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発表題目（※学会発表の場合のみ記載）	内側手綱核に強く発現する Nwd2 遺伝子の同定と機能解析

発表の概要と成果（抄録を公開している URL がある場合、「概要・成果」を記載した上で、URL を末尾に記してください。また、抄録 PDF は別途ご提出ください。なお、抄録 PDF は Web 上には公開されません。）

上記学会においてポスター形式にて発表を行った。

以下 Abstract を示す。

The habenula is a highly conserved brain region from fish to mammals as a control center of monoamine neurons regulating the firing of serotonin and dopamine neuron. In recent years, it has been shown that the disfunction of habenula is associated with the etiology of mood disorders. However, the molecular mechanisms that control the formation and function of the habenula has not been fully elucidated. We previously showed that the NACHT and WD repeat domain-containing protein 1 (Nwd1), a member of STAND family, functions in de novo purine biosynthesis during mammalian brain development. Here we identified the mouse Nwd2 gene, which is a paralog of Nwd1. Nwd2 expression was primarily confined to neurons in the medial habenular nucleus. We identified Kv channel-interacting protein 1a, which is expressed in medial habenular nucleus neurons, as a factor interacting with Nwd2 using Yeast Two-Hybrid screening. We generated the Nwd2 knockout mouse using the CRISPR-Cas9 genome editing technique of oviductal nucleic acids delivery (iGONAD). Translation stop codon was inserted in the critical exon encoding the NACHT-domain, resulting in the Nwd2 null allele. Interbreeding of the heterozygous mutant mice yielded homozygous mutant pups with the expected Mendelian ratio, indicating that Nwd2 is not essential for embryonic viability. The absence of Nwd2 expression in brains in homozygous animals was confirmed by immunostaining analysis. Nwd2 deficient adult mice showed an abnormal increase in spontaneous activity in an open field test. Nwd2-deficient mice could serve as a new model for hyperactive behavioral disorders such as attention deficit hyperactivity disorder (ADHD).

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