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Biology, CBS, 2008
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Endothelial Biology of BOECs in Caucasians and African Americans

Afflicting 1 in 300 each year, stroke is the leading cause of long-term disability and the third leading cause of death in the United States. Accounting for ninety percent of all strokes, the most common type is ischemic stroke. During this condition, the blood supply to the brain stops, causing the death or apoptosis of nerve cells and the silmutaneous release of Ca^{2+} from the mitochondria into the cytosol. This elivated intracellular calcium concentrations causes an increase in the mitochondrial membrane permeability that in turn, triggers a flood of calcium and more widespread cell death or apoptosis as triggered by the enzyme phosphoserine phosphatase. This excessive cell death results in varying severity of ischemic damage.

While the magnitude of damage varies, African Americans have an up to three times greater risk of ischemic stroke than Caucasians do, and are more likely to die of it. The physiological basis to account for this striking statistical difference is not completely understood and the genetic basis remains to be determined. My project tests the hypothesis that there is a difference in the concentration and activity of the phosphoserine phosphatase enzyme in the Blood Outgrowth Endothelial Cells (BOECs) of African Americans and Caucasians. The results of the project may reveal the root cause of the striking statistical difference in risk of ischemic stroke between African Americans and Caucasians, and may imply ways to reduce cell death in patients who have had stroke.

Tissue culturing methods and resources were used to grow out BOECs that have been collected from 10 African Americans and 11 Caucasians. These cells were then harvested and the phosphoserine phosphatase was isolated. Then, Bradford assays were used to determine the concentration of the protein while activity assays and Western blotting techniques were used to examine its gene expression.

Poster Number: Session: