SEMINAR CYCLE

of the PhD in Neuroscience of Turin

7th Appointment

Prof. Menachem Hanani

Hebrew University of Jerusalem

"Satellite Glial Cells. Two decades of Progress"

6th October, 2023 h 3:00 PM

The lecture will last 1 hour and it will be followed by discussion.

Host: Prof. Francesco Ferrini

Aula B – Anatomy Institute
C.so Massimo d'Azeglio 52

Link: https://bit.ly/3PDcGvp

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PROF. MENACHEM HANANI

Professor Menachem Hanani graduated in Physical Chemistry and received his PhD in Neurobiology at the Hebrew University (Jerusalem, Israel) in 1976 after a visiting period as PhD student at the Max Planck Institute for Biological Cybernetics (Tübingen, Germany). Then, Professor Hanani moved to USA where he worked as a postdoc at the Laboratory of Neurophysiology of the National Institute of Neurological Disorders and Stroke in Bethesda (Maryland, USA). He is now full professor at the Faculty of Medicine at the Hebrew University of Jerusalem, with an over 40 years experience in neurobiological research and several contributions in the development of new basic concepts and methodologies in the field. In the last 20 years professor Hanani focused on the contribution to chronic pain of neuron-glia interactions in sensory ganglia and was the first to focus on the function of satellite glial cells in sensory ganglia and their role in chronic pain.

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ABSTRACT

Neuronal cell bodies in sensory ganglia are surrounded completely by a special type of glial cells, known as satellite glial cells (SGCs). The distance between the neuron and surrounding SGCs is only 20 nm wide. Such an arrangement is not found in the central nervous system, and its functional significance had received little attention. We studied neuron-SGC interactions in mouse sensory ganglia, using electrophysiology, calcium imaging (in vitro and in vivo), histology, dye coupling, and behavior. We found that sensory neurons and SGCs display bidirectional communications by both chemical messengers (mainly ATP) and gap junctions. These interactions are enhanced in a variety of pain models induced by nerve injury such as inflammation and axotomy. This enhancement appears to contribute to chronic pain. For example, in a model of visceral pain, the augmented gap junctions contribute to neuronal excitability and to pain behavior. In all the models that were studied, SGCs activation (gliosis) was present, as evidenced by upregulation of glial fibrillary acidic protein (GFAP). These results correlated with recent work from other laboratories showing that chronic pain syndromes in humans may be associated with SGCs activation. In conclusion, neuron-SGC interactions are important for the function of sensory ganglia under both normal and pathological sates.

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