



The Simons Center for the Social Brain Newsletter

Fall 2025

Inside this issue

SCSB Targeted Projects	2-5
SCSB Postdoctoral Fellows	6-8
Summer 2025 MRSP Students	9-10
Fall 2025 Events Overview	11
Upcoming Funding opportunities	12



**Simons Center
for the Social Brain**

SCSB Targeted Projects - Timeline



Targeted Projects - Updates

Since its inception, SCSB has supported collaborative targeted projects to address outstanding questions in autism. In this issue, we highlight the outcomes of two recently completed SCSB targeted projects, each bringing together leading investigators across institutions. The first project, The Role of the Thalamic Reticular Nucleus (TRN), involved four labs: Matthew Wilson (Picower Institute), Dara Manoach (Harvard/MGH), Guoping Feng (McGovern Institute), and Michael Halassa (McGovern Institute). The second project, The Nature of the Pragmatic Impairment in Autism Spectrum Disorders, involved six labs: Ev Fedorenko (McGovern Institute), Rebecca Saxe (BCS), Nancy Kanwisher (McGovern Institute), Edward Gibson (BCS), Laura Schulz (BCS), and Joshua Tenenbaum (BCS).

Role of the Thalamic Reticular Nucleus in thalamocortical coordination, cognitive processing, and sleep in ASD

by members of Manoach, Wilson, Feng, and Halassa labs

In 2016, SCSB initiated this 3-year targeted project by investigators **Matthew Wilson, Dara Manoach, Guoping Feng, and Michael Halassa**. The project focused on the **thalamic reticular nucleus (TRN)**, a thin layer of neurons in the thalamus, and its role in coordinating brain activity. The TRN acts like a “gatekeeper” or “switchboard” for information entering the cerebral cortex, and disruptions here could lead to the brain being overwhelmed by sensory input. The team hypothesized that a malfunctioning TRN could be a root cause of some of the key challenges faced by individuals with ASD, including issues with sensory processing, attention, and sleep.

By combining molecular mapping, animal models, human sleep studies, and cognitive neuroscience the team attempted to reframe how scientists think about the circuit roots of autism spectrum disorder (ASD) symptoms, from sensory overload to disrupted sleep.

A major finding reported in 2020 (Li et al., Nature 2020) used multi-scale single cell analyses to systematically examine the TRN neuron diversity by integrating transcriptomic profiling, electrophysiological recording, connectivity mapping, in situ localization, and in vivo functional perturbation. They identified marker genes *Spp1* (secreted phosphoprotein 1) and *Ecel1* (endothelin converting enzyme like-1) that allowed mapping of two functional TRN subnetworks with divergent sensory and limbic/prefrontal connectivity providing cell type-specific entry points for tackling ASD related attention, sensory, and sleep challenges.

In a separate study published in 2019 (Nakajima et al., Neuron 2019), using mouse models of ASD, the researchers were able to pinpoint specific neural

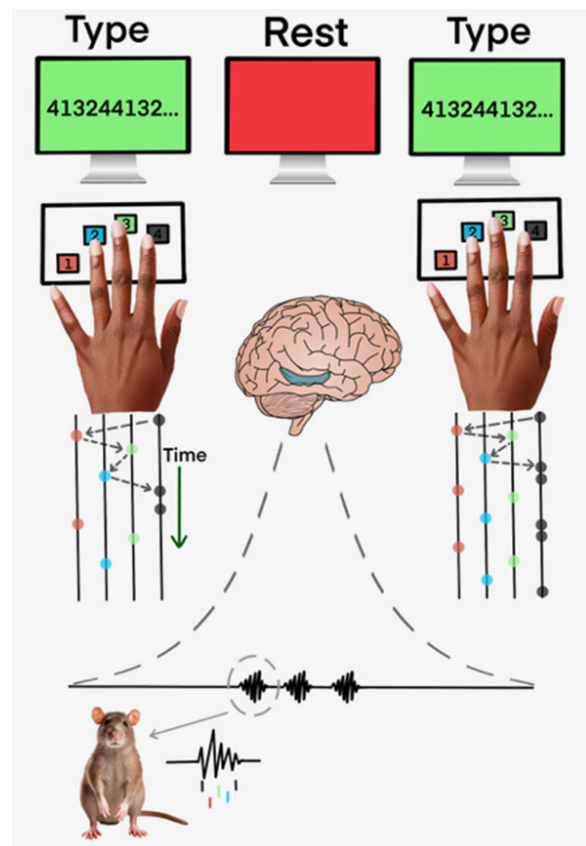


Fig. 1: While learning a typing task, epilepsy patients undergoing direct EEG monitoring of their hippocampus, got faster after brief rest breaks rather than while typing. There was a corresponding increase in hippocampal ripple rate that predicted these offline gains in speed, suggesting that ripples contribute to motor learning during wakeful rest. This study was inspired by work from the Wilson lab showing that when rats navigate a maze, they show increased hippocampal ripples during pauses, that these ripples co-occur with memory replay, and that interfering with these ripples impairs performance. The Manoach lab is now trying to detect ripple-related memory replay in humans and to determine whether it goes awry in people with neuropsychiatric disorders characterized by memory loss such as schizophrenia. Understanding this could improve our ability to treat memory loss in a range of conditions. (Image credit: Bryan Baxter and Grace Lee.)



circuits that contribute to noise hypersensitivity, a common symptom in autism. They demonstrated that by manipulating these circuits, they could effectively reverse this hypersensitivity using the procognitive drug, Modafinil, that enhances interactions between frontal thalamus and cortex along with engagement of the TRN. This finding suggests that targeting these brain networks could be a promising new avenue for therapeutic interventions.

Using simultaneously acquired MEG (magnetoencephalography) and EEG, the Manoach lab showed that learning a motor task shapes spindle activity (a read-out of TRN-mediated thalamocortical circuit function) during sleep and predicts how much people improve their performance after sleeping (Sjøgård et al., J Neurosci, 2025). They are now examining the consequences of TRN dysfunction on spindle activity in ASD and on this sleep-dependent improvement (Townsend et al., in preparation).

Additional research has extended the findings of the targeted project, providing new understanding of the brain circuits involved in autism and exploring new therapeutic strategies.

Recent work from the Manoach lab (Townsend et al., in preparation) has found a relationship between reduced sleep spindles, aberrant responses to auditory stimulation, and ASD symptom severity supporting their use as biomarkers of thalamocortical circuit dysfunction in ASD. Additionally, the Manoach lab has found that brain oscillations tied to memory (hippocampal ripples) that were studied as part of the original targeted project in rodent models, also predict learning over rest breaks in humans (**Fig. 1**; Sjøgård et al., Nat. Comm. 2025). They are now trying to identify ripples non-invasively in humans based on scalp EEG so that they can be used as a biomarker of memory.

Continued work from the Feng lab and collaborators has extended the circuit study approach to primate marmoset models (Krienen et al., Nature 2020) and has made important advances in the development of translational bridges for the study of behavioral circuits in ASD (Dragoi et al., Curr. Biol. 2025).

By integrating molecular, systems, and cognitive neuroscience approaches across species, the project created a roadmap for therapies that address the root circuitry behind core ASD symptoms.

The nature of the pragmatic impairment in autism spectrum disorders

by members of Fedorenko, Saxe, Kanwisher, Gibson, Schulz, and Tenenbaum labs

In 2015, SCSB initiated this 3-year targeted project involving 6 investigators: [Ev Fedorenko](#), [Rebecca Saxe](#), [Nancy Kanwisher](#), [Edward Gibson](#), [Laura Schulz](#), and [Joshua Tenenbaum](#). Language enables us to share knowledge, thoughts, and feelings with one another, and facilitates cooperative behaviors. One important feature of linguistic exchanges is that they often require going beyond the literal meaning. For instance, someone may describe a ruthless lawyer as a shark, or remark “Lovely weather!” when it’s pouring rain. Communicative success requires inferences about speaker goals. These inferences — **pragmatic reasoning** — was the focus of the SCSB targeted project. The project spanned three teams, all probing pragmatics using diverse approaches, including behavior, fMRI, ERPs, and computational modeling in neurotypical children and adults, and adults with autism. Below, we highlight key empirical discoveries and theoretical advances.

The language and the Theory of Mind networks jointly support pragmatic reasoning. Across several interrelated lines of work, [Fedorenko, Saxe, and Kanwisher](#) established that linguistic, social, and executive abilities—putative components of pragmatic reasoning — are supported by distinct brain networks: the language network, the Theory of Mind (ToM) network, and the Multiple Demand network, respectively. Although distinct, the language and the ToM network show some degree of synchronized activity, and both contribute to pragmatic reasoning (**Fig. 2**). Contra past claims, the Multiple Demand network does not contribute to pragmatic reasoning, or to language comprehension in general.

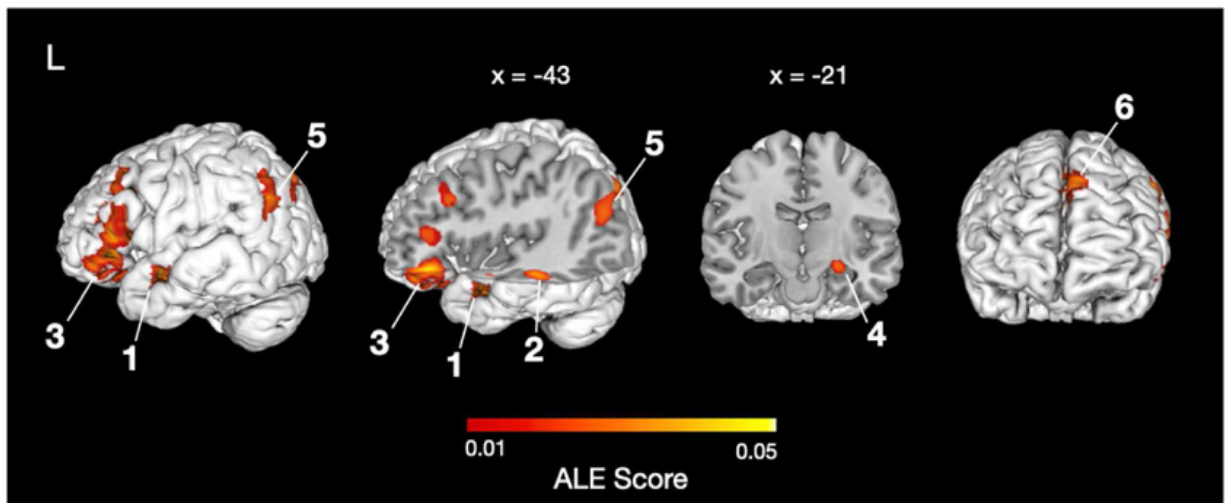


Fig. 2. Results of a meta-analysis of fMRI studies of pragmatic reasoning (Hauptman et al., Cortex 2023). By comparing these clusters against probabilistic atlases for different cognitive networks, the authors were able to conclude that the language network and the Theory of Mind network jointly support pragmatic processing, but the previously implicated executive control (Multiple Demand) network does not play a role.

Finally, Olessia Jouravlev (then a postdoc with Fedorenko and Gibson) found that these three networks are also distinct in autistic adults, but the language network is more bilateral due to stronger activity in the right-hemisphere areas, and this right-hemispheric activity is predictive of communicative impairments.

A computational framework for understanding rational actions, including speech acts. Julian Jara-Ettinger (then a postdoc with Schulz and Tenenbaum) developed a powerful framework — Naïve Utility Calculus — to explain how we understand others' actions, including critically, linguistic utterances. Language use is a communicative behavior, which is a kind of cooperative behavior, and cooperative behaviors are, in turn, a kind of social behavior. However, although this construal of language has a long history, Jara-Ettinger was the first to propose a unifying formal framework. He then systematically evaluated several critical predictions in a series of ingenious behavioral experiments with children and showed that the model accurately predicts behavior across diverse contexts.

Humans model the mental states of others. Working with Gibson and Fedorenko, Jouravlev carried out one of the first investigations of what has since become known as “altercentric cognition” — understanding how our behavior and brain responses are affected by the mere presence of others. Using ERPs, she found that when reading or listening to something that might be confusing to someone lacking the relevant context, neurotypical adults experience what appears to be empathetic confusion when another individual is present, but not when they are absent. More recently, Jouravlev found that autistic adults show this effect to a lesser degree, which may explain some of the pragmatic difficulties they experience.

Perhaps the best outcome of this project was its long-lasting impact on the PIs involved, leading to many discoveries beyond the project's timeline, new collaborations, and external funding. And the key junior scientists are now running successful labs — including Julian Jara-Ettinger (Yale), Olessia Jouravlev (Carleton), and Benjamin Deen (Tulane) — continuing to investigate questions at the intersection of language and social cognition. Thus, this project not only enabled substantial advances in our understanding of pragmatic reasoning, but also helped launch careers for several stellar young researchers.

For additional information on Targeted Projects, please visit: <http://scsb.mit.edu/research/targeted-projects/>

Postdoctoral Fellows

Welcome to new SCSB Fellows!



Rani Borbara, Ph.D.

Project: Linking Synaptic Pathology to Brain State Dynamics in Autism Spectrum Disorder Using *C. elegans* Model

Laboratory: [Steven Flavell, Ph.D.](#)

Ph.D. from: The Weizmann Institute of Science

Hobbies: Playing the Oud and hiking



Agata Wolna, Ph.D.

Project: The neural infrastructure of speech articulation in neurotypical and autistic populations

Laboratories: [Ev Fedorenko, Ph.D.](#) and [Karen Chenausky, Ph.D.](#)

Ph.D. from: Jagiellonian University in Kraków, Poland

Hobbies: Cooking, hiking, and movies



Qing Xie, Ph.D.

Project: Two-photon all-optical interrogation of synaptic plasticity in action

Laboratory: [Linlin Fan, Ph.D.](#)

Ph.D. from: Lehigh University

Hobbies: Playing Mario Kart, jogging outdoor and reading

Simons Center Postdoctoral Fellows - Alumni

We are proud to highlight two of the many remarkable alumni of the Simons Postdoctoral Fellows program [<https://scsb.mit.edu/people/scsb-postdoctoral-fellows/>] who have since launched their own independent laboratories. Their journeys reflect not only the impact of their training at MIT but also the lasting influence of the Fellows program in shaping the next generation of leaders in science.

Anila M. D'Mello, Ph.D. | Assistant Professor and Jon Heighten Scholar in Autism Research in The Department of Psychiatry and Peter O'Donnell Jr. Brain Institute at UT Southwestern; Faculty, The Department of Psychology, University of Texas at Dallas

SCSB Fellowship Project: Characterizing Neural Adaptation in Autism Spectrum Disorder

Lab: John Gabrieli, McGovern Institute

A fundamental finding in neuroscience is that repeated exposure to a stimulus results in reduced neural activity - a phenomenon referred to as 'repetition suppression'. Repetition suppression is a key marker of brain plasticity, reflecting the brain's ability to learn from repeated experience and even form predictions about regularities in the environment. Prior research in autism suggests a reduced ability to benefit from environmental regularities. This may contribute to lower-level difficulties such as sensory hypersensitivities, which can arise when the brain fails to efficiently filter or adapt to repeated, predictable inputs. An interesting question is whether difficulty suppressing neural activity for repeating or predictable inputs may also affect social communication, where challenges distinguishing what is new versus familiar could disrupt higher-order recognition of faces, voices, and people.



Current lab members in the D'Mello Lab at University of Texas Southwestern Medical Center.

I was awarded a Simons Postdoctoral Fellowship to investigate whether disruptions in repetition suppression are associated with such social difficulties in autism. My project entailed a comprehensive analysis of repetition suppression across multiple domains - faces, objects, print, and speech - using behavioral and neuroimaging measures. We found that autistic individuals showed reduced repetition suppression for faces, but not other stimulus categories, and that this reduction was correlated with greater challenges in social communication. At the systems level, reduced repetition suppression in face perception regions was linked to altered functional connections with higher-order face recognition areas, as well as microstructural differences in relevant white matter pathways. Together, these findings demonstrated that fundamental mechanisms of brain plasticity are selectively altered for face processing in autism, and contributes to an understanding of how difficulties forming stable neural representations of faces may cascade into higher-order impairments in social communication.

Notably, this Simons funded research directly motivated a number of important follow-up studies that were unrelated to the original aims. For example, during our Simons-funded project, we identified some puzzling sex differences showing that autistic females differed from autistic males even at the level of basic neural processes such as repetition suppression. This finding pushed us to intentionally recruit more autistic

females, which in turn revealed a broader and more troubling reality - that autistic females are dramatically underrepresented in research at rates far higher than autistic males. This work was highlighted by Scientific American and other media outlets, and contributed to a growing awareness of the importance of studying sex differences in autism. Another major insight that emerged from data collected as part of this project was that the spatial location of functional brain regions (such as face processing regions) is more variable in autistic than in neurotypical individuals. This observation has two important implications: it informs our understanding of how the autistic brain is organized, and it challenges the field's reliance on neuroimaging methods that average across participants. By ignoring individual variability, such methods risk obscuring meaningful differences that are central to understanding autism. Critically, much of my lab's current work has been influenced by these studies: a central goal of my research program is to develop neuroimaging and behavioral approaches designed to capture and respect individual variation rather than obscure it.

Beyond the research findings, the Simons Fellowship was transformational for my career in several ways. It provided critical data that allowed me to successfully compete for subsequent grants (including a Simons Bridge to Independence Award which has supported my transition to an independent faculty position), and it embedded me within the Simons Foundation network, which has been invaluable for collaborations, mentorship, and continued growth of my research program.

Michael Segel, Ph.D. | Assistant Professor of Stem Cell and Regenerative Biology, Harvard University

SCSB Fellowship Project: Exploiting retroelements for targeted gene insertion

Lab: Feng Zhang, Broad Institute of MIT and Harvard

After completing my PhD in developmental neuroscience and aging at the University of Cambridge, I joined Feng Zhang's group at MIT with two goals 1) To continue to explore the biology of the developing nervous system and 2) To develop new tools to bring emerging genome engineering tools to the brain.

Almost immediately, Feng got me hooked on an idea that would transform my understanding of biology: although less than 2% of the human genome encodes proteins, nearly half of it consists of genetic code from viral-like entities that have hacked their way into our genome throughout evolution. As a developmental neuroscientist by training, I wondered whether any of these viral-like genomes are turned on in the mammalian brain. What we found was quite surprising: some of these overlooked regions of the genome are not just active in the adult brain, but also that they form virus-like proteins that fulfill important roles for normal brain function.

Working with a fantastic team in Feng's lab, we were able to turn these virus-derived sequences into an entirely new gene therapy vector. We reasoned that if viruses are good at injecting genetic material into cells, maybe these native virus-like proteins could do the same thing, and, perhaps, being proteins natively found in our own tissues, would be better tolerated by our body's immune system. Since our initial publication, we've worked to scale these technologies as new gene delivery tools to be clinically useful, such as treating neurodevelopmental disorders.

Support from the Simons Center early on in this project and my academic career was critical, and I am so grateful that they took a chance on such a high-risk high-reward project — it has served as a launching point for my career. Now with continuing support through the Simons Foundation Fellows-to-Faculty Award, I have established my own independent lab at Harvard University's Department of Stem Cell and Regenerative Biology. In my own lab, we continue to study how these viral relics influence neurodevelopment and brain disorders, revealing that what was once considered "junk DNA" may actually be central to brain health and disease.



Mikey Segel, Assistant Professor of Stem Cell and Regenerative Biology.

Simons Center MSRP 2025 Summer students

The 2025 MIT Summer Neuroscience Program brought together a diverse group of undergraduate students from across the nation for an intensive 10-week research experience. Hosted by the Department of Brain and Cognitive Sciences, the Center for Brains, Minds, and Machines, and the Department of Biology, the program offered non-MIT students an opportunity to engage in hands-on research in world-class facilities. Funded in part by MIT's School of Science, the National Science Foundation, and the Simons Center for the Social Brain, the program focused on inspiring students from underrepresented backgrounds to pursue graduate studies and careers in basic research. Participants worked closely with faculty and graduate mentors on a range of cutting-edge projects, from exploring the neural mechanisms underlying behavior to investigating brain development and disorders.



Ramyla Dahmer | Rising Senior, Howard University

Laboratory: John Gabrieli, McGovern Institute for Brain Research, MIT

Project: Predictors of Improvement in Autistic Adults Following a Remote Mindfulness-Based Intervention

This summer, I was an MSRPbio student in the lab of John Gabrieli. The project I focused on investigated predictors of improvement in autistic adults following a remote mindfulness-based intervention. For some background - autism is a neurodevelopmental condition characterized by maladaptive differences in social communication and behavior, with many autistic individuals also experiencing mental health challenges such as anxiety and depression. While therapy is a common treatment for adverse symptoms associated with autism, it can be inaccessible or expensive to maintain long-term. Emerging evidence indicates that remote mindfulness-based interventions that promote mindfulness, the state of being aware of your surroundings in a nonjudgemental way, offer an accessible and cost-effective approach to alleviating negative mental health outcomes in autistic adults. However, little is known about how baseline measures of autistic individuals such as autistic traits, anxiety, stress, and trait mindfulness, predict their likelihood of improvement following a remote mindfulness-



Ramyla and her mentor, Dr. John Gabrieli.



Ramyla presenting at the MSRP Poster Session.

based intervention. In this project, we sought to reveal any significant baseline predictors through analyses of covariance and multivariate multiple regression models between baseline and change scores of collected measures. We found that a lower baseline mindfulness significantly predicts a higher improvement in mindfulness alongside anxiety, affect, and stress improvement. Most notably, we found that sex is the only demographic serving as a significant predictor of mindfulness with females predicting a higher improvement. These findings were not observed in similar experiments with non-autistic populations, suggesting an underlying relationship between mindfulness, autism, and sex. Further analyses of this relationship could contribute to more targeted, efficient, and potentially generalizable care for autistic populations with remote mindfulness-based interventions.



Noah Kabbaj | Rising Senior, Washington University in St. Louis

Laboratory: Linlin Fan, Picower Institute for Learning and Memory, MIT

Project: Uncovering Serotonergic Regulation of Plasticity Using All-Optical Physiology

This summer, I was an MSRP student in Dr. Linlin Fan's lab, where I studied how serotonin, an important neuromodulator, is involved in regulating plasticity in learning and memory. In my project, we hypothesized that serotonin encodes the salience of events and provides a modulatory signal that regulates hippocampal plasticity and learning.

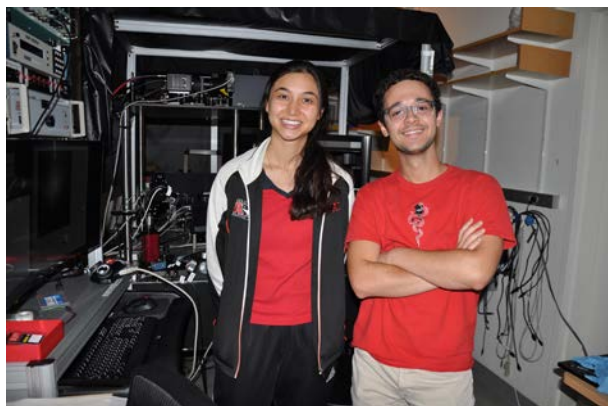
To investigate how serotonin may encode salience, we worked with mice with GPCR activation-based (GRAB) 5-HT sensors in the CA1 region of the hippocampus. Using fiber photometry, we monitored hippocampal serotonin in real time. We found increases in serotonin within the CA1 when animals were presented with sucrose or an air puff, oppositely valenced stimuli with similar salience. As a negative control, we used a mutant GRAB sensor and collected preliminary evidence indicating no change in response to sucrose or air-puff delivery. We also analyzed calcium activity of serotonergic cells in median raphe (MR) in mice exposed to two environments. In a blank virtual reality environment, we saw no change in calcium activity at the start of each trial, whereas a novel environment prompted an increase in activity at trial start.

Then, to understand how serotonin modulates CA1 pyramidal neuron physiology, we optogenetically excited serotonergic projections from the MR to the CA1. We found that this stimulation induced hyperpolarization in CA1 pyramidal neurons. We also found that stimulating non-projection-defined MR serotonergic neurons resulted in a twitch-like movement, while stimulating projection-defined MR serotonergic neurons to the CA1 had no impact on motion.

Next, we will employ all-optical physiology to simultaneously control and record membrane potential in CA1 pyramidal neurons in behaving mice. In these mice, we will also use optogenetics to excite or inhibit serotonergic release from the median raphe to the CA1. Combining these approaches, we will evaluate serotonin's impact on hippocampal plasticity and learning.



Noah with his mentor, Dr. Linlin Fan.
Photo courtesy of Mandana Sassanfar.



Noah with his supervisor, Graduate student Adrienne Kashay. Photo courtesy of Mandana Sassanfar.

Upcoming Events: Fall 2025

Colloquium Series



September 10
Carol Wilkinson, M.D., Ph.D.
Boston Children's Hospital



November 12
Uri Hasson, Ph.D.
Yale University



October 8
Carla J. Shatz, Ph.D.
Stanford University



December 10
Vikaas Sohal, M.D., Ph.D.
University of Arizona



October 29
Aakanksha Singhvi, Ph.D.
Fred Hutch Cancer Research Center

General Info

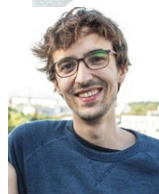
Time: 4PM–5PM, *reception to follow*

Location: Singleton Auditorium, 46-3002
registration is not required

Lunch Series



September 5
Gwangsu Kim, Ph.D.
SCSB Postdoctoral Fellow,
James DiCarlo Laboratory, MIBR



November 14
Marvin Lavechin, Ph.D.
SCSB Postdoctoral Fellow,
Computational Psycholinguistics Lab



September 19
Gabriel Stine, Ph.D.
SCSB Postdoctoral Fellow,
Mehrdad Jazayeri Laboratory, MIBR



December 5
Haoran Xu, Ph.D., Research
Scientist & **Beizhen Zhang, Ph.D.**,
Postdoctoral Fellow, Robert
Desimone Laboratory, MIBR



October 17
Caroline Robertson, Ph.D.
Associate Professor of Psychological
and Brain Sciences,
Dartmouth College

General Info

Time: 12PM–1PM

Hybrid Location: Simons Center Conference Room,
46-6011 + Zoom Meeting, *registration is not required*

*All events are open to public, please visit our website
for all upcoming events: scsb.mit.edu/events*



News & Announcements

Simons Postdoctoral Fellowship opportunities

The Simons Center has two rounds of funding annually for postdoctoral fellowships.

Fall 2025 deadline: Tuesday, September 30, 2025

Spring 2026 deadline: Tuesday, March 31, 2026

Postdoctoral Fellowships are intended for outstanding candidates with recent PhDs (please see eligibility criteria on our website) who wish to conduct autism-related research at MIT under the mentorship of MIT faculty researchers. Applicants currently completing their PhD outside MIT (external candidates), who wish to carry out postdoctoral research at MIT, are strongly encouraged to apply.

As part of the Brain & Cognitive Sciences complex at MIT, the Center offers supportive mentorship to postdoctoral researchers, an exceptional environment for scientific inquiry, and a strong commitment to an inclusive, welcoming culture. To learn more about our commitment, visit here: <https://scsb.mit.edu/our-values/>. To learn more about postdoctoral resources that support personal, family, and community life here at MIT, visit: <https://postdocs.mit.edu/>.

For information on how to apply, please visit our website at <http://scsb.mit.edu/funding/postdoctoral-fellowship-funding/>.

Support Our Research

We urge you to support the Simons Center for the Social Brain (SCSB) at MIT. Your gift will fund groundbreaking research into causes, mechanisms and treatments of neurodevelopmental disorders including autism spectrum disorders (ASD). The center supports laboratories that study the brain at multiple levels spanning molecular, circuit and computational mechanisms of brain function and cognition.

Our programs include funding for innovative, collaborative team projects and postdoctoral fellowships, and events that reach a wide audience, as well as outreach efforts within the larger community.

Please consider making a gift: Simons Center for the Social Brain - **Autism Research Fund 3836050**

Credits:

Editing and Design: Alexandra Sokhina

Cover Art: Artwork created collaboratively by children from the Cambridge Public Schools, Simons Center for the Social Brain staff and Postdoctoral Fellows, and members of Pawan Sinha Lab, MIT.

Contributing Writers: Targeted Project PIs and their laboratories, Michael Segel, Anila D'Mello, Ramyla Dahmer, Noah Kabbaj

SCSB team:

Director: Mriganka Sur | msur@mit.edu

Senior Administrative Manager &

Director of Development: Eleana MacPhail | ericci@mit.edu

Program Coordinator: Alexandra Sokhina | asokhina@mit.edu



Simons Center for the Social Brain
43 Vassar Street, Cambridge, MA 02139
<http://scsb.mit.edu>