The nervous and immune systems are traditionally thought of as two autonomous entities and are therefore generally studied separately. However, accumulating evidence suggests that the two systems often function in an integrated and coordinated manner. Mechanistically, the systems communicate through a common language of mediators and receptors which enables them to respond to the signals each produce. This interplay allows for a major bidirectional dialogue, which is adaptive in some settings, but can also become maladaptive and contribute to disease pathophysiology in others. For example, we highlighted that in the context of allergic airway inflammation, the lung sensory neurons were able to detect pro-asthmatic cytokines triggering the local release of neuropeptides. These peptides then drive the chemotaxis and activation of adaptive immune cells which leads to further release of interleukins. This series of events creates an inflammatory signaling loop that promotes allergic inflammation, something that can be abolished by silencing lung nociceptors. In lights of these findings, my future work will be devoted at unravelling how, when, and where neuro-immune interplay becomes maladaptive. I will invest immediate effort to investigate 1) the capacity of nociceptors to detect antibodies and allergens, and to examine 2) the role of nociceptors in cancer immunosurveillance. In sum, my overarching goal is to define a framework of the neuro-immune crosstalk at a system level, by deciphering how and which subpopulation of sensory neurons controls innate and adaptive immune responses, and to develop new targeted therapies for resolution of chronic inflammatory diseases.