Alexander Persson -Assistant professor

I have always held a strong interest in microbial induced inflammatory manipulation of innate immune cells. For years I have studied how *Mycobacterium tuberculosis* alters the life span of neutrophils and macrophages to be able to survive themselves. Initially, a dead neutrophil may seem like a failure but the dead cells in fact keep on combatting the infection after their death. They do so by a last effort in activating surrounding macrophages in a quite specifically targeted fashion. This is in stark contrast to the common notion that clearance of dead cells is an anti-inflammatory process, mediating resolution of inflammation. It turns out that why and how a cell dies dictates how active the legacy and after-life will be.

This work has evolved into inflammasome regulatory mechanisms and I am presently focusing on NLRP3 inflammasome regulation in neutrophils and macrophages. The “ON”-switch is quite well described in macrophages but we know little about how this work in other immune and non-immune cells. Another focus of mine is to describe the “volume-knob” of the inflammasome activity.

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