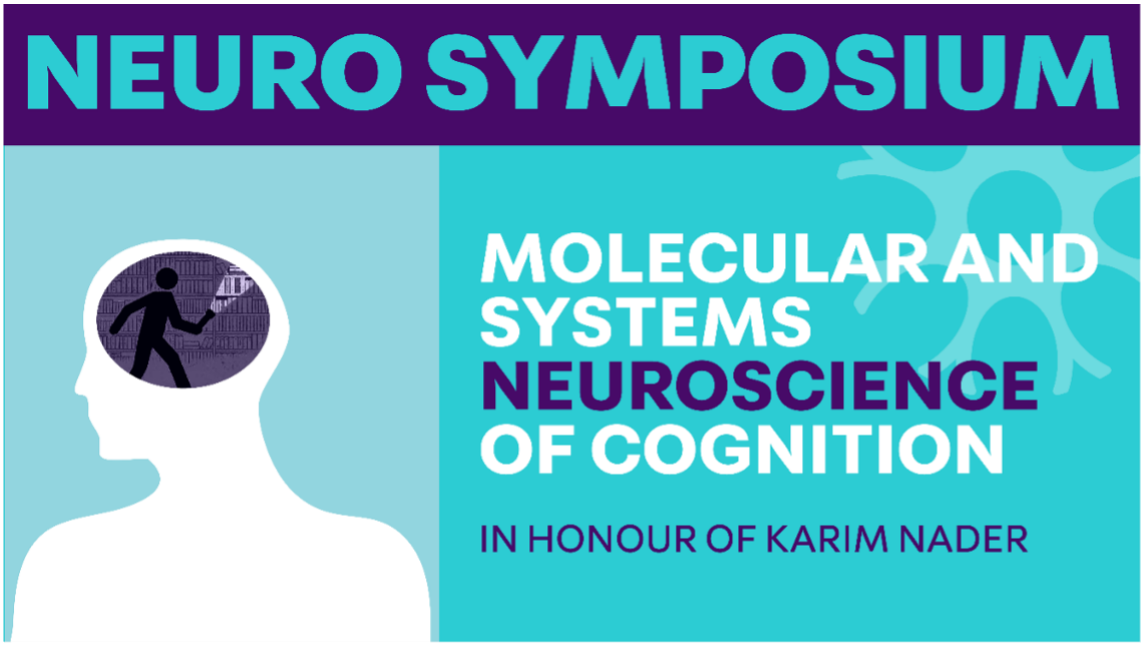

Program Booklet

August 22-23, 2022

Jeanne Timmins Amphithéâtre

The Neuro

3801 University Street

neuro
Institut-Hôpital neurologique de Montréal
Montreal Neurological Institute-Hospital

McGill

Centre universitaire de santé McGill
McGill University Health Centre

Karim Nader

Professor, James McGill Chair

Department of Psychology, McGill University

Initially labile, memory traces are thought to be stabilized into more enduring forms through a process known as consolidation. The consolidation framework guided neurobiological studies of memory in much of the 20th century. Studies in this era established that consolidation of long-term memories required *de novo* protein synthesis. However, consolidation was viewed as a one-way street: For any given memory, consolidation occurred once, and was irreversible.

This view was turned on its head by Karim Nader's seminal *Nature* paper in 2000 (cited >3000 times). Nader showed that retrieved fear memories once again became labile, requiring *de novo* protein synthesis to be re-stabilized through a process termed 'reconsolidation'. The compelling and mechanistic nature of Nader's work immediately brought reconsolidation to the fore, catalyzing studies that generalized this phenomenon across behavioral paradigms and species.

Nader's empirical and theoretical work on reconsolidation has profoundly shaped memory research in the 21st century, both in the lab and in the clinic. First, his studies highlighted that memories are not set in stone; rather, memory traces may be modified through reactivation. This dynamic view of memory aligned neurobiological studies of memory with psychological accounts that emphasize memory as a constructive process. Second, the ability to modify memories via reactivation has inspired reconsolidation-based therapies for targeting maladaptive memories.

He is a recipient of the prestigious Alfred P. Sloan, EJLB, and E.W.R Steacie Memorial Fellowships.

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Yadin Dudai
Weizman Institute (V*irtual*)

4:25 **Application of Reconsolidation Blockade to Psychiatric Treatment**
Roger Pitman
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University of Oxford

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SPEAKERS, HOSTS AND CHAIRS

PAUL FRANKLAND

Paul Frankland is a Senior Scientist in the Neurosciences & Mental Health Program at SickKids Research Institute. He holds a Canada Research Chair in Cognitive Neurobiology and is appointed as a Full Professor in the Department of Psychology, Department of Physiology, and Institute of Medical Science at the University of Toronto. He is a Fellow of the Royal Society of Canada, and a member of the Canadian Institute for Advanced Research (CIFAR) in the program for Child and Brain Development. Dr. Frankland's research program combines behavior, imaging and molecular approaches to understand the neurobiological basis of memory.

MAURO COSTA-MATTIOLI

Mauro Costa-Mattioli was the Cullen Foundation Endowed Professor in Neuroscience at Baylor College of Medicine (BCM). He was also the director of the Memory & Brain Research Center at BCM. He is currently a founding Principal Investigators at Altos Labs, Inc. Costa-Mattioli has elucidated central mechanisms underlying neurological dysfunction. He received his bachelor's degree in biology from the University of Republic (Uruguay) and PhD in microbiology from the University Nantes (France). He performed his postdoctoral training in neurobiology at McGill University (Canada). His laboratory has produced several important contributions to the understanding of the neurobiological basis of memory formation. He is best known for discovering that the protein homeostasis network dubbed the integrated stress response (ISR) is a universal regulator of long-term memory formation, and its activation the main causative mechanism underlying cognitive dysfunction in a wide range of memory disorders. His work has not only impacted the neurobiology of memory, but also industry efforts to develop drugs that target the ISR to promote brain health. More recently, Costa-Mattioli serendipitously discovered and characterized the mechanism(s) by which specific microbes in the gut modulate brain function and complex behaviors. Costa-Mattioli has received numerous awards, including the international Eppendorf & Science Prize in Neurobiology, the Searle Scholar award, the international society for neurochemistry's young investigator award, the UCSF presidential award, and serves in several editorial boards.

DEREK van der KOOY

Derek van der Kooy serves as Professor in the Department of Molecular Genetics, University of Toronto. Derek's lab works on stem cell biology and developmental biology, as well as genes important for learning and memory in worms, and the neurobiology of motivation in rodents. Of note, the lab discovered early embryonic primitive mouse brain stem cells, as well as adult mammalian retinal and pancreatic stem cells.

Derek received a M.Sc. in Psychology at the University of British Columbia, and a Ph.D in Anatomy, first at Erasmus University I in

the Netherlands, and finishing in the Department of Anatomy at the University of Toronto. He gained postdoctoral research experience at Cambridge University in England and at the Salk Institute in California.

Talk Abstract: Experiments done by Karim Nader and Tony Bechara when they were graduate students served to double dissociate two separate motivational systems in the brain. Lesions of the tegmental pedunculopontine nucleus (TPP) blocked the rewarding effects of morphine in previously drug naive animals but not in opiate dependent and withdrawn animals, whereas dopamine antagonists blocked the rewarding effects of morphine in opiate dependent and withdrawn animals but not in previously drug naive animals. This double dissociation was seen following both systemic and local brain ventral tegmental area (VTA) morphine injections. The data made a strong prediction that activation of GABA VTA neurons projecting to the TPP would be rewarding. However, other groups have shown that tonic 20 to 40 HZ or continuous optogenetic activation of VTA GABA neurons was motivationally neutral or aversive, respectively. Lyla El-Futomi has replicated these optogenetic motivational effects, but in addition demonstrated that activating VTA GABA neurons using laser pulse sequences that mimic the pattern of morphine induced action potentials elicits reward. Our results suggest that temporal coding of physiologically relevant firing patterns may be important in interpreting optogenetic neurobehavioral research.

ROGER PITMAN

Roger Pitman is a psychiatrist at Massachusetts General Hospital and Professor of Psychiatry at Harvard Medical School, Boston, MA. He served as a psychiatrist in the U.S. Navy during the Vietnam era and went on to complete a 30-year career in the Department of Veterans Affairs prior to moving to MGH. He is the recipient of the International Society for Traumatic Stress Studies' Award for Outstanding Scientific Achievement in the field of PTSD and its Lifetime Achievement Award. Dr. Pitman's research into the psychobiology of post-traumatic stress disorder (PTSD) spans more than 25 years. He has more than 100 peer-reviewed publications on PTSD and more than 200 overall publications in the general psychiatric and medical literature.

Talk Title: Application of Reconsolidation Blockade to Psychiatric Treatment

JOSEPH E LeDOUX

Joseph LeDoux is a University Professor and Henry and Lucy Moses Professor of Science at New York University, and directs the Emotional Brain Institute at NYU. His work is focused on the brain mechanisms of emotion, memory, and consciousness. LeDoux has received a number of awards for his research, and is an elected member of the American Academy of Arts and Sciences and the National Academy of Sciences USA. He is also the author of several books, including The Emotional Brain, Synaptic Self, Anxious (2016 APA William James Book Award), and The Deep History of Ourselves (finalist for the 2020 Pen America E.O. Wilson Award for Literary Science Writing). He is the 2023 President-Elect of the Association for the Scientific Study of Consciousness. As a sideline, he is the lead singer and songwriter in the rock band, The

Amygdaloids, and in the acoustic duo So We Are.

Talk Title**:** The Day I Told Karim Nader, “Don’t Do The Study”

YADIN DUDAI

Yadin Dudai is Professor of Brain Sciences (emeritus) at the Weizmann Institute of Science, Rehovot, and a Global Distinguished Professor of Neural Science at New York University. He graduated from the Hebrew University in Jerusalem in Genetics and Biochemistry with supplements in Modern History, and received his Ph.D. in Biophysics from the Weizmann Institute of Science. As a research fellow at the California Institute of Technology, Pasadena, he was among the pioneers of the field of neurogenetics of memory. Over the years he has contributed to the understanding of brain and behavioral mechanisms of learning and memory, with a focus on encoding, consolidation and modification of long-term and remote memory of naturalistic events in both animal models and in humans. In recent years he has extended his research to the investigation of the mechanisms of memory in human groups and cultures. Dudai published over 200 research papers and several books on memory. He has served as Dean of the Faculty of Biology and Chair of the Department of Neurobiology and Director of the Brain Research Centers at the Weizmann Institute, and scientific director of the Israeli Center of Research Excellence in the Cognitive Sciences. He is Chair of the Sciences Division of the Israeli Academy of Sciences and Humanities.

Talk Title and Abstract: Zeitgeists are Palimpsests of Reconsolidating Paradigms - The culture of scientific disciplines is dominated by the spirit of their time. This prevailing collection of dogmas, models, and opinions infiltrates not only the mindset of individual investigators but also that of reviewers of papers and grants, reinforcing the zeitgeist and extending its life. It takes a combination of originality, chutzpah, resilience and naiveté to disrupt this positive feedback loop. The conceptual framework of memory consolidation is just an example. When consolidation (not yet known by that name) was first proposed 2,000 years ago, it was noted that contrary to expectation, memory decay is not monotonously deterministic. Yet the re-emergence of the concept 1900 years later depicted it as a developmental program that fixates memory irreversibly. Early evidence that this not might be the case and that old memories can reconsolidate was quickly consolidated under the rug. Only the combination of a proper memory protocol, clean experimental design, a permissive research group, and the aforementioned captivating chutzpah that even open-minded colleagues could barely tolerate, renavigated the zeitgeist to a new version, possibly overenthusiastically overshooting so that the new zeitgeist now requires a new Karim to reconsolidate it. Additional tenets of memory research seem to follow the healthy path of doubt, aided by new armamentarium (e.g., optogenetics) and insights (e.g., epigenetics): Is memory indeed encoded in synaptic strength? Is memory transfer by chemical extracts a misguided target for damnatio memoriae? And, in another facet of the Science of Memory, how much of the memory of an individual is indeed stored in that individual's brain and how much in other interacting brains and artifacts? Here again, old layers of neglected data may unsettle accepted paradigms and rewrite pages in (electronic) textbooks, only to be altered again in generations to come. Consolidations never end.

SHEENA JOSSELYN

Sheena Josselyn is a Senior Scientist at The Hospital for Sick Children (SickKids) and a Professor in the departments of Psychology and Physiology at the University of Toronto in Canada. She holds a Canada Research Chair in Brain Mechanisms underlying Memory, and is a Fellow of the Royal Society of Canada. Her undergraduate degrees in Psychology and Life Sciences and a Masters degree in Clinical Psychology were granted by Queen's University in Kingston (Canada). Sheena received a PhD in Neuroscience/Psychology from the University of Toronto with Dr. Franco Vaccarino as her supervisor. She conducted post-doctoral work with Dr. Mike Davis (Yale University) and Dr. Alcino Silva (UCLA). Dr. Josselyn received several awards, including the Innovations in Psychopharmacology Award from the Canadian College of Neuropsychopharmacology (CCNP) and the Effron Award from the American College of Neuropsychopharmacology (ACNP). Dr. Josselyn is interested in understanding how the brain encodes, stores and uses information. Several human disorders (ranging from autism spectrum disorder to Alzheimer's disease) may stem from disrupted information processing. Therefore, this basic knowledge is not only critical for understanding normal brain function, but also vital for the development of new treatment strategies for these disorders. Here she will discuss her lab's work on examining the physical or functional representation of a memory in the brain (a memory trace or "engram") is formed and used in mice.

Talk Title: Reconsolidating Engrams - Understanding how the brain uses information is a fundamental goal of neuroscience. Several human disorders (ranging from autism spectrum disorder to PTSD to Alzheimer's disease) may stem from disrupted information processing. Therefore, this basic knowledge is not only critical for understanding normal brain function, but also vital for the development of new treatment strategies for these disorders. Memory may be defined as the retention over time of internal representations gained through experience, and the capacity to reconstruct these representations at later times. Long-lasting physical brain changes ('engrams') are thought to encode these internal representations. My lab studies engrams at the neuronal ensemble level. Here, I will be discussing unpublished data from Karim-inspired experiments examining engrams and memories.

WAYNE SOSSIN

Wayne Sossin received undergraduate degrees in Biology and Computer Science from MIT in 1984, a PhD at Stanford in Biological Sciences with Dr. Richard Scheller from Stanford in 1989. He did post-doctoral work with Dr. Schwartz at Columbia University in the Center for Neurobiology and Behavior before being appointed Assistant Professor at the Montreal Neurological Institute at McGill University in 1993 where he is now a James McGill Professor. Dr. Sossin has published over 125 papers on the molecular and cellular processes underlying memory formation and maintenance with a particular interest in the role of persistent protein kinases and the regulation of local translation in this process. He has proposed that specialized synapses that are particularly dependent on the continued presence of persistent protein kinases underlie the storage of memory with important implications for the fluidity of memory storage.

MICHAEL W. SALTER

Michael Salter is a Senior Scientist in the Neurosciences and Mental Health Program at SickKids Research Institute and is a Professor in the Department of Physiology at the University of Toronto. He completed his term as Chief of Research and now holds Chief of Research Emeritus with SickKids Research Institute. He received his MD at the University of Western Ontario and his PhD from McGill University. Dr. Salter is determining fundamental molecular and cellular mechanisms of normal and pathological neuroplasticity. His discoveries have broad implications for the control of cell-cell communication throughout the nervous system and his work has regularly appeared in elite journals including Nature, Science, Cell, Nature Medicine and Neuron. He is using his discoveries to design and test new types of treatment for individual suffering from a variety of disorders of the CNS. He is developing molecules that target major cell signaling pathways in neurons and in glial cells involved in pain, stroke, neurodegenerative diseases and schizophrenia. He has won numerous awards for his work, including an International Research Scholarship from the Howard Hughes Medical Institute, and he is a Fellow of the Royal Society of Canada.

Talk Title and Abstract: GRIN1 splicing and NMDAR regulation - NMDA receptors (NMDARs) are a crucial subtype of glutamate receptor playing key roles in a diversity of developmental, physiological, and pathological processes in the central nervous system. These receptors are heterotetramers comprised of two glycine-binding GluN1 subunits and two glutamate-binding GluN2 subunits. While NMDARs are well-known to function ionotropically, these receptors are increasingly being shown to also signal without ion flux. Non-ionotropic signaling by NMDARs is increasingly implicated in development, physiology, and disease, and in novel actions of CNS drugs. A major question about non-ionotropic signaling by NMDARs is what controls this signaling? Here, we investigated non-ionotropic signaling through NMDARs induced by glycine, which we refer to as ‘glycine priming’. We used recombinant NMDARs expressed in heterologous cells and tested the 8 splice variants of Grin1, the gene that encodes the GluN1 subunit. We found, unexpectedly, that not all the splice variants showed glycine priming. Rather, we discovered that glycine priming was only exhibited by the four splice variants lacking exon 5 of Grin1, an exon that encodes the N1 cassette in the GluN1 polypeptide. To determine whether this dichotomy is also seen with native NMDARs in neurons we investigated mice that we engineered to constitutively lack, or to constitutively express, exon 5. We found that wild type mice showed glycine priming of synaptic NMDARs in pyramidal neurons in the hippocampus. Glycine priming was robust in the mice in which exon 5 was excluded, but glycine priming was absent in mice in which we forced inclusion of exon 5. Together our findings reveal differential non-ionotropic signaling by specific splice variants of Grin1, which we anticipate having relevance to physiological and pathological processes in the central nervous system. This research was supported by a Canadian Institutes of Health Research Foundation grant (FDN-154336).

ENRIQUE LANUZA

Graduate in Biological Sciences from the University of València (1993), and PhD obtained in the same University in 1997, on comparative and evolutionary neurobiology, under the supervision of Fernando Martínez-García (Univ. of València) and Mimi Halpern (State University of New York). Postdoctoral training at the laboratory of Joseph E. LeDoux (Center for Neural Science, New York

University), with a Fulbright Fellowship (1999). Appointed Associate Professor of the Dept. of Cell Biology of the Univ. of València in 2002, and Full Professor in the same University since 2020. His research focuses on neural circuits underlying social and sexual behaviors, such as sexual attraction, aggression and maternal behavior, in which the amygdala plays a critical role.

Talk Title and Abstract: The neural base of territorial learning in mice - Mice are territorial animals and use urinary signals, detected by the vomeronasal system, to delimitate the boundaries of the individual territories. Consequently, learning the territorial map should incorporate the vomeronasal signals indicating individual identity (the "who" component of memory) into the hippocampal spatial representation. In this work we show that navigating a virtual environment induced synchronic activity in the vomeronasal amygdala and the dorsal CA1 of the hippocampus in the theta frequency range, and the detection of urinary signals elicited a common pattern of theta-nested gamma activity in this amygdalo–hippocampal network. The detection of urine stimuli induced synaptic plasticity (measured as long-term potentiation) in the vomeronasal pathway and the dorsal hippocampus, associated with the overexpression of pAKT and pGSK3β in the dorsal hippocampus. A newly described amygdalo-entorhino-hippocampal circuit likely underlies the formerly unknown influence of pheromonal information in hippocampal learning. We suggest that this circuit is the neural substrate of territorial behavior in mice, and it mediates the integration of social and spatial information, that is, the "who" and "where" components of episodic memory.

NATALIE TRONSON

Natalie Tronson, Ph.D. is Assistant Professor of Psychology at the University of Michigan. Her research focuses on the molecular mechanisms of learning and memory; the internal and external factors that can lead to changes in memory; and the role of memory dysregulation in psychiatric and neurological disorders including post-traumatic stress disorder, and post-operative cognitive decline. She is particularly intrigued by questions such as "Why do many patients develop post-traumatic stress disorder after heart attack?" and "What causes cognitive and memory decline after illness and surgery?" and "How do normal memories for trauma become pathological in post-traumatic stress disorder?" and "Why are women so much more susceptible to these than males?" Her lab focuses on mechanisms by which neuroimmune signaling and stress lead to enhancements or impairments in long term memory formation; in the role of memory reconsolidation after retrieval in maladaptive memory; and the similarities and differences in how males and females form and modulate memory.

Talk Title and Abstract: Memory reconsolidation and the dynamic structure of associative memories - Studies of memory reconsolidation opened the door to changing how we conceptualize memory. Most notably, this work shifted the narrative of memory as a stable entity to one that is dynamic – we now think of memory as something that can be updated, strengthened, and disrupted under some conditions, even after it is stored. But modifications of memory after retrieval are not universally observed, and across studies, some behavioral responses may be more likely to be disrupted after post-retrieval manipulations. This is commonly discussed in terms of the precise conditions required to observe manipulations of reconsolidation, but when identified within the same studies, sometimes within the same individuals, these differential vulnerabilities to post-retrieval memory disruption raise the possibility that some information – some components of memory – are more sensitive to disruption than others. Here, I will discuss how we might use and extend these data from memory reconsolidation experiments

to understand what information is learned during Pavlovian conditioning (and retrieved during recall), and thereby conceptualize a more complex structure of “simple” associative memories.

JOHANNES FELSENBERG

Johannes is a group leader at the Friedrich Miescher Institute for Biomedical Research in Basel, Switzerland. His research focuses on understanding the neural circuit mechanisms that allow to adjust learned information. To address the function of specific circuits, the Felsenberg lab uses a combination of genetic tools to manipulate neuronal activity, behavioral assays and in vivo calcium imaging. Utilizing these tools, his team aims to identify the general circuit motifs underlying memory re-evaluation in the relatively simple brain of the fruit fly Drosophila. Revealing common circuit principles in this context will help to understand how to re-write existing memories.

Talk Title and Abstract: Recovery of forgotten memory in *Drosophila* - Learned information can be forgotten. Encountering related cues, however, can reinstate the forgotten memory. How this change in accessibility of memory traces is achieved, is not understood. We developed a new paradigm to investigate the neuronal circuit mechanisms that underlie the recovery of forgotten memory in the fruit fly *Drosophila melanogaster*. Re-exposing flies to training-related reminder cues recovers forgotten aversive memories. In this process, the presence of contextual information that match the learning situation seem to be crucial. Addressing the circuits that underlie this process reveals that output from two different olfactory centers, the lateral horn and the mushroom body, seem to recruit a single pair of dopamine neurons to recover forgotten memory. The capacity of these two dopamine neurons to reinstall learned avoidance seem to depend on odor specific plasticity established during initial learning. This is in line with the finding that the identified dopamine pathway is known to strengthen learning in repeated experiences but not in initial training trials. Comparing the recovered memory with the initial memory reveals differences in strength and retrieval circuitry, suggesting that the recovery process can implement changes in the stored information. Indeed, we find evidence that changing the reminder settings has direct impact on the expression and the valence of the recovered memory. Based on these findings I will argue that recovering forgotten memories is a dopamine driven process with the potential to change the recovered memory.

JONATHAN PHILLIP BRITT

Jonathan Britt is an Associate Professor at McGill University. He earned his PhD at the University of Chicago under the mentorship of Dan McGehee and completed postdoctoral research at NIDA in the lab of Antonello Bonci. His research examines how striatal circuitry contributes to reward learning and psychopathology.

Talk Title and Abstract: Striatal circuit disruptions and compulsive grooming behaviour - Obsessive-compulsive disorder is associated with aberrant corticostriatal activity. In mice, stimulation of corticostriatal fibers for 5 minutes a day elicits compulsive grooming behaviour, which is a hallmark of mouse models of this pathology. Here, we show that stimulation of thalamic, but not hippocampal, inputs to the ventral striatum also induce compulsive grooming behaviour that persists for weeks. With the goal of informing therapeutic deep brain stimulation-based interventions, we are now testing in vivo synaptic plasticity protocols and trying to identify behavioural and physiological measures of pathway-specific synaptic strength in vivo.

JEFF MOGIL

Jeffrey S. Mogil is currently the E.P. Taylor Professor of Pain Studies and the Canada Research Chair in the Genetics of Pain at McGill University, and the past Director of the Alan Edwards Centre for the Study of Pain. Dr. Mogil has made seminal contributions to the field of pain genetics and is the author of many major reviews of the subject, including an edited book, The Genetics of Pain (IASP Press, 2004). He is also a recognized authority in the fields of sex differences in pain and analgesia, and pain testing methods in the laboratory mouse. Dr. Mogil is the author of over 270 journal articles and book chapters since 1992 and has given almost 400 invited lectures in that same period. His h-index is currently 91. He has trained almost 30 graduate students, postdocs, and visiting scholars, over 170 undergraduate research assistants, and lectures on pain to over 400 undergraduates every year. He is the recipient of numerous awards, including: the Neal E. Miller New Investigator Award from the Academy of Behavioral Medicine Research; the Patrick D. Wall Young Investigator Award from the International Association for the Study of Pain; the SGV Award from the Swiss Laboratory Animal Science Association; the Donald O. Hebb Award from the Canadian Psychological Association; the John C. Liebeskind (early career) and Frederick W.L. Kerr (lifetime achievement) awards from the American Pain Society; and Early Career, Distinguished Career, and Outstanding Mentorship awards from the Canadian Pain Society. He recently served as a Councilor at IASP and was the Chair of the Scientific Program Committee of the 13th World Congress on Pain.

Talk Title and Abstract: Pain, Sex, and Death - With the implementation of sex-as-a-biological-variable policies in funding agencies around the world, more and more evidence of sex differences in the biological mechanisms underlying pain is being uncovered. We have been performing experiments investigating the interaction of sex with another often-ignored factor of great relevance to pain: time. Evidence will be provided suggesting that the long-term sequelae of painful nerve injuries are sex-dependent in mice, and that pain affects lifespan in a sex-specific manner, likely due to telomere dysfunction-induced cellular senescence in spinal cord microglia. Our data reinforce the importance of performing all preclinical experiments on both sexes, and suggest that “chronic” pain in mice occurs over a much longer time span than is addressed by the current literature.

SZU-HAN WANG

Szu-Han Wang is an Alzheimer’s Research UK Senior Research Fellow and a senior lecturer with Centre for Clinical Brain Sciences, University of Edinburgh, UK. She leads a team and aims to understand the behavioural and biological mechanisms underlying memory persistence or decline in anxiety, in aging, or in dementia. She received training from Prof Karim Nader and obtained PhD from McGill University, Canada. Her postdoc positions were with Prof Paul Frankland in the Hospital of Sick Children, Toronto and with Prof Richard Morris in the University of Edinburgh. She received awards and supports from Caledonian Research fellowship (Royal Society of Edinburgh), Biotechnology and Biological Sciences Research Council new investigator grant, and Alzheimer’s Research UK fellowship.

Talk Title and Abstract: Reconsolidation: taming aversive memory and facilitating appetite memory -

Reactivating a memory can render the memory labile and susceptible to disruption. This has been demonstrated by Prof Karim Nader and colleagues since their Nature article in 2000 which reinvigorated reconsolidation research. Interfering reconsolidation that leads to the weakening of fear memory provides promising therapeutics for post-traumatic stress disorders. In this talk, I will cover work demonstrating a boundary condition of reconsolidation due to the strength of the fear memory and the correlated receptor mechanism - work that was developed in the Nader lab. I will then cover evidence demonstrating 'boosting' memory reconsolidation in improving persistence of appetitive memories which has implication in cognitive aging and in dementia.

PETER FINNIE

Peter Finnie is an Associate Research Fellow at the University of Toronto, Canada. He completed his BA in Psychology from McGill University in 2006. He then received a PhD in Psychology from McGill University in 2016. He worked as a Postdoctoral Research Fellow in the Bear Lab at the Massachusetts Institute of Technology. He began his research pursuits in the field of drug addiction and relapse, before transitioning to the development, implementation, and evaluation of clinical therapies in children diagnosed with a range of comorbid behavioural and/or developmental disorders.

SATOSHI KIDA

Satoshi Kida was an undergraduate at the University of Tokyo in 1989. He then received a Ph.D from the University of Tokyo in 1994. He worked in the Institute of Molecular and Cellular Biosciences in the University of Tokyo and then moved to Cold Spring Harbor Laboratory as a postdoctoral fellow. In 1997, he joined the Tokyo University of Agriculture as an associate professor and then became a professor in 2008. In 2019, he became a professor at the Graduate School of Agriculture and Life Sciences, the University of Tokyo. He is the President of Molecular and Cellular Cognition Society-Asia. He has focused on understanding the mechanisms of learning and memory and tried to develop methods to improve brain disorders such as PTSD. He also investigated roles of nutrient factors in brain function.

Talk Title and Abstract: Active transition of fear memory phase from reconsolidation to extinction through ERK-mediated prevention of reconsolidation - The retrieval of fear memory induces two opposite memory process, i.e., reconsolidation and extinction. Brief retrieval induces reconsolidation to maintain or enhance fear memory, while prolonged retrieval extinguishes this memory. Although the mechanisms of reconsolidation and extinction have been investigated, it remains unknown how fear memory phases are switched from reconsolidation to extinction during memory retrieval. Here, we show that an ERK-dependent memory transition process after retrieval regulates the switch of memory phases from reconsolidation to extinction by preventing induction of reconsolidation in an inhibitory avoidance (IA) task in male mice. First, the transition memory phase, which cancels the induction of reconsolidation, but is insufficient for the acquisition of extinction, was identified after reconsolidation, but before extinction phases. Second, the reconsolidation, transition, and extinction phases after memory retrieval showed distinct molecular and cellular signatures through CREB and ERK phosphorylation in the amygdala, hippocampus, and medial prefrontal cortex (mPFC). The reconsolidation phase showed

increased CREB phosphorylation, while the extinction phase displayed several neural populations with various combinations of CREB and/or ERK phosphorylation, in these brain regions. Interestingly, the three memory phases, including the transition phase, showed transient ERK activation immediately after retrieval. Most importantly, the blockade of ERK in the amygdala, hippocampus, or mPFC at the transition memory phase disinhibited reconsolidation-induced enhancement of IA memory. These observations suggest that the ERK signaling pathway actively regulates the transition of memory phase from reconsolidation to extinction and this process functions as a switch that cancels reconsolidation of fear memory.

AMY MILTON

Amy L Milton is a University Associate Professor at the University of Cambridge, UK. Her research focuses on the neurochemical and molecular mechanisms of memory reconsolidation, with a strong translational interest in the application of reconsolidation-based approaches for the treatment of mental health disorders including post-traumatic stress disorder, drug addiction and obsessive-compulsive disorder.

Talk Title and Abstract: Catching a fleeting moment: the challenges of memory destabilisation - Reconsolidation-based approaches to disrupting maladaptive emotional memories hold great promise for mental health disorders. However, data on the efficacy of reconsolidation-based interventions has been mixed even in animal models, with some studies finding long-term reductions in the behaviour of interest, and others finding no effect of amnestic agents. One major challenge for reconsolidation-based interventions, relevant to these apparent discrepancies, is demonstrating that the target memory has, in fact, been successfully destabilised by the memory reactivation procedure. Using examples of the reconsolidation of fear and drug memories in our rodent behavioural models, this talk will consider potential accounts for apparent failures to replicate, and the opportunities for both future research and the development of novel treatments.

CRISTINA MARIA ALBERINI

Cristina Alberini received her PhD in Immunological Sciences from the University of Genoa (Italy), and then trained in neurobiology as a post-doctoral fellow at Columbia University. From 1997-2000 she was an Assistant Professor in the Department of Neuroscience at Brown University and then Associate and Full Professor at Mount Sinai School of Medicine in New York from 2001 to 2011. In 2011 she joined the Center for Neural Science at New York University where she is currently a Professor in Neuroscience. Prof. Alberini's research focuses on understanding the molecular and cellular mechanisms underlying the consolidation and strengthening of long-term memories, as well as memory retrieval and reconsolidation. Her studies target different ages of the lifespan and different brain cell types. The results of her studies provide information for improving brain functions and developing potential therapeutics against cognitive impairments and psychopathologies. Prof. Alberini received the Hirschl-Weill Career Scientist Award, NARSAD Independent Investigator Award, Golgi Medal, Athena Award, MERIT Award and the 2018 Jacob K. Javits NYU Award, NYU Silver professorship, and is a Member of the American Academy of Arts and Sciences.

Talk Title and Abstract: From Reconsolidation to Autophagy in Memory formation and its Dysregulation in Cognitive Impairments - Prof. Alberini will briefly summarize the work of her laboratory in memory

reconsolidation, inspired by the studies of Karim Nader. She will then present and discuss some recent work of her lab on the role of autophagy in memory formation, in particular its link to protein synthesis. Finally, she will provide evidence on how disruption of autophagy is associated to cognitive impairments using a model of neurodevelopmental disorder.

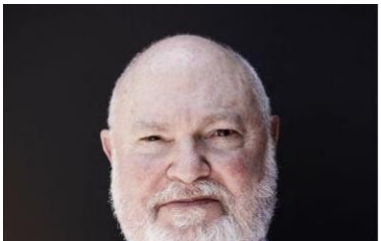
JOSUE HAUBRICH

After completing his PhD with Prof. Jorge Quillfedt and Prof. Lucas Alvares at UFRGS (Brazil) in 2017, Josue Haubrich spent the next 4 years working as a postdoc with Prof. Karim Nader and Prof. Oliver Hardt at McGill University. Currently, he is conducting a second postdoc with Prof. Denise Manahan-Vaughan at Ruhr University Bochum (Germany). Talk Title and Abstract: Memory consolidation and reconsolidation of severe fear memories - Manipulations targeting the reconsolidation process can alter a memory trace, by enhancing, impairing, or updating its content. However, severe fear learning can result in pathological memories that are resistant to change. Dr. Haubrich will be talking about his previous research at the Nader lab where he studied the differences in how normal and severe fear memories are formed and recalled.

CATHARINE RANKIN

Catharine Rankin is a professor in the Department of Psychology and a member of the Centre for Brain Health at the University of British Columbia. Dr Rankin is internationally recognized for her work using an invertebrate model system to address fundamental psychological questions about the effects of experience on the nervous system and behaviour. She was the first to show that the nematode *Caenorhabditis elegans* is capable of learning and memory, and has uncovered genes that play important roles in learning and memory. Her research is beginning to shed light on the cellular mechanisms of habituation, the simplest form of learning.

Talk Title and Abstract: Neuroligin Plays a Role in Ethanol-Induced Disruption of Memory and Corresponding Modulation of Glutamate Receptor Expression - Exposure to alcohol causes deficits in long-term memory formation across species. Using a long-term habituation memory assay in *Caenorhabditis elegans*, the effects of ethanol on long-term memory (> 24 hours) for habituation were investigated. An impairment in long-term memory was observed when animals were trained in the presence of ethanol. Cues of internal state or training context during testing did not restore memory. Ethanol exposure during training also interfered with the downregulation of AMPA/KA-type glutamate receptor subunit (GLR-1) punctal expression previously associated with long-term memory for habituation in *C. elegans*. Interestingly, ethanol exposure alone had the opposite effect, increasing GLR-1::GFP punctal expression. Worms with a mutation in the *C. elegans* ortholog of vertebrate neuroligins (*nlg-1*) were resistant to the effects of ethanol on memory, as they displayed both GLR-1::GFP downregulation and long-term memory for habituation after training in the presence of ethanol. These findings provide insights into the molecular mechanisms through which alcohol consumption impacts memory.



DAVID GLANZMAN

David Glanzman graduated from Indiana University with a B.A. in psychology in 1973. He completed his Ph.D. in psychology at Stanford University in 1980. Afterwards, he did postdoctoral research in neurobiology and behavior with Frank Krasne in the Department of Psychology at UCLA, and with Eric Kandel at the Howard Hughes Medical Institute at Columbia University. In 1990 he returned to UCLA as an Assistant Professor. Currently, he is a Distinguished Professor in the Departments of Integrative Biology and Physiology, UCLA College, and Neurobiology, in the David Geffen School of Medicine at UCLA. In addition, he is a Council Member of the Integrative Center for Learning and Memory of UCLA's Brain Research Institute.

Talk Title and Abstract: Potential encoding of memory by nuclear mechanisms in Aplysia - Recent evidence from Aplysia indicates that memory can persist after learning-induced synaptic changes have been erased. Furthermore, epigenetic changes, particularly DNA methylation, are critical for the persistence of long-term memory (LTM) in Aplysia. This suggests that LTM in Aplysia may be maintained by nuclear changes in neurons. A potential nuclear change that might be critical for encoding memory is retrotransposition. To test the possibility that LTM in Aplysia depends on retrotransposition, we examined the effect of inhibitors of reverse transcriptase (RT)—the enzyme that synthesizes DNA from RNA—on serotonin (5-HT)-induced facilitation of Aplysia sensorimotor synapses in dissociated cell culture. Five spaced pulses of 5-HT (5X5-HT training) induce facilitation of sensorimotor synapses that persists for ≥ 24 h (long-term facilitation or LTF). Treatment with either the nucleoside RT inhibitor lamivudine or the non-nucleoside RT inhibitor rilpivirine blocked LTF. Neither drug altered baseline sensorimotor synaptic transmission. In addition, we tested whether either RT inhibitor altered short-term facilitation (STF) of in vitro sensorimotor synapses due to a single, brief (2-min) application of 5-HT. Surprisingly, both lamivudine and rilpivirine impaired STF. The rapid time course of the effects of the RT inhibitors on STF appears inconsistent with disruption of retrotransposition; thus, RT may be critical for a retrotransposon-independent mechanism of synaptic plasticity. Taken together, our results imply that RT plays a necessary, heretofore unsuspected, role in both short-term and long-term memory in Aplysia.

SCOTT WADDELL

Scott Waddell studied biochemistry as an undergraduate at the University of Dundee, and researched cancer biology for his Ph.D. at the University of London. After postdoctoral study in the Department of Brain and Cognitive Sciences at Massachusetts Institute of Technology he spent 10 years leading a research group in the Department of Neurobiology at the University of Massachusetts Medical School. Scott moved to Oxford as a Professor of Neurobiology and founding member of the Centre for Neural Circuits & Behaviour in November 2011. His group studies neural circuit properties of memory-directed behaviour, using the fruit fly. Scott is a Wellcome Trust Principal Research Fellow in Basic Biomedical Science, a Fellow of the Academy of Medical Sciences, a member of EMBO, and was awarded the 2014 Liliane Bettencourt Prize for the Life Sciences.

Talk Title and Abstract: Multisensory learning binds modality-specific neurons into a cross-modal memory engram - Associating multiple sensory cues with objects and experience is a fundamental brain process that improves object recognition and memory performance. However, neural mechanisms that bind sensory features during learning and augment memory expression are unknown. Here we demonstrate multisensory appetitive and aversive memory in *Drosophila*. Combining colors and odors

improved memory performance, even when each sensory modality was tested alone. Temporal control of neuronal function revealed visually-selective mushroom body Kenyon Cells (KCs) to be required for both enhancement of visual and olfactory memory after multisensory training. Voltage imaging in head-fixed flies showed that multisensory learning binds activity between streams of modality-specific KCs, so that unimodal sensory input generates a multimodal neuronal response. Binding occurs between regions of the olfactory and visual KC axons, which receive valence-relevant dopaminergic reinforcement, and is propagated downstream. Dopamine locally releases GABA-ergic inhibition to permit specific microcircuits within KC-spanning serotonergic neurons to function as an excitatory bridge between the previously 'modality-selective' KC streams. Cross-modal binding thereby expands the olfactory memory engram by recruiting visual path KCs to become odor responsive. This broadening of the engram improves memory performance after multisensory learning and permits a single sensory feature to retrieve the memory of the multimodal experience.

NAHUM SONENBERG

Nahum Sonenberg received his Ph.D. in Biochemistry from the Weizmann Institute of Science (Rehovot, Israel) in 1976. He joined the Roche Institute of Molecular Biology in Nutley, New Jersey as a Chaim Weizmann postdoctoral fellow with Aaron Shatkin. In 1979 he moved to Montreal to become a Professor in the Department of Biochemistry at McGill University. Currently he is a Gilman Cheney Chair in the Department of Biochemistry and the Rosalind and Morris Goodman Cancer Research Centre at McGill. Dr. Sonenberg studies the molecular basis of the control of protein synthesis in eukaryotic cells and its importance in diseases such as cancer, obesity, diabetes and neurological diseases. He made seminal discoveries demonstrating that control of translation initiation is implicated in cancer, learning and memory, autism and fragile X-syndrome. For his research he received, among many others, the Wolf Prize in Medicine in 2014. He is elected member of many prestigious societies: The Royal Society of London; he is Associate Member of the EMBO, and a Foreign Associate of the National Academy of Sciences of the USA and Member of the National Academy of Medicine, USA.

Talk Title and Abstract: Astrocyte-dependent mnemonic processes via eIF2-mediated translational control - The integrated Stress Response (ISR) signaling pathway plays a critical role in cellular stresses by modulating mRNA translation. Genetic or pharmacological manipulations to suppress ISR in mice promote long-lasting memory and correct memory deficits in aged animals and models of Alzheimer's disease. We used molecular genetics to dissect the role of the ISR in astrocytes and the mechanisms by which the astrocyte-specific ISR manipulation affects cognitive processing. We found a reduction in eIF2α phosphorylation (p-eIF2α) on Ser51 in astrocytes following learning. Moreover, selective reduction of p-eIF2α in astrocytes in the CA1 region of the hippocampus in young mice enhances spatial memory in the Morris water maze (MWM) task and lowers the threshold for the induction of L-LTP by enhancing neuronal excitability. The results demonstrate that ISR activity via p-eIF2α phosphorylation in astrocytes affect cognitive processing. This provides a basis to search for modifiers of this pathway to combat cognitive impairments associated with aberrant eIF2 signaling such as aging, Alzheimer's disease, and other neurodegenerative disorders.

SHERNAZ BAMJI

Shernaz Bamji is a Professor in the Department of Cellular and

Physiological Sciences, the Associate Director of the Center for Brain Health at UBC, and President elect of the Canadian Association for Neuroscience. She received her Ph.D. at McGill University with Dr. Freda Miller studying the molecular mechanisms underlying apoptosis in the nervous system and did her postdoctoral training at the University of California, San Francisco with Dr. Lou Reichardt studying the role of adhesion molecules in synapse formation. Dr. Bamji has a long-standing interest in understanding the molecular and cellular mechanisms underlying neural connectivity and synaptic plasticity. Her work has provided valuable information about fundamental mechanisms underlying learning and memory, as well as how these processes are perturbed in diseased states.

Talk Title and Abstract: Dynamic Palmitoylation of Synaptic Proteins in Learning and Memory - Palmitoylation is the most common post-translational lipid modification in the brain and has been shown to be important for neurite outgrowth, spine and synapse formation, synaptic transmission, and synaptic plasticity. While ~10% of all proteins in the genome are substrates for palmitoylation, a striking 41% of all synaptic proteins can be palmitoylated. Enzymes that mediate palmitoylation have been shown to be associated with brain disorders, further underscoring the importance of palmitoylation in brain development and function. My talk will demonstrate the importance for palmitoylation in regulating synapse formation and plasticity.

CATIA TEIXEIRA

Catia M. Teixeira conducted her PhD research in the laboratory of Dr. Paul Frankland at the University of Toronto. There she focused on the cellular and molecular mechanisms of long-term memory formation. She earned her PhD in 2007. She then joined the laboratory of Dr. Eduardo Soriano in Barcelona where she studied how the dysregulation of certain genes involved in neuronal development affect adult behavior. She continued her post-doctoral training at Columbia University with Dr. Mark Ansorge where her studies and research focused on the developmental critical periods during which exposure to commonly prescribed/abused drugs affect adult-behavior. In 2015, she started her independent research efforts at Nathan Kline Institute/New York University where she focuses on how developmental manipulations lead to long-lasting alterations in circuit function and behavior. Her research is funded by the NIH.

Talk Title and Abstract: Role of PFC activity in the behavioral deficits induced by maternal separation: Environmental factors have profound influence on brain development during early-life. Maternal care arguably has the greatest influence on this process, with neglect resulting in an array of mental health issues in adolescents and adults. Neural activity in early life shapes circuit development and can have lifelong consequences on brain function. Our recent work revealed that maternal presence/absence from the nest regulates the activity of the PFC of rat pups at postnatal day 11 via the serotonergic system. Here I will present how we are exploring the causal relationship between PFC activity during early-life and serotonergic function and behavioral expression in adulthood.

KARL PETER GIESE

Karl Peter Giese completed his PhD in Neurobiology at the ETH Zurich in 1992. He worked from 1993 until 1998 in Alcino Silva's lab at Cold Spring Harbor Laboratory. In 1998 he started his own lab at University College London as Lecturer and later Reader in Neurogenetics. In 2006 he joined King's College London as Professor of Neurobiology. He studies memory mechanisms in health and disease, using primarily mouse models. He has published more than 100 papers and his most highly cited work is the demonstration that the autophosphorylation of alphaCaMKII at T286 is required for LTP and spatial learning (Giese et al., 1998, Science).

Talk Title and Abstract: Generation of multi-input synapses as novel memory mechanism - Multi-input synapses are synapses that connect three or more neurons. Independent presynaptic inputs converge on one post-synaptic spine. For long it was thought that multi-input synapses are 'left-over' from neuronal development. However, we have found that contextual conditioning can lead to their generation in hippocampal area CA1. We found this first in alphaCaMKII-T286A mutants, which lack NMDA receptor-dependent LTP in CA1, but still form contextual memory. Next, we showed that aged mice (18-month-old) do not induce structural LTP in CA1 but generate multi-input synapses after contextual conditioning. Further, blocking the generation of multi-input synapses, with an inhibitor of PSD95/nNOS interaction, impairs specifical contextual memory in aged, but not young-adult mice. Thus, we conclude that the synaptic basis of contextual memory storage changes with ageing. Further, we found that contextual memory reconsolidation is impaired in aged mice, suggesting that multi-input synapse-dependent memory storage is impaired in updating upon retrieval.

TODD SACKTOR

Todd C. Sacktor is a Professor of Physiology and Pharmocology and Professor of Neurology at the SUNY Downstate Medical Center. His research has focused on how the brain stores longterm memory. He discovered a brain-specific form of PKC called PKMzeta and his lab has demonstrated that PKMzeta was both necessary and sufficient for maintaining long-term potentiation—the leading candidate mechanism for memory in the brain. In 2006, the editors of *Science* highlighted Sacktor's work on PKMzeta and memory as one of the top ten "Breakthroughs of the Year." In 2009, his contributions were featured on the front page of *The New York Times*.

Talk Title and Abstract: Persistently active molecules store long-term memory by sustaining potentiation of activated synapses in neural networks. But how activated synapses continually target these molecules remains unclear. The autonomously active PKC isoform, PKMzeta, maintains potentiation exclusively at activated synapses in the synaptic model of memory, long-term potentiation (LTP). However, LTP induction, like memory formation, increases the synthesis of PKMzeta widely in neurons. Here we show that KIBRA (kidney and brain expressed protein/WWC1), a postsynaptic scaffolding protein genetically linked to human memory performance, continually anchors PKMzeta function to activated synapses. Synaptic activation drives KIBRA and PKMzeta to postsynaptic sites, where they maintain as complexes in late-LTP. To test the function of this persistent KIBRA-PKMzeta coupling, we decoupled the molecules using antagonists of KIBRA-binding in PKMzeta and, conversely, PKMzeta-binding in KIBRA. Neither

decoupling agent measurably affects basal synaptic transmission. However, both reverse established late-LTP. Therefore, KIBRA targets PKMzeta action exclusively at activated synapses. Both agents disrupt maintenance of spatial memories. The zeta-antagonist does not disrupt compensatory PKMzeta-independent maintenance mechanisms in PKMzeta-knockout mice, thus controlling for off-target effects. Therefore, two persistent processes sustain LTP and long-term memory: 1) synaptic potentiation by PKMzeta, and 2) anchoring of this action to activated synapses by KIBRA.

BONG-KIUN KAANG

Bong-Kiun Kaang is an Endowed Chair Professor of neurobiology at School of Biological Sciences, Seoul National University (SNU). He joined SNU as a faculty member since 1994. He obtained B.S. at SNU in 1984. He obtained Ph.D. at Columbia University, in 1992 (Supervisor: Eric R. Kandel). His research focuses on molecular mechanisms underlying synaptic plasticity. He has used cellular, molecular, electrophysiological and behavioral techniques to understand the molecular and cellular mechanisms underlying learning and memory and brain disorders using marine snail and rodents as experimental models. He won the Kyung Ahm Prize (2012) from the Kyung Ahm Foundation, the National Academy of Sciences Award of Korea (2016), Korea Best Scientist & Engineer Award (2018) from the Korean Government, and the Samsung Hoam Prize (2021). He is currently a National Honor Scientist and a Fellow of the Korean Academy of Science and Technology.

Talk Title: Synaptic Reconsolidation - Memory needs to be updated and reorganized to meet the changing world. According to the reconsolidation theory, reactivated memory becomes labile and modifiable, and has to undergo some new process of stabilization depending on protein synthesis. Previously we have shown that protein degradation might be involved in the process by which consolidated state becomes labile state during memory retrieval. We found that some of the specific synaptic proteins were highly polyubiquitinated one hour after the retrieval of contextual fear conditioning in mice. This reduction was blocked by a proteasome inhibitor, suggesting that proteasome induced the labile state and then this labile state was reconsolidated by protein synthesis. We also found that after memory retrieval, behavioral long-term sensitization in Aplysia becomes labile via ubiquitin/proteasome-dependent protein degradation and is reconsolidated by means of de novo protein synthesis. In parallel, we found that the same synapses that store the long-term memory trace encoded by changes in the strength of synaptic connections critical for sensitization are disrupted and reconstructed after signal retrieval. In this talk, I will discuss the synaptic changes underlying memory reconsolidation.

MEREL KINDT

Merel Kindt is Professor of Experimental Clinical Psychology at the University of Amsterdam. The aim of her research programme is to advance current interventions for fear and anxiety related disorders. Her research focuses on the neurobiological and psychological processes of fear learning and memory to understand the mechanisms of change. The potential of her research program lies in the unique bidirectional translational approach: It builds on fundamental insights from animal and human neuroscience literature as well as on clinical science.
Talk Title and Abstract: Karim's shoulders - Current psychological and pharmacological treatments for disorders of emotional memory only dampen the affective response while

leaving the underlying fear memory intact. Under adverse circumstances, these original memories regain prominence, causing relapses in many patients. Observations of post-retrieval amnesia for learned fear in animals have generated a novel and influential hypothesis on the plasticity of memory, generally coined as memory reconsolidation. The clinical potential of pharmacologically disrupting the process of memory reconsolidation has sparked a wave of interest into whether this phenomenon can also be demonstrated in humans, and ultimately harnessed for therapeutic purposes. Here I will outline how the work of Karim Nader and others have moved the field forward: from a focus on extinction learning to the prospect of a revolutionary treatment for emotional memory disorders. Instead of multiple or prolonged sessions of cognitive behavioural treatment or daily drug intake with a gradual decline of symptoms, it involves just one single administration of a very common drug (i.e., propranolol) - given in conjunction with memory retrieval (i.e., brief exposure) during a specific time window - that leads to a sudden (but delayed) decline in fear. Even though basic science in animals and humans suggests that we are on the verge of a breakthrough in fundamentally changing emotional memories, the necessary and boundary conditions for targeting and disrupting memory reconsolidation in clinical practice are largely unknown. Understanding the critical conditions to trigger memory reconsolidation in clinical practice is one of the greatest challenges to be addressed before we can witness a paradigm shift in the treatment of emotional memory disorders.

BRIAN WILTGEN

Dr. Wiltgen received his B.S. in Psychology at the University of Iowa while working in the laboratory of Dr. Isidore Gormezano. He went to graduate school at UCLA where he received his Ph.D. with Dr. Michael Fanselow and then did post-doctoral research with Dr. Alcino Silva at the same institution. He is currently an Associate Professor in the Center for Neuroscience at the University of California, Davis. His laboratory studies neurobiological mechanisms of learning and memory in the rodent hippocampus. Talk Title and Abstract: Phasic activity in the locus coeruleus enhances aversive learning by increasing dopamine release in the hippocampus - The locus coeruleus (LC) supports a wide array of cognitive functions by modulating brain-wide arousal states and responding to salient events in the environment. It is thought to facilitate learning and memory by releasing norepinephrine, a neuromodulator that enhances synaptic plasticity and intrinsic excitability. However, recent work suggests that the LC can also enhance memory by releasing dopamine. In fact, dopamine release from LC terminals in the hippocampus appears to be more important for spatial learning than the release of norepinephrine. In the current experiments, we examined the dynamics of catecholamine release during learning using a hippocampus-dependent trace conditioning task. Unlike spatial learning tasks, trace conditioning involves discrete, well-controlled stimuli that can be time-locked to LC activity and the release of norepinephrine/dopamine. We first examined the response of LC neurons, and their terminals in the dorsal hippocampus, using fiber photometry and GCaMP6. We found clear, learning related changes in the phasic responses of these cells, consistent with their role in signaling the salience of environmental stimuli. When we amplified these phasic responses with optogenetic stimulation, it led to robust enhancements in long-term memory. Follow-up experiments with fluorescent catecholamine sensors revealed that LC stimulation induced large increases in both norepinephrine and dopamine in the dorsal hippocampus. Despite this fact, only dopamine was needed to enhance memory. The implications of these findings for LC function, aversive learning, and dopamine release in the hippocampus will be discussed.

MELANIE SEKERES

Melanie Sekeres received her PhD in Physiology from the University of Toronto under Dr. Sheena Josselyn, and completed postdoctoral training at the Rotman Research Institute under the supervision of Drs. Cheryl Grady and Morris Moscovitch. From 2016-2020, she was Assistant Professor in the department of Psychology & Neuroscience at Baylor University, and moved her lab to the University of Ottawa's School of Psychology in Summer of 2020 where she is now a Canada Research Chair and Assistant Professor. Dr. Sekeres' research takes a cross-species approach to understanding how the brain creates episodic memories, and how these memories transform over time. She investigates the networks of brain regions involved in memory formation, reorganization, and storage, and the underlying molecular mechanisms that regulate memory stabilization in the healthy brain, and in various disease conditions including cancer and chemotherapy-related cognitive impairment.

Talk Title and Abstract: Chemobrain and hippocampal neurotoxicity: Implications for episodic memory consolidation and recollection network disruptions following chemotherapy treatment: Memory disturbances are amongst the most reported symptoms by cancer survivors experiencing 'chemobrain'. The hippocampus, which plays an essential role in episodic memory processing, is highly sensitive to structural and functional disruptions following chemotherapy treatment. Even subtle hippocampal disruption resulting from chemotherapy-induced neurogenic suppression or hippocampal atrophy may underlie deficits in the retrieval of episodic elements of an event memory. To date, the few investigations of complex event memory, including autobiographical memory, in cancer survivors have lacked the rigor to objectively assess the subdomains of episodic memory retrieval, and have not accounted for the differential effects of chemotherapy-mediated disruptions along a temporal gradient. These are important considerations, as hippocampal network disruptions resulting from chemotherapy-induced neurotoxicity may differentially impair retrieval of context-specific and perceptual components of an event memory, while sparing the more general, or schematic components of the retrieved memory. Further, given the process of memory transformation and retrieval network reorganization that occurs over time even in the healthy brain, it is plausible that deficits in retrieval of complex event memories in chemotherapy-treated individuals will be less evident for more remote memories due to the natural forgetting of episodic details, and their reduced reliance on the hippocampus. Taken together, identifying changes to hippocampal networks following chemotherapy treatment is essential to understanding nuanced memory deficits in encoding and retrieval of personally experienced events across the lifespan in cancer survivors. Given the importance of autobiographical memory to a person's history, sense of identity, and ability to plan for the future, an underappreciation of how this memory domain is affected by standard cancer treatments may be discounting its negative impact on the quality of life of cancer survivors.

MARCO LEYTON

Marco Leyton completed his doctoral training with Jane Stewart in Concordia University's Center for Studies in Behavioral Neurobiology. As a post-doc, he transitioned to research in humans, immersing himself first in psychosocial research approaches and then the tools of clinical neuroscience. Currently, he is a Professor in McGill University's Department of Psychiatry, co-Director of Continuing Medical Education, and a past President of the Canadian College of Neuropsychopharmacology. His

research focuses on the causes of addictions and their commonly comorbid conditions*. Thank you, Karim, for convincing me to apply memory reconsolidation principles to the treatment of substance use disorders.*

Talk Title and Abstract: A Stress-Based Intervention to Reduce Cigarette Use in Non-Treatment Seeking Smokers –

Background: Tobacco use is the leading cause of preventable mortality worldwide. Since current smoking cessation aids show only modest efficacy, new interventions are needed. Given the evidence that stress is a potent trigger for smoking, the present randomized clinical trial tested whether stress could augment the effects of a memory updating (retrieval-extinction) intervention.

Methods: Non-treatment seeking smokers (34M/28F; 35.8±13.0 years old; 19.7±14.1 years smoking) were assigned to one of four conditions composed of either a stressful or non-stressful psychosocial challenge followed by either smoking or neutral cues. Ten minutes after this manipulation, all underwent a 60-minute extinction procedure during which they viewed smoking-related videos and images and manipulated smoking paraphernalia. Cue reactivity test sessions took place 24 hours, two weeks and six weeks following the intervention.

Results: Across test sessions, all groups showed (1) significantly increased motivation to quit smoking and smoking cue-induced heart rate responses ($P \leq 0.040$), and (2) significantly decreased cigarette dependence scores and smoking cue-induced skin conductance ($P \leq 0.001$). Compared to participants who were not exposed to the laboratory stressor, the stressor-exposed groups exhibited greater psychophysiological (skin conductance, heart rate, blood pressure and craving) responses during their intervention (all Ps ≤ 0.025) and greater decreases in cigarette use at two- and six-weeks (Ps ≤ 0.040) follow-up independent of smoking cue exposure.

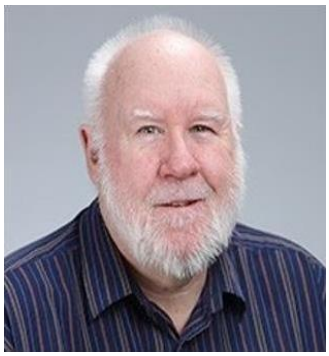
Conclusions: Together, these findings suggest that the ability of stress to activate cigarette seeking processes can be exploited to decrease cigarette use. With replication, the stress-based intervention could become a novel strategy for decreasing cigarette use in non-treatment seeking smokers.

DANIELA SCHILLER

Daniela Schiller is a Professor in the Department of Psychiatry, the Nash Family Department of Neuroscience, and the Friedman Brain Institute, at the Icahn School of Medicine at Mount Sinai. Her research is focused on how the brain represents and modifies emotional memories. Schiller got her PhD in Tel Aviv University where she developed a laboratory model for negative symptoms of schizophrenia. She then continued to do a postdoctoral fellowship at New York University where she examined methods for emotional memory modification in the human brain. Schiller joined Mount Sinai in 2010 and has been directing the laboratory of affective neuroscience since. Her lab has delineated the neural computations of threat learning, how the brain modifies emotional memories using imagination, and the dynamic tracking of affective states and social relationships. Schiller is a Fulbright Fellow and a Kavli Frontiers of Science Fellow, and has been the recipient of many awards, including the New York Academy of Sciences' Blavatnik Award, and the Klingenstein-Simons Fellowship Award in the Neurosciences.

Talk Title and Abstract: Memory retrieval facilitates suppression and reconsolidation update at different temporal scales - Memory reactivation renders consolidated memory fragile and preludes memory reconsolidation, via which the original memory can be modulated or even erased. However, research in declarative memory suggests that memory reactivation also facilitates suppression strategies distinct from memory reconsolidation and yields a more immediate amnesic effect. These findings point to the

intriguing possibility that memory reactivation may prompt memory manipulations with different temporal dynamics. The talk will describe two experiments: First, we demonstrate that memory reactivation is required to prevent the return of fear shortly after extinction training and such effect is cue independent, consistent with suppression. Furthermore, memory reactivation triggers fear memory reconsolidation through extinction training and produces cue-specific amnesia at a longer and separable timescale. These temporal dynamics triggered by fear memory reactivation link memory suppression and reconsolidation under a unified framework for memory updating. These findings open-up new theoretical and clinical avenues for the treatment of persistent maladaptive memory.



RICHARD BROWN

Richard Brown was born in Victoria, BC, Canada and received a BSc (Honours) from the University of Victoria, an MA and PhD in Psychology and Physiology from Dalhousie University and was a Post-Doctoral Fellow in Zoology at Oxford University. Since 1978, he has been a professor in Psychology and Neuroscience at Dalhousie University in Canada. His research has been on the behaviour of rats and mice and over the years this has encompassed studies on sexual behaviour, olfaction, social behaviour, ultrasonic vocalizations, development and aging. He is currently examining sensory, motor, cognitive and affective behaviour over the lifespan of mouse models of Alzheimer's disease and Autism Spectrum Disorder. He has taught IBRO courses in neuroscience in Kenya, Nigeria, Uganda and Cameroon since 2003 and has a long-standing interest in the history of neuroscience. He has published over 200 papers and two books:Social odours in mammals (1985) and An Introduction to Neuroendocrinology (1994; 2015). His interest in apathy derived from the unusual behaviour of the triple transgenic mice in studies of reward-based learning and memory.

Talk Title and Abstract: Not your father's synapse: revising the Hebb synapse for the 21st century - In 1949, Donald O. Hebb developed his neuropsychological postulate, which involved three neural processes: synaptic modifications (i.e., the Hebb Synapse), the cell assembly, and the phase sequence. The Hebb synapse describes how activity at pre- and post-synaptic neurons modulates the strength of a synapse (and synaptic networks). The discovery of long-term potentiation and long-term depression provided evidence for the physiology of the Hebb synapse. However, new findings suggest that the concept of the Hebb synapse needs revision. We propose a 'hepta-partite' model of the synapse to account for the role of astrocytes, oligodendrocytes, microglia, the extracellular matrix (ECM), and the neurovascular unit (NVU) in the regulation of synaptogenesis and modulation of synaptic activity/plasticity. Based on this new information, we revise Hebbian theories of synaptic plasticity, the cell assembly, and phase sequence underlying learning and memory to reflect the current understanding of synaptic plasticity, while upholding the legacy of Donald Hebb.

Poster Abstracts

1. Revising the Hebb Synapse for the 21st Century

Richard Brown, Bayla Dolman
Department of Psychology and Neuroscience Dalhousie University, Halifax Nova Scotia, Canada

In 1949, Donald O. Hebb developed his neuropsychological postulate, which involved three neural processes: synaptic modifications (i.e., the Hebb Synapse), the cell assembly, and the phase sequence. The Hebb synapse describes how activity at pre- and post-synaptic neurons modulates the strength of a synapse (and synaptic networks). The discovery of long-term potentiation and long-term depression provided evidence for the physiology of the Hebb synapse. However, new findings suggest that the concept of the Hebb synapse needs revision. We propose a 'hepta-partite' model of the synapse to account for the role of astrocytes, oligodendrocytes, microglia, the extracellular matrix (ECM), and the neurovascular unit (NVU) in the regulation of synaptogenesis and modulation of synaptic activity/plasticity. Based on this new information, we revise Hebbian theories of synaptic plasticity, the cell assembly, and phase sequence underlying learning and memory to reflect the current understanding of synaptic plasticity, while upholding the legacy of Donald Hebb.

2. Astrocyte-dependent mnemonic processes via eIF2-mediated translational control

Vijendra Sharma[1,2,9] Rapita Sood[1,2,9], Abdessar Khlaifia[3,4], Danning Lou[1,2,9], Tzu-Yu Hung[1,2,9], Noah Cohen[1,2,9], Po-Chieh Cheng[1,2,9], Randal J. Kaufman[5], Mauro Costa-Mattioli[6], Arkady Khoutorsky[7,8,9], Jean-Claude Lacaille[3], Kobi Rosenblum[10,11], Nahum Sonenberg[1,2,9]
[1]Department of Biochemistry
[2]Rosalind and Morris Goodman Cancer Research Centre
[3]Centre for Interdisciplinary Research on Brain and Learning, University of Montreal, Montreal, QC, Canada
[4]Department of Psychology, University of Toronto Scarborough, Toronto, ON, Canada
[5]Degenerative Diseases Program, Sanford-Burnham-Prebys Medical Discovery Institute, La Jolla, CA, USA
[6]Department of Neuroscience, Baylor College of Medicine, Houston, TX, USA
[7]Department of Anesthesia
[8]Faculty of Dentistry
[9]McGill University, Montreal, QC, Canada
[10]Sagol Department of Neurobiology
[11]Centre for Gene Manipulation in the Brain
[12]University of Haifa, Haifa, Israel

The integrated Stress Response (ISR) signaling pathway is an essential mechanism that controls cells' ability to cope with different cellular stresses by modulating the production of new proteins. Genetic or pharmacological manipulations suppressing ISR promote long-lasting memory in healthy mice and correct memory deficits in aged animals and animal models of Alzheimer's disease (AD). Here we used molecular genetics to dissect the role of the ISR in astrocytes and the mechanisms by which the astrocyte-specific ISR manipulation affects cognitive processing. We found a reduction in the levels of eIF2α phosphorylation (p-eIF2α) on Ser51 in astrocytes following learning. Moreover, selective reduction of p-eIF2α in astrocytes of the CA1 region of the hippocampus in young adult mice enhances spatial memory in the Morris water maze (MWM) task and lowers the threshold for the induction of L-LTP by enhancing neuronal excitability. The results suggest that ISR activity and p-eIF2α levels in astrocytes affect neuronal functions, are implicated in cognitive processing and provide a solid basis to prevent cognitive impairments in conditions associated with aberrant eIF2 signalings such as aging, AD, and other neurodegenerative disorders.

3. Gestalt plasticity: neurobiological mechanisms recruited to encode a visual stimulus depend on temporal structure of experience

Peter Finnie, Dustin Hayden, Samuel Cooke, Mark Bear
Picower Institute for Learning and Memory, Massachusetts Institute of Technology, Cambridge, MA, USA

In memory research, a longstanding puzzle concerns how mental representations are formed for individual sensory features embedded with an ongoing stream of stimuli. Does the brain link otherwise discrete representations for each stimulus, or does it encode the sensory pattern as a conjunctive entity? Here we dissected the neurobiological mechanisms mediating long-term plasticity in the mouse primary visual cortex following repeated exposure to either a single visual grating stimulus, or a temporal sequence of stimuli. Mechanisms required to encode a single stimulus are not necessary to learn a sequence of stimuli. Conversely, manipulations with no impact on encoding of a stimulus presented in isolation cause severe impairments for those presented sequentially. Paradoxically, this includes the recognition of each constituent image in addition to their temporal organization. Thus, mechanisms recruited to form a neural representation for a given visual stimulus are dictated by its predictable arrangement relative to other sensory inputs.

4. Implications for studies on autism and migraines with a focus on cortical spreading depression

Faly Golshan, Marla Mickleborough
University of Saskatchewan, Canada

There is a rapid increase in autism spectrum disorder (ASD) with a great number of comorbid neurological complications including migraines. Some previous studies suggest that developmental experience of migraine is relatively similar to ASD and there are speculations that autistic individuals are more highly at risk of migraine development throughout their lives. Yet we still need to learn if there exists a comparable trend of cortical responses in these two conditions. In this literature review, we focus on multiple similar characteristics of migraines and ASD followed by a further emphasis on cortical similarities between the two conditions especially focusing on visual hypersensitivity disorder. Secondly, we suggest how future studies on cortical spreading depression (CSD) could consider the existence of similar cortical hyperexcitability as a result of CSD in autism and if this information could be used for predicting migraine headaches in individuals with ASD.

5. Translation Initiation Factor eIF4G1 Controls Oxidative Phosphorylation, Neuronal Morphogenesis, and Learning and Memory

Sunghoon Kim
McGill University, Montreal, QC, Canada

mRNA translation initiation plays a critical role in cognitive function. The eIF4F complex, composed of the cap-binding protein eIF4E, ATP-dependent RNA helicase eIF4A, and scaffold protein eIF4G, is a major factor in the initiation process. eIF4G1, the most abundant paralog of three eIF4G family proteins, is indispensable for development, but its function in learning and memory is unknown. To study the role of eIF4G1 in cognition, we utilized an eIF4G1 haploinsufficient (eIF4G1-1D) mouse model. The mice are healthy but impaired in hippocampus-dependent learning and memory. Translatome profiling showed that the translation of mitochondrial oxidative phosphorylation (OXPHOS) genes was reduced in the eIF4G1-1D brain, and OXPHOS activity was decreased in eIF4G1-silenced cells. The axonal arborization of eIF4G1-1D primary hippocampal neurons was disrupted, which postulates deficient neural transmission in the eIF4G1-1D brain. These results demonstrate that eIF4G1-

mediated mRNA translation is crucial for cognitive function, which is dependent on OXPHOS and neuronal morphogenesis.

6. Astrocytes involvement in the effects of early-life stress on cognitive dysfunction

Anthony Bosson[1], Ifeoluwa Adedipe[1,2], Lewis Depaauw-Holt[1,2], Ciaran Murphy-Royal[1,2]
[1]Centre de recherche du Centre Hospitalier de l`Université de Montréal (CRCHUM)
[2]Université de Montréal

Adverse childhood experiences are associated with enhanced susceptibility to develop mood disorders later in life. The lateral amygdala (LA) is important for emotive and cognitive behaviors and vulnerable to early-life stress (ELS). However, the mechanisms by which ELS impairs behavior are poorly defined and mainly focused on the underlying neuronal mechanisms despite the active regulation of synaptic transmission by glial cells. Hence, we aimed to identify the role of astrocytes in the effects of ELS LA-dependent behavior.
Using a rodent model of maternal abuse and neglect, we showed that ELS impaired threat-detection as well as long-term potentiation (LTP) in LA. Genetically induced astrocyte network and activity dysfunctions impaired threat-detection and LTP. Additionally, deleting astrocyte glucocorticoid receptors enhanced cognitive function in both ELS and naïve mice. Our results suggest that astrocytes are central regulators of the effects of ELS in LA and highlights astrocytes as potential therapeutic targets.

7. The role of ICAM1 in glioblastoma tumor associated macrophages under hypoxic conditions.

Kaviya Devaraja[1], Hafsah Ali[2], Sheila Mansouri[3], Gelareh Zadeh[4]
[1]University of Toronto - Institute of Medical Science, University Health Network - Princess Margaret Cancer Research Center, Toronto, ON, Canada

Glioblastoma (GBM) is an aggressive, highly fatal brain cancer in adults. Existing treatment methods are ineffective, therefore, new treatments that extend the overall survival and improve quality-of-life are needed. Intracellular adhesion molecule 1 (ICAM1) is a cell adhesion molecule expressed by tumor associated macrophages (TAMs) in GBM. TAMs enhance tumor growth and proliferation within the characteristic hypoxic tumor microenvironment (TME) of GBM. I hypothesize that the expression of ICAM1 on TAMs contributes to GBM cell invasiveness in the hypoxic TME. The expression of ICAM1 is enhanced when macrophages are treated with tumor cell-conditioned medium and further exacerbated upon incubation in hypoxia. The migration levels and proliferation rate of macrophages is higher in wild type cells than in ICAM1 deficient cells and increases upon co-culturing with tumor cell condition media. It is evident that the hypoxic tumor microenvironment increases the expression of ICAM1 in macrophages and GBM cell invasiveness and migration.

8. Morphometric analysis of the human striatum: a study on striatal patch-matrix organization

Gregory Mikerov, Jemal Yesuf, Vladimir Rymar, Abbas Sadikot
Department of Neurology and Neurosurgery, Montreal Neurological Institute, Montreal, QC, Canada

Histochemically distinct domains, namely the matrix and patches (striosomes), have been suggested to differentially contribute to physiological and pathophysiological cognitive processes in the striatum, however many aspects of their function are yet to be described in humans. The current study sought to morphometrically characterize the human striatal patch-matrix system using postmortem brain tissue. Immunohistochemical methods and unbiased stereological tools were used to visualize, measure, and describe the patch-matrix domains. Immunohistochemical analyses using calbindin as a regional marker revealed that in the caudate nucleus, striosomes occupy 17% of regional volume, and compared to the matrix, have a lower neuronal density and higher glia : neuronal cell ratio. Similar morphometric differences were also observed in rostromedial putaminal patch-matrix domains. Due to lack of clear patch-matrix definition with anti-calbindin staining at dorsolateral aspects of the putamen, a future work is designed to employ additional markers to identify and similarly quantify the putaminal patch-matrix organization.

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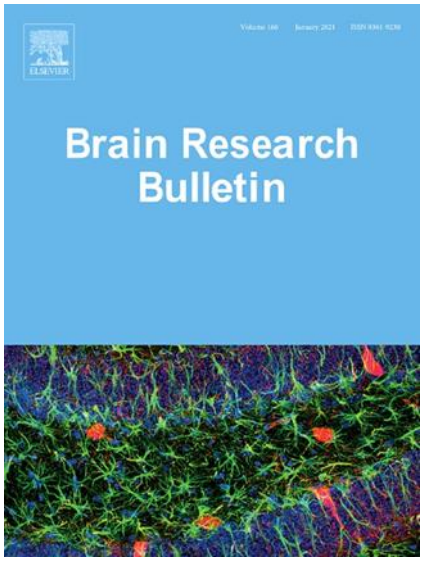


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