Symposium | Nurodevelopmental disorder：[Symposium 106] Oral Splint Therapy for Tourette Syndrome: Bridging Dentistry and Psychiatry

Sun. Sep 28, 2025 2:50 PM - 4:20 PM JST | Sun. Sep 28, 2025 5:50 AM - 7:20 AM UTC Session Room 7 (Conference Room C)

[Symposium 106] Oral Splint Therapy for Tourette Syndrome: Bridging Dentistry and Psychiatry

Moderator: Yuki Oda (Hiroshima Oral Health Center)

[SY-106-03] Therapeutic Effects and Underlying Mechanisms of Oral Splint Use for Ameliorating Tic Symptoms in Tourette Syndrome

*Yoshihisa Tachibana[1] (1. Department of Physiology and Cell Biology, Kobe University Graduate School of Medicine (Japan))

Keywords：basal ganglia、insula、striatum、intralaminar thalamic nuclei、tic

Tourette syndrome (TS) is a neurodevelopmental disorder characterized by motor and vocal tics. TS is often accompanied by cognitive and emotional dysfunctions, including obsessive-compulsive disorder and attention-deficit/hyperactivity disorder. We previously reported that a removable oral splint ameliorates both motor and vocal tics in TS patients. However, the mechanism by which the oral splint exerts these effects remains unclear. To address this issue, we first examined the ascending pathways of the masticatory muscle spindles, which are activated by the insertion of the oral splint. We identified that proprioceptive information originating from these spindles is ultimately transmitted to the insular cortex via the supratrigeminal nucleus and the caudo-ventromedial region of the ventral posteromedial thalamic nucleus. We then investigated why modulation of orofacial proprioceptive input to the insula alleviates tic symptoms. Previous imaging studies have reported abnormal activity in the striatum and insula in TS patients. Given that dysfunction of the cortico-basal ganglia-thalamocortical circuits has been implicated in TS, we hypothesized that this network plays a critical role in symptom generation. To test this, we developed a drug-induced mouse model of tics by unilaterally injecting bicuculline, a GABA receptor antagonist, into the striatal motor region. In these mice, c-Fos immunoreactivity revealed neuronal activation in limbic structures (insular cortex, cingulate cortex, and amygdala) as well as in motor regions (M1, globus pallidus, and subthalamic nucleus) but also. Using anterograde and retrograde viral tracers, we mapped the anatomical route from basal ganglia output structures to limbic cortices and identified the intralaminar thalamic nuclei as key hubs linking these regions. Finally, we demonstrated that chemogenetic inhibition of the insular cortex or its thalamocortical input significantly reduced tic-like behaviors in the mouse model. These findings suggest that aberrant neuronal processing within both motor and limbic domains of the cortico-basal ganglia-thalamocortical circuits contributes to tic generation in TS.