A Novel Application of Microfluidic Assay to Evaluate the Role of Calponin in Platelet Function and Clot Formation

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The World Health Organization states that cardiovascular disease is the leading cause of death worldwide. Fourteen million people die annually from cardiovascular disease and stroke. Antiplatelet medications such as Aspirin are used in the treatment and prevention of these diseases. However, antiplatelet drugs can cause life threatening bleeding complications. Calponin is an actin binding protein that converts external mechanical signals into intracellular chemical signals. My hypothesis was that if Calponin in platelets is inhibited, then platelet activation and spreading will be delayed without an increase in bleeding time because Calponin targets the actin cytoskeleton. The use of microfluidic assays represents a promising novel method to research platelet adhesion as they bridge the gap between in vivo and in vitro environments by controlling shear flow. Using a novel approach, I studied morphological changes between wild type platelets and KO (knock out) platelets that had been genetically modified to remove Calponin. Using DIC microscopy, I evaluated platelet spreading to determine whether the inhibition of Calponin affected spreading on collagen. To evaluate platelet spreading, I recorded the total number of platelets adhered and total area covered by adhered platelets at each time point. I also analyzed the fluorescence images of adherent platelets in microfluidic channels using Bioflux Montage image analysis software. My study found that Calponin inhibition does in fact delay platelet activation and spreading without an increase in bleeding time. These findings will aid in the creation of a new class of antiplatelet medications that help prevent and treat heart disease and stroke without additional bleeding complications, ultimately saving millions of lives.

Awards Won:

Fourth Award of $500