The Control of Reproduction by a Blood Meal: The Mosquito as a Model for Vector Endocrinology

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In this Symposium we are considering various consequences of blood-feeding in insect vectors. We have considered digestion of the blood meal and the membrane barriers through which the digested products must pass. I plan to discuss some of the physiological consequences of blood-feeding, once the components of the blood meal enter the haemolymph and move toward their ultimate destination, the oocytes. Mosquitoes are among those insects that produce many eggs simultaneously and continue to produce batches of eggs throughout their lifetime. But females can produce multiple batches only if they have the ability to feed as adults and acquire certain essential nutrients, particularly amino acids (Lea et al., 1958). After one batch of eggs matures and is laid, there is the necessity of finding another blood meal, and because success is variable, the female must be physiologically ready to begin oocyte development as soon as a host is located. But until the eggs are laid, there must be a mechanism for blocking further egg development, so that, if the female takes blood again before ovipositing, she does not become overloaded with eggs and be unable to reach a favorable oviposition site.

Mosquitoes make good models in which to study how an insect that produces multiple batches of eggs and has specialized oviposition requirements regulates its reproductive physiology. Most of the data to which I will refer has been obtained from microsurgical experiments with Aedes aegypti, and much of it has been verified in other aedine species. Present evidence indicates that hormones from the corpora allata (CA), neurosecretory system, and the ovary regulate major steps in maturation of the oocyte.

Follicles of the newly emerged female must complete a certain amount of growth and differentiation before they are capable of undergoing vitellogenesis. This growth is dependent on CA hormone (Gwadz & Spielman, 1973). A. aegypti females, allatectomized at emergence and subsequently fed blood, rarely deposit any yolk, although in other species the CA apparently is active before emergence and some yolk is deposited (Lea, 1963; Lea, 1969). Once the ovary has reached
the so-called resting stage, allatectomy does not alter the steps which lead to egg maturation. In addition to its influence on the oocytes, CA hormone has other functions in preparing the female for reproduction. CA hormone must be secreted if the female is to become receptive to copulation with males (Lea, 1968; Gwadz & Craig, 1968), and in Calliphora erythrocephala CA hormone stimulates the production of neurosecretory material by the medial neurosecretory cells (MNC) in the protocerebrum (Lea & Thomsen, 1969).

Gillett (1956) and Clements (1956) showed by decapitating females at intervals after blood feeding that the head played an essential role in initiating maturation of the oocytes. Gillett's haemolymph exchange experiment (1958) demonstrated that a humoral factor, which he suggested was a neurosecretory hormone from the brain, was the controlling mechanism. His interpretation was verified by MNC extirpation and implantation experiments (Lea, 1967, 1972). This neurosecretory hormone is produced in the MNC and stored in their axon terminals in the wall of the aorta (Meola & Lea, 1971). The complex of hormone-laden axons in the aorta wall, lying on the dorsal plate of the pharyngeal sucking pump, is referred to as the corpus cardiacum CC (S. Meola & Lea, 1972).

For many years it was assumed, without experimental evidence, that in mosquitoes the neurosecretory hormone was released by some nerve stimulus from the midgut which had been stretched with the recently ingested blood meal, as Wigglesworth described in Rhodnius (Wigglesworth, 1934). However, my experiments have indicated that the nervous system is not involved, and that the cue for release from the CC is a haemolymph-borne stimulus (Lea, 1972). In any event, extirpation of the MNC at emergence blocked egg development in the females subsequently fed blood. If the MNC were removed several days after emergence, then the corpus cardiacum had to be removed as well as the MNC perikarya in order to suppress oogenesis. This evidence supported the view that a neurosecretory hormone, produced in the MNC perikarya soon after emergence, moves along the axons of these cells, through the brain and out into the wall of the aorta where it remains until the appropriate stimulus after ingestion of blood initiates release.

To explain the function of the neurosecretory hormone I must describe briefly some recent experiments of Henry Hagedorn at the University of Massachusetts. Hagedorn has found that after a blood meal, the fat body makes a female specific protein termed vitellogenin (Hagedorn & Judson, 1972). His evidence comes from in vitro incubation of fat bodies, from females fed blood 18 h earlier, that were incubated in a medium containing labelled phenylalanine. After incubation, vitellogenin was precipitated by the addition of antibody to vitellogenin and the labelling of vitellogenin was counted. Fat body from
females ovariectomized before the blood meal could not make vitellogenin but did so if an ovary was implanted before the blood meal (HAGEDORN & FALCON, 1973). Therefore, HAGEDORN concluded that a factor from the ovary of a blood-fed female stimulated the fat body to produce vitellogenin. This factor has now been identified as α-ecdysone (FALCON et al., 1974). Evidence for the occurrence of ecdysone in blood-fed mosquitoes has already come from SCHLAEGER et al. (1974), who also corroborated the report of SPIELMAN et al. (1971) that injection of β-ecdysone into non-blood fed A. aegypti stimulated vitellogenesis.

HAGEDORN and I now have preliminary evidence that extirpation of the neurosecretory system will prevent the ovary from secreting ecdysone after a blood meal, and as a consequence the fat body will not make vitellogenin (LEA and HAGEDORN, unpublished observation). Furthermore, by incubating pieces of brain containing the MNC with ovaries and fat body from unfed females, we have detected the synthesis of vitellogenin. We therefore suggest that the neurosecretory hormone, produced in the MNC and released from the CC, is an ecdysiotrophic hormone stimulating the secretion of ecdysone by the ovary.

While secretion of ecdysone by the ovary may possibly be novel to the mosquito, there is prior evidence that the ovary is an endocrine tissue. ADAMS et al. (1968) describe the production of an "oostatic" hormone from developing oocytes that blocked vitellogenesis in the less advanced penultimate oocytes of Musca domestica. R. MEOLA and LEA (1972) described a similar follicle-inhibiting factor in mosquitoes which were retaining eggs. The follicle inhibiting factor prevents the female from maturing additional eggs which would overload her and make her unflightworthy in the event she encounters a source of blood before a suitable oviposition site is available.

In summary, it appears that once the female takes a blood meal, several events occur almost simultaneously. The peritrophic membrane is formed; water is removed from the blood meal and excreted; proteolytic enzymes begin to be secreted; ecdysiotrophic hormone is released from the CC; and vitellogenesis begins. While all vectors may not regulate their reproductive physiology in exactly this way, the mosquito has proven to be an unusually responsive model in which a variety of surgical manipulations and experimental techniques have yielded new information on vector endocrinology.

References