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Different Mechanisms of Action of Phospholipase Isoforms May Predict Effects on Neointimal Formation after Overstretch Injury to the Carotid Artery in Mice

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Recent research into why vascular disease is worse in people with diabetes suggests that the phospholipase A2 (PLA2) family of enzymes may be involved. We hypothesized that the calcium-dependent PLA2 alpha isoform (cPLA2α), which catalyzes the liberation of arachidonic acid from fatty acid substituents leading to formation of vasoactive prostaglandins and leukotrienes, may be more important than calcium independent isoforms (iPLA2) β and γ in the vascular response. To test this, we utilized a surgical technique that mimicked balloon angioplasty to damage the common carotid artery of cPLA2α, iPLA2β and iPLA2γ KO and WT mice in order to compare the relative effects of each isoform on vascular remodeling and neointimal thickening of the vessel wall over 3 weeks. Serial cross-sections of the vessel were then analyzed to determine the intima to media (I/M) ratio as a measure of neointimal thickening. We expect to see the least neointimal thickening in the iPLA2β and γ KO mice because of their role in membrane and mitochondrial metabolism. We expect to see the greatest neointimal thickening in cPLA2α KO since this isoform is most likely to be responsible for production of inflammatory prostaglandins, which can then potentiate smooth muscle cell migration and proliferation. The results will be important to determine whether drugs to inhibit one or more of these enzymes could decrease vascular disease progression after angioplasty in diabetic patients.