

CONTRIBUTION OF CANCER MITOCHONDRIA TO ONCOGENIC PROPERTIES

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Previous studies demonstrated that cancerous mitochondria could affect cellular activities. We hypothesize that mitochondria from cancer cells can regulate nuclear gene expression and contribute to oncogenic properties. To validate our hypothesis, mitochondrial functions and oncogenic properties were examined in transmitochondrial cybrids containing mitochondria derived from tumorigenic (MDA-MB-231 and MDA-MB-468) or non-tumorigenic breast cancer cell lines (MDA-MB-436 and MDA-MB-453), and normal breast epithelial cell line (MCF-10A) in the same nuclear background from mitochondria-depleted rho zero cells derived from osteosarcoma cell line 143B.TK.

In this report, we provided evidences that cancerous mitochondria demonstrated reduced respiratory chain function and oxidative phosphorylation activities. In addition, mitochondria derived from highly metastatic breast cancer cell lines contributed to cell proliferation and other oncogenic properties. Gene expression profiles of the cybrids revealed differential gene expression. Our results suggested that mitochondria-nuclear cross-talk play an important role in breast cancer development.