



## Postdoctoral Fellow – PRRs in CNS autoimmunity

**Job Description:** Post-doctoral positions in neuroimmunology are available for enthusiastic, hard-working individuals to study “Innate immune pattern recognition receptors (PRRs) in CNS autoimmunity” in Dr. Mari Shinohara’s laboratory at Duke University School of Medicine at Durham, North Carolina. Expected date of start is early 2021. The successful applicant will investigate how the innate immune system impacts neuroinflammation in multiple sclerosis (MS) by using its animal model EAE. This project will emphasize the use of complex mouse models, molecular and biochemical approaches, and immunological techniques. For more information on the Shinohara lab, please visit: <https://sites.duke.edu/shinoharalab/>

Here are relevant publications from our laboratory:

- Deerhake ME, Danzaki K, Inoue M, Cardakli ED, Nonaka T, Aggarwal A, Barclay WE, Ji RR, Shinohara ML. 2020. **Dectin-1 limits central nervous system autoimmunity through a non-canonical pathway.** *bioRxiv*. <https://www.biorxiv.org/content/10.1101/2020.05.06.080481v1.full.pdf>
- Deerhake ME, Biswas DD, Barclay WE, Shinohara ML. **Pattern Recognition Receptors in Multiple Sclerosis and Its Animal Models.** *Front. Immunol.* 12 November 2019. <https://doi.org/10.3389/fimmu.2019.02644>
- Barclay W, Shinohara ML 2017. **Inflammasome activation in multiple sclerosis and experimental autoimmune encephalomyelitis (EAE).** *Brain Pathol.* 27(2):213-219.
- Inoue M, Chen PH, Siecinski S, Li QJ, Liu C, Steinman L, Gregory SG Benner E, Shinohara ML. 2016. **An interferon- $\beta$ -resistant and NLRP3 inflammasome-independent subtype of EAE with neuronal damage.** *Nat Neurosci.* 19(12):1599-1609.
- Inoue M, Williams KL, Oliver T, Vandenabeele P, Rajan JV, Miao EA, Shinohara ML. **Interferon- $\beta$  therapy against EAE is effective only when development of the disease depends on the NLRP3 inflammasome.** *Sci. Signaling.* 5, ra38.
- Inoue M, Williams K, Gunn MD, Shinohara ML. 2012. **NLRP3 inflammasome enhances T helper cell migration to the CNS during EAE progression.** *Proc. Natl. Acad. Sci. USA.* 109:10480-5. PMC3387125.

### Requirements:

Self-motivated individuals who have a PhD and/or MD. The ideal candidates should have a strong background in immunology and mouse disease models. Additional expertise in molecular biology, experimental neuroscience, or genomics is greatly advantageous. Experience in EAE is desirable.

Candidates will also develop skills in mouse models of autoimmunity and neurological disorders, immune function assays, flow cytometry, microscopy, biochemistry and molecular biology techniques. The candidate will also gain considerable experience writing manuscripts, reviews and grants along with oral presentations. Additional duties will include mouse colony management, training of undergraduate and graduate students, participation in department activities, presentations at lab, local and national meetings and any other assignments that the PI may request. Collaboration and working on multiple projects are highly encouraged.

Salary will be based on the NIH guideline.

To apply, please email cover letter (less than a page) with a short summary of research interests, CV, and the contact information of 3 professional references to Dr. Mari Shinohara ([mari.shinohara@duke.edu](mailto:mari.shinohara@duke.edu)).

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