The shelf-life of platelet concentrates for transfusion purposes is currently limited due to safety and quality reasons based on the risk of bacterial growth as well as a reduction of platelet functionality – called platelet storage lesion (PSL) - during blood bank storage, respectively. Pathogen reduction technologies (PRT) have shown to dramatically reduce the risk of infectious contamination of blood components; however, several in vitro studies have revealed a negative impact on the quality of platelet concentrates after PRT treatment which is in part explained as a general acceleration of the PSL. In order to minimize this undesired side effect, it is necessary to understand the underlying molecular mechanisms triggered by the PRT treatment.

We use combinations of biochemical and proteomic approaches to unravel alterations in the platelet proteome after MIRASOL (riboflavin/UV) treatment, a commercial pathogen reduction technology. Our recent studies have revealed some insights into potential molecular mechanisms triggered by the PRT treatment discovering an acceleration of the actin rearrangement in platelets during storage compared to a non-irradiation control. Furthermore, a phospho-protein kinase array unraveled the activation of certain protein kinases such as p38 MAPK during the PRT treatment. This finding was confirmed in an inhibitor study targeting p38 MAPK along with the striking observation that this inhibitor improves the in vitro platelet quality measures. However, the UV-triggered signaling mechanism is still unknown, but evidence points towards a link to apoptosis development. We have demonstrated for the first time that platelets exhibit a functional Fas receptor and biochemical analyses revealed its activation during Mirasol treatment.

These findings provide a potential signaling model for the underlying molecular mechanism in blood platelets triggered by the MIRASOL treatment and identify potential targets for intervention.