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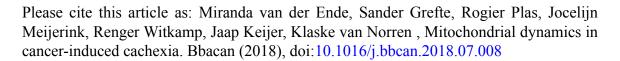
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Mitochondrial dynamics in cancer-induced cachexia

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Abstract

Cancer-induced cachexia has a negative impact on quality of life and adversely affects therapeutic outcomes and survival rates. It is characterized by, often severe, loss of muscle, with or without loss of fat mass. Insight in the pathophysiology of this complex metabolic syndrome and direct treatment options are still limited, which creates a research demand. Results from recent studies point towards a significant involvement of muscle mitochondrial networks. However, data are scattered and a comprehensive overview is lacking. This paper aims to fill existing knowledge gaps by integrating published data sets on muscle protein or gene expression from cancer-induced cachexia animal models. To this end, a database was compiled from 94 research papers, comprising 11 different rodent models. This was combined with four genome-wide transcriptome datasets of cancer-induced cachexia rodent models. Analysis showed that the expression of genes involved in mitochondrial fusion, fission, ATP production and mitochondrial density is decreased, while that of genes involved ROS detoxification and mitophagy is increased. Our results underline the relevance of including post-translational modifications of key proteins involved in mitochondrial functioning in future studies on cancer-induced cachexia.

Keywords

Mitochondria Mitochondrial dynamics Cancer-induced cachexia Muscle Animal models

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