



# CDB SEMINAR

Speaker: **Takahiro Ohyama**

< Molecular Development, House Ear Institute >

Title: **“Ear or skin: Wnt signaling mediates a placode–epidermis fate decision”**

Date: Wednesday, December 14

Time: 16:00-17:00

Place: 1F Auditorium of Building C, CDB

Takahiro Ohyama, a post-doc in the Lab of Andy Groves, has been using a conditional *Cre* line to knockout or over-express molecules in the mouse inner ear. His work has been answering fundamental questions on the induction of the mouse inner ear and enables us to reconcile previously conflicting data from other species.

## Summary:

The otic placode, the anlagen of the inner ear, develops from an ectodermal field characterized by expression of the transcription factor *Pax2*. Previous fate mapping studies suggest that these *Pax2*<sup>+</sup> cells will give rise to both otic placode tissue and epidermis (Streit, *Dev. Biol.* 2002; Ohyama and Groves, *Genesis* 2004), but the signals that divide the *Pax2*<sup>+</sup> field into placodal and epidermal territories are unknown. We analyzed a reporter strain that carries six copies of TCF/Lef binding sites (Mohamed et. al., *Dev. Dyn.* 2004), which revealed that the canonical Wnt signaling pathway is normally activated in a subset of *Pax2*<sup>+</sup> cells. We also performed conditional inactivation of beta-catenin in these cells and found an expansion of epidermal markers at the expense of the otic placode. Conversely, conditional activation of beta-catenin in *Pax2*<sup>+</sup> cells causes an expansion of the otic placode at the expense of epidermis, and the resulting otic tissue expresses exclusively dorsal otocyst markers. Together these results suggest that Wnt signaling acts instructively to direct *Pax2*<sup>+</sup> cells to an otic placodal, rather than an epidermal fate, and promotes dorsal cell identities in the otocyst. Based on our present study, we propose a new model of inner ear induction that reconciles conflicting data from recent studies.

Host **Raj Ladher** <Sensory Development, CDB>

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