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NEURO2026 Overview

Attending NEURO2026, represented an incredible milestone. The event served as a joint meeting of the 49th Annual Meeting of the Japan Neuroscience Society, the 69th Annual Meeting of the Japanese Society for Neurochemistry, and the 36th Japanese Neural Network Society under the inspiring theme "Let Future Neuroscientists Take Off!" Held from July 30 to August 2, 2026, at the Kobe Convention Center in Kobe, Japan, the conference was co-organized by Prof. Hiroyuki Kamiguchi (RIKEN Institute), Prof. Seiji Hitoshi (Shiga University of Medical Science), and Prof. Saori C. Tanaka (Nara Institute of Science and Technology, NAIST).

This year's edition successfully combined exceptional scientific research with the dynamic integration of findings in neuroscience, neurochemistry, neural network research, and related fields. It also provided a forum for cross-disciplinary interactions with areas such as immunology, oncology, and genomics. Ultimately, the unique synergy between historical cultural immersion in Kobe and transformative neuroscience dialogue offered an unforgettable platform for dynamic learning, networking, and energizing a shared passion to pioneer the next generation of global brain research.

This spirit of collaboration and cross-disciplinary exchange defined my experience at NEURO2026, which I attended as an International Young Scientist Travel Award recipient with additional support from the Tianqiao & Chrissy Chen Institute through the Chen Science Writer Fellowship. Beyond providing travel and registration funding for early-career researchers, this fellowship extends the impact of scientific conferences by synthesizing discussions, highlighting core themes, and amplifying innovative presentations for a broader audience.

Scientific Topics

A great diversity of topics was explored during NEURO2026, encompassing multiple methodological approaches, models, and exciting research questions. Being surrounded by world-class researchers and emerging scientific leaders created an electric atmosphere packed with groundbreaking discoveries, cutting-edge methodologies, and vibrant global collaborations. Engaging directly with plenary lectures and interactive sessions across diverse brain science disciplines provided immense motivation to rethink traditional models and push personal research limits.

Although every presentation was unique and of high quality, several timely topics consistently emerged across the outstanding plenary and special talks.

For instance, Pieter Roelfsema (Netherlands Institute for Neuroscience) presented breakthrough research in humans on the neural mechanisms of conscious visual perception and perceptual organization. His work highlights a major frontier in vision restoration: projecting visual information directly into the brain to bypass damaged eyes—a long-standing

goal in neuroscience that offers a viable solution for patients with irreversible optic nerve damage.

Bridging biological and computational insights, Alison Gopnik (UC Berkeley, USA) presented work on "Empowerment and Causal Learning: A Bridge Between Reinforcement Learning and Bayesian Model Building," detailing empirical studies on how children and adults utilize empowerment cues to infer causal relations and design effective interventions.

Expanding on cell-cell communication, Michelle Monje (Stanford University / Howard Hughes Medical Institute, USA) demonstrated that neuron-glial and neuron-glioma interactions crucial for normal circuit function also drive glial cancers—mechanisms with far-reaching relevance beyond neuro-oncology.

Glial biology—and microglial research in particular—stood out as a highlight of the meeting. In the plenary sessions, Li Gan (Weill Cornell Medicine, USA) addressed how to reprogram microglia to prevent neuronal injury while preserving their homeostatic functions, utilizing a humanized platform to study glia-neuron interactions in neurodegeneration. Sonia Garel (Collège de France/IBENS, France) brought equal focus to early development, revealing how microglia shape brain morphogenesis and serve as a protective shield against fetal brain stress. Furthering this translational vision, Shuichi Koizumi (University of Yamanashi) examined how microglial dysregulation drives long-term pathology due to their capacity for local self-renewal, showcasing a novel intranasal transplantation method that non-invasively replaces aberrant microglia with healthy populations to ameliorate disease.

This focus on microglial biology was deepened during the symposium, *"Cutting-Edge Research on Neuroimmunology: Diversity and Therapeutic Targeting of Brain Macrophage."* Speakers traversed the full spectrum of macrophage and microglia biology across the lifespan. Rosa Paolicelli (University of Lausanne) illustrated how impaired microglial function during early maturation leads to persistent deficits in neuronal function and behavior. Qingyun Li (Washington University in St. Louis) introduced an inducible Clec7a-CreERT2 driver line to track, label, and isolate reactive microglial subpopulations across development, aging, and disease contexts. Takahiro Masuda (Kyushu University) addressed regional heterogeneity, contrasting parenchymal microglia with CNS border-associated macrophages (CAMs) at meningeal and perivascular interfaces as novel therapeutic targets. Finally, Hiroaki Wake (Nagoya University) explored the neuro-immune-vascular interface in Alzheimer's disease models, detailing microglial synapse elimination via CCL3/4-CCR5 signaling, interactions between Axl-expressing microglia and astrocytic Gas6 in regulating blood-brain barrier permeability, and the role of subarachnoid macrophages in cerebral amyloid angiopathy.

Complementing these mechanistic sessions, specialized symposia highlighted emerging translational frameworks and interdisciplinary tools. An industry-academia collaboration symposium addressed the human-specific limitations of mouse models by presenting astrocyte-enriched cortical organoids generated via transcription factor overexpression to model human glial maturation. Other cross-species dynamic sessions merged neurotechnologies, neuromodulation, and artificial intelligence to examine brain dynamics across species, alongside interdisciplinary models combining virtual reality and AI to probe mathematical cognition. Lastly, sessions on emerging frontiers in neurochemistry highlighted

the role of selective autophagy in modulating neuronal activity and synaptic transmission, uncovering new regulatory pathways in neurodegenerative diseases.

Conference Highlights

A standout element of the conference was the strong collaborative spirit among Japanese universities, featured prominently in high-quality poster sessions covering diverse neuroscience topics with a remarkable emphasis on tangible industrial applications, practical translational work, and patentable discovery potential. Of special importance was how NEURO2026 actively incentivized the participation and leadership of young national and international researchers. The meeting recognized their innovative research and exceptional communication skills during interactive poster sessions and oral presentations. These platforms allowed them to exchange ideas, receive feedback, formulate new hypotheses, and establish new collaborations.

Exhibits from major, well-known international companies specializing in live neuroimaging, AI-driven data analytics, and the advanced neural interface and neurochemistry sectors further illustrated the practical intersection of industry and academia. These displays featured advanced technologies designed to elucidate brain mechanisms at scales ranging from single cells to entire neural networks. Alongside them, global life science brands, reagent suppliers, and distributors showcased precision cellular markers, imaging dyes, and neuroscience-focused antibodies.

Poster presentation

With the support of the NEURO2026 Travel Award and the Chen Science Writer Fellowship, funded by the Tianqiao and Chrissy Chen Institute, I had the privilege of presenting my research poster at NEURO2026. Participating in this conference enabled me to present my research on microglia–neuron interactions in health and disease to a global audience of neuroscientists, engage in high-level discussions with leading experts in the field, and significantly elevate the visibility and impact of my findings. Beyond sharing my work, this experience facilitated invaluable professional networking and exposed me to cutting-edge methodologies that will directly inform the next phase of my research.

Specifically, our recent findings address the spatial heterogeneity of the brain response to amyloid-beta (Aβ) accumulation. While the cortex undergoes heavy Aβ plaque deposition, the cerebellum remains remarkably sparse in plaque burden. Nevertheless, emerging clinical evidence indicates that in Alzheimer's disease (AD) patients, late-stage disease progression (Braak Stage IV) and worsened clinical outcomes correlate with cerebellar deterioration and physiological alterations. Parallel studies demonstrate that microglia also exhibit regional heterogeneity in their transcriptomic and functional profiles. Consequently, we investigated the pathophysiological microenvironments of cortical and cerebellar microglia to identify the cellular mechanisms underlying cerebellar resilience to Aβ plaque formation.

Despite high expression of mutant human *APP* and *PSEN1*, aged 5xFAD mice (an AD mouse model) show minimal Aβ plaque deposition in the cerebellum relative to the cortex. This resistance correlates with a distinct homeostatic state in cerebellar microglia, characterized by lower expression of activation markers such as IBA1, CD68, TREM2, and CD36. Rather than undergoing the hyper-inflammatory, neurotoxic polarization typical of cortical disease-

associated microglia, cerebellar microglia maintain efficient clearance capabilities while avoiding dystrophic VGLUT1-positive synaptic damage.

Finally, the extracellular matrix acts as a crucial mediator in cerebellar resilience. Levels of key extracellular matrix chondroitin sulfate proteoglycans—specifically brevican and aggrecan—are elevated in the 5xFAD cerebellum. Cerebellar microglia demonstrate enhanced internalization and degradation of brevican co-aggregated with small Aβ assemblies compared to their cortical counterparts. This rapid uptake by microglia of matrix-associated Aβ complexes provides a mechanical pathway by which the cerebellum neutralizes oligomeric toxic seeds before macro-plaques can form.

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