During my visit to the Miles Lab at the SCRIPPS Institute in La Jolla, California, I was able to characterise the expression of a novel plasminogen receptor, Plg-R_{KT}, on the surface of mouse platelets. Confocal microscopy allowed visualisation of Plg-R_{KT} on the platelet surface, which was found to be primarily associated with PS-negative (spread) platelets but was also observed in the "cap" of PS-positive platelets. Plg-R_{KT} was observed to co-localise with platelet-derived plasminogen on the platelet surface confirming that endogenous plasminogen interacts with Plg-R_{KT}.

Flow cytometry analysis revealed a significant decrease in plasminogen exposure on the platelet surface upon thrombin stimulation in platelets isolated from Plg-R_{KT} KO mice compared to WT. Interestingly, platelets derived from plasminogen KO mice had significantly less Plg-R_{KT} on their surface than their WT littermates, suggesting that plasminogen and Plg-R_{KT} retention on the platelet surface augment one another.

Differences in plasminogen and Plg-R_{KT} exposure between sexes was identified. Males were found to have significantly increased levels of both plasminogen and Plg-R_{KT} on their platelet surface compared to females. Further analysis revealed that the additional pool of plasminogen in males was retained via Plg-R_{KT}. This was also reflected in plasmin activity assays, where males had a 10-fold increase in plasmin activity compared to females.

Our data indicates that Plg-R_{KT} is important in anchoring platelet-derived plasminogen on the activated platelet membrane and may therefore play a role in plasminogen activation on the cell surface.