Platelets accumulate at the site of vessel injury and prime hemostasis, an event that may occur excessively in atherosclerotic lesions, leading to acute ischemic events. Current antiplatelet drugs are the cornerstone of the treatment of this widespread disease but their clinical benefit is limited by an increased risk for bleeding. Research towards new targets that would optimally balance antithrombotic effects and hemorrhagic risk is therefore mandatory.

In the present seminar, I will report a specific role for Panx1 channels in the signaling pathway leading to collagen-induced platelet aggregation. The importance of these ATP release channels is further supported by the association between a Panx1 400A>C genetic polymorphism and collagen-induced platelet reactivity. This gain-of-function Panx1 polymorphism may be of importance in future atherothrombotic risk assessment and prediction of efficacy of upcoming drugs targeting the collagen receptor GPVI.