The Implications of Hemorrhagic Shock on the Expression of Sirtuin 1 and Sirtuin 3

Griffith, Kira

Trauma is very commonly encountered by people across the globe. In one out of every five cases it takes the form of hemorrhagic shock. It is theorized that in order to reverse this process or detour it, the active work of the mitochondria, regardless of disturbances in the body, would need to occur. Within the mitochondria, there would need to be a high expression of the enzyme sirtuin 3 whose primary purpose is to deacetylate other enzymes that regulate oxygen levels in the blood. In order for this sirtuin to be activated, its activator sirtuin 1 would also have to be high in expression. This study was designed to discover how hemorrhagic shock affects the expression of sirtuin 1 and sirtuin 3 in liver tissue samples. The methodology for this experiment included the preparation of tissues from 30 individual rats induced with hemorrhagic shock for a traditional western blot with chemiluminescence. Mitochondria were isolated to measure the expression of sirtuin 3 and tissue lysates were prepared to measure the expression of sirtuin 1. It was hypothesized that the expression of both sirtuin 1 and sirtuin 3 would increase as the tissue progressed through the stages of hemorrhagic shock. We concluded that sirtuin 1's expression steadily increased throughout hemorrhagic shock. Sirtuin 3 however was unaffected by hemorrhagic shock. My hypothesis was partially accepted by these results and leads me to ponder and investigate why sirtuin 3 was unaffected by hemorrhagic shock.

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