Wolbachia variability and cytoplasmic incompatibility in *Culex*

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The maternally inherited bacterial symbiont *Wolbachia*, found in many mosquito species, is responsible for the induction of crossing sterilities known as cytoplasmic incompatibility (CI). *Wolbachia*-modified sperm are unable to complete fertilization of uninfected ova, but a rescue function allows infected eggs to develop normally. By providing a reproductive advantage to infected females, *Wolbachia* can rapidly invade uninfected populations, and this could provide a mechanism for transgene drive. CI can also occur between *Wolbachia*-infected populations which is usually associated with the presence of different *Wolbachia* strains. In the *Culex pipiens* group an unusual degree of complexity of *Wolbachia*-induced crossing-types has been reported, with partial or complete CI that can be unidirectional or bidirectional, yet no *Wolbachia* strain variation was found. We have sequenced the genome of the *w*Pip strain of *Wolbachia* from *C. quinquefasciatus*, currently in assembly (http://www.sanger.ac.uk/Projects/W_pipientis/). It contains a large number of ankyrin repeat-encoding (ANK) genes, which in other systems can be involved in modification of the activity of cell-cycle regulating proteins. Because CI seems to involve disruption of the timing of the host cell cycle at karyogamy, ANK genes are candidates for control of the phenotype. Significant levels of variation were found between incompatible *Culex* strains in two *Wolbachia* ANK genes, one of which also showed different patterns of expression between strains. Both of these ANK genes are associated with a *Wolbachia* prophage region, and this integrated prophage is capable of producing phage particles. Genome introgression experiments by backcrossing have also revealed a direct effect of the host nuclear genome on CI rescue. The implications of these results will be discussed with respect to unravelling *Culex* crossing type complexities and mechanisms of CI.