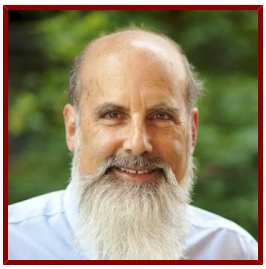
Stanford



Matthew P. Scott

Professor of Developmental Biology, Emeritus

Department of Developmental Biology

Curriculum Vitae available Online

Bio

BIO

Dr. Scott is Emeritus President of the Carnegie Institution for Science (carnegiescience.edu), and is emeritus at Stanford. The Scott lab's research was aimed at learning fundamental molecular mechanisms of development, including gene regulation and cell-cell signaling. He also studied the formation and function of brain circuitry. He worked with cultured cells, Drosophila, and mice to investigate how normal embryos grow and what goes wrong in birth defects, cancer, and neurodegenerative disease. A major goal was to identify and explore new genes and proteins that control development. The lab group investigated the development of the nervous system, especially the cerebellum, using cell and tissue culture, genomics, and transgenic animals. Cells were grown on controlled and patterned surfaces to govern neurite outgrowth. To investigate signal transduction between and within cells, the group studied regulators that control cell morphology and intracellular trafficking. Time-lapse video of engineered proteins was combined with genetic modifications that alter cell-cell signaling and the assembly and transport of organelles. Imaging and image processing were important tools. Collaborative engineering projects included the invention of an embryo sorting instrument and the development of new injection methods applicable to high-throughput screens of gene functions.

Dr. Scott did undergraduate and graduate work at M.I.T., with Prof. Mary Lou Pardue as his Ph.D. thesis advisor. He moved to Indiana University for his postdoctoral work as a Helen Hay Whitney fellow with Profs. Thomas Kaufman and Barry Polisky. He then set up his own lab at the University of Colorado, Boulder. Scott came to Stanford in 1990 to join the newly formed Department of Developmental Biology, and the Department of Genetics. His research focus is on genes that control development, and how damage to them leads to birth defects, cancer, and neurodegeneration. He discovered the "homeobox", an evolutionarily conserved component of many genes that control development. His lab group discovered the genetic basis of the most common human cancer, basal cell carcinoma, and of the most common childhood malignant brain tumor, medulloblastoma. He served as Associate Chair and Chair of the Department of Developmental Biology for a total of six years. He chaired the Bio-X program from 2001-2007. He is presently co-chair of the Center for Children's Brain Tumors. He has been recognized by election to the American Academy of Arts and Sciences, the National Academy of Sciences, and the National Institute of Medicine, and served as President of the Society for Developmental Biology. His awards include the Passano Award (1990), the Conklin Medal of the Society for Developmental Biology (2004), and the Pasarow Award in Cancer Research (2013). He is now at the Carnegie Institution for Science, based at Stanford and in Washington D.C., Baltimore, and Pasadena.

Dr. Scott is married to Margaret Fuller, who is Professor of Developmental Biology and Genetics at Stanford. Together with their children, they enjoy exploring wild places around the world by foot, on bicycles, and underwater. Dr. Scott spends a lot of time looking through cameras: matthewscottphotography.com.

ACADEMIC APPOINTMENTS

- Emeritus Faculty, Acad Council, Department of Developmental Biology
- Member, Bio-X
- Member, Maternal & Child Health Research Institute (MCHRI)
- Member, Wu Tsai Neurosciences Institute

ADMINISTRATIVE APPOINTMENTS

- Chair, Developmental Biology, (1996-1998)
- Associate Chair, Developmental Biology, (1999-2002)
- Co-Chair, Center for Children's Brain Tumors, (2005- present)
- Chair, Bio-X Program, (2001-2007)

HONORS AND AWARDS

- Helen Hay Whitney Postdoctoral Fellow, Helen Hay Whitney Foundation (1980-1983)
- Searle Scholar's Award, Searle Foundation (1985-1989)
- Passano Young Investigator Award, Passano Foundation (1990)
- Investigator, Howard Hughes Medical Institute (1993-)
- Fellow, American Academy of Arts and Sciences (1996)
- President, Society for Developmental Biology (1997-1998)
- Member, National Academy of Sciences (1999-)
- Conklin Medal, Society for Developmental Biology (2004)
- Member, National Institute of Medicine (2007-)
- Einstein Professor, Chinese Academy of Sciences (2008)
- Excellence in Teaching, School of Medicine, Stanford University (2008-2012)
- Pasarow Prize in Cancer Research, Pasarow Foundation (2013)
- President, Carnegie Institution for Science (2014-2017)

BOARDS, ADVISORY COMMITTEES, PROFESSIONAL ORGANIZATIONS

- Member, Institute of Medicine (2007 - present)
- Member, National Academy of Sciences (1999 - present)
- Member, American Academy of Arts and Sciences (1996 - present)

PROFESSIONAL EDUCATION

- Ph.D., M.I.T. , Biology (1980)
- B.S., M.I.T. , Biology (1975)

COMMUNITY AND INTERNATIONAL WORK

- Patan Academy of Health Sciences, Nepal, Kathmandu, Nepal

Research & Scholarship

CURRENT RESEARCH AND SCHOLARLY INTERESTS

Early embryonic development is governed by an exquisite interplay of genes that organizes cells as they proliferate. Signals flow between cells to control their fates; information inherited by the cells influences their responses to the signals. Transcription factors necessary for forming particular parts of the bodysuch as head-to-tail differences, heart, eyes, or nervous systemhave remained dedicated to those tasks through evolution. Similarly, the genes and proteins that code for signals, signal receptors, and information transfer within the cell have been preserved. We study evolutionarily conserved regulators in flies and in mice to learn how the embryo is constructed and how pattern-organizing genetic programs arose, function, and change. Genetic damage to developmental regulators can lead to cancer, birth defects, and neurodegeneration; we study all of these processes in the context of the development of the mammalian cerebellum.

The Hedgehog Signaling System in Development and Cancer

The evolutionarily conserved Hedgehog (Hh) signaling system is used in most animals to control the embryonic development of numerous tissues, such as brain and spinal cord, limbs, skeleton, and skin. We study how the Hh protein signal is received, transduced, and interpreted, using mice and flies. We study how Hh signaling controls cell differentiation and patterning in structures as diverse as the fly wing and the developing mammalian cerebellum. Mutations in human PATCHED (PTCH), which encodes the Hh receptor, cause birth defects and medulloblastoma of the cerebellum, the most common childhood malignant brain tumor. PTCH mutations caused by UV lead to basal cell carcinoma of the skin, the most common human cancer. We are using mutant ptc mouse models to investigate how normal cerebellum cells become tumor cells. Using high-throughput sequencing and chromatin analyses, we have identified batteries of genes that are directly regulated by Hh signals in normal and cancerous cells. We are studying mechanisms of selective target gene control, and how the targets contribute to normal development or cancer. We are studying detailed mechanisms of Hh signal transduction, particularly the roles of primary cilia, remarkable organelles where Hh signals are transduced. We have identified new components of Hh signaling and are investigating their roles. In collaboration with Professor Josh Elias, we are using mass spectrometry to characterize early events in responses to Hh signaling. In collaboration with Professor W.E. Moerner, we are analyzing the movements of single protein molecules during Hh transduction. In collaboration with researchers at BGI and with Professor Serafim Batzoglou we are analyzing genome alterations that occur during the growth of mouse and human cancers, employing single-cell exome sequencing.

Biology of the Niemann-Pick type C syndrome, a neurodegenerative and lysosome storage disorder

Children mutant in either of the two NPC genes undergo neurodegeneration and usually die by the teenage years. Mutant cells accumulate masses of sterols in aberrant organelles due to defective intracellular trafficking. In humans and mice, NPC disease causes the death of the Purkinje neurons of the cerebellum and damage to other tissues. We have engineered mice in which labeled, functional NPC proteins can be provided to specific cell types in otherwise mutant mice, thus allowing analyses of cell type-specific components of the overall pathology, and analyses of the movements and functions of NPC proteins within cells. We have created two different models of the disease in Drosophila in order to apply genetics to identifying and analyzing interacting genes. Our goals are to learn the mechanisms of NPC protein actions, and to learn how NPC proteins preserve functions and survival of specific cell types. We are using Drosophila genetics to identify interacting genes that may affect NPC protein functions.

Teaching

GRADUATE AND FELLOWSHIP PROGRAM AFFILIATIONS

- Bioengineering (PhD Program)

- Biology (School of Humanities and Sciences) (PhD Program)
- Cancer Biology (PhD Program)
- Developmental Biology (PhD Program)
- Genetics (PhD Program)
- Neurosciences (PhD Program)