Blood-feeding cause functional changes in *Aedes aegypti* mitochondria

Sales-Gonçalves RL, Oliveira PL,Vannier-Santos MA, Galina A, Oliveira MF

1-IBqM, UFRJ; RJ; 2-CPqGM, FIOCRUZ, Salvador, BA.

The mosquito Aedes aegypti is the vector of dengue virus and females ingest blood to meet their energy requirements. Blood digestion poses a challenge to this organism since heme release is associated to many deleterious effects. An hypothesis raised by our group suggest that hematophagous organisms shift their energy metabolism from aerobic to fermentative during blood digestion as a preventive antioxidant defence, avoiding oxygen utilization and reactive oxygen species generation in mitochondria. To test this hypothesis, we investigated here the respiratory functions of mitochondria isolated from thoraxes of blood-fed (BF) and non blood-fed (NBF) adult *A. aegypti* females. Blood-feeding causes drastic changes on some mitochondrial parameters independent of the site of electron entry. We observed a significant reduction in state 3 respiration induced by either pyruvate-proline (Pyr-pro) or glycerol 3-phosphate (G3P) as substrates. Cytochrome c oxidase activity was also significantly reduced in blood fed mosquitoes but NADH-cytocrome c reductase activity and G3P-cytocrome c reductase activity were unchanged. Measurements of membrane potential (??m) in mitochondria from NBF revealed that pyr-pro were capable to induce hyperpolarization. However, when G3P is used, higher concentrations of this substrate were needed to generate levels of ??m comparable to those produced by pyr-pro, indicating that G3P respiration contributes less to ??m. Together, these results indicate that blood feeding promotes an overall reduction in the oxygen consumption capacity of *A. aegypti* thorax mitochondria. **Support: WHO-TDR-SSI; CNPq; FAPERJ; HHMI.**