

Abstract

Title – O-GlcNAcylation and Activity of Succinate Dehydrogenase

Program of Study – Biochemistry and Molecular Biology

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Subtype – Basic

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This presentation will look at how O-GlcNAcylation affects the activity of succinate dehydrogenase and how it may be involved in metabolic diseases such as diabetes and obesity. O-linked N-acetylglucosamine Transferase (OGT) is an enzyme that glycosylates proteins on serine and threonine residues. O-GlcNAcylation has been shown to play a prominent regulatory role in a variety of cellular processes, and aberrant O-GlcNAc signaling has been implicated in human diseases such as obesity and Type II diabetes (1). High glucose levels have been shown to increase O-GlcNAcylation, which has been connected to diabetes (2). One way in which excess O-GlcNAcylation may be involved in diabetes is by causing decreased metabolic function. O-GlcNAcylation has been shown to occur on complexes I, III, and IV of the electron transport chain in the mitochondria, and as glucose levels and O-GlcNAcylation increased, mitochondrial function was impaired (3). This presentation will explore whether this type of modification has effects on a principal mitochondrial enzyme, succinate dehydrogenase (SDH), which is involved both in catalyzing the conversion of succinate to fumarate in the TCA cycle and is a part of complex II in the electron transport chain. Key O-GlcNAcylation sites on SDH were mutated, then transfected into HeLa cells. An enzymatic assay was performed to examine the effects of O-GlcNAcylation on SDH activity by using the reduction of DCPIP. The SDH with a double mutation appeared to have decreased activity, suggesting that some O-GlcNAcylation is required for the function of SDH.

References

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