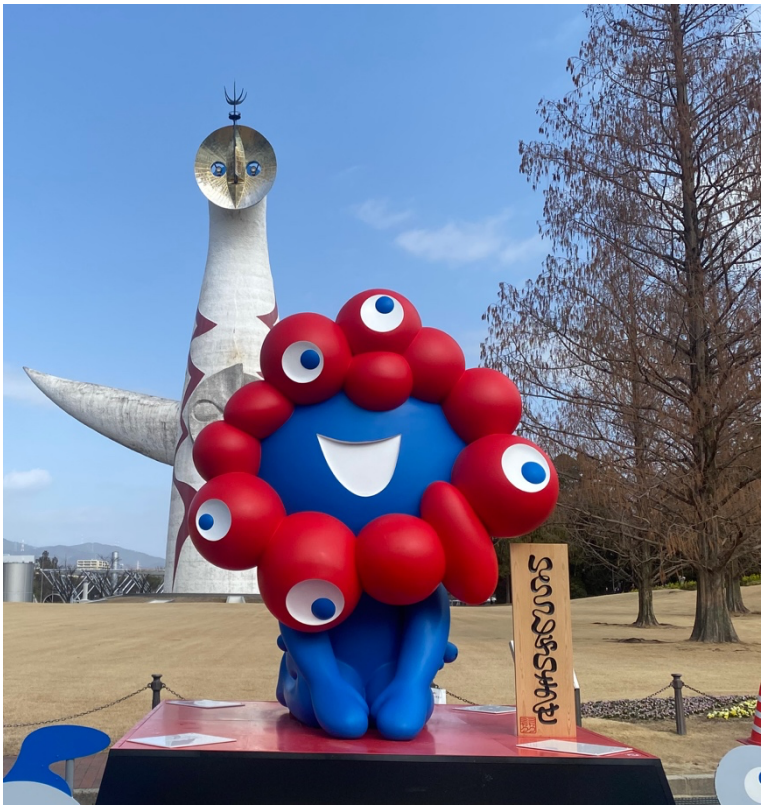
The International Symposium on Oral Neuroscience 2025

April 4th, 2026



Program & Abstract

大阪大学 THE UNIVERSITY OF OSAKA

Challenge to Intractable Oral Diseases

Venue: Yumikura Memorial Hall (Building F, 5F)
Graduate School of Dentistry, The University of Osaka, Osaka, Japan

Remarks

All attendance including oral presenters, chairmans and staffs, please attend with wearing casual (informal) clothes (e.g., no jacket, no tie).

The International Symposium on Oral Neuroscience 2025

Program & Abstract

April 4th, 2026

Graduate School of Dentistry, The University of Osaka

Osaka, Japan

Organizing Committee

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Cosponsor:

"Challenge to Intractable Oral diseases" from Graduate School of Dentistry, The University of Osaka

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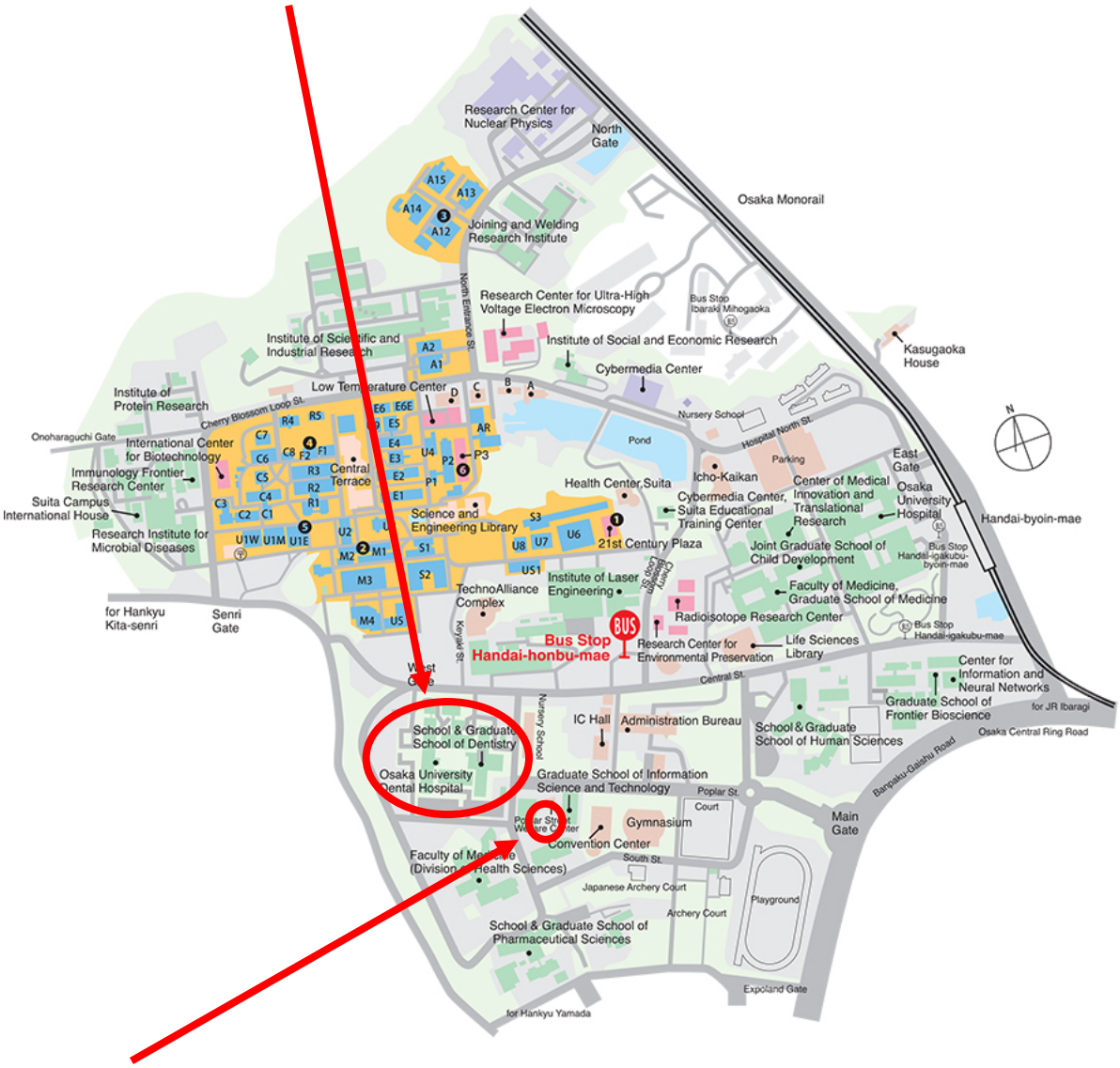
Access

Oral Session (13:30–)

Yumikura memorial hall (Dental Faculty Building F, 5F)

Graduate School of Dentistry, The University of Osaka

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



Information exchange meetings (17:00–)

Restaurant Couleur, (Welfare Building, Poplar Street, 1F)

Suita Campus, The University of Osaka

1-12 Yamadaoka, Suita, Osaka, Japan 565-0871

(about a few minutes' walk from Dental faculty building)

Information

Accommodation

The registration desk will open from 12:30 PM to 4:00 PM that day. Please make sure to receive your name tag.

General (not including speakers): 3,000 yen

Student: free

Social gathering @ Restaurant Couleur

The party will be held from 5:00 PM to 7:00 PM. Please pay the fee at the registration desk.

Buffet style, some foods & drinks (beer, beverage, coffee, etc.)

General (not including speakers): 6,000 yen

Student (not including speakers): 3,000 yen

Internet Service

Wireless internet access is unavailable in the conference room. Please take your mobile Wi-Fi router.

If you have an “eduroam” ID issued by your university or institute, you can connect to the eduroam Wi-Fi network during your visit to Osaka University.

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Presentation information for speakers

As you prepare for your oral presentation at Oral Neuroscience 2025, please find important information concerning your oral presentation.

* Please bring your presentation on your PC.
* Please check the monitor output terminal of your PC. The PC terminal in the conference room is HDMI-type. If your PC does not have its type, please bring a conversion adaptor.
* Be sure to bring an AC adaptor.
* Please adjust your computer settings so it does not revert to screensaver or energy-saving mode during your presentation.
* If your presentation data includes links to movies, graphs, or similar data, please be sure to save these files and check their operation in advance. (checking time: 12:45-13:15 and resting time before your presentation)
* Please bring your PC to the staff at break times or below and check your file before your presentation.

Presentation time

Please confirm the presentation time you use.

Short talk: The time allowed for the slide presentation is 15 minutes, including 5 minutes for discussion.

Mini-review: The time allowed for the slide presentation is 30 minutes, including 5 minutes for discussion.

Oral Neuroscience 2025　Program

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

12:30–16:00　Registration

13:30–13:35　Opening Remarks　　Chair Kazuhiro Takuma

Mini Review 1 (13:35–14:05)	Chair	Kazuhiro Takuma (UOsaka)

13:35–　PACAP–PAC1 signaling in stress responses: circuit mechanisms and the antidepressant action of PAC1 antagonism

Atsuko Hayata-Takano (Department of Pharmacology, Graduate School of Dentistry, The University of Osaka)

Interval (14:05-14:10)

Short Talk (14:10–15:25)	Chair	Soju Seki (UOsaka)

14:10–　Respiratory effort increases before sleep-related masticatory muscle activity in sleep bruxism subjects: A chest–abdominal respiratory analysis

Kento Hata (Department of Regenerative Prosthodontics Graduate School of Dentistry, The University of Osaka)

14:25–　Effects of hypoxia-induced respiratory effort on jaw muscle activity in urethane-anesthetized rats

Cao Jiyuan (Department of Regenerative Prosthodontics Graduate School of Dentistry, The University of Osaka)

14:40–　Input and output patterns of non-nociceptive information in the caudal subnucleus of the spinal trigeminal nucleus

Risa Kimura (Department of Systematic Anatomy and Neurobiology, Graduate School of Dentistry, The University of Osaka)

14:55– Sensory neuron dysfunction in dorsal root ganglia during disease onset in the SOD1G93A mouse model for ALS

Akira Nishiura (Department of Oral and Maxillofacial Surgery, Graduate School of Dentistry, The University of Osaka)

15:10– Clonidine attenuated masticatory muscle activity elevation across sleep-wake cycle induced by acute foot-shock stress

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

Coffee Break (15:25-15:40)

Mini Review 2 (15:40-16:40)	Chair	Chiho Kudo (UOsaka)

15:40– Wasabi ingredient, hexaraphane, promotes dentin formation via SLC4As-NHE-NCX-PMCA signaling axis

Maki Kimura (Department of Physiology, Tokyo Dental College)

16:10– Thalamo-insular pathway regulates tic generation via motor-limbic crosstalk

Yoshihisa Tachibana (Department of Physiology and Cell Biology, Kobe University Graduate School of Medicine)

16:40– Closing Remarks

17:00– Information exchange meeting at the Restaurant Couleur, Welfare Building 1F.

Abstract

Mini Review 1 (13:35–14:05)

PACAP–PAC1 signaling in stress responses: Circuit mechanisms and the antidepressant action of PAC1 antagonism

Atsuko Hayata-Takano[1,2,3], Yusuke Shintani[4], Chiaki Asaka[2], Hiroki Toyoda[5], Yukio Ago[6], Takafumi Kato[3,7], Kazuhiro Takuma[1,3], Hitoshi Hashimoto[2,3]

[1]Department of Pharmacology, Graduate School of Dentistry, The University of Osaka, Suita, Osaka, Japan; [2]Laboratory of Molecular Neuropharmacology, Graduate School of Pharmaceutical Sciences, The University of Osaka, Suita, Osaka, Japan; [3]United Graduate School of Child Development, Molecular Research Center for Children's Mental Development, The University of Osaka, Suita, Osaka, Japan; [4]Department of Physiology and Cell Biology, Kobe University School of Medicine, Kobe, Hyogo, Japan; [5]Department of Physiology, School of Dentistry, Aichi Gakuin University, Nagoya, Aichi, Japan; [6]Department of Cellular and Molecular Pharmacology, Graduate School of Biomedical and Health Sciences, Hiroshima University, Hiroshima, Japan; [7]Department of Oral Physiology, Graduate School of Dentistry, The University of Osaka, Suita, Osaka, Japan

Stress-related psychiatric disorders, including major depression and post-traumatic stress disorder (PTSD), impose a substantial global burden, and current pharmacotherapies often show limited remission rates, leaving significant unmet clinical needs. Pituitary adenylate cyclase–activating polypeptide (PACAP) and its receptor PAC1 have been implicated in stress responses and in the pathophysiology of anxiety, depression, and PTSD based on both human genetic and animal studies. In this study, we evaluated the therapeutic potential of PA-915, a non-peptidergic, high-affinity PAC1 receptor antagonist, using multiple animal models, and further investigated PACAP-associated neural circuits involved in stress regulation.

A single intraperitoneal administration of PA-915 ameliorated anxiety- and depression-like behaviors in mice subjected to repeated social defeat stress (RSDS), producing rapid and sustained antidepressant effects. PA-915 also normalized the reduced dendritic spine density and the decreased excitation–inhibition (E/I) ratio of layer V pyramidal neurons in the medial prefrontal cortex (mPFC) of RSDS mice. The mPFC is a brain region involved in emotional processing and cognitive functions, and accumulating evidence indicates structural and functional abnormalities in patients with depression. Therefore, we used chemogenetic approaches to selectively suppress mPFC-projecting PACAP neurons. In RSDS-exposed mice, inhibition of this pathway ameliorated anhedonia and depressive-like behavior as assessed by sucrose preference and forced swim tests. Retrograde tracing revealed that PACAP-expressing inputs to the mPFC predominantly originate from the amygdala and specific thalamic nuclei. Circuit-specific activation using Cre-dependent and Flp-based expression systems increased immobility in the forced swim test and elevated corticosterone levels.

These findings indicate that PAC1 receptor antagonism represents a promising therapeutic strategy for stress-related disorders and that mPFC-projecting PACAP circuits critically

contribute to stress responses, potentially through modulation of hypothalamic−pituitary–adrenal (HPA) axis activity.

Key words: PACAP, stress-related psychiatric disorders, prefrontal cortex (mPFC), chemogenetic techniques

Short Talk-1 (14:10–14:25)

Respiratory effort increases before sleep-related masticatory muscle activity in sleep bruxism subjects: A chest–abdominal respiratory analysis

Kento Hata[1,2], Ryota Takaoka[1], Masahiro Nishimura[1], Takafumi Kato[2]

[1] Department of Regenerative Prosthodontics, Graduate School of Dentistry, The University of Osaka, Suita, Osaka, Japan; [2] Department of Oral Physiology, Graduate School of Dentistry , The University of Osaka Suita, Osaka, Japan

Previous studies have reported a slight decrease in blood oxygen saturation prior to rhythmic masticatory muscle activity (RMMA) in individuals with sleep bruxism (SB), and mandibular advancement devices have been shown to reduce RMMA frequency. These findings suggest that increased upper airway resistance may contribute to RMMA generation. Because increased airway resistance requires greater respiratory effort to maintain ventilation, this study investigated whether respiratory effort increases prior to RMMA onset. Polysomnographic recordings were obtained from ten healthy volunteers in their twenties (4 males and 6 females; mean age 23.1 ± 1.19 years; BMI 19.6 ± 1.7 kg/m²) selected based on an apnea–hypopnea index ≤5 and an RMMA index ≥4 episodes/hour. Twenty-second segments immediately preceding RMMA and non-specific masticatory muscle activity (NSMMA) episodes were extracted, excluding segments containing masseter activity or apnea/hypopnea events. A total of 236 RMMA segments and 326 NSMMA segments were analyzed, along with 236 artifact-free segments during quiet sleep. Chest and abdominal respiratory waveforms were plotted as Lissajous figures. We defined the Respiratory Phase Difference Index (RPDI) as the average perpendicular distance from the Lissajous curve to its regression line, calculated every 0.1 seconds. Median RPDI values were calculated for each subject. RPDI values preceding RMMA and NSMMA were significantly higher than those during quiet sleep ($P = 0.0059$), with no significant difference between RMMA and NSMMA ($P = 0.3926$). These findings indicate that both RMMA and NSMMA are preceded by increased respiratory effort, suggesting that respiratory dynamics may represent a common physiological background underlying sleep-related masticatory muscle activity.

Key words: sleep bruxism, RMMA, respiratory effort

Short Talk-2 (14:25–14:40)

Effects of hypoxia-induced respiratory effort on jaw muscle activity in urethane-anesthetized rats

Cao Jiyuan, Yiwen Zhu, Ayano Katagiri, Takafumi Kato

Department of Oral Physiology, Graduate School of Dentistry , The University of Osaka
Suita, Osaka, Japan

Objective: In obstructive sleep apnea, increased respiratory effort is often accompanied by jaw muscle activation with arousals. This study examined whether enhanced respiratory effort under hypoxia is associated with changes in jaw muscle activity.
Methods: Adult Sprague–Dawley rats were used. Under urethane anesthesia, electrodes were implanted to record electroencephalography (EEG), masseter, digastric and diaphragmatic electromyographic (EMG) activity. The head was fixed in a stereotaxic frame, and transcutaneous pulse oximetry was applied to a hindlimb to measure oxygen saturation and pulse rate. Animals inhaled gas through a fitted mask, and inspired oxygen concentration was reduced from 21% to 10% by mixing nitrogen. During normoxia and hypoxia, diaphragmatic EMG bursts were analyzed for peak amplitude and inter-burst interval. Masseter and digastric EMG signals were averaged and aligned to diaphragmatic bursts to evaluate phase relationships. Mean EMG activity of all muscles was quantified during hypoxia.
Results: Lower inspired oxygen shortened diaphragmatic inter-burst intervals and increased mean diaphragmatic activity. Oxygen saturation decreased and pulse rate increased with hypoxia. EEG power spectral showed no differences between normoxic and hypoxic conditions. Diaphragmatic EMG activities were synchronized with digastric EMG activities but not with masseter EMG activities. Mean digastric and masseter EMG activity were unchanged by hypoxia.
Conclusion: Under urethane anesthesia, hypoxia-induced increases in respiratory effort characterized by diaphragmatic drive but do not alter EEG or jaw muscle activity. These results suggest a dissociation between respiratory load–related drive and jaw motor activation under anesthetized conditions.

Key words: respiratory effort, jaw muscle activity, arousal

Short Talk-3 (14:40–14:55)

Input and output patterns of non-nociceptive information in the caudal subnucleus of the spinal trigeminal nucleus

Risa Kimura, Aya Takenaka, Takahiro Furuta

Department of Systematic Anatomy and Neurobiology, Graduate School of Dentistry, The University of Osaka, Suita, Osaka, Japan

Sensory information from the orofacial region is entered to the principal trigeminal nucleus (PrV) and the spinal trigeminal nucleus, which are relay nuclei of the trigeminal system. The spinal trigeminal nucleus is divided into three parts: oral subnucleus (Sp5O), interpolar subnucleus (Sp5I), and caudal subnucleus (Sp5C).
Noxious information of the orofacial region is transmitted from primary neurons to the layer 1 and layer 2 of Sp5C. And then, secondary neurons relay the information to the parabrachial nucleus (PBN) and the ventral posteromedial thalamic nucleus (VPM). On the other hand, non-nociceptive information from the orofacial region is entered mainly to PrV firstly, and then relayed to VPM. Additionally, some reports suggest that non-nociceptive information is also entered to Sp5C. However, it remains unclear to which layer of Sp5C the information will be entered and after that which part of the brain it will be transmitted. To clarify these questions anatomically, I conducted experiments using electrophysiological techniques and anterograde tracer BDA. From the experiments, I found non-nociceptive information is entered to layer 3 of Sp5C and is sent by layer 3 neurons to PrV.

Key words: the caudal subnucleus of the spinal trigeminal nucleus, non-nociceptive information, neuroanatomy

Short Talk-4 (14:55–15:10)

Sensory neuron dysfunction in dorsal root ganglia during disease onset in the SOD1G93A mouse model for ALS

Akira Nishiura[1], Soju Seki[1], Takuhiro Kobayashi[1], Sou Kawata[1,2], Yoshihiro Kitaoka[3], Susumu Tanaka[1]

[1]Department of Oral and Maxillofacial Surgery, Graduate School of Dentistry, The University of Osaka. Suita, Osaka, Japan; [2]Department of Dentistry and Oral Surgery, Yao Municipal Hospital, Yao, Osaka, Japan; [3]Laboratory of Neuropharmacology, Section of Biosystems and Function, School of Dentistry, University of California, Los Angeles, Los Angeles, CA, USA.

Amyotrophic lateral sclerosis (ALS) has traditionally been regarded as a motor neuron disease; however, sensory abnormalities are increasingly recognized in patients. The pathological mechanisms underlying primary sensory neuron dysfunction remain unclear. In this study, we investigated dorsal root ganglia (DRG) in SOD1G93A mice at disease onset using integrated RNA sequencing, immunohistochemical, and electrophysiological analyses.

RNA-seq revealed significant upregulation of genes associated with oxidative stress and immune-related pathways, with the phagosome pathway showing the strongest enrichment. Comparative transcriptomic analysis demonstrated both shared and DRG-specific molecular alterations relative to spinal motor neurons. Immunohistochemical analyses showed significant soma atrophy in both A-fiber–DRG neurons. Notably, colocalization ratios of Nav1.7 and Nav1.8 were significantly increased in A-fiber neurons.

Electrophysiological recordings demonstrated enhanced membrane excitability in A-fiber neurons of SOD1G93A mice, characterized by depolarized resting membrane potential, increased spike height, elevated firing frequency, and a trend toward reduced rheobase.

Collectively, these findings indicate that ALS pathology extends beyond motor neurons and affects primary sensory neurons at molecular, structural, and functional levels from disease onset. Nav channel–associated hyperexcitability, particularly in A-fiber DRG neurons, may contribute to sensory dysfunction in ALS and represent a potential therapeutic target.

Key words: ALS、DRG、Sensory neuron

Short Talk-5 (15:10–15:25)

Clonidine attenuated masticatory muscle activity elevation across sleep-wake cycle induced by acute foot-shock stress

Yiwen Zhu, Ayano Katagiri, Takafumi Kato

Department of Oral Physiology, Graduate School of Dentistry, The University of Osaka, Suita, Osaka, Japan

In humans, sleep bruxism (SB) is characterized by elevated masticatory muscle activity (MMA) during sleep, and stress is recognized as a major risk factor. In our previous research, the elevation of MMA across sleep-wake cycle was successfully induced by inescapable foot-shock (IFS) stress. Because central noradrenaline system involved in the regulation of sleep-wake cycle and stress response, it may be important in the genesis of stress-related MMA elevation. In this study, we investigated the role of central noradrenaline system by administrating clonidine, an α2-adrenergic agonist, .

Electrodes for EEG, ECG, and EMGs of the neck and masseter muscles were surgically implanted to monitor sleep, MMA, and heart rate (HR) in three groups: Sham; IFS + saline; and IFS + clonidine. For sham condition, rats were placed in the IFS box without stimulation. Clonidine (0.1 mg/kg, i.p.), or saline was administered prior to acute IFS stress, followed by electrophysiological recording in the dark phase. Acute IFS stress induced elevated MMA across the sleep-wake cycle, accompanied by sleep disruption and sympathetic activation. Clonidine markedly attenuated MMA elevation across the sleep-wake cycle, along with suppression of cortical and sympathetic activity.

In other independent 3 groups of rats, open field test (OFT) and immunohistochemistry of the locus coeruleus and limbic system were conducted to confirm the stress condition and neural activation. Clonidine significantly altered stress-related behavior in OFT, reduced immunoreactivity in stress-related brain regions.

In conclusion, the elevation of MMA during sleep-wake cycle was suppressed by clonidine along with suppression of cortical and sympathetic activity, suggesting the noradrenergic system was involved in the genesis of stress-related MMA during sleep-wake cycle.

Key words: stress, masticatory muscle activity, noradrenaline

Wasabi ingredient, hexaraphane, promotes dentin formation via SLC4As-NHE-NCX-PMCA signaling axis

Maki Kimura[1], Yoshiaki Furusawa[2], Isao Okunishi[3], Takehito Ouchi[1], Ryuya Kurashima[1], Tomoe Katou-Yamada[3], Hidetaka Kuroda[4], Nanae Iwasawa[1,5], Makoto Sugita[6], Masahiro Furusawa[2], Yoshiyuki Shibukawa[1]

[1]Department of Physiology, Tokyo Dental College, Tokyo, Japan; [2]Department of Endodontics, Tokyo Dental College, Tokyo, Japan; [3]Kinjirushi Co., Ltd., Nagoya, Aichi, Japan; [4] Department of Dental Anesthesiology, Kanagawa Dental University, Yokosuka, Kanagawa, Japan; [5]Tokyo Dental Junior College, Tokyo, Japan; [6]Department of Physiology and Oral Physiology, Hiroshima University, Hiroshima, Japan.

Various stimuli to the dentin surface are detected by odontoblasts, resulting in reactionary dentin formation. High-pH dental materials, such as mineral trioxide aggregate (MTA) and calcium hydroxide, induce dentin formation. We have previously reported that high-pH stimulation activates transient receptor potential ankyrin subfamily member 1 (TRPA1) channels in odontoblasts. The TRPA1 channels are also activated by isothiocyanate compounds. Among the compounds, allyl isothiocyanate, 6-methylsulfinylhexyl isothiocyanate (6-MSITC; hexaraphane), and 6-methylthiohexyl isothiocyanate are contained in Wasabi (Eutrema japonicum). We thus examined the effect of the isothiocyanate compounds on dentin formation and its regulatory mechanism in human odontoblasts. In mineralization assay, 6-MSITC (40 or 400 μM) promoted mineralization by human odontoblasts. 6-methylsulfonylhexyl isothiocyanate, a similar compound of 6-MSITC, also promoted mineralization. Addition of 6-MSITC (40 or 400 μM) increased pH of culture medium. 6-MSITC-induced mineralization was suppressed by a carbonic anhydrase (CA), plasma membrane Ca2+-ATPase (PMCA), or the solute carrier family 4 (SLC4A)/Cl- channel inhibitor, whereas Na+-Ca2+ exchanger (NCX) inhibitor had no effect. In immunostaining analysis, SLC4A1, SLC4A2, SLC4A3, SLC4A4, SLC4A8, SLC4A9, CA I, and CA II were expressed in human odontoblasts. In gramicidin-perforated patch-clamp recording, we obtained whole-cell current-voltage relationships by application of voltage ramp protocol (ranging from −100 to +100 mV) at holding potential of -70 mV. 6-MSITC increased outward currents. The 6-MSITC-induced outward currents were inhibited by application of a CA, Na+-H+ exchanger (NHE), or NCX inhibitor, but not by SLC4A/Cl- channel inhibitor. In the mandibular first molars of Wistar rats, application of 6-MSITC increased reactionary dentin formation, collagen production, and calcium deposition. In real-time RT-PCR experiments, 6-MSITC increased expression levels of the mRNAs encoding SLC4A1, SLC4A2, and CA II. These results suggest that 6-MSITC enhances reactionary dentin formation by SLC4As-NHE-NCX-PMCA signal coupling in odontoblasts.

Key words: odontoblasts, dentin formation, 6-methylsulfinylhexyl isothiocyanate

Mini Review 2-2 (16:10–16:40)

Thalamo-insular pathway regulates tic generation via motor-limbic crosstalk

Yoshihisa Tachibana

Department of Physiology and Cell Biology, Kobe University Graduate School of Medicine, Kobe, Japan.

Tourette syndrome (TS) is a neurodevelopmental disorder characterized by motor and vocal tics, often accompanied by cognitive and emotional comorbidities such as obsessive-compulsive disorder (OCD) and attention-deficit hyperactivity disorder (ADHD). Although behavioral, pharmacological, and surgical interventions are currently applied, their therapeutic efficacy remains limited. We have reported that motor and vocal tics in TS patients can be ameliorated by a removable oral splint (Murakami et al., Mov Disord, 2019). This observation raised the possibility that proprioceptive signals from masticatory muscle spindles may influence neuronal circuits involved in tic generation.

Anatomical studies revealed that afferent signals from masticatory muscle spindles ascend to the insular cortex (IC), a region previously implicated in TS pathophysiology by human imaging studies showing abnormal activity in both the striatum and IC (e.g., Bohlhalter et al., Brain, 2006). Because abnormal information processing within cortico-basal ganglia-thalamo-cortical loops has been proposed to underlie tic disorders, we investigated the neuronal circuits responsible for tic generation using a mouse model of tic-like movements induced by unilateral striatal injection of bicuculline, a $GABA_A$ receptor antagonist.

This model induced robust c-Fos activation in both motor and limbic structures, including IC. Fiber photometry recordings revealed tic-associated activity in IC as well as the primary motor cortex (M1). Viral tracing demonstrated that basal ganglia outputs from the substantia nigra pars reticulata reach IC via the intralaminar thalamic nuclei (ITN). Furthermore, chemogenetic inhibition of IC or the thalamo-insular pathway suppressed tic-like behaviors and reduced tic-associated cortical activity.

Together, these findings identify the IC as a critical node involved in tic generation and highlight the thalamo-insular pathway as a circuit linking motor and limbic networks. These results provide a potential neuronal substrate through which oral-splint-induced proprioceptive signals may modulate tic symptoms and suggest thalamo-insular signaling as a candidate therapeutic target for TS.

Key words: Tourette syndrome, basal ganglia, thalamo-insular pathway

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