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Alteration of mitochondrial dynamics and PTEN-induced kinase 1 expression in adipocyte differentiation

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Mitochondrial dysfunction is suggested to participate in the pathogenesis of type 2 diabetes mellitus (T2DM). PTEN-induced kinase 1 (PINK1) is the study focus among the mitochondria-related studies. Our previous study demonstrated that full-length PINK1 is processed to smaller fragments during adipocyte differentiation (adipogenesis), while inhibiting adipogenesis attenuates PINK1 proteolysis. The present study aimed at investigating the underlying mechanism. Our results indicated that levels of presenilin associated rhomboid like protein (PARL), a protease with PINK1-cleaving activity, and Drp1, the protein required for mitochondrial fission, were increased during adipogenesis. The proteolytic PINK1 fragments were released to the cytosol and associated with Parkin for inhibiting mitophagy. It suggests that full-length PINK1 is the guard against adipogenesis, which must be processed to allow the cells enter differentiation. Meanwhile, mitochondria were first lengthened by fusion to ensure their enhanced function for meeting the energy demands at early phase of adipogenesis, then switched to Drp1-dependent fragmentation at late stage for keeping the energy consumption as low as possible in the mature adipocytes for their energy reservoir nature. Furthermore, hampering both regulation of mitochondria dynamics and elimination of damaged mitochondria impaired insulin-dependent glucose uptake. In summary, PINK1-mediated mitochondrial dynamics may play an important role in the regulation of glucose homeostasis and mediating the bioenergetic adaptation during 3T3-L1 differentiation.

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