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Areas of Interest: Neurodegenerative diseases, Amyotrophic lateral sclerosis (ALS) and Frontotemporal dementia (FTD)

Postdoctoral Training:

Baylor College of Medicine, Houston, TX, USA (Nov 2023- April 2026)
Johns Hopkins University, School of Medicine, Baltimore, MD USA (Nov 2018- Nov 2023)
Stanford University, School of Medicine, San Francisco, California USA (Dec 2017- Oct 2018)

Education:

PhD: Cytogenetics Laboratory, Department of Zoology, Banaras Hindu University (BHU), Varanasi India.

Research Interest:

- The role of defective nucleocytoplasmic transport in ALS/FTD
- Pathophysiology of mislocalization and aggregation of RNA-binding proteins in motor neurons
- Nuclear protein quality control in neurons during aging and disease conditions

Personal statement:

We use molecular and cell biology approaches in order to gain a mechanistic understanding of nucleocytoplasmic transport defects and protein aggregation in neurodegenerative diseases. We are studying how mutations in ALS-causing genes disrupt nucleocytoplasmic transport machinery using expansion microscopy to perform super-resolution imaging of the nuclear pore complex in *Drosophila*, induced Pluripotent Stem Cell-derived motor neurons (iPSC-MNs) from ALS patients and human patient samples. Our long-term goal is to investigate the RNA metabolism and protein quality control pathways in ALS.

Additionally, we are interested in studying environmental factors as potential risk factors for ALS/FTD.

Models: *Drosophila*, induced Pluripotent Stem Cell-derived motor neurons (iPSC-MNs) from ALS/FTD patients and human patient samples

Publication:

- 1- **Dubey SK**, Chaubey D, Ikenaga C, Lin W, Bellen HJ, Lloyd TE. Aberrant nuclear pore complex degradation contributes to neurodegeneration in VCP disease. **Neuron** 2026, DOI: 10.1016/j.neuron.2025.11.017.
- 2- **Dubey SK**, Bellen HJ. A neuroprotective role of polyunsaturated fatty acids in C9orf72-ALS/FTD. **Nature Neuroscience** 2025, DOI: 10.1038/s41593-025-01920-7.
- 3- **Dubey SK**, Lloyd TE, Tapadia, MG. Disrupted nuclear import of cell cycle proteins in mHtt/PolyQ diseases cause neurodevelopment defects in cellular and *Drosophila* model. **Heliyon** 2024, DOI: 10.1016/j.heliyon.2024.e26393.
- 4- **Dubey SK**, Maulding K, Sung H, Lloyd TE, Nucleoporins are degraded via upregulation of ESCRT-III/Vps4 complex in *Drosophila* models of C9-ALS/FTD. **Cell Reports** 2022, DOI: 10.1016/j.celrep.2022.111379.
- 5- Lee H, Lee JJ, Park NY, **Dubey SK**, Kim T, Ruan K, Lim SB, Park SH, Ha S, Kovlyagina I, Kim KT, Kim S, Oh Y, Kim H, Kang SU, Song MR, Lloyd TE, Maragakis NJ, Hong YB, Eoh H, Lee G. Multi-omic analysis of selectively vulnerable motor neuron subtypes implicates altered lipid metabolism in ALS. **Nature Neuroscience** 2022, DOI: 10.1038/s41593-021-01000-6.
- 6- Cunningham KM, Maulding K, Ruan K, Senturk M, Grima JC, Sung H, Zuo Z, Song H, Gao J, **Dubey S**, Rothstein JD, Zhang K, Bellen HJ, Lloyd TE. TFEB/Mitf links impaired nuclear import to autophagolysosomal dysfunction in C9-ALS. **eLife** 2020, DOI: 10.7554/eLife.59419.
- 7- **Dubey SK** and Tapadia, M.G. Yorkie regulates neurodegeneration through canonical pathway and innate immune response. **Molecular Neurobiology** 2018, DOI: 10.1007/s12035-017-0388-7.