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UBIAD1 is an antioxidant enzyme that regulates NO signaling by CoQ10 synthesis

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Protection against oxidative damage caused by excessive reactive oxygen species (ROS) by an anti- oxidant network is essential for the health of tissues, especially in the cardiovascular system. Here, we identified a gene with important antioxidant features by analyzing a null allele of zebrafish ubiad1, called barolo (bar). Bar mutants show specific cardiovascular failure due to oxidative stress and ROS-mediated cellular damage. Human UBIAD1 is a non-mitochondrial prenyltransferase that synthesizes CoQ10 in the Golgi membrane compartment. Loss of UBIAD1 reduces the cytosolic pool of the antioxidant CoQ10 and leads to ROS-mediated lipid peroxidation in vascular cells. Surprisingly, inhibition of eNOS prevents Ubiad1-dependent cardiovascular oxidative damage, suggesting a crucial role for this enzyme and non-mitochondrial CoQ10 in NO signaling. These findings identify UBIAD1 as a non-mitochondrial CoQ10-forming enzyme with specific cardiovascular protective function via the modulation of eNOS activity.

Biography

Massimo M. Santoro obtained is Master Diploma from University of Turin, Italy and his Ph.D. in Molecular and Cellular Biology from the Open University, London, UK. In 2001 he received his first post-doctoral training at University of Piemonte Orientale, and then he moved to University of California, San Francisco, USA, where he became interested in vascular development and in health and diseased angiogenesis. In 2008, he moved back to Italy at University of Turin where he established his laboratory of Cardiovascular Biology as Assistant Professor. In 2013 he was appointed VIB Group Leader and Professor at Vesalius Research Center, University of Leuven, Belgium. He is holding an HFSP Career Developmental Award and Marie Curie Reintegration Grant. His aim is to expand the current knowledge of how metabolism regulates endothelial redox homeostasis and vice-versa in healthy and diseased conditions. To accomplish this goal his lab has taken advantage of the innovative genetic and imaging technology as well as new molecular and biochemical approaches in vertebrate models, including zebrafish and mouse. Using a set of new cellular, molecular, and genetic approaches as well as advanced microscopy techniques he is willing to identify new redox mechanisms involved in vascular metabolism and homeostasis.

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