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The context-dependence of neuro-immune interplay

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The sensory nervous and immune systems, historically considered autonomous, actually work in concert to promote host defense and tissue homeostasis. These systems interact with each other through a common language of cell surface G protein-coupled receptors and receptor tyrosine kinases as well as cytokines, growth factors, and neuropeptides. While this bidirectional communication is adaptive in many settings, helping protect from danger, it can also become maladaptive and contribute to disease pathophysiology. The fundamental logic of how, where, and when sensory neurons and immune cells contribute to either health or disease remains, however, unclear. Our lab and others have begun to explore how this neuro-immune reciprocal dialog contributes to physiological and pathological immune responses and sensory disorders. The cumulative results collected so far indicate that there is an important role for nociceptors (noxious stimulus detecting sensory neurons) in driving immune responses, but that this is highly context-dependent. To illustrate this concept, our findings in a model of airway inflammation, in which nociceptors seem to have major involvement in type 2 but not type 1 adaptive immunity.

Biography

Talbot's laboratory is at the inter-phase of neuroscience and immunology, a novel and highly dynamic field, and combines techniques of molecular and cell biology, optogenetics, tissue clearance and imaging, electrophysiology, neuroanatomy, behavior, and genetics. His aim is at identifying the mechanisms and molecules that regulate the interplay between the immune and sensory nervous systems in physiology and pathology. Ultimately, his aim is the defining a framework of the neuro-immune interplay at the system level, to decipher how and which sub-population of sensory neurons controls innate and adaptive responses, and to develop new targeted therapies for resolution of chronic inflammatory diseases.

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