

JNK Signaling as a Mediator for Gene Differentiation in Human Umbilical Vein Endothelial Cells Exposed to High Glucose

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Diabetes is a growing and deadly epidemic around the world and particularly in the United States. Complications associated with diabetes arise from prolonged high blood glucose concentrations, known as hyperglycemia. These complications commonly include neuropathy, nephropathy, retinopathy, and foot disease. All of these listed complications are directly associated with the vascular system. Endothelial cells line the walls of the vascular system and experience direct contact with the glucose in the bloodstream. A stress signaling pathway, JNK, is known to cause apoptosis when exposed to high blood glucose. However, the mechanisms and changes in gene expression beyond apoptosis mediated by the JNK pathway under high glucose exposure has yet to be thoroughly investigated. This paper investigates the effects of short term high glucose exposure mediated by the JNK pathway on Human Umbilical Vein Endothelial Cells (HUVEC) using RNA sequencing analysis. The RNA sequencing analysis revealed differential expression of various genes crucial for energy production, neurological development, mitochondrial function, and the relationship between T-lymphocytes and endothelial cells. Further Gene Ontology (GO) analysis of differentially expressed genes dependent on JNK signaling in a high glucose environment revealed broader themes of JNK mediating differences in cell division and mitotic sister chromatid segregation. Ultimately, hundreds of differentially expressed genes were revealed as dependent on the JNK signaling pathway in the presence of high glucose. This is crucial for understanding the mechanisms behind the detrimental and deadly vascular complications of diabetes.