INSULIN MODULATES MITOCHONDRIAL STRUCTURAL AND FUNCTIONAL MOSAICISM IN BROWN ADIPOCYTES

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Since the discovery of the thermogenic role of brown adipocytes, there was consensus that the biochemical and metabolic function of their mitochondria is uniform. By switching the ATP production between glycolytic pathway and oxidative phosphorylation, brown adipocytes are able to produce heat in mitochondria through uncoupling protein 1 (UCP1). Thermogenically active brown adipocyte's mitochondria are characterized by clear morphological features (long, tightly packed cristae). The process of their biogenesis includes an increased number of mitochondria (by division), increase of their surface area, and incorporation of UCP1 as well as specific structural organization of the cristae. But, is it true that all BA mitochondria within one cell are structurally and functionally the same? Do they harbor the same set of enzymes? Actually, the very first cell mosaicism, e.g. Harlequin appearance was shown in brown adipose tissue. This unique uneven UCP1 expression suggests that brown adipocyte's mitochondria may be heterogeneous regarding production of ATP (bioenergetic) vs. heat (thermogenic) role. This presentation deals with structural and functional mitochondrial mosaicism and changes caused by insulin.

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