"Suppression of the innate immune response by a relative of the plague bacterium"

Millions of years of coevolution explain why many pathogens could develop intricate molecular mechanisms to suppress the human immune response. Central to the immunosuppressive activities of the plague agent Y. pestis and its enteropathogenic relatives Y. pseudotuberculosis and Y. enterocolitica is a molecular machine that injects bacterial effectors into immune cells. One group of these effectors subverts crucial immune cell functions such as downstream signaling of toll-like-receptors. However, sometimes bacterial effector activities conflict with the intended immunosuppression, i.e. because they downregulate the immunosuppressive Interleukin-10 or activate the inflammasome. To counteract these unwanted “side effects” of their own effectors, yersiniae have developed a second group of effectors, which i.e. “rescue” Interleukin-10 production.

I am a medical and cellular microbiologist heading an Institute at the University Medical Centre Hamburg-Eppendorf. In my talk I will address the cell biological effects and structural features of the Y. enterocolitica “rescue” effector YopM and how it modulates the inflammatory response in human macrophages.