Cold acclimation promotes an uncoupling activity modulated by fatty acid and nucleotide in goldfish white muscle mitochondria: presence of a UCP?

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Uncoupling protein (UCP) is an integral membrane protein that is located in inner mitochondrial membrane and mediates a regulated leak of protons. Although UCP homologous were already identified in cyprinids (commom carp and zebrafish), their functions are not cleary understood. Goldfish is a cyprinid able to maintain itself active even in cold temperatures due to metabolic changes in skeletal muscle. We previously observed an increased oxidative capacity accompanied by a higher proton leak in goldfish white muscle mitochondria after cold exposure. Thus, we aim to study whether this proton leak could be promoted by an UCP activity and if it is modulated by cold acclimation. To investigate this question we measured the effect of both activator (palmitate) and inhibitors (GDP and BSA) of UCPs upon to the remaining oligomycin-inhibited respiration rate (state 4o) in permeabilized fibers from white muscle. Palmitate addition promoted a dose-dependent increase in state 4o, which was partially inhibited by GDP. Cold-acclimated permeablilized fibers show an increased state 4o (2.7-fold higher than warm-acclimated ones), indicating a higher proton leak. Addition of GDP and BSA was able to inhibit oxygen consumption in cold-acclimated but not in warm-acclimated permeabilized fibers. Curiously, palmitate was able to promote a rise in state 4o rate in warm-acclimated goldfish to a similar extent to state 4o level in cold-acclimated goldfish, which in turn was not affected by palmitate addition. These results suggest that goldfish white muscle mitochondria possess an UCP with similar properties of mammalian UCPs. Furthermore, this UCP may play an important role during cold acclimation process in goldfish.

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