

Mechanisms underlying the fitness costs imposed by malaria infection and immune stimulation.

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Malaria infection is known to impose fitness costs upon its mosquito vector. These costs include an increase in early mortality when infections are heavy and a decrease in fecundity and fertility that occurs even when infections are light. Stimulation of both the humoral and cellular immune response has also been associated with a reduction in egg production. We are investigating the sequence of events that occurs in the ovaries of immune stimulated or infected *Anopheles gambiae* in an attempt to determine whether egg production is inhibited by the same mechanism in each case. A melanization response was generated by the injection of a C-25 or G-25 Sephadex bead into the haemocoel and an antibacterial response by inoculation of lipopolysaccharide. The rodent malaria, *Plasmodium yoelii nigeriensis* was used to infect mosquitoes. Ovaries were examined 18 hours post infection / injection.

In all three cases, cells in the follicular epithelium of a proportion of ovarian follicles were undergoing apoptosis. This was determined by the detection of caspase-like activity. Previous work has shown that, in malaria-infected mosquitoes, apoptosis of patches of follicular epithelial cells results in resorption of developing oocytes and thus a reduction in fecundity. At the time of examination, a significantly higher proportion of follicles were undergoing apoptosis in malaria-infected mosquitoes than in immune stimulated ones but all treatments induced a significantly higher incidence of apoptosis than sham injected controls.

We propose the trade-off between reproductive fitness and immune defence in *An. gambiae* operates via the induction of apoptosis in ovarian follicles and that different immune responses impose costs via the same pathway. Whether malaria infection induces fecundity reduction indirectly, via the activation of an immune response will be discussed.