Heredity (2009) 103, 194–195

www.nature.com/hdy

NEWS AND COMMENTARY

Essential *Wolbachia*

Essential but unhelpful wasp *Wolbachia*

AA Hoffmann

Heredity (2009) **103,** 194–195; doi:10.1038/hdy.2009.50; published online 10 June 2009

Kremer *et al*. describe a strain of the maternally inherited bacterium *Wolbachia* that induces parthenogenesis in its host, the wasp *Asobara japonica*; in certain populations females only produce female offspring when they are infected. A related *Wolbachia* strain in another *Asobara* wasp species (*Asobara tabida*) has quite a different effect—this other strain is essential for successful oogenesis but it does not cause parthenogenesis. Two related *Wolbachia* have, therefore, diverged and become essential for their wasp hosts, but without providing them any fitness benefit.

Wolbachia are well-known bacteria that live inside the reproductive tissue and other tissues of their insect hosts. The bacteria spread across generations by vertical transmission through females. The presence of these bacteria is associated with a range of host effects—from cytoplasmic incompatibility (where infected males prevent or reduce the production of viable embryos when they mate with uninfected females), through to feminization (genetically programmed males turning into females) and parthenogenesis (where *Wolbachia* cause females to produce only female offspring). Recently, it has become apparent that *Wolbachia* infections influence the fitness of their hosts in diverse ways, by altering patterns of reproduction, resistance to microbial infections and the provision of nutrients (Hedges *et al*., 2008; Teixeira *et al*., 2008; Ghedin *et al*., 2008). Changes in *Wolbachia* or their host genomes in the same species have now been documented (Hornett *et al*., 2006; Weeks *et al*., 2007) and suggest that evolutionary shifts in *Wolbachia*–host interactions (and their host effects) can be rapid. *Wolbachia* can therefore mediate rapid evolutionary shifts in host reproductive patterns and fitness effects.

Parasitoid wasps reproduce by producing offspring that then develop in the immature stages of moths, spiders, aphids and other arthropods. Both wasps, and their host arthropods, are often infected by *Wolbachia* and the bacterial infections are known to influence the fitness, cytoplasmic incompatibility and parthenogenesis of their wasp hosts. Parasitoid wasps may even facilitate the horizontal spread of *Wolbachia* across different arthropods as they attack a range of host species, although this is likely to be a rare event (Haine *et al*., 2005; Baldo *et al*., 2008).

Kremer *et al*. now provide evidence for multiple phenotypic effects that seems to have evolved in *Wolbachia* residing in parasitoids of the genus *Asobara*. In an earlier work on the species *A. tabida*, it was discovered that *Wolbachia* controls oogenesis by influencing programmed cell death (Pannebakker *et al*., 2007). When *Wolbachia* were removed from strains after an antibiotic treatment, there was no oogenesis and therefore no offspring production. *Wolbachia* have therefore become essential for one component of *Asobara* reproduction. In this paper, Kremer *et al*. describe a different strain of *Wolbachia* isolated from a Japanese population of the species *A. japonica*. This population was known to produce only female offspring through parthenogenesis. The authors show that parthenogenesis no longer occurs once the *Wolbachia* are removed because male offspring are then produced. These male offspring court and attempt to mate with females, but the females from this population are no longer receptive to the males. The *Wolbachia* have therefore become essential for the continued persistence of this *A. japonica* population, although other populations around Japan that have not been infected by *Wolbachia* continue to exist and exhibit sexual reproduction. Unlike the *Wolbachia* strain found in *A. tabida*, the new strain of *Wolbachia* is not essential for oogenesis, because offspring are produced when *Wolbachia* are removed through antibiotics.

To test the origin of the new *Wolbachia* strain from *A. japonica*, Kremer *et al*. sequence a series of *Wolbachia* genes used earlier in phylogenetic analyses. They find that the new strain is closely related to the strain from *A. tabida* that is essential for oogenesis, and not necessarily related closely to strains of *Wolbachia* from other hosts where the infection results in parthenogenesis. Therefore, two related strains of *Wolbachia* that occur in *Asobara* species have quite different effects, presumably because of recent evolutionary divergence in a few genes responsible for triggering different host phenotypes. However, it is also possible that the host genome dictates the expression of the phenotype induced by *Wolbachia*—this could be tested by transfering different *Wolbachia* strains across populations and species.

The spread of *Wolbachia* in insect populations depends on host effects as well as the fidelity of *Wolbachia* transmission across generations. For *Wolbachia* causing parthenogenesis to spread at the expense of other strains they need to provide an advantage to their hosts in terms of female offspring production, and exhibit a low rate of leakage of the infection when transmitted from parents to offspring (Stouthamer, 1997). However, in the case of some populations of *A. japonica*, it seems that the *Wolbachia* have basically become essential for the wasps, because females are no longer able to mate successfully with uninfected males. Presumably, there has been DNA sequence decay in the genes controlling female components of the mating interaction. Therefore, even if *Wolbachia*-infected strains no longer have a reproductive advantage, the infection is likely to persist in these populations of *A. japonica*, at least in the short term, and perhaps until they are invaded by sexually reproducing *A. japonica* or a close competitor. *Wolbachia*–host associations in parasitoids might then represent evolutionarily transient phenomena; they may develop rapidly, but then be prone to displacement, unless a fitness benefit for the host has meanwhile evolved.

Professor AA Hoffmann is at the Centre for Environmental Stress and Adaptation Research, Department of Genetics, University of Melbourne, Parkville 3010, Australia.

e-mail: ary@unimelb.edu.au

Baldo L, Ayoub NA, Hayashi CY, Russell JA, Stahlhut JK, Werren JH (2008). Insight into the routes of *Wolbachia* invasion: high levels of horizontal transfer in the spider genus Agelenopsis revealed by *Wolbachia* strain and mitochondrial DNA diversity. *Mol Ecol* **17**: 557–569.

Ghedin E, Daehnel K, Foster J, Slatko B, Lustigman S (2008). The symbiotic relationship between filarial parasitic nematodes and their *Wolbachia* endosymbionts—A resource for a new generation of control measures. *Symbiosis* **46**: 77–85.

Haine ER, Pickup NJ, Cook JM (2005). Horizontal transmission of *Wolbachia* in a *Drosophila* community. *Ecol Entomol* **30**: 464–472.

Hedges LM, Brownlie JC, O'Neill SL, Johnson KN (2008). *Wolbachia* and virus protection in insects. *Science* **322**: 702.

Hornett EA, Charlat S, Duplouy AMR, Davies N, Roderick GK, Wedell N, Hurst GDD (2006). Evolution of male-killer suppression in a natural population. *PLoS Biol* **4**: 1643–1648.

Pannebakker BA, Loppin B, Elemans CPH, Humblot L, Vavre F (2007). Parasitic inhibition of cell death facilitates symbiosis. *Proc Natl Acad Sci USA* **104**: 213–215.

Stouthamer R (1997). *Wolbachia*-induced parthenogenesis. In: O'Neill SL, Hoffmann AA, Werren JH (eds). *Influential Passengers: Inherited Microorganisms and Arthropod Reproduction*. Oxford University Press: New York, pp 102–124.

Teixeira L, Ferreira A, Ashburner M (2008). The bacterial symbiont *Wolbachia* induces resistance to RNA viral infections in *Drosophila melanogaster*. *PLoS Biol* **6**: 2753–2763.

Weeks AR, Turelli M, Harcombe WR, Reynolds KT, Hoffmann AA (2007). From parasite to mutualist: rapid evolution of *Wolbachia* in natural populations of *Drosophila*. *PLoS Biol* **5**: 997–1005.

Editor's suggested reading

White JA, Kelly SE, Perlman SJ, Hunter MS (2009). Cytoplasmic incompatibility in the parasitic wasp *Encarsia inaron*: disentangling the roles of *Cardinium* and *Wolbachia* symbionts. *Heredity Online* 10.1038/hdy.2009.5.

Clark ME, Bailey-Jourdain C, Ferree PM, England SJ, Sullivan W, Windsor DM, Werren JH (2008). *Wolbachia* modification of sperm does not always require residence within developing sperm. *Heredity* **101**: 420–428.

Sheeley SL, McAllister BF (2009). Mobile male-killer: similar *Wolbachia* strains kill males of divergent *Drosophila* hosts. *Heredity* **102**: 286–292.

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