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Wolbachia and Virus Protection in Insects

Lauren M. Hedges, Jeremy C. Brownlie, Scott L. O'Neill, Karyn N. Johnson*

W olbachia pipientis are matemally transmitted, Gram-negative, obligate intracellular bacteria found in filarial nematodes, crustaceans, arachnids, and at least 20% of all insect species. Many *Wolbachia* bacteria increase their prevalence in populations by manipulating host reproductive systems (1). Insects are also commonly infected with viruses, and, considering the shared intracellular location, it is possible that *Wolbachia* may influence the outcome of virus infection in an insect host.

Drosophila melanogaster is commonly infected with Wolbachia and is a powerful model for studying host-pathogen interactions and antiviral responses (2). Drosophila C virus (DCV), a member of the Dicistroviridae family, is a natural pathogen of D. melanogaster and is found in 30 to 40% of both laboratory and wild-caught populations (3, 4). Infection of adult Drosophila with DCV by injection can result in 100% mortality within 3 to 4 days. Although variation in susceptibility of fly strains to DCV-induced mortality has been recorded (3), the underlying basis for this variation has not been determined.

We compared the survival of flies infected with DCV in the presence or the absence of Wolbachia infection (Fig. 1 and fig. S1) (5). In flies from the standard laboratory strain, Oregon RC, Wolbachia infection delayed DCV-induced mortality compared with Oregon RC flies cured of Wolbachia infection (Fig. 1A). The delay in mortality corresponded with a delay in virus accumulation in Wolbachia-infected flies (fig. S2). The experiment was repeated with the fly strain w^{1118} with similar results observed (Fig. 1B). The survival curves of Oregon RC and w^{1118} Wolbachiafree flies were similar to those of two wild-type laboratory populations (Champetières and Oregon R) that are naturally uninfected with Wolbachia (compare Fig. 1, A and B, with fig. S1). Oregon RC and w^{1118} flies are infected with two closely related strains of Wolbachia, wMelCS and wMelPop, respectively (6). These results indicate that these strains of Wolbachia, in different genetic backgrounds of Drosophila, have an antiviral effect.

Two further viruses were tested with use of the survival bioassay: cricket paralysis virus (CrPV; Dicistroviridae), a natural Drosophila pathogen,

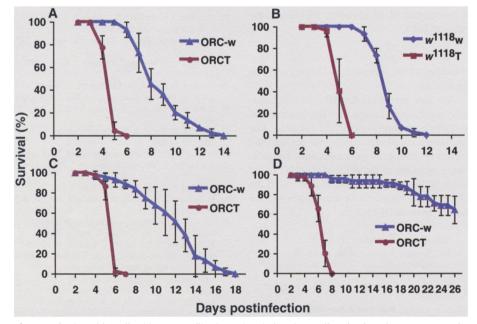


Fig. 1. Infection with *Wolbachia* protects flies from virus-induced mortality. The data shown represent the mean of triplicates, and the bars indicate standard error. The survival curves were significantly different for *Wolbachia* infected versus uninfected flies (Kaplan-Meier analysis, P < 0.0001 in each case). (**A**) Comparison of the survival of *Wolbachia*-infected (ORC-w) and uninfected Oregon RC (ORCT) flies after challenge with DCV. (**B**) Comparison of the survival of *Wolbachia*-infected (W) and uninfected (T) w^{1118} flies after challenge with DCV. (**C**) Comparison of the survival of *Wolbachia*-infected (ORC-w) and uninfected Oregon RC (ORCT) flies after challenge with CrPV. (**D**) Comparison of the survival of *Wolbachia*-infected (ORC-w) and uninfected Oregon RC (ORCT) flies after challenge with FHV.

and Flock House virus (FHV; Nodaviridae). The latter is unrelated to DCV and CrPV and is pathogenic in adult flies (7), although natural infections have not been reported. Like DCV, both CrPV and FHV induce rapid mortality when injected into adult Drosophila. All Oregon RC flies infected with Wolbachia and CrPV died within 17 days postinfection (Fig. 1C). In contrast, the Wolbachiafree Oregon RC flies died within 7 days of infection. Similarly, Wolbachia-free flies challenged with FHV died within 8 days of infection, whereas 26 days postinfection only 35% of the Wolbachiainfected flies had succumbed to FHV-induced mortality (Fig. 1D). These results indicate that the antiviral effect observed in Wolbachia-infected Drosophila functions to protect flies from diverse RNA viruses.

Typically Wolbachia manipulate host reproductive systems to increase the number of infected hosts within a population. However, Wolbachia strains that infect D. melanogaster do not induce these parasitic traits under field conditions at levels sufficient to invade host populations (8). Theory predicts that in the absence of strong reproductive parasitism Wolbachia should confer a fitness benefit to the host, but for D. melanogaster no such benefit has been identified in nature (8). Because both DCV and Wolbachia are common in wild Drosophila populations, the association of Wolbachia with a robust antiviral effect may confer a positive selective advantage to flies. If generalized, the antiviral protection associated with Wolbachia infection might be exploited in future strategies to reduce insect-transmitted diseases.

References and Notes

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 Materials and methods are available as supporting
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Supporting Online Material

www.sciencemag.org/cgi/content/full/322/5902/702/DC1 Materials and Methods

Figs. S1 and S2

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