ASHBi SEMINAR

Pain changes the brain; the brain changes the pain

—Synaptic plasticity in the central amygdala underlies nociplastic pain

Lecturer: Fusao Kato Dr.Med.Sci, Dr.Pharm.Sci
Professor Emeritus, Center for Neuroscience of Pain
Jikei University School of Medicine

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Venue Conference Room
B1F, Faculty of Medicine Bldg. B

Register here

Pain, particularly chronic pain, is a serious global health issue. Undoubtedly, the biological (i.e., evolutionary) purpose of pain is to enhance survival by enabling individuals to avoid potentially life-threatening situations. To achieve this goal, animals have evolved a system capable of (1) detecting harmful or aversive conditions within the body and the environment and (2) orienting the individual to effectively avoid such risks across various timescales. This latter process is often accompanied by the assignment of emotional valence to the situation and the active modulation of the sensitivity of the detection system.

Over the past 20 years, my group has focused on the central amygdala (CeA) as a core structure underlying these processes, as it receives nociception-specific information and exhibits robust neuroplasticity in states of persistent pain. We demonstrated robust synaptic potentiation at the synapses between neurons in the parabrachial nucleus (PBN)—a major convergence site for nociceptive inputs from the spinal cord and trigeminal nucleus—and the CeA, a site involved in assigning emotional valence to sensory information, in various rodent models of persistent pain (Kato et al., 2018). More recently, we found that activation of this pathway alone induced robust mechanical sensitization across widespread body regions, even in the absence of tissue injury or nerve damage (Sugimoto et al., 2021). This behavioral outcome aligns well with the recently proposed third mechanistic category of pain—“nociplastic pain”—which refers to pain arising from plastic changes in the pain processing network in the absence of ongoing nociceptive input or nerve damage. Based on these findings, we propose that the CeA serves as the core brain site for the biologically relevant pro-survival functions of pain, whereby pain induces passive changes in this network, leading to its chronicity. Moreover, these changes—or the activation of this network by other factors—can, in turn, actively alter the sensitivity of this system, which may underpin the total experience of pain.

Organizer : Institute for the Advanced Study of Human Biology (WPI-ASHBi)

Contact: Tadashi Isa
[E-mail] isa.tadashi.7u@kyoto-u.ac.jp