

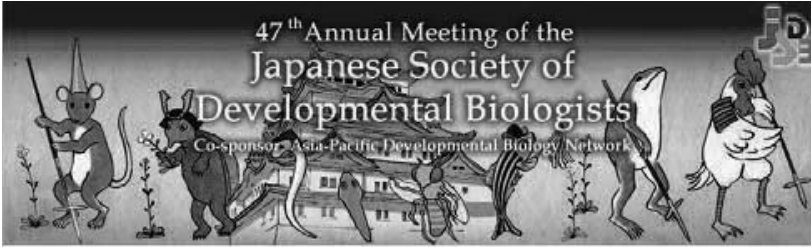
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47th annual meeting organizing committee


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47th Annual Meeting of the
Japanese Society of
Developmental Biologists
Co-sponsor: Asia-Pacific Developmental Biology Network

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Schedule and Program		President Masahiko Hibi (Nagoya University)
Instruction for Presentations		Language May 27 (in Japanese and English) May 28-30 (in English)
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Title click !

Plenary Lectures

May 20 (Fri) 9:00-11:30 Room A
Plenary Lectures
Chairpersons: Tesuya Tabata (Univ. of Tokyo), Shiro Nakagawa (RIKEN)

PL-01: **Hedgehog Signaling in Development and Disease**
09:00-10:15 Matthew Scott (Stanford University, Center for Neurobiology)

PL-02: **The development of the hindbrain and zebrafish retinas**
10:15-11:30 William Harris (University of Cambridge)

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Symposia

DATE: May 19 (Thu) 9:00~11:30 Room A
Symposium1: Neural Development: from circuits to behavior

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[PL-01]

Hedgehog Signaling in Development and Disease

*Matthew Scott
(Stanford University School of Medicine)

The development of numerous tissues and organs depends on Hedgehog (Hh) protein signals that influence gene expression in target cells. Defective Hh signaling leads to birth defects and cancer. We are investigating Hh signal transduction and gene expression mechanisms in the context of cultured fibroblasts and cerebellum development. The Hh protein signal has many unique features. First, Hh is secreted as a pro-peptide and is processed into mature Hh. Second, Hh binds to its receptor, Patched 1 (Ptch1), on the cell surface. Third, Hh binding to Ptch1 causes the recruitment of Smoothened (Smo) to the cell surface. Fourth, Smo is a transmembrane protein that is normally excluded from the cell surface. In the presence of Hh, Smo is able to activate Gli transcription factors, which in turn control target gene expression. Using tagged proteins, and mutants that affect Hh signaling, we are exploring the mechanisms of protein trafficking and target gene activation. We are characterizing direct Hh target genes in responsive cerebellum granule neuron precursors and in the medulloblastoma tumors that arise from the precursors when Ptch function is reduced.

Signaling in development

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