

Sameer Aryal, Ph.D.

CONTACT INFORMATION	Stanley Center for Psychiatric Research Broad Institute of MIT and Harvard Cambridge, MA	Phone: (413) 347-9726 E-mail: sameer.aryal@gmail.com LinkedIn: linkedin.com/in/sameer-aryal-nyu Website: www.sameeraryal.com
PROFESSIONAL SUMMARY	Computational and experimental neurobiologist with 10+ years of experience investigating molecular and cellular mechanisms of neuropsychiatric and neurodevelopmental disorders. Expertise spans synaptic biology, proteomics, transcriptomics, single-cell genomics, and integrative analysis of human and mouse multi-omics data, with particular depth in RNA biology of the brain, including mRNA translation, alternative splicing, and isoform regulation. Experienced in leading cross-disciplinary research programs, directly supervising research staff, developing scalable experimental and analytical workflows, and translating large-scale molecular findings into experimentally testable mechanisms.	
CORE SKILLS	• Integrative neurobiology: molecular and cellular mechanisms of psychiatric and neurodevelopmental disease; human–mouse and cross-model multi-omics integration • Synaptic biology and proteomics: biochemical synapse purification; quantitative mass spectrometry (DDA, DIA); network and pathway analysis • Transcriptomics and RNA biology: bulk and single-nucleus RNA-seq (10X, split-pool); mRNA translation, ribosome profiling, alternative splicing, isoform regulation, and activity-dependent gene expression • Computational biology: Python, R, MATLAB, Bash; WGCNA, graph modeling, and meta-analysis; high-performance computing (Slurm/SGE) • Experimental neurobiology: primary neuronal culture, viral perturbation, molecular validation, immunoblotting, imaging, and mouse pharmacology	
PROFESSIONAL EXPERIENCE	<i>Stanley Center for Psychiatric Research</i>, Broad Institute, Cambridge, MA <i>Research Scientist II</i> July 2026 – present • Lead cross-disciplinary computational and experimental studies across genetic mouse models of schizophrenia and bipolar disorder to identify convergent synaptic, cellular, and gene-regulatory mechanisms. • Directly supervise research associates and coordinate projects spanning mouse genetics, proteomics, transcriptomics, computational biology, and experimental neurobiology. • Perform cross-model meta-analyses of synaptic proteomic and transcriptomic datasets and integrate mouse findings with human postmortem brain data to identify conserved pathways, candidate biomarkers, and therapeutic hypotheses. • Translate large-scale molecular findings into mechanistic experiments addressing neuronal activity, synapse organization and function, cellular composition, RNA processing, and protein synthesis. <i>Research Scientist I</i> January 2024 – June 2026 • Led integrative bulk RNA-seq, single-nucleus RNA-seq, alternative-splicing, and synaptic proteomic analyses of <i>Srrm2</i> haploinsufficient mice. • Identified selective loss of SynGAP-γ, conserved <i>Agap3</i> mis-splicing, and reductions in mature oligodendrocytes and myelin-associated programs, validated using orthogonal molecular and imaging approaches. • Developed scalable experimental and computational workflows for whole-brain single-nucleus RNA-seq and directed mechanistic studies spanning RNA processing, synaptic biology, neuronal activity, cellular phenotypes, and pharmacological treatment. • Presented the <i>Srrm2</i> program as an invited speaker at the 2024 Stanley Center Scientific Advisory Board meeting.	

Postdoctoral Associate, Advisor: Morgan Sheng, M.D., Ph.D. October 2020 – December 2023

- Conducted WGCNA and differential analyses of human postmortem synaptic proteomics, revealing reduced synaptic protein networks and increased autophagy- and protein-homeostasis-associated signatures in schizophrenia and bipolar disorder.
- Led cross-species analyses of human and mouse synaptic proteomes to identify molecular pathway alterations shared between psychiatric disease and genetic model systems.
- Led quantitative proteomic analysis of postsynaptic density-enriched synaptic fractions from *Akap11* and *Grin2a* mutant mice. Identified convergent alterations in synaptic, ribosomal, mitochondrial, and RNA-processing pathways, and integrated proteomic findings with human synapse proteomics and mouse transcriptomic data to distinguish shared disease-associated pathways from model-specific changes.

Center for Neural Science, New York University, New York, NY

Graduate Research Assistant, Advisor: Eric Klann, Ph.D.

June 2014 – June 2020

- Investigated the RNA biology of fragile X syndrome, focusing on dysregulated mRNA translation using ribosome profiling, TRAP-Seq, primary neuronal cultures, molecular assays, and mouse genetics.
- Developed computational pipelines to quantify ribosome dynamics, coding-sequence-length-dependent translational changes, and protein synthesis rates from genome-wide datasets.
- Demonstrated that genetic removal of p70 S6K1 corrects coding-sequence-length-dependent translational dysregulation in fragile X syndrome mice.
- Contributed molecular and computational analyses linking cell-type-specific cortico-striatal circuit dysfunction to repetitive behavior in a fragile X syndrome model.

EDUCATION

Ph.D., Basic Medical Science (Neurobiology, Genomics), New York University, 2020 — Advisor: Eric Klann, Ph.D.

Dissertation: “Molecular and computational examination of *de novo* protein synthesis in fragile X syndrome”

B.A., Biology (Honors) & Economics, Williams College, 2012 — Advisor: Tim J. Lebestky, Ph.D.

Honors Thesis: “The role of DopR neuronal circuits in regulating endogenous arousal in *D. melanogaster*”

SELECTED PUBLICATIONS

- **Aryal, S.**, Geng, C., Kwon, M. J., Farsi, Z., Goble, N., Asan, A. S., Brenner, K., Shepard, N., Seidel, O., Wang, Y., et al. Srrm2 haploinsufficiency drives SynGAP- γ reduction, Agap3 mis-splicing, and oligodendrocyte deficits in a genetic mouse model of schizophrenia. *Cell Reports*, 45(6), 2026.
- **Aryal, S.**, Bonanno, K., Song, B., Mani, D. R., Keshishian, H., Carr, S. A., Sheng, M., Dejanovic, B. Deep proteomics identifies shared molecular pathway alterations in synapses of schizophrenia and bipolar disorder patients and mouse model. *Cell Reports*, 42(5), 2023.
- Longo, F., **Aryal, S.**, Anastasiades, P. G., Maltese, M., Baimel, C., Albanese, F., Tabor, J., Zhu, J., Oliveira, M. M., Gastaldo, D., et al. Cell-type-specific disruption of cortico-striatal circuitry drives repetitive patterns of behavior in fragile X syndrome model mice. *Cell Reports*, 42(8), 2023.
- **Aryal, S.**, Longo, F., Klann, E. Genetic removal of p70 S6K1 corrects coding sequence length-dependent alterations in mRNA translation in fragile X syndrome mice. *Proceedings of the National Academy of Sciences USA*, 118(18):e2001681118, 2021.
- Ho, J., Tumkaya, T., **Aryal, S.**, Choi, H., Claridge-Chang, A. Moving beyond P values: data analysis with estimation graphics. *Nature Methods*, 16(7):565–566, 2019.
- **Aryal, S.** & Klann, E. Turning up translation in fragile X syndrome. *Science*, 361(6403):648–649, 2018.

Full CV and publication list available at www.sameeraryal.com