## Neurobiology and anatomy

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We are interested in how brains are developed and why neurological diseases occur. In order to understand neural development and neurological diseases at molecular, cellular, and neuronal circuit levels, we use interdisciplinary approaches. In most of our projects, we use mutant mice and/or genetically modified mice. Since neuroscience is multidisciplinary field, we are collaborating with many researchers, such as molecular biologists, biochemists, physiologists, geneticists, and pathologists.



## Research interests

- Neural development: molecular mechanism of differentiation from neural stem cells to specific neurons and glial cells. We are especially interested in oligodendrocyte development.
- 2. Molecular and cellular mechanisms of neurological diseases (dystonia, demyelinating disease, motoneuron disease, and epilepsy).
- 3. Development of a new treatment for neurological diseases.

## Materials and methods for collaborations

- 1. Histological methods: immunohistochemistry, *in situ* hybridization, etc.
- 2. Cell culture experiments including primary culture of neural stem cells.
- 3. Mutant mice and genetically modified mice; we have several model mice for neurological disease.

## Links to additional info

- 1. Takebayashi H & Ikenaka K. Oligodendrocyte generation during mouse development. Glia. 63(8):1350-1356, 2015.
  - https://www.ncbi.nlm.nih.gov/pubmed/26013243
- 2. Horie M, et al. Characterization of novel *dystonia musculorum* mutant mice: Implications for central nervous system abnormality. Neurobiol Dis. 96:271-283, 2016.
  - https://www.ncbi.nlm.nih.gov/pubmed/27693510
- Hayakawa-Yano Y, et al. An RNA-binding protein, Qki5, regulates embryonic neural stem cells through pre-mRNA processing in cell adhesion signaling. Genes Dev. 31(18):1910-1925, 2017. <a href="https://www.ncbi.nlm.nih.gov/pubmed/29021239">https://www.ncbi.nlm.nih.gov/pubmed/29021239</a>
- 4. Lab HP (Japanese). <a href="https://www.med.niigata-u.ac.jp/an2/index.html">https://www.med.niigata-u.ac.jp/an2/index.html</a>