### **BIOGRAPHICAL SKETCH**

Provide the following information for the Senior/key personnel and other significant contributors. Follow this format for each person. **DO NOT EXCEED FIVE PAGES.** 

#### NAME: Madhav Sharma

#### eRA COMMONS USER NAME (credential, e.g., agency login):

#### POSITION TITLE: Ph.D. Candidate

EDUCATION/TRAINING (Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)

INSTITUTION AND LOCATION	DEGREE (if applicable)	Completion Date MM/YYYY	FIELD OF STUDY
Delhi University, India	Bachelor of Science (B.Sc.)	July, 2017	Microbiology
Indian Institute of Science Education and Research, Kolkata India	Integrated Ph.D. (IPh.D.)	-	Biological Science

#### A. Personal Statements

I completed my Bachelor of Science (B.Sc.) from Delhi University, India in the field of Microbiology and Cleared Joint Admission Test for Masters (JAM) exam with a good rank. I moved to Indian Institute of Science Education and Research, Kolkata, India as Integrated Ph.D. student (MS+Ph.D.) for further studies where I joined Prof. Jayasri Das Sarma Lab because of my interest in Viral-induced neuroinflammation and neurodegeneration mechanisms and their potential therapeutic strategies. Viral infection initiates activation of Cellular sensors like TLRs, RLRs which in turn induce Interferons and Interferon stimulated genes like Ifit2. Though the role of Ifit2 in mounting the acute neuroinflammation is much studied, the impact of Ifit2 deficiency on virus spread to the spinal cord, and its associated chronic progressive neuroinflammatory demyelination upon RSA59 infection is much warranted. My studies showed Ifit2 deficiency causes uncontrolled viral replication and spread in the grey and white matter of the spinal cord whereas in WT mice RSA59 is restricted to grey matter and grey-white matter junction. Reduced number of CD4<sup>+</sup>T and effector CD8<sup>+</sup> T cells were observed in Cervical-lymph-node of these mice in addition to limited permeability of Blood-Brain-Barrier. The reductionist approach showed upregulation of Ifit2 mRNA in primary astrocytes upon RSA59 infection. ZO-1, a tight junction protein, expression suggest that Blood-brain-barrier is less permeable in Ifit2<sup>-/-</sup> mice compared to WT mice. Therefore, Ifit2 is not only required for efficient T cell priming in the CLN but also protects mice from developing severe chronic neuro-inflammatory demyelination. Overall, my research showed dual role of Ifit2 in not only restricting viral replication but also in preventing chronic phase demyelination.

### **B.** Positions and Honors

- Dr. SS Parmar Poster Award for best poster at the XXXVIX Annual E-Conference of Indian Academy of Neuroscience jointly organized by the Indian Institute of Science Education and Research Kolkata (IISER-K), in coordination with Netaji Subhas Open University (NSOU) and CSIR-Indian Institute of Chemical Biology (IICB), 15th-19th Dec 2021
- **Participated** in **Frontiers in Modern Biology (FIMB) symposium** 2020 organized by Department of biological science, IISER Kolkata, Nadia, west Bengal, 741246. 28<sup>th</sup> to 29<sup>th</sup> February 2020
- Presented a poster entitled as "The anti-viral role of Ifit2 in murine CoV Mouse Hepatitis Virus induced neuroinflammation" at the XXXVIII Annual Conference of Indian Academy of Neuroscience jointly organized by the Department of Animal Biology, School of Life Sciences, University of Hyderabad and the National Institute of Pharmaceutical Education and Research (NIPER), Hyderabad from 4th-7th October 2020.

## C. Contributions to Science

I am always keen to study Viral-induced neuroinflammation and neurodegeneration mechanisms and their potential therapeutic strategies. My current research is focused on the understanding mechanism of Mouse hepatitis virus (MHV) induced acute neuroinflammation and chronic demyelination, which mimics specific pathologies of human neurological disease Multiple Sclerosis. Inflammation triggered by MHV causes an increase in pro-inflammatory cytokines during the acute phase of infection, including interferons. Interferon-induced gene Ifit2 has been shown to play a critical role in countering numerous virus infections. My studies emphasized on the regulatory role of cellular protein Ifit2 in restricting murine- $\beta$ -coronavirus replication in brain and Spinal cord.

# D. Additional Information: Research Support and/or Scholastic Performance

1. IIT JAM, 2017- For admission into the Integrated Ph.D. course. All India Rank: 33.

2. CSIR-UGC NET DECEMBER 2019: <u>Subject</u>: Life Sciences. All India <u>Rank</u>: 60.