Loss of O-GlcNAc Transferase Alters Mitochondrial Function

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Mitochondrial dysfunction is a common characteristic of many diseases, such as Alzheimer's disease, diabetes, and cancer. Normal mitochondrial function is regulated by post-translational modifications of proteins, which alter protein function. One post-translational modification that plays a key role in mitochondrial protein function is the attachment of a single N-acetylglucosamine (O-GlcNAc) sugar to proteins. This process, known as O-GlcNAcylation, is catalyzed by the enzyme O-GlcNAc transferase (OGT). Two experimental SH-SY5Y human neuroblastoma cell lines with shRNA mediated knockdown of OGT were compared to a control group to determine the effect of the loss of O-GlcNAc on mitochondrial function. Western blot analysis showed changes in the expressions of various mitochondrial proteins, suggesting that the reduction of O-GlcNAc transferase and the resulting decreased O-GlcNAc levels alter the mitochondrial proteome. Both oxidative phosphorylation and glycolytic rates were decreased in the OGT knockdown cell lines when compared to the control group. Changes in the enzyme O-GlcNAc transferase heavily impact normal mitochondrial function; therefore, further study of the role of O-GlcNAcylation in regulating mitochondria is necessary to understand more about this essential biological function.

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