Our research team makes fundamental discoveries in cardiovascular research and translates these into improved clinical management of patients. Our core work on how high intraluminal pressure and lipids cause vascular inflammation, including their impact on monocyte and macrophage subsets, differentiation and polarization, as well as on endothelial activation and phenotype, is based on the central tenet that these mechanistic alterations occur via the cell membrane structural site, the caveola and its associated protein, caveolin-1. These concepts not only have discovery impact, but also clinical relevance in the context of patients with high blood pressure, hypercholesterolaemia, and coronary artery disease.

**Research Projects**

1. Patient induced algorithms of pulsatile pressure on vascular inflammation
2. Imaging and tracking monocytic-like cells in hypertensive zebrafish
3. Bioenergetics of monocyte/macrophage subsets in patients with dyslipidemia

**Selected significant publications:**


