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Inflammation can be seen as a double-edged sword, where this fundemental process initiates a plethora of effector mechanisms that aids in the elimination of microbes and other exposures that are harmful to the host. However, this same process can, if not properly regulated, lead to tissue damage and immune dysfunction, which sets the scene for other types of diseases such as chronic inflammatory disorders and cancer.

I started my research as a PhD student at the division for Medical Microbiology at Linköping University, where I studied the interaction between human macrophages and *Mycobacterium tuberculosis*. My special emphasis was on the role of the so called inflammasome, which plays a central role during inflammatory reactions. We could show that polymorphisms that increase inflammsome activity in macrophages also increased their capacity to kill intracellular bacteria.

After defending my thesis ”*Mycobacterium tuberculosis* and the human macrophage – shifting the balance through inflammsome activation” in December 2013, I was recruited as a post doc to the unit for Autoimmunity and Immune regulation at Linköping university, where I studied both anti-inflammatory mechanisms during pregnancy as well as the neuroinflammatory disease multiple sclerosis.

As of January, I work as an assistant professor at iRiSC, in the recently launched Synergy-project, which aims at studying the role and function of the inflammsome in a range of disorders and combining this knowledge in an interdisciplinary manner. My main interests is in studying the interaction between the inflammsome and metabolic pathways and try to understand how this interplay affects the outcome of different hyperinflammatory states such as sepsis and trauma-induced inflammation