CDB SEMINAR

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15:00~16:00 C1F CDB Auditorium

Establishing Cell Fate in the Cerebral Cortex

Summary

The mammalian cerebral cortex is a highly ordered structure that consists of neurons with distinct molecular identities, morphologies and axonal projection patterns. Developmentally, these neurons arise from common progenitors within the cortical ventricular zone that sequentially produce the neurons that populate the deep through superficial cortical laminae. Previous work addressing the contribution of intrinsic versus extrinsic cues in specifying neurons within the cortex demonstrated that there is a progressive restriction of competence in cortical progenitors, in which earlier progenitors can adopt a later fate but not the converse. However the molecular mechanisms involved in this process has remained elusive. The focus of this talk is to understand the identity of the progressive restriction during earliest corticogenesis, when cortical progenitors switch from generating the earliest-class of neurons, the Cajal-Retzius (CR) cells to deep-layer projection neurons that arise next during development. In this regard, Foxg1, a winged helix transcriptional repressor expressed in the telencephalon, plays a critical role in determining the fate switch from production of CR cells to deep-layer neurons. Foxg1 functions during early phases of cortical neurogenesis to prevent the generation of CR neurons after their normal birthdate. Furthermore, recent reports have suggested that CR cells arise from multiple regions within the telencephalon, in particular the dorsomedial signaling center. This is notable as Foxg1 plays a pleiotropic role as a transcriptional repressor, and one of its functions is to antagonize the dorsomedial signaling center. We have therefore investigated whether Foxg1 suppresses CR cell fate primarily by antagonizing dorsomedial signaling or by inhibiting the generation of CR cells directly. To distinguish between these possibilities we both genetically ablated the BMP signaling in the dorsomedial cortex, as well as the entire dorsomedial cortex itself. Surprisingly in both contexts the normal cohort of CR cells is produced. These results suggest that the the dorsomedial signaling center is dispensable for the generation of CR cells, while Foxg1 is required for suppressing the earliest cell fate in all contexts.

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