Platelets play important roles in coagulation by interacting with many plasma proteins and releasing thrombin and adenosine diphosphate (ADP) upon activation. Shear stress (SS), and secondhand smoke (SHS) are both known risk factors for platelet activation and cardiovascular disease. Interestingly enough it has been observed that platelets treated with both shear stress and secondhand smoke showed no difference in aggregation compared to samples treated with shear stress alone, at a level much lower than that observed with untreated platelets. We hypothesized that minor aggregation had occurred during shear stress treatment and ADP released from platelets could be a mediator. Ten different experimental conditions were applied to platelets: Untreated (used as control), SHS (1 cigarette/5 L), constant shear stress at 10 dyn/cm² with or without SHS (1 hour), constant shear stress at 30 dyn/cm² with or without SHS, normal pulsatile shear stress (maximum shear at 10 dynes/cm²) with or without SHS, and elevated pulsatile shear stress (maximum shear at 60 dynes/cm²) with or without SHS. Aggregation occurred during shear stress exposure was measured at 620 nm in a microplate reader. Platelet aggregation following treatments was induced by thrombin receptor activation peptide (TRAP) and measured in an aggregometer. A standard ADP assay was used to measure plasma ADP level. Preliminary results indicate that low level aggregation occurred when platelets were exposed to shear stress (especially under elevated shear), which might have caused the reduced aggregation induced by TRAP. This low level aggregation can potentially cause a person to hemorrhage when a wound is inflicted due to platelets not being able to properly clot. No significant changes were observed in plasma ADP concentration, suggesting ADP was not involved. Further investigation is needed to determine if shear stress has reduced TRAP-induced platelet aggregation by modifying platelet surface thrombin receptors.