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Endocrine Control Of Fat Hypertropy In The Diapause Mosquito Culex pipiens

This proposal focuses on the neuroendocrine basis for diapause (dormancy) in the northern house mosquito, Culex pipiens. The definition of diapause is the delay in development in response to regularly and recurring periods of adverse environmental conditions, which is a key characteristic for disease vectors to survive during adverse environment.

For example, the diapause-programmed mosquitoes do not seek blood meals but instead feed on nectar and other carbohydrate sources that are converted into glycogen and lipid stores in the fat body, and these sequesters are then used as the energy source for survival during the long winter. Hypertrophy of the fat body and elevation of fat reserves occur after adult eclosion; within a week, females programmed for diapause accumulate more than twice the lipid reserves of their nondiapausing counterparts, and these reserves are mainly consumed during diapause. When the mosquitoes have consumed their glycogen energy sources (after the first month of diapause), the energy demands are fueled from lipid stores in the fat body. The next spring, surplus lipids that remain with the mosquito after diapause termination are readily used for subsequent egg production. Since West Nile virus, carried within the mosquito Cx. pipiens, is passed from parents to eggs by transovarial transmission, understanding the endocrine regulation of fat hypertrophy is thus critical not only for understanding the energetic architecture of diapausing mosquitoes but also for identifying key steps of nutrient metabolism that may be vulnerable to disruption of the arboviral disease transmission.

Insulin-like peptides (ILPs) and adipokinetic hormone (AKH) are counter-endocrine systems that regulate carbohydrate in hemolymph and lipid levels in the fat body of many insects. Thus we propose that these two endocrine systems are the primary regulators for adaptive nutrient homeostasis, and that these two systems work closely together to regulate genes involved in energy metabolism.