



# **Autophagy: Chapter 10. Mitophagy Plays a Protective Role in Fibroblasts from Patients with Coenzyme Q10 Deficiency**

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Coenzyme Q10 (CoQ) deficiencies are clinically and genetically heterogeneous diseases that can occur due to defects of ubiquinone biosynthesis (primary deficiencies) or other causes (secondary deficiencies). Radical oxygen species (ROS) production and oxidative stress is a common consequence of dysfunctional mitochondria and CoQ deficiency. Mitochondrial damage induced by ROS can trigger mitochondrial permeability transition (MPT) by opening of non-specific high conductance permeability transition pores in the mitochondrial inner membrane. This, in turn, leads to a simultaneous collapse of mitochondrial membrane potential and the activation of selective elimination of depolarized and dysfunctional mitochondria by mitophagy. In this respect, mitophagy could be considered as a protective mechanism for elimination of potential harmful mitochondria. Mitophagy must be accompanied by mitochondrial biogenesis activation to compensate the mitochondrial loss. However, massive and persistent mitophagy may impair cell bioenergetics, autophagy flux, and mitochondrial biogenesis, and eventually may cause cell death.

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