2025 Brain Sciences Special Symposium

Molecular Neurobiology of Memory

2025.10.22 (WED) 13:00 - 18:00

E8-312 (학술정보관 세미나실)

SPEAKER 1

Dr. Judy Illes

University of British Columbia (UBC)

Title: Environmental Change and Global Brain Health

SPEAKER 2

Dr. Toru Takumi

Kobe University School of Medicine

Title: Towards understanding the pathophysiology of neurodevelopmental disorders

SPEAKER 3

Dr. Andrew Holmes

National Institutes of Health (NIH)

Title: Exploring novel mechanism mediating fear memory

SPEAKER 4

Dr. Satoshi Kida

Tokyo University

Title: Molecular and cellular mechanism of PTSD

SPEAKER 5

Dr. Karl Peter Giese

King's College London

Title: Role for increased protein synthesis in Alzheimer's disease

DGIST 대구경북과학기술원 Daegu Gyeongbuk Institute of Science & Technology

Department of Brain Sciences

2025 Brain Sciences Special Symposium

Molecular Neurobiology of Memory

2025.10.22 (WED) 13:00 – 18:00

E8-312 (학술정보관 세미나실)

DGIST 대구경북과학기술원 Daegu Gyeongbuk Institute of Science & Technology

Department of Brain Sciences

Speaker

SPEAKER 1

Dr. Judy Illes

University of British Columbia (UBC)

Title: Environmental Change and Global Brain Health

SPEAKER 2

Dr. Toru Takumi

Kobe University School of Medicine

Title: Towards understanding the pathophysiology of neurodevelopmental disorders

SPEAKER 3

Dr. Andrew Holmes

National Institutes of Health (NIH)

Title: Exploring novel mechanism mediating fear memory

SPEAKER 4

Dr. Satoshi Kida

Tokyo University

Title: Molecular and cellular mechanism of PTSD

SPEAKER 5

Dr. Karl Peter Giese

King's College London

Title: Role for increased protein synthesis in Alzheimer’s disease

Program

10.22 (Wed)

Time	Title		Speaker
13:00 - 14:00	Registration		
14:00 - 14:10	Opening Remarks		Dr. Hyosang Lee DGIST
14:10 - 14:50	Session 1	A Neuroethics Lens on the Exposome: Environmental Change and Global Brain Health	Dr. Judy Illes University of British Columbia (UBC)
14:50 - 15:30	Session 2	Towards understanding the pathophysiology of neurodevelopmental disorders	Dr. Toru Takumi Kobe University School of Medicine
15:30 - 15:40	Coffee break		
15:40 - 16:20	Session 3	Exploring novel mechanism mediating fear memory	Dr. Andrew Holmes National Institutes of Health (NIH)
16:20 - 17:00	Session 4	Molecular and cellular mechanism of PTSD	Dr. Satoshi Kida Tokyo University
17:10 - 17:50	Session 5	Role for increased protein synthesis in Alzheimer's disease	Dr. Karl Peter Giese King's College London
17:50 - 18:00	Closing Rmarks		Dr. Yong-Seok Oh DGIST
18:00 -	Dinner with Faculty members		

Session 1

Judy Illes, CM, Ph.D., FRSC, FCAHS

Professor of Neurology and Director, Neuroethics Canada
Division of Neurology, Department of Medicine
University of British Columbia (UBC)
Email: jilles@mail.ubc.ca

POSITIONS AND DEGREES

- 2007 - present Professor, University of British Columbia (UBC)
- 2003 - 2007 Senior Research Associate and Director, Program in Neuroethics
- 1983 - 1987 Ph.D., Hearing and Speech Sciences, Neuropsychology, Stanford University
- 1981 - 1983 M.A., Psychology, McGill University
- 1977 - 1981 B.A., Psychology, Brandeis University

PUBLICATIONS

1. Rotenberg, A., Gunning, M., Nadler, R. M., Kiss, Z. H. T., & **Illes, J**. (2025). A liability framework for high-risk neural devices. *Science (New York, N.Y.)*, *388*(6752).
2. Perreault, M.L., Taylor-Bragge, R., McLachlan, A.D., Gregory, T.R., Khalid, R., Bassil, K., Svalastog, A-L., Velarde, M.L., **Illes, J**., Indigenous representation in neuroscience scholarship, teaching, and care, Nature Reviews Neuroscience, 2025.
3. Coates McCall, I., Lau, C., Minielly, N., & **Illes, J**. (2019). Owning Ethical Innovation: Claims about Commercial Wearable Brain Technologies. *Neuron*, *102*(4), 728–731.
4. **Illes, J**., Weiss, S., Bains, J., Chandler, J. A., Conrod, P., De Koninck, Y., Fellows, L. K., Groetzinger, D., Racine, E., Robillard, J. M., & Sokolowski, M. B. (2019). A Neuroethics Backbone for the Evolving Canadian Brain Research Strategy. *Neuron*, *101*(3), 370–374.
5. **Illes, J**., Blakemore, C., Hansson, M. G., Hensch, T. K., Leshner, A., Maestre, G., Magistretti, P., Quirion, R., & Strata, P. (2005). International perspectives on engaging the public in neuroethics. *Nature reviews. Neuroscience*, *6*(12), 977–982.

Session 1

Abstract

Title

A Neuroethics Lens on the Exposome:
Environmental Change and Global Brain Health

Changes to the built and natural environments – from neuroarchitecture to neurotoxins – all affect well-being across the human lifespan. In this presentation, I will examine examples of such changes and their impacts on human neurological and mental health through the lens of environmental neuroethics, a subspecialty of the field of neuroethics that is designed to explicitly align human values with advances in neuroscience. I will consider social and cultural factors in addition to cognitive ones, and the means that global advocacy through evidence can mitigate harm and promote benefits for brain health on global scale.

Session 2

Toru Takumi, M.D., Ph.D

Professor, Department of Physiology and Cell Biology
Kobe University School of Medicine
Email: takumit@med.kobe-u.ac.jp

POSITIONS AND DEGREES

- 2019 - present Professor, Kobe University School of Medicine
- 1996 - 2001 Associate Professor, Kobe University School of Medicine, Japan
- 1994 - 1996 Assistant Professor, Osaka University Medical School, Japan
- 1991 - 1994 HFSP fellow, Whitehead Institute for Biomedical Research, Massachusetts Institute of Technology, USA
- 1991 - 1991 JSPS fellow, Kyoto University Faculty of Medicine, Japan
- 1986 - 1990 Ph.D., Kyoto University Graduate School of Medicine, Japan
- 1980 - 1986 M.D., Fukui Medical School, Japan

PUBLICATIONS

1. S. Fujima, M. Sato, N. Nakai and **T. Takumi**. (2025). Parvalbumin interneurons in the insular cortex control social familiarity and emotion recognition. *Cell Rep*., 13, 116085.
2. J. Nomura, A. Zuko, K. Kishimoto, H. Mutsumine, H. Maegawa, K. Fukatsu, Y. Nomura, X. Liu, N. Nakai, ES library team (C. Maeda, Y. Kusakari, T. Arai, I. Shibasaki, A. Homma, K. Yanaka, K. Matsuno, E. Bergoglio, Y. Sakai, Q. Eusuf, M. Seki, R. T. Fresia, S. Furukawa, K. Yamamoto, P. Carninci, S. Kuraku, T. Yamamoto), E. Takahashi, T. Kouno, J. W. Shin and **T. Takumi**. (2025). ESC models of autism with copy number variations reveal cell-type-specific translational vulnerability. *Cell Genom*, 5, 100877.
3. T. Kaizuka, T. Suzuki, N. Kishi, K. Tamada, M. W. Kilimann, T. Ueyama, M. Watanabe, T. Shimogori, H. Okano, N. Dohmae and **T. Takumi**. (2024). Remodeling of the postsynaptic proteome in male mice and marmosets during synapse development. *Nat Commun*., 15, 2496.
4. N. Nakai, M. Sato, Y. Sekine, X. Fu, O. Yamashita, A. Zalesky and **T. Takumi**. (2023). Virtual reality-based real-time imaging reveals abnormal cortical dynamics during behavioral transitions in a mouse model of autism. *Cell Rep*., 42, 112258.
5. C.-W. Lin, D.E. Septyaningtrias, H-W. Chao, M. Konda, K. Atarashi, K. Takeshita, K Tamada, J. Nomura, Y. Sassagawa, K. Tanaka, I. Nikaido, K. Honda, T.L. McHugh and **T. Takumi**. (2022). A common pathologic mechanism across cellular origins underlies systemic immune dysregulation in an idiopathic autism mouse model. *Mol Psychiatry*, 27, 3343-3354, 2022.

Session 2

Abstract

Title

Towards understanding the pathophysiology of neurodevelopmental disorders

Substantial evidence indicates that chromosomal abnormalities, including copy number variations (CNV), contribute to autism risk. The duplication of human chromosome 15q11-13 is known as the most common cytogenetic abnormality in autism spectrum disorder (ASD). We modeled this genetic change in mice by chromosome engineering, creating a 6.3-Mb duplication of the conserved region on mouse chromosome 7. Mice with paternal duplication exhibit autistic-like behaviors, including reduced social interactions, behavioral inflexibility, and abnormal ultrasonic vocalizations. This chromosome-engineered mouse model for ASD (15q dup mouse) appears to replicate several aspects of human autistic traits and supports the relevance of the human CNV. It is the first CNV model of ASD and serves as a founder mouse for the forward genetics of a developmental brain disorder. Our multidimensional approach reveals that 15q dup mice exhibit impaired spine phenotypes, serotonin abnormalities, and an imbalance between excitatory and inhibitory neurons. Using synapse phenotypes, we identified a key gene (Necdin) within the duplication. To systematically study CNV pathology, we developed a comprehensive library of ES cell models with CNVs. Additionally, to explore the neural networks underlying social behavior, we created a mouse VR system to monitor cortical network dynamics during behavior. I will present our multidisciplinary approach to understanding the pathophysiology of ASD.

Session 3

Andrew Holmes, Ph.D.

Chief, Laboratory of Behavioral and Genomic Neuroscience,
National Institute on Alcohol Abuse and Alcoholism, National Institutes of Health
Email: holmesan@mail.nih.gov

POSITIONS AND DEGREES

- 2011 - present Laboratory Chief, National Institute on Alcohol Abuse and Alcoholism, NIH, USA
- 2003 - 2010 Section Chief, National Institute on Alcohol Abuse and Alcoholism, NIH, USA
- 1999 - 2003 Visiting Fellow, National Institute of Mental Health, National Institutes of Health (NIH), USA
- 1995 - 1998 Ph.D., University of Leeds, UK
- 1992 - 1995 B.A., University of Newcastle upon Tyne, UK

PUBLICATIONS

1. Silverstein, S. E., O'Sullivan, R., Bukalo, O., Pati, D., Schaffer, J. A., Limoges, A., Zsembik, L., Yoshida, T., O'Malley, J. J., Paletzki, R. F., Lieberman, A. G., Nonaka, M., Deisseroth, K., Gerfen, C. R., Penzo, M. A., Kash, T. L., & **Holmes, A**. (2024). A distinct cortical code for socially learned threat. *Nature*, *626*(8001), 1066–1072.
2. Hagihara, K. M., Bukalo, O., Zeller, M., Aksoy-Aksel, A., Karalis, N., Limoges, A., Rigg, T., Campbell, T., Mendez, A., Weinholtz, C., Mahn, M., Zweifel, L. S., Palmiter, R. D., Ehrlich, I., Lüthi, A., & **Holmes, A**. (2021). Intercalated amygdala clusters orchestrate a switch in fear state. *Nature*, *594*(7863), 403–407.
3. Gunduz-Cinar, O., Castillo, L. I., Xia, M., Van Leer, E., Brockway, E. T., Pollack, G. A., Yasmin, F., Bukalo, O., Limoges, A., Oreizi-Esfahani, S., Kondev, V., Báldi, R., Dong, A., Harvey-White, J., Cinar, R., Kunos, G., Li, Y., Zweifel, L. S., Patel, S., & **Holmes, A**. (2023). A cortico-amygdala neural substrate for endocannabinoid modulation of fear extinction. *Neuron*, *111*(19), 3053–3067.e10.
4. Sengupta, A., & **Holmes, A**. (2019). A Discrete Dorsal Raphe to Basal Amygdala 5-HT Circuit Calibrates Aversive Memory. *Neuron*, *103*(3), 489–505.e7.
5. Bukalo, O., Pinard, C. R., Silverstein, S., Brehm, C., Hartley, N. D., Whittle, N., Colacicco, G., Busch, E., Patel, S., Singewald, N., & **Holmes, A**. (2015). Prefrontal inputs to the amygdala instruct fear extinction memory formation. *Science advances*, *1*(6), e1500251.

Session 3

Abstract

Title

Exploring novel mechanisms mediating fear memory

Neural systems mediating responses to previously encountered threats are critical to animal's adaptive success and, ultimately, survival. It is known that memory for learned threats is underpinned by neural representations in the basolateral amygdala (BLA), but the contribution of non-neuronal cells, including astrocytes, to this process remains unclear. In this presentation, we will show how, using in vivo calcium (Ca2+) imaging, together with chemogenetic manipulations, BLA astrocytes dynamically track fear state and causally contribute to fear memory retrieval. We will also show how combining astrocyte manipulations with in vivo cellular-resolution Ca2+ imaging and electrophysiological recordings of BLA neurons, demonstrates that astrocyte Ca2+-signaling enables population-level neuronal representation of fear state that is readout through a BLA-prefrontal circuit to support memory performance. The findings of this presentation will reveal a key role for astrocytes in the generation of behaviorally-relevant neural representations, revising current neurocentric models of amygdala function.

Session 4

Kida Satoshi, Ph.D.

Professor, Department of Department of Applied Biological Chemistry,
Graduate School of Agriculture and Life Sciences,
The University of Tokyo
Email: akida@g.ecc.u-tokyo.ac.jp

POSITIONS AND DEGREES

- 2009 - present Professor, Department of Applied Biological Chemistry, The University of Tokyo
- 1996 - 1997 Post doctoral fellow, Dr. Alcino Silva's laboratory, Cold Spring Harbor Laboratory, USA
- 1994 - 1996 Post doctoral fellow, University of Tokyo, Japan
- 1991 - 1994 Ph.D., University of Tokyo, Japan
- 1989 - 1991 M.S., University of Tokyo, Japan
- 1985 - 1989 B.A., University of Tokyo, Japan

PUBLICATIONS

1. Hori, H., Fukushima, H., Nagayoshi, T., Ishikawa, R., Zhuo, M., Yoshida, F., Kunugi, H., Okamoto, K., Kim, Y., & **Kida, S.** (2024). Fear memory regulation by the cAMP signaling pathway as an index of reexperiencing symptoms in posttraumatic stress disorder. Molecular psychiatry, 29(7), 2105–2116.
2. Fukushima, H., Zhang, Y., & **Kida, S.** (2021). Active Transition of Fear Memory Phase from Reconsolidation to Extinction through ERK-Mediated Prevention of Reconsolidation. The Journal of neuroscience : the official journal of the Society for Neuroscience, 41(6), 1288–1300.
3. Hasegawa, S., Fukushima, H., Hosoda, H., Serita, T., Ishikawa, R., Rokukawa, T., Kawahara-Miki, R., Zhang, Y., Ohta, M., Okada, S., Tanimizu, T., Josselyn, S. A., Frankland, P. W., & **Kida, S.** (2019). Hippocampal clock regulates memory retrieval via Dopamine and PKA-induced GluA1 phosphorylation. Nature communications, 10(1), 5766.
4. **Kida, S.**, & Kato, T. (2015). Microendophenotypes of psychiatric disorders: phenotypes of psychiatric disorders at the level of molecular dynamics, synapses, neurons, and neural circuits. Current molecular medicine, 15(2), 111–118.
5. Fukushima, H., Zhang, Y., Archbold, G., Ishikawa, R., Nader, K., & **Kida, S.** (2014). Enhancement of fear memory by retrieval through reconsolidation. eLife, 3, e02736.

Session 4

Abstract

Title

Molecular and Cellular Mechanisms of PTSD

Post-traumatic stress disorder (PTSD) is a psychiatric disorder associated with traumatic memory, yet its etiology remains unclear. Reexperiencing symptoms are specific to PTSD compared to other anxiety-related disorders. Importantly, reexperiencing can be mimicked by retrieval-related events of fear memory in animal models of traumatic memory. We first demonstrate the tight linkage between facilitated cAMP signaling and PTSD by analyzing loss- and gain-of-cAMP signaling effects on fear memory in mice and the transcriptomes of fear memory-activated mice and PTSD patients with reexperiencing symptoms. Integrative mouse and human transcriptome analysis revealed reduced mRNA expression of phosphodiesterase 4B (PDE4B), an enzyme that degrades cAMP, in the peripheral blood of PTSD patients exhibiting more severe reexperiencing symptoms, as well as in the mouse hippocampus after fear memory retrieval. In line with these observations, pharmacological and optogenetic upregulation or downregulation of cAMP signaling transduction enhanced or impaired the retrieval and subsequent maintenance of fear memory in mice. These findings raise the possibility that the facilitation of cAMP signaling mediating the downregulation of PDE4B expression enhances traumatic memory, thereby playing a key role in the reexperiencing symptoms of PTSD patients as a functional index of these symptoms.

Session 5

Karl Peter Giese, Ph.D.

Professor of Neurobiology of Mental Health,
Department of Basic and Clinical Neuroscience Institute of Psychiatry,
Psychology and Neuroscience
King's College London
Email: karl.giese@kcl.ac.uk

POSITIONS AND DEGREES

- 2006 – present Professor, Institute of Psychiatry, King's College London, UK.
- 1998 - 2004 Lecturer, University College London, UK.
- 1993 - 1998 Postdoctoral Fellow with Alcino J. Silva, Cold Spring Harbor Laboratory, USA.
- 1989 - 1992 Ph.D. in Neurobiology, Federal Institute of Technology (ETH) Zurich, Switzerland.
- 1983 - 1989 Study of Chemistry and Diploma in Chemistry, Ruhr-University Bochum, Germany.

PUBLICATIONS

1. Ghosh, A., Mizuno, K., Tiwari, S. S., Proitsi, P., Gomez Perez-Nievas, B., Glennon, E., Martinez-Nunez, R. T., & **Giese, K. P**. (2020). Alzheimer's disease-related dysregulation of mRNA translation causes key pathological features with ageing. Translational psychiatry, 10(1), 192.
2. Aziz, W., Kraev, I., Mizuno, K., Kirby, A., Fang, T., Rupawala, H., Kasbi, K., Rothe, S., Jozsa, F., Rosenblum, K., Stewart, M. G., & **Giese, K. P**. (2019). Multi-input Synapses, but Not LTP-Strengthened Synapses, Correlate with Hippocampal Memory Storage in Aged Mice. Current biology : CB, 29(21), 3600–3610.e4.
3. Tiwari, S. S., Mizuno, K., Ghosh, A., Aziz, W., Troakes, C., Daoud, J., Golash, V., Noble, W., Hortobágyi, T., & **Giese, K. P**. (2016). Alzheimer-related decrease in CYFIP2 links amyloid production to tau hyperphosphorylation and memory loss. Brain : a journal of neurology, 139(Pt 10), 2751–2765.
4. Radwanska, K., Medvedev, N. I., Pereira, G. S., Engmann, O., Thiede, N., Moraes, M. F., Villers, A., Irvine, E. E., Maunganidze, N. S., Pyza, E. M., Ris, L., Szymańska, M., Lipiński, M., Kaczmarek, L., Stewart, M. G., & **Giese, K. P**. (2011). Mechanism for long-term memory formation when synaptic strengthening is impaired. Proceedings of the National Academy of Sciences of the United States of America, 108(45), 18471–18475.
5. Engmann, O., Hortobágyi, T., Pidsley, R., Troakes, C., Bernstein, H. G., Kreutz, M. R., Mill, J., Nikolic, M., & **Giese, K. P**. (2011). Schizophrenia is associated with dysregulation of a Cdk5 activator that regulates synaptic protein expression and cognition. Brain : a journal of neurology, 134(Pt 8), 2408–2421.

Session 5

Abstract

Title

Role for increased protein synthesis in Alzheimer's disease

Alzheimer's disease (AD), the most prevalent form of dementia, has a very complex phenotype, which includes amyloid pathology including amyloid plaques, tau pathology including neurofibrillary tangles, neuro-inflammation, synapse loss that precedes neuronal loss, memory impairment, and other cognitive deficits. Amongst the earliest causes of AD is abnormal processing of the amyloid precursor protein (APP). Interestingly, App mRNA can be locally translated under regulation of FMRP. Therefore, we studied whether FMRP and its regulators CYFIP1/2 are dysregulated in post-mortem AD brain, and we found down-regulation of CYFIP2 expression by about 50%. We modelled the impact of this down-regulation in heterozygous CYFIP2 knockout mice and found that this results in increased synaptic protein synthesis, abnormal APP processing, tau hyperphosphorylation, neuroinflammation, synapse loss, and memory loss in ageing-dependent manner. This indicates that elevation of protein synthesis is sufficient to cause some of the complexity of AD. To investigate the causes of elevated protein synthesis in early AD we exposed primary neurons with amyloid-beta oligomers and found an increase in de novo protein synthesis. We performed de novo proteomic studies and identified changes in various cellular mechanisms. Further, we assessed synapse changes after expansion microscopy (as it allows to study multi-synapses) and, surprisingly, revealed that low concentrations of amyloid-beta oligomers are synaptogenic. These synaptogenic effects are consistent with an increase in local APP expression. Interestingly, these early synaptogenic effects can be prevented with a clinically approved cancer drug. The implications of our findings will be discussed.

MEMO

MEMO