Developmental Biology 319 (2008) 489

Contents lists available at ScienceDirect

Developmental Biology

journal homepage: www.elsevier.com/developmentalbiology

Abstracts

Plenary session III

Program/Abstract # 71

Regulatory logic of neuronal diversity: Neuronal selector genes and selector motifs

Oliver Hobert

Columbia University Medical Center, New York, NY, USA HHMI, USA

The Selector Gene concept initially introduced in 1975 by Garcia-Bellido defines selector genes as genes that control the formation and identity of a specific region in a developing organism. The absence of the selector gene leads to loss of the region ("region-specific selector gene") or organ ("organ-specific selector gene"). I apply this concept here to the acquisition of terminal neuronal identity. My lab has identified 3 separate selector genes (1 is a complex of 2 selector genes) that each act to define the identity of one individual neuron class and which we call "terminal neuronal selector genes". Absence of the respective selector gene causes a complete loss of the identity of that neuron type. We have shown that all 3 selector genes (complexes) each act through a very simple *cis*-regulatory logic, by each binding to a simple, small *cis*-regulatory motif that we propose to term "selector motif". This surprisingly simply regulatory logic may be an underlying theme in determining neuronal identity. It has the advantage to quickly evolve, thereby providing a conceptual framework for how cellular complexity does evolutionarily arise in a nervous system.

doi:10.1016/j.ydbio.2008.05.080

Program/Abstract # 72

The Mütter Museum of the College of Physicians of Philadelphia: An introduction to its history and resources for the teaching of human developmental biology

Gerald B. Grunwald

Department of Pathology, Anatomy and Cell Biology, Thomas Jefferson University, Philadelphia, PA, USA

The College of Physicians of Philadelphia was founded in 1787 and is the oldest professional medical organization in the United States. The College is today an important resource for the professional medical community and for public education in the history, art and science of medicine. Today, housed in a Georgian Beaux Arts style building dating to 1909, the College is also home to the Mütter Museum, an internationally renowned medical museum whose exhibits include many unique anatomical and pathological specimens. The museum began with the donation to the College in 1858 of the anatomical teaching collection of Thomas Dent Mütter, M.D. (1811–1859), formerly Professor and Chair of Surgery at Philadelphia's Jefferson Medical College. The museum was then important in educating physicians about normal and abnormal human anatomy, and the collection today continues to grow, including over 20,000 medically-related objects including dry and fluid-preserved specimens, medical instruments and apparatus, anatomical models, and memorabilia of scientists and physicians. Today the collection includes many specimens reflecting normal and abnormal human development, including conjoined and parasitic twins, and examples of skeletal, limb, neural tube and craniofacial defects. The museum, first opened in 1863, today draws thousands of visitors each year. This includes an annual visit of students from the "Mechanisms of Birth Defects" course in our Cell and Developmental Biology graduate program, for which the museum collection provides a valuable educational resource.

doi:10.1016/j.ydbio.2008.05.081

Program/Abstract # 73

Fibrodysplasia ossificans progressiva (FOP) — How does one tissue become another?

Eileen M. Shore

Department of Orthopaedics, University of Pennsylvania, Philadelphia, PA, USA

Department of Genetics, University of Pennsylvania, Philadelphia, PA, USA

Center for Research in FOP and Related Disorders, University of Pennsylvania, Philadelphia, PA, USA

Harry E., born in Philadelphia in 1933 with bent great toes, had a normal and active early childhood. However, by the age of ten, Harry had painful swellings that developed into hard bumps. Harry was forming bone within his muscles — first in his upper back and neck, then through his arms and legs. Harry was experiencing the typical progression of a rare genetic disease called fibrodysplasia ossificans progressiva (FOP). When Harry died at 40 years of age, an extensive network of extra-skeletal bone had caused his near total immobilization. Two characteristics define classic FOP: congenital malformation of the great toes and progressive formation of extra-skeletal (or heterotopic) bone in skeletal muscle and other soft connective tissues. The heterotopic bone forms independently of the skeleton and is normal by physiological criteria. It is the time and place of bone formation that is not normal. In FOP, the cellular mechanisms that regulate cell fate decisions to the chondro/osseous lineages are altered by a genetic mutation. We recently discovered that FOP is caused by heterozygous mutation of the ACVR1/Alk2 gene, which encodes a bone morphogenetic protein (BMP) type I receptor, and that this mutation causes enhanced BMP signaling, revealing ACVR1 as a critical regulator for normal skeletal development and for cell differentiation decisions in adult tissues. Further, this signaling pathway is now a therapeutic target not only for FOP, but also is an important new focus for developing treatments for other more common conditions of too much and too little bone formation.

doi:10.1016/j.ydbio.2008.05.082