Interactions of human malaria parasites, *Plasmodium vivax* and *P.falciparum*, with the midgut of *Anopheles* mosquitoes

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Abstract. Present understanding of the development of sexual stages of the human malaria parasites *Plasmodium vivax* and *P.falciparum* in the *Anopheles* vector is reviewed, with particular reference to the role of the mosquito midgut in establishing an infection. The sexual stages of the parasite, the gametocytes, are formed in human erythrocytes. The changes in temperature and pH encountered by the gametocyte induce gametogenesis in the lumen of the midgut. Macromolecules derived from mosquito tissue and second messenger pathways regulate events leading to fertilization. In *An.tessellatus* the movement of the ookinete from the lumen to the midgut epithelium is linked to the release of trypsin in the midgut and the peritrophic matrix is not a firm barrier to this movement. The passage of the *P.vivax* ookinete through the peritrophic matrix may take place before the latter is fully formed. The late ookinete development in *P.falciparum* requires chitinase to facilitate penetration of the peritrophic matrix. Recognition sites for the ookinetes are present on the midgut epithelial cells. N-acetyl glucosamine residues in the oligosaccharide side chains of *An.tessellatus* midgut glycoproteins and peritrophic matrix proteoglycan may function as recognition sites for *P.vivax* and *P.falciparum* ookinetes. It is possible that ookinetes penetrating epithelial cells produce stress in the vector. Mosquito molecules may be involved in oocyst development in the basal lamina, and encapsulation of the parasite occurs in vectors that are refractory to the parasite. Detailed knowledge of vector–parasite interactions, particularly in the midgut and the identification of critical mosquito molecules offers prospects for manipulating the vector for the control of malaria.

Key words. *Anopheles*, *Plasmodium vivax*, *P.falciparum*, midgut glycoproteins, trypsin, gametocytes, ookinetes.