Blood-Feeding Induces Reversible Functional Changes in Flight Muscle Mitochondria of *Aedes aegypti* Mosquito

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Hematophagy poses a challenge to blood-feeding organisms since products of blood digestion process (BDP), as heme and iron, exert cellular deleterious effects. A number of adaptations were identified in hematophagous arthropods after ingestion of vertebrate blood including mechanisms involved in detoxification of blood-derived products. Mitochondrial function is a key element in this context as both redox and energy-transducing pathways converge to this organelle and that would be physiologically relevant for hematophagous . Here, we investigated the effects of blood-feeding (BF) on flight muscle (FM) mitochondria from the mosquito *Aedes aegypti*, a vector of dengue and yellow fever diseases. We observed that 24 hours after blood meal (ABM) the cytochromes c and a+a3 levels and cytochrome c oxidase activity were specifically reduced. Mitochondrial oxygen consumption and hydrogen peroxide (H_2O_2) generation were both reverisbly time-dependent inhibited in all metabolic states evaluated and paralell to the BDP. These changes were particularly evident at 24 hours ABM, the peak of BDP, when oxygen consumption was inhibited by 68 % and H_2O_2 formation by 51 % at state 3 respiration using NAD+-linked substrates. Morphometric analyses of FM demonstrate that BF increased mitochondrial area which was paralell to a reduction of mitochondria density, suggesting fusion. Analysis of the supramolecular organization of mitochondrial inner membrane proteins reveled that BF causes a transient re-organization of the mitochondrial supercomplexes along the BDP. Collectively, these results indicate that blood meal induce reversible functional and structural changes in *A. aegypti* FM mitochondria which are parallel to BDP and may represent an important adaptive mechanism to hematophagy.

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