Oral Neuroscience 2022

March 4th, 2023

Program & Abstract

Venue
Osaka University
Graduate School of Dentistry
Osaka, Japan

大阪大学 OSAKA UNIVERSITY

Challenge to Intractable Oral Diseases

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Organizing Committee

Hitoshi Niwa*
Dept. of Dental Anesthesiology, Osaka Univ. Grad. Sch. of Dentistry

Kazuhiro Takuma
Dept. of Pharmacology, Osaka Univ. Grad. Sch. of Dentistry

Takafumi Kato
Dept. of Oral Physiology, Osaka Univ. Grad. Sch. of Dentistry

Susumu Tanaka
First Dept. of Oral and Maxillofacial Surgery, Osaka Univ. Grad. Sch. of Dentistry

Takahiro Furuta
Dept. of Oral Anatomy and Neurobiology, Osaka Univ. Grad. Sch. of Dentistry

Shinsuke Ohba
Dept. of Oral Anatomy and Developmental Biology, Osaka Univ. Grad. Sch. of Dentistry

Chizuko Inui
Dept. of Oral Anatomy and Developmental Biology, Osaka Univ. Grad. Sch. of Dentistry
* Chair

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“Challenge to Intractable Oral diseases” from Osaka University Graduate School of Dentistry

Contact

Department of Dental Anesthesiology, Osaka University Graduate School of Dentistry

1-8 Yamadaoka, Suita, Osaka, Japan 565-0871 Tel +81-6-6879-2913 Fax +81-6-6879-2914

Access

Yumikura memorial hall (Building F, 5F)

Osaka University Graduate School of Dentistry

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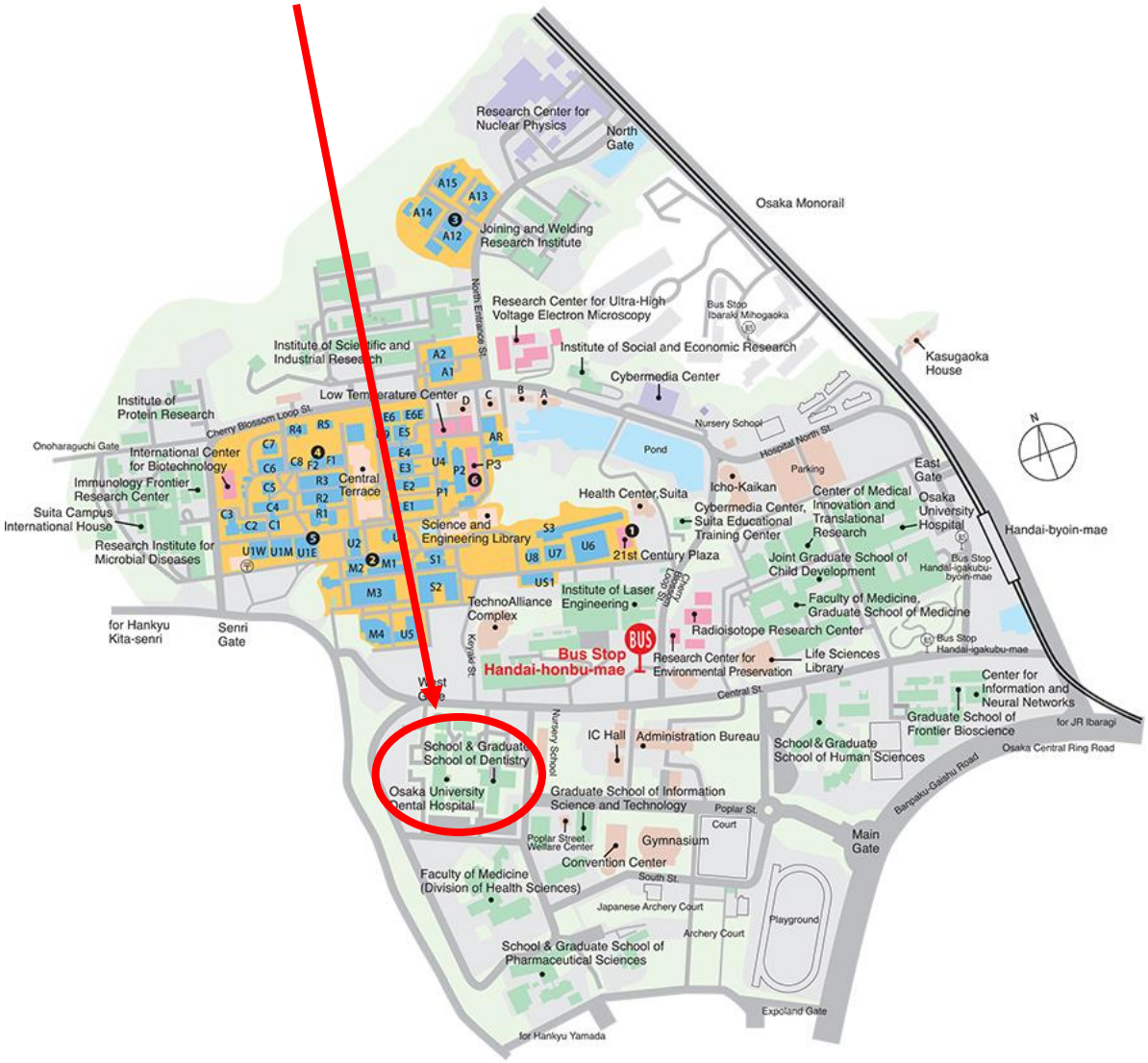

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- ✓ The time allowed for the slide presentation of plenary lecture is 60 minutes including 10 minutes for discussion.
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Oral Neuroscience 2022

Yumikura memorial hall (Dental faculty building F, 5F)

13:00- Opening Remarks Hitoshi Niwa

Plenary Lecture (13:05-14:05)	Chair	Hitoshi Niwa

13:05- Development of therapeutic strategies to repair neuronal network for the central nervous system diseases
Toshihide Yamashita (Dept. of Molecular Neuroscience, Osaka Univ. Grad. Sch. of Medicine)

Coffee Break (14:05-14:15)

Session 1 (14:15-15:15)	Chair	Chiho Kudo

14:15- [Mini review-1] Temporally-selective reconfiguration of cortical synapses underlies acquisition of proficient motor skill
Jaerin Sohn（Dept. of Oral Anatomy and Neurobiology, Osaka Univ. Grad. Sch. of Dentistry)

14:45- [Short talk-1] An electromyographic analysis of jaw-closing muscle activities during suckling and mastication in developing rats
Masaharu Yamada (Dept. of Oral Physiology, Osaka Univ. Grad. Sch. of Dentistry)

15:00- [Short talk-2] Analysis of feeding behavior characteristics of ALS model mice
Yoshihiro Kitaoka (First Dept. of Oral and Maxillofacial Surgery, Osaka Univ. Grad. Sch. of Dentistry)

Coffee Break (15:15-15:25)

Session 2 (15:25-16:25)	Chair	Ayano Katagiri

15:25- [Mini review-2] Central mechanisms related to sensorimotor function and dysfunction of the tongue in humans
Hitoshi Maezawa (1. Faculty of Rehabilitation, Kansai Medical Univ., 2. Department of Neurological Diagnosis and Restoration, Osaka Univ. Grad. Sch. of Medicine)

15:55- [Short talk-3] Involvement of TRPM2 in orofacial neuropathic pain
Chiaki Yoshikawa (Dept. of Dental Anesthesiology, Osaka Univ. Grad. Sch. of Dentistry)

16:10- [Short talk-4] Somatosensory Receptors of Rat Chorda Tympani Geniculate Ganglion Neurons
Namiki Kishigami (First Dept. of Oral and Maxillofacial Surgery, Osaka Univ. Grad. Sch. of Dentistry)

Coffee Break (16:25-16:35)

Session 3 (16:35-17:05)	Chair	Yusuke Yokota

16:35- [Short talk-5] NK-1 and GABAB receptors mediate the long-term plasticity in the GABAergic synapses of the rat insular cortex
Kiyofumi Yamamoto (Dept. of Pharmacology, Nihon Univ. Sch. of Dentistry)

16:50- [Short talk-6] The mossy fiber pathway conveys orofacial proprioceptive signals from the supratrigeminal nucleus to the cerebellar cortex
Yumi Tsutsumi (Dept. of Oral Anatomy and Neurobiology, Osaka Univ. Grad. Sch. of Dentistry)

17:05- Closing Remarks Susumu Tanaka

Plenary Lecture

Development of therapeutic strategies to repair neuronal network for the central nervous system diseases

Toshihide Yamashita

Department of Molecular Neuroscience, Graduate School of Medicine, Osaka University

Initial behavioral deficits resulting from brain injury are frequently followed by spontaneous recovery of function, although this recovery is quite limited. It has been noted that synaptic plasticity in pre-existing pathways and the formation of new circuits through collateral sprouting of lesioned and unlesioned fibers are important aspects of the spontaneous recovery process. Although reorganization of the neural network is considered to contribute to this recovery, behavioral plasticity is not fully understood. Furthermore, the molecular mechanism of this phenomenon is poorly understood. We aim to elucidate the mechanisms underlying this plasticity, knowledge of which will contribute to enhancement of functional recovery after injury to the central nervous system. We have explored the mechanism of this behavioral plasticity, and more importantly, we have obtained evidence to show that immune modulation, inflammation-induced neovessels, and some types of microglia enhance plasticity and survival of neurons by secreting trophic factors. Disorders of the central nervous system, such as cerebrovascular diseases, cerebrospinal trauma, and encephalomyelitis, often cause spatiotemporal changes in the nervous system and in various biological systems, such as the immune system and vascular system. We analyzed the mechanism by which the spatiotemporal dynamics in those biological systems control a series of processes. Additionally, we aimed to elucidate the principle involved in the operation of living organisms with neural network disorders within the central nervous system by observing such disorders and their functional recovery process with respect to the dynamics of the entire biological system and by conducting a comprehensive analysis of the association between each system. These immune cells, neovessels, and microglia may prove to be drug targets for the treatment of CNS injuries, CNS inflammation, and neurodegenerative diseases. I will talk about our recent findings that uncover the molecular mechanism of formation and restoration of neuronal network in the CNS.

Key Words: neural network, central nervous system, regeneration

Abstract
Oral Session

Temporally-selective reconfiguration of cortical synapses underlies acquisition of proficient motor skill

Jaerin Sohn

Department of Oral Anatomy and Neurobiology, Graduate School of Dentistry, Osaka University

The architecture of neural circuits that underlies a variety of brain functions involves spatially-selective synaptic connections among neurons. The wiring diagram, “connectome”, composed of diverse cell types in the neocortex has been described anatomically, and we have also revealed the spatial selectivity of the subcellular synaptic configuration in single cells with fixed brain samples. This neural circuit map of the neocortex, however, lacks the information about the role that each wiring plays in a particular function of animals. Observation of synaptic dynamics may allow us to unveil “functional connectome” involved in animal’s behavior in vivo.

We therefore focused on synaptic remodeling in motor skill learning. Learning novel motor skills rewires neuronal connections by generating new synapses in the motor cortex. Of particular importance are the spines on pyramidal cell dendrites where synapses are potentiated, formed and maintained during learning. Since both corticocortical and thalamocortical fibers converge on the apical tufts of pyramidal cell dendrites, we characterized the presynaptic axon terminals innervating newly-formed spines. We observed spine dynamics under a two-photon microscope while Thy1-eGFP-M mice learned a forelimb motor task, and subsequently fixed the brains to prepare thin sections for immunohistochemistry. Post hoc characterization of the presynaptic axon terminals on new spines revealed that motor skill improvement coincided with selective formation of spines innervated by corticocortical axons. Thalamocortical synapses were generated fewer during motor learning but survived longer and increased spine size more than new corticocortical synapses. The input-dependent reconfiguration of learning-related new spines suggests that transient corticocortical synaptogenesis contributes to skill improvement while new thalamocortical connections are sustained for retention of the acquired motor memory. This temporally-selective dual spine supervision may govern diverse skill learning in the neocortex.

Key Words: Neocortex, neural circuit, synaptic dynamics

An electromyographic analysis of jaw-closing muscle activities during suckling and mastication in developing rats

Masaharu YAMADA[1,2], Ayano KATAGIRI[1], Yuji MASUDA[3], Hiroki TOYODA[1], Hitoshi NIWA[2], Takafumi KATO[1]

[1]. Department of Oral Physiology, Osaka university Graduate school of Dentistry
[2]. Department of Dental Anesthesiology, Osaka university Graduate school of Dentistry
[3]. Divison of Oral and Maxillofacial Biology, Institute for Oral Science, Matsumoto Dental University

[Introduction]
After birth suckling is only motor strategy for ingestion. Suckling is replaced by mastication for ingesting solid food at weaning. Both behaviors are characterized by rhythmic movements of the jaw. This study attempted to investigate developmental changes of jaw-closing muscle activities in suckling and mastication longitudinally in rats.
[Method]
Rat pups underwent a surgery to install the electrodes for electromyograms (EMG) of the masseter and the temporalis on the postnatal day 10 (P10). On P14, EMG for suckling was recorded. Rat pups were weaned on P21. From P21 to P49, EMG for mastication was recorded while chewing pellets and biting pasta sticks. For suckling and mastication (i.e., pellet chewing and pasta biting), Burst rhythm and, time-lag and correlation between the masseter and temporalis activities were calculated.
[Result]
On P14, burst rhythm of suckling including nipple attachment and rhythmic sucking were significantly slower than that of pellet chewing and pasta biting at P21. Time-lag and correlation differed between the suckling at P14 and mastication at P21. Burst rhythm for pasta biting and pellet chewing increased linearly with growth. For pellet chewing, the correlation increased from P21 to P24 significantly, and time-lag decreased from P21 to P24 significantly, but these variables were stable after P24. For pasta biting, correlation and time-lag did not change from P21 to P49.
[Conclusion]
The results demonstrate that 1) the distinct patterns of rhythmic jaw-closing muscle activities emerge before weaning, and 2) the developmental processes of jaw-closing motor dynamics would differ between chewing and biting after weaning.
Key Words: suckling, mastication, Development

Analysis of feeding behavior characteristics of ALS model mice

Yoshihiro Kitaoka, Soju Seki, Sou Kawata, Akira Nishiura, Kohei Kawamura, Shin-ichiro Hiraoka, Susumu Tanaka

First Department of Oral and Maxillofacial Surgery, Osaka University Graduate School of Dentistry

[Introduction] Amyotrophic lateral sclerosis (ALS) is an intractable disease with severe muscle weakness, and as the disease progresses, eating disorders are observed, hastening the time of death. Recent studies have indicated that the mesencephalic trigeminal neuron (MesV), the center of mastication, shows abnormality from neonatal ALS model mice, but the timing of the onset of mastication disorder are unknown. In this study, we created an AI model to detect the mastication movement of mice based on videos of their feeding behavior, and verified the mastication movements over time. We also analyzed electrophysiological modulation in MesV of the mature ALS model mice.

[Methods] We observed feeding behaviors of ALS model mice (SOD1-G93A; mSOD1) and wild-type mice (WT) from neonatal, and created an AI model to detect the opening and closing mouth movements of mSOD1 using a Single Shot Multibox Detector (SSD) based on the video information captured. In addition, electrical recordings were performed from MesV of mature mSOD1 mice using the patch clamp method.

[Results] In mSOD1 group, the opening phase duration was significantly prolonged after 15 weeks of age. Body weight was decreased at 12 weeks of age, and the correlation was found between weight loss and prolongation of opening phase duration. In the mSOD1 group at 12 weeks of age, the percentage of MesV with continuous firing activity showed a decreasing trend. In addition, a significant decrease in the duration of firing activity and an increase in spike frequency were observed.

[Conclusion:] In this study, modulation of masticatory movements and weight loss in ALS model mice were observed from the same period, and electrophysiological modulation in MesV of the mature ALS model mice. We intend to continue this study to elucidate the eating disorders observed in ALS and to develop therapeutic agents.

Key Words: Amyotrophic lateral sclerosis (ALS), AI, mastication

Session 2: Mini review-2

Central mechanisms related to sensorimotor function and dysfunction of the tongue in humans

Hitoshi Maezawa

1. Faculty of Rehabilitation, Kansai Medical University
2. Department of Neurological Diagnosis and Restoration, Graduate School of Medicine, Osaka University

The tongue plays an important role in a variety of critical sensorimotor functions in humans, including mastication, swallowing and speech production. Magnetoencephalography (MEG) is a powerful tool for elucidating cortical activity with high spatial and temporal resolution. However, MEG signal without artifact contamination is difficult during tongue stimulation and movement because of the proximity between the tongue and brain. Recent technological advances, such as motion capture systems and stimulus electrodes, successfully reduce the artifacts from the oral region. In the presentation, we provide a overview of central mechanisms related to tongue sensorimotor functions in humans, based on the findings of recent MEG studies. In addition, we review the clinical applications of MEG to evaluate sensory disturbances of the tongue.

First, I will introduce novel corticokinematic coherence (CKC) analysis that combines MEG measurement and deep-learning-assisted capture motion systems[1]. This CKC approach has the advantage of being magnetic, noise-free, movement-free, and risk-free, because no recording devices are placed on the tongue. The CKC approach may aid in revealing the pathophysiology of motor dysfunction of the tongue, such as lingual dystonia[2]. Furthermore, I will introduce a clinical approach for the quantitative evaluation of sensory disturbance of the tongue by measuring the somatosensory-evoked magnetic fields, following tongue stimulation with pin electrodes. Increased knowledge of the physiological mechanisms underlying tongue sensorimotor processing may improve our understanding of the cortical entrainment of stomatognathic function[3].

1. Maezawa H, et al., Sci Rep. 2022. 10;12(1):388. doi: 10.1038/s41598-021-04469-0.
2. Maezawa H, et al., Toxins (Basel). 2022. doi: 10.3390/toxins14110751.
3. Maezawa H. Front Hum Neurosci. 2017. 28;11:134. doi: 10.3389/fnhum.2017.00134

Key Words: stomatognathic function, magnetoencephalography, oscillation

Session 2: Short talk-3

Involvement of TRPM2 in orofacial neuropathic pain

Chiaki Yoshikawa, Hiroharu Maegawa, Hitoshi Niwa

Department of Dental Anesthesiology, Osaka University Graduate School of Dentistry

【Purpose】TRPM2 (transient receptor potential melastatin-2) is a member of the TRP family, and suggested to be involved in neuropathic pain. TRPM2 is also expressed in the microglia which has important role of pathogenesis of neuropathic pain. The aim of this study is to reveal the role of TRPM2 on orofacial neuropathic pain.
【Methods】We produced chronic constriction injury of infraorbital nerve (ION-CCI) rats as orofacial neuropathic pain model. After ligation, fluorescent immunostaining for ionized calcium-binding adaptor molecule-1 (Iba1) and TRPM2 was performed in the trigeminal spinal subnucleus caudalis (Vc). Next, miconazole, an antagonist of TRPM2, was administered intraperitoneally to the rats in two ways, as a single dose or as a 4-day series. Then, mechanical threshold to the whisker pad was measured by von Frey test, and immunostaining for phosphorylated extracellular signal-regulated kinase (pERK) was performed in the Vc. Immunostaining for CD68 was also performed.
【Results】TRPM2 was confirmed to be expressed on microglia in the Vc. Immunoreactivity of Iba1 and TRPM2 of 3 days after the ligation was significantly increased compared to that of 1 and 7 days after the ligation. Immunoreactivity of those was significantly increased in 3 and 7 days after the ligation compared to sham. ION-CCI rats with miconazole showed a significant increase in threshold. Compared to the vehicle group, ION-CCI rats with miconazole showed significant reductions in the number of pERK- and CD68-immunoreactive cells compared to ION-CCI with vehicle.
【Discussion】TRPM2 is suggested to be involved in orofacial neuropathic pain. Miconazole can have an inhibitory effect on mechanical hypersensitivity induced by infraorbital nerve injury.

Key Words: TRPM2, microglia, neuropathic pain

Somatosensory Receptors of Rat Chorda Tympani Geniculate Ganglion Neurons

Namiki Kishigami, Karen Yamauchi, Susumu Tanaka and Yusuke Yokota

First Department of Oral and Maxillofacial Surgery, Osaka University Graduate School of Dentistry, Suita, Osaka, Japan.

Chorda tympani nerve (CT) fibers have cell bodies in the geniculate ganglion and respond to taste stimuli of the anterior tongue, meanwhile lingual nerve responds to thermal and mechanical properties of oral stimuli. However, CT has also been known to be involved in somatosensory perception such as temperature and touch sensation (Yokota and Bradley. 2017). In addition, expressions of TRPV1, TRPA1, and TRPM8 channels have been reported in rat geniculate ganglion neurons (Katsura et al. 2006), which would suggest that CT has also other functions like mechano-sensor. In the present study, to investigate the involvement of TRPV1, TRPA1, and TRPM8 channels as somatosensory receptors, we applied each channel blockers to the lingual artery of rats in vivo. Adult female Sprague-Dawley rats (body weight 200 g; N = 13) were used for the experiments. Cold and tactile stimuli were applied to the anterior tongue, and action potentials were recorded from CT with Ag/AgCl electrode. TRP channel blockers were selectively injected into the lingual artery with intra-arterial administration, then the thermal and mechanical stimuli were applied again. With administration of TRPV1 blocker (SB-366791), responses to tactile stimuli were significantly decreased ($p = 0.049$). After injection of TRPA1 blocker (HC-030031), responses to cold stimuli were significantly decreased ($p = 0.015$), and responses to tactile stimuli were also decreased ($p = 0.041$). With administration of TRPM8 blocker (TRPM-8 antagonist 2), responses to cold stimuli were significantly decreased ($p = 0.018$). Our findings suggest that TRPA1 and TRPM8 channels on rat tongue dorsum play a role in CT thermal responses, and TRPV1 and TRPA1 channels play a role in CT tactile responses, respectively.

Key Words: chorda tympani, somatosensory receptor, TRP channel

NK-1 and GABAB receptors mediate the long-term plasticity in the GABAergic synapses of the rat insular cortex

Kiyofumi Yamamoto[1], Masayuki Kobayashi[1]
[1]Department of Pharmacology, Nihon University School of Dentistry

Several GABAergic non-pyramidal cell subtypes are identified, especially fast-spiking neurons (FSN), have many inhibitory synaptic connections to adjacent neurons. Long-term synaptic plasticity, such as long-term potentiation (LTP) and depression (LTD), have been well studied in the hippocampal and cerebrocortical excitatory synapses, and various mechanisms have been hypothesized. Although long-term plasticity in the inhibitory synapses between interneurons was reported, the detailed mechanisms underlying the induction of LTP/LTD have been still unknown. To examine whether LTP/LTD is induced in inhibitory synaptic connections of FSN ➜pyramidal neuron (PN) and to examine the mechanism, we applied the quadruple whole-cell patch-clamp and recorded unitary IPSCs (uIPSCs) in response to the paired-pulse stimulations to the presynaptic FSN, which enabled to calculate the paired-pulse ratio (PPR). θ-burst stimulation (TBS) to the presynaptic FSN induced changes in synaptic responses, including LTP/LTD. Changes in uIPSC amplitudes in response to TBS depended on PPR obtained before TBS. Bath application of a GABAB receptor (GABABR) antagonist and intracellular perfusion of a GTPase inhibitor into presynaptic FSN blocked to induce LTP. Surprisingly, a low concentration of baclofen (1 μM), a GABABR agonist, induced LTP-like synaptic potentiation in FSN ➜ PN synapses. Because U73122, added into presynaptic FSN, suppressed LTP induction, there is a possibility that phospholipase C (PLC) takes part in LTP induction. In addition, we tested the effects of SR140333, a selective NK1 receptor (NK1R) antagonist, and found that all FSN➜PN connections were enhanced by TBS, suggesting that the Substance P (SP)-NK1R pathway mediates LTD induction. A nitric oxide (NO) synthase inhibitor also suppressed LTD, which indicates that NO participates in LTD induction. These results suggest that PPR determines the direction of plasticity, and LTP and LTD depend on the presynaptic GABABR-PLC and SP-NK1R-NO pathways, respectively.

Key Words: insular cortex, parvalbumin, IPSC

Session 3: Short talk-6

The mossy fiber pathway conveys orofacial proprioceptive signals from the supratrigeminal nucleus to the cerebellar cortex

Yumi Tsutsumi[1], Yayoi Morita[1,2], Fumihiko Sato[1], Takahiro Furuta[1], Jaerin Sohn[1], Katsuro Uchino[3], Masayuki Moritani[4], Yong Chul Bae[5], Takafumi Kato[6], Yoshihisa Tachibana[7], Atsushi Yoshida[1,8]

[1]Dept. of Oral Anatomy and Neurobiology, Osaka Univ. Grad. Sch. of Dentistry

[2]Dept. of Dental Anesthesiology, Osaka Univ. Grad. Sch. of Dentistry

[3]Dept. of Acupuncture, Faculty of Health Care Science, Takarazuka Univ. of Medical and Health Care

[4]Dept of Physical Therapy, Faculty of Health Science, Morinomiya Univ. of Medical Sciences

[5]Dept of Anatomy and Neurobiology, Sch. of Dentistry, Kyungpook National Univ.

[6]Dept. of Oral Physiology, Osaka Univ. Grad. Sch. of Dentistry

[7]Div. of System Neuroscience, Kobe Univ. Grad. Sch. of Medicine

[8]Dept. of Oral Health Care, Faculty of Health Care Science, Takarazuka Univ. of Medical and Health Care

Proprioceptive signals from muscle spindles are essential for the regulation of motor functions. However, little is known about the motor control regions in the cerebellar cortex that receive proprioceptive signals from muscle spindles distributed throughout the body, including the orofacial muscles. We performed three experiments to reveal the projection patterns in the rat cerebellar cortex from the supratrigeminal nucleus (Su5), which conveys orofacial proprioception arising from jaw-closing muscle spindles (JCMSs). (1) We made injections of an anterograde tracer biotinylated dextranamine (BDA) into the Su5 identified electrophysiologicaly by responses to stimulation of the masseter nerve and to sustained jaw-opening movements. Many bilateral axon terminals (rosettes) were distributed in the granular layer of the hemisphere of the cerebellar cortex (including the simple lobule B, crus II, and flocculus). (2) We recorded JCMS proprioceptive signals in these cerebellar cortical areas (the simple lobule B, crus II, and flocculus) during stimulation of the masseter nerve and sustained jaw-opening movements. Then we injected a retrograde tracer Fluorogold or cholera toxin B subunit into these recording sites. Many retrogradely labeled cells were observed in the Su5 and other precerebellar nuclei. (3) We injected BDA into the external cuneate nucleus (ECu), which receives proprioceptive signals arising from forelimb and neck muscle spindles to clarify differences between the Su5- and ECu-projection patterns in the cerebellar cortex. The labeled terminals from the ECu were distributed predominantly in the vermis of the cerebellar cortex. Almost no overlap was seen in the terminal distribution of the Su5 and ECu projections. Our findings demonstrated that each proprioceptive signal arising from orofacial and other body-part muscles inputs different cerebellar cortical region and may be differentially processed in segregated domains of cerebellar functions. (COI : Non)

Key Words: Cerebellar cortex, Muscle spindle, Trigeminal

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