一般口演

[3O04m2]神経変性疾患 1

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2022年7月2日(土) 10:00 〜 11:00 第4会場 (沖縄コンベンションセンター 会議場B5~7)

[3O04m2-03]パーキンソン病に着目したレム睡眠行動障害のメカニズムの解明

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キーワード：REM sleep behavior disorder, Parkinson's disease, α-synuclein, Synucleinopathy

During rapid eye movement (REM) sleep, the cerebral cortex becomes activated and produces vivid dreams. Yet, it normally does not lead to motor output owing to expression of loss of muscle tone (muscle atonia). However, patients with REM sleep behavior disorder (RBD) exhibit impaired muscle atonia during REM sleep and frequently act out of their dreams. For example, the patients talk loudly or exhibit violent movements such as hitting and kicking, which may injure their bed partner. Moreover, patients often wake up following such movements, which can lead to reduced sleep quality and excessive sleepiness or fatigue during the daytime.

A majority of RBD patients co-suffer from synucleinopathies including Parkinson’ s disease and dementia with Lewy bodies or eventually develop these diseases within 10-14 years. Thus, RBD is considered as a prodromal of synucleinopathies. In synucleinopathies, α-synuclein accumulates and causes nerve cell death, which leads to motor symptoms such as tremor, bradykinesia, and rigidity as well as non-motor symptoms including sleep disturbances, cognitive impairments, hallucinations and depression.

Here, we aimed to understand the mechanisms underlying the link between RBD and Parkinson’ s disease, and establish an RBD mouse model for future development of effective treatment for RBD. A rare G51D α-synuclein mutation is observed in familial Parkinson’ s disease, and it leads to the production of toxic α-synuclein fibrils and rapid progression of Parkinson’ s disease.

We produced and injected G51D α-synuclein fibrils into the brainstem pontine tegmental area in mice, and examined the effects on muscle atonia during REM sleep, behavior and motor symptoms across several months. We expect that this study will provide important insight to how RBD develops at the early stages of Parkinson’ s disease.