2002 Grand Prize Winner

Anjen Chenn was born in Taipei, Taiwan, and grew up in Marion, Ohio. Dr. Chenn received his bachelor's degree in biochemical sciences from Harvard University in 1990. He went on to graduate studies in the Medical Scientist Training Program at Stanford University where he joined Dr. Susan McConnell's laboratory and studied mammalian cerebral cortical development. His research on asymmetric divisions in mammalian neurogenesis was published in a first-author paper in *Cell* and was featured on the cover. He received his Ph.D. in neurosciences from Stanford University in 1996 and his M.D. in 1997. From there Dr. Chenn moved to residency training in clinical pathology at the Brigham and Women's Hospital in Boston and became board-certified in clinical pathology in 2000. During his residency training, Dr. Chenn was awarded a Howard Hughes Physician Postdoctoral Fellowship and pursued postdoctoral research in Dr. Christopher A. Walsh's laboratory at the Beth Israel Deaconess Medical Center. His postdoctoral research on genetic regulation of cerebral cortical size resulted in a first-author research article and cover figure in *Science*. Dr. Chenn is now an assistant professor in pathology at the Feinberg School of Medicine at Northwestern University in Chicago where his laboratory continues to pursue research in mammalian neural development.

Finalists

Liqun Luo, for his essay, "From Single Neuron to Neural Circuits," reporting research carried out in the Department of Biological Sciences at Stanford University. Dr Luo grew up in Shanghai, China, and earned his bachelor's degree in molecular biology from the University of Science and Technology of China. After obtaining his Ph.D. from Brandeis University and pursuing postdoctoral studies at the University of California, San Francisco, Dr. Luo started his own laboratory in December 1996. Work in his laboratory focuses on using genetic tools to understand the logic of brain wiring. Dr. Luo also teaches a course in molecular and cellular neurobiology to Stanford University undergraduate and graduate students.

Lisa Stowers, for her essay, "How Mice Detect and Respond to Pheromones," based on work done in Dr. Catherine Dulac's laboratory at Harvard University. Dr. Stowers was born in Petaluma, California, and received a B.A. in bacteriology from the University of California at Davis. In 1997 she was awarded a Ph.D. in molecular and cellular biology from Harvard University for work in Dr. John Chant's laboratory characterizing signal transduction components of mammalian cell polarity. As a postdoctoral fellow in the laboratory of Dr. Dulac, she used a molecular genetic approach to study the neurobiology of mouse behavior. This work identified the sensory neurons that respond to pheromones and illuminated the influence of the chemical environment on both the social behavior and the neuroendocrine response of the mouse. She is currently continuing this work as an assistant professor of cell biology at The Scripps Research Institute.

Thomas Thannickal, for his essay, "Human Narcolepsy as a Neurodegenerative Puzzle," based on postdoctoral research done in Dr. J. M. Siegel's laboratory at the Sepulveda VA Medical Center and University of California, Los Angeles. Dr. Thannickal was a Ph.D. student in the laboratory of Dr. V. C. Thomas at the Mahatma Gandhi University, Kerala, India, and received his Ph.D. in 1995. In 1996 he joined Dr. V. Mohan Kumar's laboratory at the All India Institute of Medical Sciences, New Delhi, where he studied the basic mechanism of sleep. Dr. Thannickal moved to the United States in 1999 and, working with Dr. Siegel, investigated the neurophysiological basis of human narcolepsy. Their work indicated that narcolepsy is associated with a neurodegenerative process that causes significant loss of hypocretin neurons.

The full text of essays by the finalists and information about applying for next year's awards can be viewed on *Science* Online at www.sciencemag.org/feature/data/prizes/eppendorf/eppenprize.shtml

Our findings support evidence suggesting that epithelial architecture and proteins at adherens junctions regulate growth control and cell proliferation (*23*). Disruptions of adherens junctions may cause misregulation and accumulation of cytoplasmic β-catenin. Our findings that β-catenin signaling can regulate the decisions of neural precursors to reenter or exit the cell cycle lend support to the possibility that β-catenin signaling may mediate the loss of growth control when adherens junctions are disrupted.

It has been proposed that subtle regulation of the decision of neural precursors to divide or differentiate can underlie the expansion of the precursor population without changing the thickness of the cortex (*5*, *24*). We found that β-catenin activation can regulate the size of the neural precursor pool by influencing the decision to divide or differentiate without increasing cell cycle rate, decreasing cell death, or grossly altering neuronal differentiation. Although larger brains can be generated in different ways as well (*25*–*29*), our findings suggest that subtle changes in the decisions of neural precursors to reenter or exit the cell cycle result in horizontal expansion of the surface area of the developing cerebral cortex without increased cortical thickness. Many questions remain about how β-catenin might regulate neural precursor decisions, and whether selective β-catenin inheritance plays a role in cell fate determination. Given its function in a wide variety of tissues, the role of β-catenin in cortical progenitors is likely to be complex. Further understanding of how the decision to divide or differentiate is regulated by β-catenin and other molecules during evolution can provide insight into the mechanisms that underlie the growth of the cerebral cortex in higher mammals.

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