論 文 審 査 の 結 果 の 要 旨

報　告　番　号	甲　第　1301　号	氏　　名	Pang Bo
論 文 審 査 担 当 者	主　　査　　関島　良樹 副　　査　　新藤　隆行、古庄　知己　　・		

（論文審査の結果の要旨）

Salt-inducible kinase 1 (SIK1) is an AMP-activated protein kinase (AMPK) with inducible expression in the adrenal cortex in response to the salt intake or adrenocorticotropic hormone (ACTH). Six mutations in the salt-inducible kinase 1 (SIK1)-coding gene have been identified in patients with early infantile epilepticencephalopathy (EIEE-30) accompanied by autistic symptoms. To study the effects of these mutations on epilepsy, NMDA or PTZ was injected into SIK1-MT males to induce epileptic seizures. I studied these epilepsy model by focusing on the susceptibility to epileptic seizures and adrenocorticotropic hormone therapy for infantile spasms and found following:

(1) Seizure susceptibility induced by both NMDA and PTZ was enhanced in SIK1-MT mice.

(2) Distinct brain regions were activated in NMDA-induced seizures.

(3) No microglial activation was detected in NMDA-induced seizures.

(4) SIK1-MT canceled the effect of ACTH treatment on NMDA-induced seizures.

(5) Distinctive neurons within the cortical layer formation were activated in NMDA- or PTZ-induced seizures.

The committee chair and vice chairs evaluated that the thesis deserved a doctoral dissertation.