



CDB SEMINAR

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Molekulare Biologie neuronaler Signale

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16:30~17:30 C1F CDB Auditorium

Molecular control of cell fate specification in the cerebral cortex

Summary

During the development of the mammalian neocortex, neuronal progenitors located in the ventricular and subventricular zones of the dorsal telencephalon give rise to multiple projection neurons that are arranged in six cortical layers in the mature brain. Neurons within each layer are generated at similar times and share similar morphology and patterns of connectivity. The molecular determinants of the fate of these cells are still elusive.

In recent years our research was focused on identification and characterization of genes that control cell fate specification in the cerebral cortex. One of the genes we identified, *Satb2* is crucial for postmitotic specification of callosally projecting upper layer cortical neurons. Another transcription factor we identified several, *Sip1* was shown to be the cause of Mowat-Wilson syndrome in humans. We showed that in the hippocampus *Sip1* controls non-canonical Wnt signaling by suppressing *Sfrp1* gene expression. Inactivation of *Sip1* in the hippocampus induces *Sfrp1* activation, that in turns leads to inactivation of Wnt/JNK signaling, elevated cell death and subsequent degeneration of hippocampal formation. In the neocortex *Sip1* inactivation induces premature and excessive production of upper layer neurons at the expense of deep layer neurons. Furthermore, it causes precocious generation of glial cells at late corticogenesis. Molecular basis of *Sip1* and *Satb2* action will be discussed

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