Bryostatin-1 and Its Effect on Neutrophil Transmigration Through the Endothelial Layer in Liver and Kidney Ischemia-Reperfusion Injury Models

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Currently, there is a growing demand for organ transplants and an inability to meet such demand. Policies put in place by the United Network for Organ Sharing, or UNOS, restrict the acceptance of faulty organs in an attempt to reduce the likelihood of organ rejection in the recipient patient. These policies drastically reduce the availability of organs for transplantation. The purpose of this research project is to determine if liver and kidney organs can be treated with Bryostatin-1 in order to reduce the likelihood of Ischemia-Reperfusion Injury, which is one cause of organ rejection. In order to test this, Human Hepatic Sinusoidal Endothelial Cells (HHSEC) and Human Renal Glomerular Endothelial Cells (HRGEC) were cultured in order to be used for in vitro studies. These cells were also transformed into immortalized versions in order to extend their viability. Firstly, MTT assays were performed in order to determine if there was a variance in cell growth speed between both normal cell lines as well as between the transformed versions. Once it was confirmed that the transformed cells grow fastest, a western blot was performed probing for Large T-antigen SV40 in order to prove that the immortalization process was successful. Finally, transmigration experiments were set up in order to determine if Bryostatin-1 was an effective inhibitor of neutrophil transmigration. It can be concluded that Bryostatin-1 successfully reduces transmigration of neutrophils in HHSEC, HRGEC, and their immortalized counterparts and therefore may also reduce the risk of Ischemia-Reperfusion Injury in these organs.

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