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Book of Abstracts

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Endogenous Coenzyme Q content and exogenous Coenzyme Q bioavailability in D. melanogaster.

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Coenzyme Q (CoQ) performs important functions in organisms: it is essential in cellular energy metabolism and as antioxidant. Secondary CoQ deficiency is related to both pathological conditions, associated with increased oxidative stress, and physiological conditions, characterized by decreased biosynthesis observed in senescent organisms. Therefore, the CoQ status in aging research is of particular interest. Despite, *Drosophila melanogaster* represents an established model in aging studies, currently, detailed information on the CoQ status in *Drosophila* is missing.
In the present study, we characterized CoQ9 and CoQ10 distribution in *D. melanogaster* in the three body segments at different life stages in both sex. Subsequently, we evaluated the efficacy of the supplementation of the two main CoQ isoforms in increasing endogenous CoQ levels.
The results show that CoQ9 represents the prevalent isoform in every phase of flies' life cycle, with the highest content in the thorax. Although the CoQ content increases during the first part of life starting from the larval stages, distinct trends were observed during aging in relation to the sex and the body segments (head, thorax, and abdomen), suggesting a tissue-specific role of CoQ.
The 2 weeks supplementation of two weeks old flies with different concentrations of CoQ9 and CoQ10 (15M and 75M) induced segment-specific CoQ uptake, higher in females. In particular, 75□M CoQ10 supplementation led to significant increase of CoQ10 content in all female segments, similarly to CoQ9 content although to a lower extent. While in males both isoforms induced limited variation. Despite the supplementation with 75M CoQ10 was the condition more effective in modulating the CoQ status of the flies, lifelong treatment didn't affect longevity. Further studies are required to investigate whether the supplementation could minimize senescence associated oxidative stress and cellular dysfunction.