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A comparative analysis of reproductive biology of insect vectors of human disease

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Studying the reproductive strategies of insect species that transmit diseases to humans can identify new exploitable targets for the development of vector control methods. Here we describe shared characteristics and individual features of the reproductive biology of three major disease vectors: *Anopheles gambiae*, *Aedes aegypti* and *Glossina morsitans*. Current studies are identifying i) species-specific molecular cascades that determine female monandrous behavior, ii) core aspects of egg development that could be disrupted for controlling natural populations, and iii) the increasingly apparent role of resident microbiota in shaping reproductive success and disease transmission potential. The recent completion of multiple genome sequencing projects is allowing comparative genomics studies that not only increase our knowledge of reproductive processes but also facilitate the identification of novel targets for vector control.

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Introduction

The global burden of diseases spread by the biting of insect vectors is a heavy one: more than 17% of all infectious diseases are transmitted by vectors, and it is estimated over half the world's population is at risk, with more than one million deaths every year (World Health Organization; URL: www.who.int). Blood feeding is necessary for insect vectors to obtain nutrients required for energy and reproduction, and various viruses and parasites have evolved to exploit this requirement as a way to move between hosts. Current vector control strategies heavily rely on insecticides, which are nevertheless thwarted by the spread of resistance alleles in insect populations. Studying the basic reproductive biology of insect vectors of human disease can identify broad-ranging or species-specific reproductive targets that can be exploited for the development of novel control methods as alternatives or to complement the use of insecticides.

In this review, we will focus on recent findings concerning the reproductive biology of disease vectors from the genera *Anopheles* (malaria), *Aedes* (yellow fever, dengue fever, chikungunya), and *Glossina* (human African trypanosomiasis), which account for the vast majority of global vector-borne mortality. Crucial aspects of mating, egg development and symbiotic relationships will be discussed with the end goal to highlight possible weak links in these life cycles that can be exploited for disease control.

Mating strategies and post-mating behavior of insect vectors

Insect vectors show various mating strategies depending on species-specific behaviors and ecologies. Mosquitoes (suborder Nematocera) are distant relatives of the tsetse flies (suborder Brachycera). Nevertheless, their mating behaviors bear some similarities (Table 1). Most *Anopheles* species mate in crepuscular swarms formed over particular markers on the ground [1–3], where males gather at dusk and attract females by as yet unknown mechanisms, probably based on visual and chemical cues. *Aedes* mosquitoes, although they may show swarming behavior, prefer instead to mate in proximity to the hosts on which they feed [3]. Similarly, tsetse flies mate in close proximity to their vertebrate hosts and utilize visual cues to identify mating partners. Mating begins once a contact-based pheromone on the female is detected by the male [4] and pairs must remain coupled for 1.5–2 h for the pairing to be successful [5]. By contrast, mosquito matings are short (10–20 s).

Regardless of the mating strategy, in all three genera sperm transferred during mating are stored in a dedicated sperm storage organ: a single spermatheca in *Anopheles*, two in tsetse, and two spermathecae and a bursa inseminalis in *Aedes*. Males of most Anopheles species are exceptional in the coagulation of their seminal fluid to

Table 1

Comparison of the mating biology of major disease vectors.

Mating biology	*Anopheles*	*Aedes*	*Glossina*
Mating in swarms	Yes	Possible	No
Mating near host	No	Yes	Yes
Mating duration	15–20 s	10–15 s	90–120 min
Coagulated seminal fluids (SF)	Yes	No	Yes
Identified SF components	20E, TG3	JH	None
Post-mating changes			
Female monandry	Yes	Yes	Yes
Fecundity increased	Yes	Yes	No
Ovulation induced	Yes	Yes	Yes
Sperm storage	Yes	Yes	Yes

form a mating plug, a gelatinous rod rich in proteins, lipids and steroid hormones produced in the male accessory glands (MAGs) which upon sexual transfer is processed in the female reproductive tract [6–9,10••,11••]. In *An. gambiae*, the major malaria vector, transfer of the mating plug is linked to sperm storage, as females that do not receive a plug do not store sperm in their spermatheca [7]. *Aedes* seminal fluid is not coagulated but nevertheless contains a complex mix of bioactive peptides [12,13].

Seminal fluids in *Glossina* are transferred to the female reproductive tract, where they coagulate into a structure called a spermatophore, which also contains the sperm bundle [14]. After mating, the spermatophore is broken down over 24 h and sperm migrate to the spermathecae. The constituent proteins and chemical moieties associated with this structure remain however undefined.

Despite wide evolutionary distance, *Anopheles*, *Aedes* and *Glossina* share a female monandrous behavior (i.e. the occurrence of a single mating event during the female's lifespan). This mating strategy could potentially be targeted using chemical analogs that mimic monandry-inducing factors, preventing virgin females from mating, thereby decreasing vector populations. The male triggers of monandry have been recently identified in *An. gambiae*: high titers of the steroid hormone 20-hydroxyecdysone (20E) transferred to the female atrium (uterus) within the mating plug contribute to switching the female to a mated status, rendering her refractory to further copulation (among other physiological changes — see below) [10••]. As discussed later, both 20E and its precursor ecdysone (E) are also produced by the female after a blood meal, where they are essential for egg development.

Conversely, the molecular basis of monandry is unknown in *Aedes*, although a number of early studies suggest a role for peptides synthesized in the MAGs [15]. MAG protein extracts were able to induce mating refractoriness and oviposition when injected into virgin female mosquitoes [16,17]. Recent work aimed at molecularly characterizing components of *Aedes* seminal fluid has identified a number of male proteins that are transferred to the female, so that the specific factors required to induce monandry in these mosquitoes may be pinned down in the near future [12,13].

Female tsetse flies also become refractory to further copulation after sex [18], a behavior that starts 24 h after mating [19,20]. Injection of MAG extracts can induce mating refractoriness [21], suggesting that factors produced by the male glands are the trigger of this behavior. Spermatophore digestion over 24 h correlates with the initiation of refractoriness behavior in females, but the nature of the molecular triggers is not known.

Egg development is a conserved process in different vectors

Much of what we know of the molecular mechanisms of oogenesis comes from studies in *Ae. aegypti*. Here, egg development is triggered by both the nutritional status of the female and the taking of a blood meal (reviewed in [22]). After emergence, the sesquiterpene juvenile hormone (JH) is secreted by the corpora allata in the brain and coordinates the maturation of multiple tissues. As JH levels increase and peak over the first two days of adulthood, the multifunctional fat body, with roles in nutrient storage, detoxification and protein synthesis, undergoes structural remodeling and large JH-dependent changes in gene expression that render this tissue competent to respond to ecdysone produced by the ovary after blood feeding [23•]. JH also causes the pre-vitellogenic development and maintenance of ovarian follicles, which accumulate lipids and transcripts for key proteins involved in uptake of yolk protein precursors (YPPs) [24,25]. Moreover, JH delivered by *Ae. aegypti* males during mating [26] also increases female fecundity [17,27] by directing available nutrient resources toward reproduction, enlarging ovarian follicles and preventing follicle resorption [28•].

After taking a blood meal *Aedes* mosquitoes develop eggs over 2–3 days. The mosquito brain stops JH synthesis and releases the ovarian ecdysiotropic hormone (OEH) [29], triggering the ovaries to produce the steroid hormone ecdysone [30]. Ecdysone is hydroxylated in turn to 20E in the fat body. Through the 20E receptor EcR/USP and early-acting genes E74, E75 and Broad [31–33], 20E stimulates the transcription of YPPs, such as vitellogenin and lipophorin, which are released into the hemolymph and taken up by the ovaries by receptor-mediated endocytosis [34–36]. Additionally, levels of extracellular amino acids released by blood meal digestion trigger YPP production via the TOR-signaling pathway [37].

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