Oral Neuroscience 2023

Program & Abstract

April 20th, 2024

Osaka University Graduate School of Dentistry

Osaka, Japan

大阪大学 OSAKA UNIVERSITY

Challenge to Intractable Oral Diseases

Venue
Osaka University
Graduate School of Dentistry
Osaka, Japan

Organizing Committee

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Hitoshi Niwa
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Cosponsor

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

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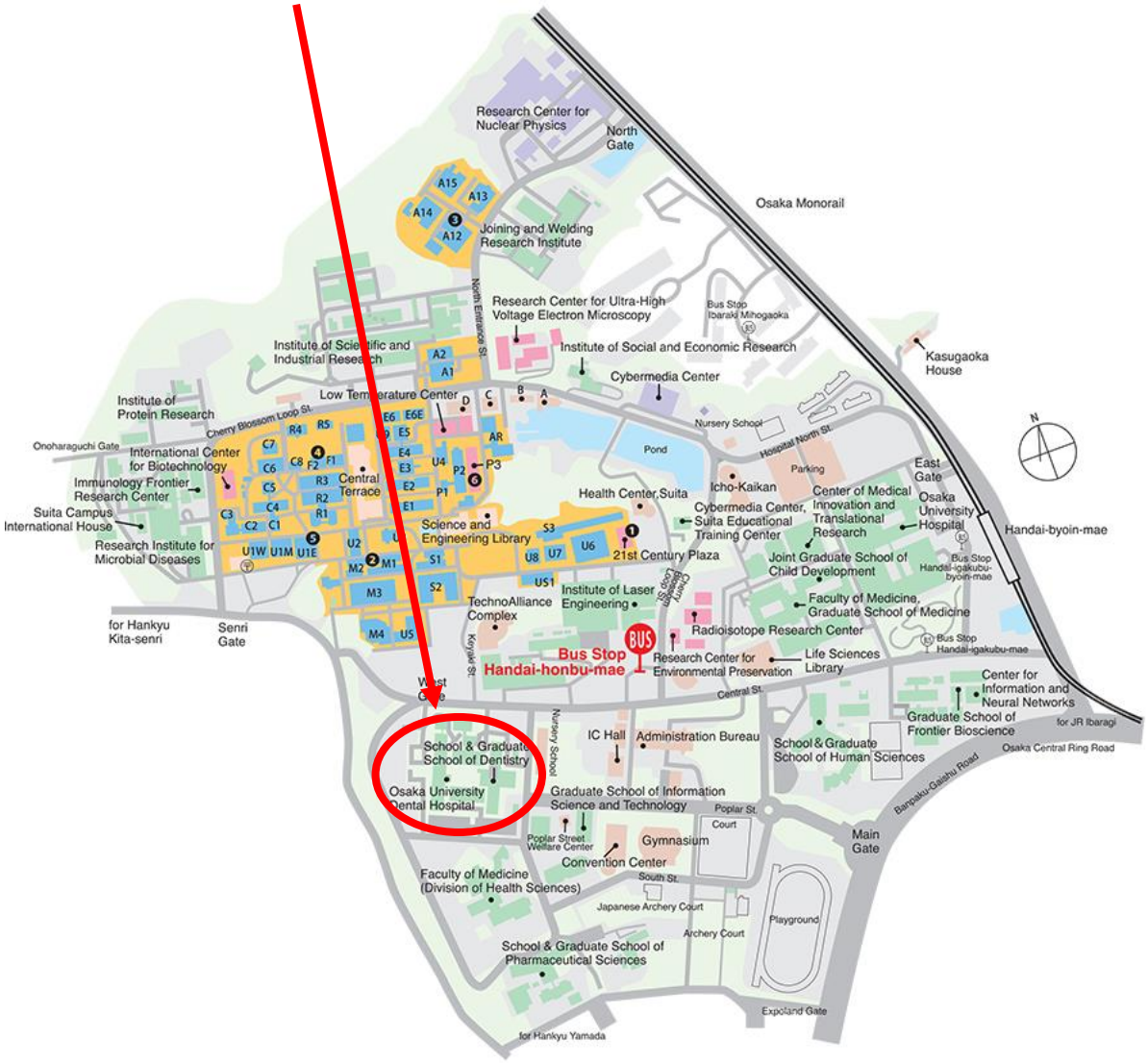
Access

Yumikura memorial hall (Building F, 5F)

Osaka University Graduate School of Dentistry

1-8 Yamadaoka, Suita, Osaka, Japan 565-0871



Oral presentation information

As you prepare for your oral presentation at Oral Neuroscience 2023, please find important information concerning your oral presentation.

Presentation time

- ✓ The time allowed for the slide presentation of Short Talk is 15 minutes including 5 minutes for discussion.
- ✓ The time allowed for the slide presentation of Plenary Lecture are 75 minutes including 15 minutes for discussion.

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Oral Neuroscience 2023

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

13:00- Opening Remarks Susumu Tanaka

Plenary Lecture (13:05-14:15)	Chair	Susumu Tanaka

13:05- Development of synthetic non-psychoactive cannabinoids for treatment of chronic pain
Igor Spigelman (Laboratory of Neuropharmacology, Section of Biosystems and Function, School of Dentistry, University of California, Los Angeles, CA, USA)

Coffee Break (14:15-14:30)

Session 1 (14:30-15:15)	Chair	Hiroki Toyoda

14:30- [Short talk-1] Correlation of chronic pain related behavior and microglia in trigeminal spinal subnucleus caudalis
Toru Yamamoto (Division of Dental Anesthesiology, Faculty of Dentistry & Graduate School of Medicine and Dental Sciences, Niigata University, Niigata, Japan)

14:45- [Short talk-2] Hormonal mechanisms of the paraventricular nuclei in the hyperalgesia in the Parkinson's disease model rats
Yang Shengsen (Department of Dental Anesthesiology, Osaka University Graduate School of Dentistry)

15:00- [Short talk-3] Preservation of Masticatory Function in ALS: Insights from a Longitudinal Study on Mouse Models
Sou Kawata (Department of Oral and Maxillofacial Surgery, Graduate School of Dentistry, Osaka University,)

Coffee Break (15:15-15:30)

Session 2 (15:30-16:00)	Chair	Soju Seki

15:30- [Short talk-4] Cell Type-Dependent Activity Modulation of Movement-Related Layer 5 Neurons in the Motor Cortex

Koshi Irisa (Department of Physiological Sciences, Graduate University of Advanced Studies, SOKENDAI, Department of Systematic Anatomy and Neurobiology, Graduate School of Dentistry, Osaka University)

15:45- [Short talk-5] Involvement of axon initial segment (AIS) abnormality in attention-deficit hyperactivity disorder (ADHD)-like behaviors of mice

Misaki Iwasaki (United Grad Sch of Child Dev, Osaka Univ, Dept Pharmacol, Grad Sch Dent, Osaka Univ, Mol Res Ctr Child Mental Dev, United Grad Sch Child Dev, Osaka Univ,)

Session 3 (16:05-16:35)	Chair	Atsuko Hayata

16:05- [Short talk-6] Effects of systematic yohimbine administration on masticatory muscle activities during sleep-wake states in freely moving rats

Yiwen Zhu (Department of Oral Physiology, Graduate School of Dentistry, Osaka University)

16:20- [Short talk-7] The mossy fiber pathway conveys orofacial proprioceptive signals from the supratrigeminal nucleus to the cerebellar nuclei

Yumi Tsutsumi (Dept. of Oral Anatomy and Neurobiology, Osaka Univ. Grad. Sch. of Dentistry)

16:40- Closing Remarks Takafumi Kato

Plenary Lecture

Development of synthetic non-psychoactive cannabinoids for treatment of chronic pain

Igor Spigelman

Laboratory of Neuropharmacology, Section of Biosystems and Function, School of Dentistry, University of California, Los Angeles, CA, USA

Synthetic and naturally occurring cannabinoids are a focus of social, legal and medical controversy concerning their therapeutic utility, yet studies show that cannabinoid-based analgesics reduce persistent pain of inflammatory and neuropathic origin in humans and animals. Also, cannabinoids are effective in alleviating chronic pain symptoms after repeated treatment, unlike opioids, which have limited long-term effectiveness. However, cannabinoids also exhibit side effects, the most troubling of which are mediated by activation of cannabinoid 1 receptors (CB1Rs) within the central nervous system (CNS). These psychotropic CNS effects also account for the abuse potential of plant-based and synthetic cannabinoids. CB1Rs and their endogenous ligands (endocannabinoids) are widely distributed in the CNS and peripheral tissues where they participate in various physiological functions including pain modulation. A major component of pain relief after administration of cannabinoids is mediated by peripheral CBRs. I will describe our efforts in the development of synthetic peripherally-restricted cannabinoids (PRCBs) focusing on the preclinical pharmacology of PRCBs in oral and bone cancer, chemotherapy-induced neuropathy, peripheral nerve injury, and migraine-like pain. Due to their lack of appreciable CNS penetration PRCBs represent an alternative to the psychoactive components of medicinal cannabis, as well as opioid abuse and overdose by chronic pain patients. Clinical implementation of PRCBs for treatment of targeted patient populations would also provide relief of their pain and suffering without affecting mental acuity, motor coordination, or memory.

Key Words: Oral cancer, chronic pain, cannabinoid

Abbreviated Curriculum Vitae

Igor Spigelman

Affiliation: Professor, Laboratory of Neuropharmacology,
Chair, Section of Biosystems and Function,
School of Dentistry, University of California, Los Angeles

DEGREES OBTAINED:

Type	Institution	Specialty	Date
B.Sc.	University of Toronto	Biology	Nov. 1983
M.Sc.	The University of British Columbia	Neuroscience	May 1986
Ph.D.	The University of British Columbia	Pharmacology	July 1988
Postdoctoral	Toronto Hospital & Univ. of Toronto	Neuropharmacology	1988-1991

EXPERTISE: Neuropharmacology of chronic pain, epilepsy, alcoholism, and traumatic stress. Developmental and disease-induced alterations in the function of voltage- and ligand–gated ion channels. Development of non-addictive analgesics (e.g., peripherally restricted cannabinoid receptor agonists and gene therapy targeting selective sodium channels). Most studies are multidisciplinary efforts involving collaborators within and outside of UCLA.

TEACHING: Dental, medical, and graduate students in various aspects of drug action, abuse, overdose, and dependence. This includes cardiovascular and gastrointestinal drugs, analgesics, anticonvulsants, and psychoactive drugs (e.g., lithium salts, antidepressants, antipsychotics, etc.).

CONSULTANT: In the past 14 years served as a pharma industry consultant and provided expert testimony for nine US law firms and the Department of Justice, Health & Welfare, Canada. This included comprehensive case evaluation and deposition related to dental & medical malpractice (plaintiff and defendant) in general pharmacology and toxicology, prescription errors, pharmacology of drug abuse, chronic pain, neurological disorders & trauma.

MEMBERSHIP IN PROFESSIONAL SOCIETIES:

Position	Society	Member since
Member	Society for Neuroscience	1987
Member	International Association for the Study of Pain	1989
Member	UCLA Brain Research Institute	1991
Member	International Association for Dental Research	2000
Member	American Society for Pharmacology & Experimental Therapeutics	2004
Member	Research Society on Alcoholism	2006
Member	International Cannabinoid Research Society	2018

PEER-REVIEW PUBLICATIONS: **> 90** **BOOK CHAPTERS:** **>10**

REVIEWER FOR: National Institutes of Health, Department of Veterans Affairs, Alzheimer's Association, New Zealand Neurological Foundation, U.S. Civilian Research & Development Foundation, Portuguese Ministry of Science and Technology, >30 RESEARCH JOURNALS SINCE 1992, Senior Editor for Neuropharmacology.

RESEARCH MENTOR: 15 pre-college students, >90 undergraduate students, 10 dental students, 7 MS graduates, 7 PhD graduates, 18 post-doctoral fellows, 9 staff researchers, 4 visiting scientists.

Abstract
Oral Session

Correlation of chronic pain related behavior and microglia in trigeminal spinal subnucleus caudalis

Toru Yamamoto [1], Mitsuhiro Yoshida [2], Yuhei Koyama [1], Eiji Imado [2], Naotaka Kishimoto [1], and Kenji Seo [1]

[1]. Division of Dental Anesthesiology, Faculty of Dentistry & Graduate School of Medicine and Dental Sciences, Niigata University, Niigata, Japan
[2]. Division of Oral and Maxillofacial Surgery and Oral Medicine, Department of Dental Anesthesiology, Hiroshima University Hospital, Hiroshima, Japan

The aim of this study was to investigate the mechanisms underlying carrageenan-induced chronic hyperalgesia and the potential preventive effect of meloxicam. Rats were injected with 3% carrageenan into the masseter muscle and exhibited acute and chronic hyperalgesia to mechanical stimuli for 6 weeks after injection. Pre-treatment with meloxicam prevented carrageenan-induced chronic hyperalgesia. Furthermore, carrageenan in combination with the steroid, but not with acetaminophen, suppressed hyperalgesia in the chronic phase. Microglial activation in the trigeminal spinal subnucleus caudalis (Vc) was assessed by immunohistology 3 days after treatment. The reactivity of microglial cells in the Vc was increased in carrageenan-treated rats compared to sham (control) rats. Subcutaneous administration of meloxicam prior to carrageenan injection significantly suppressed the carrageenan-induced increase in Vc microglial reactivity. These results suggest that early prevention of peripheral and spinal inflammation may suppress microglial reactivity in the Vc and the development of chronic pain.

Key Words: Carrageenan, Muscle chronic pain, Microglia

Session1: Short talk-2

Hormonal mechanisms of the paraventricular nuclei in the hyperalgesia in the Parkinson's disease model rats

Yang Shengsen, Usami Nayuka, Maegawa Hiroharu, Toyama Midori, Shigemasa Hiroaki, Ueda Mayuka, Kudo Chiho, Niwa Hitoshi

Department of Dental Anesthesiology, Osaka University Graduate School of Dentistry

[Objective]
Hyperalgesia in Parkinson's disease (PD) model rats has been reported in prior studies, but the mechanism remains unclear. We have suggested that hyperalgesia in PD model rats may be related to changes in neuronal activity of oxytocin (OXT)-producing cells in the paraventricular nucleus (PVN). In the present study, we investigated the relationship between the arginine vasopressin (AVP) and corticotropin releasing hormone (CRH), which are regulated by the PVN in addition to OXT, and hyperalgesia in PD.
[Methods & Results]
PD model rats were made by injecting 6-hydroxydopamine into the left medial forebrain bundle of male Wistar rats, and the rotation test was performed two weeks later. The subcutaneous injection of 5% formalin solution was injected into the left upper lip of the PD model and Sham rats under anesthesia, and blood was drawn from the heart 5 or 15 minutes later, followed by perfusion fixation. PD model and Sham rats with no injection were used as control groups. Immunostaining with antibody against tyrosine hydroxylase was performed, and immunoreactivity in the left substantia nigra and striatum was found to be decreased in PD model rats. Immunostaining of the trigeminal spinal subnucleus caudalis with antibody against p-ERK revealed increased number of p-ERK-immunoreactive cells in PD model rats, indicating hyperalgesia. Double staining with each antibody against OXT, AVP and CRH in combination with antibody against p-ERK in the PVN was also performed. The OXT, AVP and CRH levels in serum without and after formalin injection were measured with ELISA kits, and the levels of OXT and CRH were significantly decreased in PD model rats than Sham rats after formalin injection.
[Conclusion]
In PD model rats, the functions related to OXT and CRH in the PVN were altered, suggesting that this may have affected the hyperalgesia to injection of formalin.

Key Words: Hyperalgesia, Paraventricular nuclei, Parkinson's disease

Session1: Short talk-3

Preservation of Masticatory Function in ALS: Insights from a Longitudinal Study on Mouse Models

Sou Kawata[1], Soju Seki[1], Akira Nishiura[1], Yoshihiro Kitaoka[1], Mikihiko Kogo[1], Susumu Tanaka[1]

[1]. Department of Oral and Maxillofacial Surgery, Graduate School of Dentistry, Osaka University

In our study, we investigated the impact of Amyotrophic Lateral Sclerosis (ALS), a severe neurodegenerative disorder targeting motor neurons, on masticatory function compared to limb motor function. Utilizing ALS model mice (SOD1G93A) and wild-type controls, we measured changes in the masseter (a major masticatory muscle) and limb skeletal muscle volume from 4 to 16 weeks of age, alongside assessing muscle function and conducting detailed tissue analyses. Our findings revealed that, unlike the limb muscles, the masseter muscle did not exhibit a significant reduction in volume or function in ALS model mice over time. While limb muscles showed marked atrophy and functional decline, the masseter muscle maintained its volume and showed no significant functional impairment throughout the study period. Intriguingly, we observed an increase in the number of satellite cells in the masseter muscle at later stages of ALS in the model mice, indicating a potentially higher regenerative capacity of this muscle compared to limb muscles.

These results suggest that masticatory muscles, specifically the masseter, may possess unique protective mechanisms against the degenerative effects of ALS, maintaining function and muscle volume even in later stages of the disease. The observed increase in satellite cells further supports this notion, hinting at an enhanced regenerative response. Understanding these protective mechanisms in the masseter muscle could provide crucial insights into the pathology of ALS and open new avenues for developing therapeutic strategies aimed at mitigating muscle atrophy and preserving motor function in ALS patients. This study underscores the importance of exploring muscle-specific responses in neurodegenerative diseases, which could lead to muscle-specific therapeutic interventions.

Key Words: Carrageenan, ALS, SOD1G93A mouse, masticatory muscle

Session2: Short talk-4

Cell Type-Dependent Activity Modulation of Movement-Related Layer 5 Neurons in the Motor Cortex

Koshi Irisa[1,2], Jaerin Sohn[2], Takuma Tanaka[3], Takahiro Furuta[2]

[1]. Department of Physiological Sciences, Graduate University of Advanced Studies, SOKENDAI
[2]. Department of Systematic Anatomy and Neurobiology, Graduate School of Dentistry, Osaka University
[3]. Graduate School of Data Science, Shiga University

The primary motor cortex (M1) plays a critical role in the control of voluntary movements. Previous in vivo electrophysiological studies have revealed the presence of neurons exhibiting activity modulation during movement within M1. For example, the neuronal activity in the vibrissa area of M1 (vM1) of rodents is also known to be modulated during rhythmic back-and-forth vibrissa movement, known as “whisking”. However, the precise functioning of these movement-related neurons in motor control has not been fully understood, in part due to limited knowledge about where their signals are directed. Layer 5 (L5), the output layer of the neocortex, in M1 contains two distinct classes of pyramidal cells with unique projection targets: pyramidal tract (PT) cells and intratelencephalic (IT) cells. In the present study, we explored the activity profiles of these pyramidal cell subclasses as well as putative inhibitory neurons during whisking to gain insights into the distinct roles played by different cell types in motor control. We performed juxtacellular recordings in vM1 under high-speed video monitoring of self-initiated whisker motion in head-fixed awake mice. The recordings identified a variety of neuronal types with unique firing properties during whisking; a small subset of neurons displayed a pronounced peak precisely coinciding with the initiation of whisking (“Pre-onset type”), while half of L5 neurons were persistently activated (“UP type”) or inactivated (“DOWN type”) during the rhythmic whisking period. Cell identification by juxtacellular labeling combined with immunohistochemistry for chicken ovalbumin upstream promotor transcription factor-interacting protein 2 (Ctip2), which is a marker for PT cells, revealed markedly different proportions of those electrophysiologically defined cell types in PT and IT neurons. This study suggests that individual M1 neurons participate in the regulation of rhythmic movements in a projection type-dependent manner.

Key Words: Primary motor cortex, Whisker system, Juxtacellular recording

Session2: Short talk-5

Involvement of axon initial segment (AIS) abnormality in attention-deficit hyperactivity disorder (ADHD)-like behaviors of mice

Misaki Iwahashi[1,2,4], Takeshi Yoshimura[1], Wakana Harigai[1], Kazuhiro Takuma[2,3], Hitoshi Hashimoto[3-7], Taiichi Katayama[1], Atsuko Hayata-Takano[2-4]

[1]. United Grad Sch of Child Dev, Osaka Univ
[2]. Dept Pharmacol, Grad Sch Dent, Osaka Univ
[3]. Mol Res Ctr Child Mental Dev, United Grad Sch Child Dev, Osaka Univ
[4]. Lab Mol Neuropharmacol, Grad Sch of Pharmaceut Sci, Osaka Univ
[5]. Inst Open Transdiscip Res Initiatives, Osaka Univ
[6]. Inst Datability Sci, Osaka Univ
[7]. Dept Mol Pharmaceut Sci, Grad Sch Med, Osaka Univ

Attention-deficit hyperactivity disorder (ADHD) is a neurodevelopmental disorder (NDD) with three characteristics: hyperactivity, inattention, and impulsivity. These symptoms can cause difficulties with patient's social life. However, molecular mechanisms underlying these symptoms remain unclear. Axon initial segment (AIS) is a region of the proximal axon, which is responsible for action potential initiation. Recent studies have shown that the structure of AIS can be altered based on the input to the neuronal circuit, and the alteration can impact neural activity. In addition, we have recently found an abnormality of the AIS length in cortical regions of rodent models of ADHD and autism spectrum disorder. However, the relationship between AIS length abnormality and NDD pathology is still unknown.

In this study, we examined whether AIS length alteration occurs in pituitary adenylate cyclase-activating polypeptide (PACAP)-deficient ($PACAP^{-/-}$) mice, which display ADHD-like behaviors. We also examined the effects of atomoxetine, an ADHD drug, on the AIS length in $PACAP^{-/-}$ mice. And then, we found that $PACAP^{-/-}$ mice exhibited a longer AIS length in layer 2/3 neurons of the primary somatosensory barrel field (S1BF) compared with wild-type mice. In addition, repeated treatments of atomoxetine improved AIS abnormality along with hyperactivity in $PACAP^{-/-}$ mice. These results suggest that AIS abnormality is associated with the phenotypes of the ADHD mouse model, that is, AIS length abnormality may be closely related to NDD pathology.

Key Words: Attention-deficit hyperactivity disorder (ADHD), pituitary adenylate cyclase-activating polypeptide (PACAP), axon initial segment

Session3: Short talk-6

Effects of systemic yohimbine administration on masticatory muscle activities during sleep-wake states in freely moving rats

Yiwen Zhu[1], Ayano Katagiri[1], Hiroki Toyoda[1], Masaharu Yamada[1,2], Makoto Higashiyama[1], Takafumi Kato[1,3,4]

[1]. Department of Oral Physiology, Graduate School of Dentistry, Osaka University
[2]. Department of Dental Anesthesiology, Graduate School of Dentistry, Osaka University
[3]. United Graduate School of Child Development, Osaka University
[4]. Sleep Medicine Center, Osaka University Hospital

[Background]
Systemic yohimbine (YOH) administration has been shown to induce stress/anxiety conditions manifested as sleep disruption and increase in sympathetic activity which are the potential factors of masticatory muscle activity fluctuation during sleep-wake states.
[Method]
In eighteen Sprague-Dawley rats, electrodes for electroencephalogram (EEG) and electromyogram (EMG) of neck muscle were surgically implanted for sleep recording, along with those for electrocardiogram (ECG) to monitor heart rate and EMG of left masseter muscle to track masticatory muscle activity. Sleep recordings over two days included control data from ZT4 to ZT17 on the first day and baseline data from ZT4 to ZT6 on the next. Following injection at ZT6, the recording was continued until ZT17. Rats were divided into three groups and intraperitoneally injected with YOH at three different doses (1.0, 2.0, and 3.0 mg/kg). Vigilance states (wakefulness, non-rapid eye movement [NREM] sleep, and rapid eye movement [REM] sleep), heart rate and masticatory muscle activity were scored for every 10-s epoch. After minimum value was subtracted, integrated EMG activity for each epoch was normalized by that during chewing.
[Results]
During light phase, the percentage of wakefulness increased dose-dependently while those of NREM and REM sleep decreased after YOH injection. YOH also reduced the number of NREM sleep episodes dose-dependently during light phase. During both light and dark phase, heart rate significantly increased in a dose-dependent manner after YOH injection. There is no significant difference in EMG activity of masseter muscle before and after YOH injection.
[Conclusion]
Acute systematic YOH altered sleep-wake states and elevated sympathetic activities in a dose-dependent manner while having no effect on masticatory muscle activity.

Key Words: Sleep, masticatory muscle activity, yohimbine

Session3: Short talk-7

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

Yumi Tsutsumi[1], Yayoi Morita[2], Fumihiko Sato[1], Takahiro Furuta[1], Jaerin Sohn[1], Katsuro Uchino[3], Masayuki Moritani[4], Hitoshi Niwa[2], Yoshihisa Tachibana[5], Atsushi Yoshida[1,6]

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[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]. Div. of System Neuroscience, Kobe Univ. Grad. Sch. of Medicine
[6]. 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 nuclei 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 nuclei 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 electrophysiologically by responses to stimulation of the masseter nerve and to sustained jaw-opening movements. BDA-labeled axon terminals were observed bilaterally in the cerebellar nuclei, especially in the dorsolateral hump (IntDL) of the cerebellar interposed nucleus and the dorsolateral protuberance (MedDL) of the cerebellar medial nucleus. (2) We recorded JCMS proprioceptive signals in these cerebellar nuclear areas (the IntDL and MedDL) during stimulation of the masseter nerve and sustained jaw-opening movements. Then we injected a retrograde tracer Fluorogold 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 nuclei. The projection from the ECu to the cerebellar nuclei was very weak and was ipsilaterally found in the dorsomedial crest of the cerebellar interposed nucleus. These results indicate that the cerebellar nuclei receive proprioceptive signals from masticatory muscle

spindles and may be involved in masticatory motor regulation. Furthermore, these results suggest that the orofacial and body movements are regulated in different areas of the cerebellar nuclei. (COI : Non)

Key Words: Cerebellar nuclei, Muscle spindle, Trigeminal

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