The P-selectin/Triggering Receptor Expressed on Myeloid cells (TREM) Like Transcipt-1 (TLT-1) Double Null mouse as a Model for Cardiovascular Disease

Atherosclerosis is a cardiovascular disease (CVD) characterized by the buildup of plaque on artery cell walls. The formation and subsequent rupture of this atherosclerotic plaque leads to myocardial infarction and strokes. Anticoagulants such as aspirin or Plavix reduce platelet activation and the risk but leave patients with a propensity to bleed. Research is still ongoing to find alternative forms of treatment that do not compromise an individual's ability to clot. In recent years, a platelet receptor called Triggering Receptor Expressed on Myeloid cells (TREM) Like Transcript-1 (TLT-1) has been discovered to play a major role in hemostasis. Studies have shown that TLT-1 null (treml1$^{-/-}$) mice have reduced platelet aggregation and increased bleeding times, making it a possible therapeutic target. Another receptor found in platelets that is being evaluated is P-selectin, an adhesion molecule that mediates interactions with leukocytes. Similar to treml1$^{-/-}$ mice, P-selectin knockouts have shown to affect hemostasis. In order to evaluate TLT-1 and P-selectin as targets for the treatment of CVDs, the P-selectin/TLT-1 double null mouse will be developed and characterized in aspects associated to inflammation, hemostasis and thrombosis.