Program Booklet

August 22-23, 2022

Jeanne Timmins Amphithéâtre

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 S(head c)4 (G)2 (i)6 (s)4 (he

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PROGRAM

Monday, August 22

9:00 Memory Reconsolidation and the Dynamic Structure of Associative Memories

12:30 Lunch – Poster Session

Session 3: (Chair: Satoshi Kida, University of Tokyo)

1:30 Astrocyte- Dependent Mnemonic Processes via eIF2-Mediated Translational Control
Nahum Sonenberg
McGill University

1:45 Dynamic Palmitoylation of Synaptic Proteins in Learning and Memory
Shernaz Bamji
University of British Columbia

2:00 Role of PFC Activity in the Behavioral Deficits Induced by Maternal Separation
Catia Teixeira
New York University

2:15 Generation of Multi-Input Synapses as Novel Memory Mechanism
Karl Peter Giese
Kings College London

2:30 KIBRA Maintains LTP and Long-Term Memory by Perpetually Targeting PKMzeta
Todd Sacktor
SUNY Downstate Medical Centre

2:45 Synaptic Reconsolidation
Bong-Kiun Kaang
Seoul National University

3:00 Refreshment Pause

Session 4: (Chair: Cathy Rankin, University of British Columbia)

3:30 Karim's shoulders
Merel Kindt
University of Amsterdam

3:45 Phasic activity in the locus coeruleus enhances aversive learning by increasing dopamine release in the hippocampus
Brian Wiltgen

4:30 A Stress-Based Intervention to Reduce Cigarette Use in Non-Treatment Seeking Smokers
Marco Leyton
McGill University

4:45 tr-<</]TJ Motivation [(Sa6.48)]TJ/Reduce/(a)-1r()Tj-4E(M)CTwP3<62384m)Tj-0.0004Tw0(.)3Bj0n

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.9 (.)846T

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 (VTATd ( )Tj -0.013 T7 (A)( )Tj -0.2 Tw 0.283v72004 Tw 0 (at)4.r6 (e)19 (or)

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

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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 neurlt c

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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.

Talk Title: Gestalt Plasticity: The Temporal Structure of Experience Dictates Low-Level Visual Encoding Mechanisms

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

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

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

SHERNAZ BAMJI

Shernaz Bamji is a Professor in the Department of Cellular and

KARL PETER GIESE

Karl Peter Giese completed his PhD in Neurobiology at the ETH Zurich in 1992. He worked from 1993 until

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

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 in clinical practice.

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intriguing possibility that memory reactivation may prompt memory manipulations with different temporal

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, 12.739 0 Td [(t)-6ity]yaepreson, wc 0.016

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 entmodc(fe)qen-2 (pa)10.5 (-2 (e d)-6.6 (a-1.6 (ay2.7 (i)2.6 (.)P)16 (m)10.e d)dox.5 (e)-6.6
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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 fo Td [(r)-6e]/Su01 105 Two Td [(r)13 (t)-6.6 (i)2.6 (s)v2 (e)]0.6 (f)-

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

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