MAPing Out the Road for Preserving Donor Liver Viability from Ischemia-Reperfusion Injury: Unlocking the Secrets of the MAP Kinase Pathway in a Rat Model

Vasudev, Krish (School: University School of Milwaukee)

Background: Ischemia-reperfusion injury (IRI) is a significant challenge in organ transplantation, as restoring blood flow to an organ post-transplantation can cause further damage and threaten its viability. This study aimed to understand the cellular pathways involved in IRI to improve the outcomes of liver transplantation. The experiment focused on determining the phosphorylation of the MAP Kinase extracellular signal-regulated kinase 1/2 (ERK1/2) and related proteins p38 and JNK during IRI in rat liver samples that underwent IRI injury. Methods: Using a rat model of in vivo liver IRI, samples were obtained from control and IRI conditions by a qualified lab member, with 60-minute ischemia and 1-hour reperfusion time, and stored for analysis. Proteins were then extracted from snap-frozen liver samples and underwent protein quantification. ERK1/2, p38, JNK, phosphorylated ERK1/2, p38, and JNK proteins were identified by Western Blot and SDS-PAGE Polyacrylamide gel electrophoresis and confirmed by densitometry analysis. Results: An increase in phosphorylated ERK1/2, JNK, and p38 in liver samples that underwent IRI was found compared to normal liver samples and the GAPDH reference protein. Conclusions: Phosphorylation of MAP kinase proteins and activation of this pathway are involved in the cellular response to IRI in the rat liver. Understanding the mechanisms of IRI and how to prevent or mitigate this injury can help improve the success of liver transplantation.

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