EPITHELIAL RESPONSES OF THE *A. GAMBIAE* MIDGUT TO *PLASMODIUM* INVASION.

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Plasmodium causes cell damage during invasion leading to apoptosis. Cells undergoing apoptosis are nitrated in a two-step process involving the induction of an epithelial peroxidase. Parasite-induced damage is repaired faster in the *A. gambiae* refractory (R) L35 strain than in the susceptible unselected G3 strain, probably due higher systemic levels of hydrogen peroxide (H_2O_2) in the R strain. This accelerated nitration may mediate refractoriness. Two other peroxidases that mediate refractoriness to *Plasmodium* were identified and their mechanism of action is under investigation. Higher basal levels of H_2O_2 in the R strain confer greater resistance to both *Plasmodium* and bacterial infections. The expression of catalase, an enzyme involved in H_2O_2 detoxification is induced in midgut in response to blood feeding, but is no longer induced in *Plasmodium*-infected midguts. Limiting H_2O_2 clearance appears to be part of an anti-parasitic response, as reducing catalase expression by dsRNA knockdown further decreases *Plasmodium* infection. The role of catalase in female fecundity was also investigated. Two catalase alleles were identified: females highly susceptible to *Plasmodium* infection (S strain), which retain higher fecundity with aging, are homozygous for the Serine (high activity) catalase, while G3 females are homozygous for the Tryptophan (low activity) allele. Our studies also show that, *A. gambiae,* catalase accumulates in the developing oocyte and is essential to protect the embryo from oxidative damage.
Keywords: *Plasmodium, A. gambiae*, catalase.