

Postdoctoral Position: Complement Biology in Neuroinflammation

Laboratory of Complement Research and the
Immunology Center
National Heart, Lung and Blood Institute

Translational Neuroradiology Section
National Institute of Neurological Disorders
and Stroke

National Institutes of Health (NIH)

Dr. Claudia Kemper's laboratory, in the National Heart, Lung and Blood Institute (NHLBI), and Dr. Daniel Reich's laboratory in the National Institute of Neurological Disorders and Stroke (NINDS), both of which are located in the NIH Clinical Center in Bethesda, Maryland, are jointly recruiting a postdoctoral researcher to work in the area of complement biology in neuroinflammation.

Dr. Kemper's lab focuses on the new and noncanonical roles of intracellularly functioning innate immunity in the regulation of both innate and adaptive immune responses, with emphasis on mouse models and human samples. For example, the Kemper lab has previously shown that complement proteins, which normally circulate in the blood and detect and destroy bloodborne pathogens, also operate within immune cells. Such intracellularly active complement (the complosome) directly controls basic cell physiological pathways, such as mitochondrial activity, general metabolic reprogramming, and immune cell survival and effector function (Liszewski et al., *Immunity* 2013; Kolev et al., *Immunity* 2015; Arbore et al., *Science* 2016; Kolev et al., *Immunity* 2020; Niyonzima et al., *Sci Immunol* 2021). Recent exciting unpublished data from the Kemper lab indicate that the complosome also plays a central role in the cells conferring protective meningeal immunity.

Dr. Reich's lab studies the pathogenesis of multiple sclerosis (MS), with a particular focus on the etiology, detection, and monitoring of chronic neuroinflammation in human beings and nonhuman primates. Chronic neuroinflammation is thought to be a major determinant of long-term clinical disability progression in MS, and at present there are no effective treatments for this aspect of the disease. The Reich lab recently used MRI-informed single-nucleus RNA sequencing to uncover a central role for the early complement pathway in linking adaptive and innate immune activation in chronic white matter lesions of MS (Absinta et al., *Nature* 2021). The Reich lab is also leading several cutting-edge clinical trials, the goal of which is to ameliorate chronic neuroinflammation and kick-start tissue repair in MS.

We are now seeking a creative, skilled, collaborative, and ambitious postdoctoral fellow to join our labs to further study mechanisms by which complement dysregulation impacts neuroinflammation. The fellow will have access to a variety of experimental systems and models as well as state-of-the-art methodologies, ranging from single cell biology to metabolomics to intravital and magnetic resonance imaging. The ideal candidate will demonstrate a deep background in immunology and immunological methods, ideally in T cell biology and/or innate sensor systems; a good balance between collaboration and independence; and a strong publication record. Substantial experience with mouse and/or human cellular systems is important. A PhD and/or MD degree is required.

The candidate will be supported by the excellent Intramural NIH fellowship in a stimulating and interactive research environment, national and international collaborators, and an engaged and collaborative atmosphere in the Kemper and Reich labs.

To apply, email your CV and biosketch, as well as names of and contact information for three references, to:

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