INSULIN AND ITS ROLE IN INTESTINAL ALKALINIZATION OF *LUTZOMYIA LONGIPALPIS*

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Lutzomyia longipalpis is an insect belonging to the family Psychodidae. This insect is also the primary vector of *Leishmania infantum* in the Americas, the etiological agent of American visceral leishmaniasis. When this insect feeds on blood, large changes occur in the intestinal pH value. This pH change influences the production and activity of digestive enzymes, as well as the developmental process of *L. infantum*. In *L. longipalpis* females fed on sucrose, the luminal pH is observed to be around 6.0. However, when females feed on blood, the intestinal pH increases, reaching values close to 8.1. Three main mechanisms have been identified as responsible for this pH elevation: the first is related to the release of CO_2 from the ingested blood; the second involves hormonal signaling triggered during blood feeding; and the third consists of a transport system in which certain amino acids are co-transported with H^+ ions from the intestinal lumen to the enterocyte cytoplasm. The present study aims to identify and describe intra- and extracellular steps of the alkalinization mechanism associated with hormonal signaling after blood feeding. It has been demonstrated that the influx of Ca^{2+} into enteroendocrine cells of *L. longipalpis* females leads to the release of a molecule into the insect's hemolymph, which induces intestinal alkalinization in a 100% of tested midguts. Furthermore, the ex vivo application of bovine insulin to the intestines of *L. longipalpis* females reproduces the same effects as hormones released by enteroendocrine cells, with all midguts showing alkalization response. Gene expression results for insulin and its receptor (LLONM1_008043 and LLONM1_009708, respectively) confirm the presence of these genes in the midgut of *L. longipalpis* females. Although not significant, blood-fed females showed a 4.6-fold increase in insulin receptor expression. These findings indicate that insulin is the hormone responsible for intestinal alkalinization.

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