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Dr. Shatz's research aims to understand how early developing brain circuits are transformed into adult connections during critical periods of development. Her work, which focuses on the development of the mammalian visual system, has relevance not only for treating disorders such as autism and schizophrenia, but also for understanding how the nervous and immune systems interact. Dr. Shatz graduated from Radcliffe College in 1969 with a B.A. in Chemistry. She was honored with a Marshall Scholarship to study at University College London, where she received an M.Phil. in Physiology in 1971. In 1976, she received a Ph.D. in Neurobiology from Harvard Medical School, where she studied with Nobel Laureates David Hubel and Torsten Wiesel. During this period, she was appointed as a Harvard Junior Fellow. From 1976 to 1978 she obtained postdoctoral training with Dr. Pasko Rakic in the Department of Neuroscience, Harvard Medical School. In 1978, Dr. Shatz moved to Stanford University, where she attained the rank of Professor of Neurobiology in 1989. In 1992, she moved her laboratory to the University of California, Berkeley, where she was Professor of Neurobiology and an Investigator of the Howard Hughes Medical Institute. In 2000, she assumed the Chair of the Department of Neurobiology at Harvard Medical School as the Nathan Marsh Pusey Professor of Neurobiology. She returned to Stanford in 2007, where she directs Bio-X, Stanford's pioneering biosciences program that brings together faculty from across the entire university- Clinicians, Biologists, Engineers, Physicists, Computer Scientists- to unlock the secrets of the human body. Dr. Shatz has received many awards including the Gill Prize in Neuroscience in 2006. In 1992, she was elected to the American Academy of Arts and Sciences, in 1995 to the National Academy of Sciences, in 1997 to the American Philosophical Society, and in 1999 to the Institute of Medicine. In 2009 she received the Mika Salpeter Lifetime Achievement Award from the Society for Neuroscience. Most recently, Dr. Shatz was elected to the Royal Society of London, England.

Releasing the Brake on Synaptic Plasticity: Immune genes moonlighting in neurons

Connections in the adult visual system are highly precise, but they do not start out that way. Precision emerges during critical periods of development as synaptic connections remodel, a process requiring neural activity and involving regression of some synapses and strengthening and stabilization of others. We discovered, unexpectedly, that MHC Class I genes and an innate immune receptor, PirB, are involved in this process. In mice lacking expression of specific MHCI proteins, synapse regression in the developing visual system fails to occur, synaptic strengthening is *greater* than normal in adult hippocampus, and ocular dominance (OD) plasticity in visual cortex is *enhanced*. Plasticity in mutant mice lacking PirB is also enhanced. Thus, MHCI ligands signaling via PirB receptor may function to “brake” activity- dependent synaptic plasticity. Together, results imply that these molecules, thought previously to function only in the immune system, also act at neuronal synapses to limit how much- or perhaps how rapidly- synapse strength changes in response to new experience. Changes in the function of these molecules may also contribute to developmental disorders such as autism and schizophrenia.