual and asexual populations.
Table S1 Endosymbiont-induced parthenogenesis in haplo-diploid species (or haplo-haploid in the case of *Brevipalpus*), with confirmed cases highlighted in grey.
Table S2 List of parthenogenetic species with gamete duplication and where parthenogenesis is due to genetic causes. For species in brackets, gamete duplication is not formally confirmed but likely.
Table S3 Species with a firm demonstration of endosymbiont-induced parthenogenesis, and with reproductive polymorphism.
Table S4 Haplo-diploid parthenogens in which parthenogenesis is due to genetic causes, and where the cellular mechanisms underlying parthenogenesis are studied (all species except the last one in the list are hymenopterans. *Heliothrips haemorrhoidalis* is a thrips species).
Appendix S1 References.

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