PTEN, MIR, ROS and NSC: Studies of Acronyms in Autism

September 4

Tuesday, 12:30 pm

Weekly Colloquium

Billings Building
Rosedale Conference Room

Speaker: Harley Kornblum M.D., Ph.D.
Professor - Departments of Psychiatry, Pharmacology and Pediatrics, David Geffen School of Medicine at UCLA
Associate Director - UCLA Intellectual and Developmental Disabilities Research Center, Semel Institute for Neuroscience and Human Behavior

Host: Rajiv R. Ratan, M.D., Ph.D.

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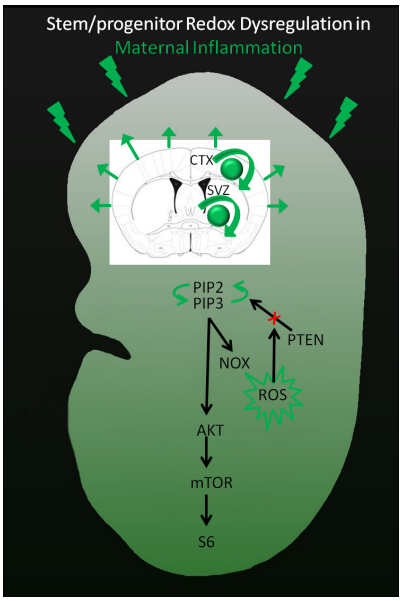
Abstract

The loss of Pten function results in macrocephaly and autism. We have been exploring the role of Pten in the genesis of normal neural stem cells. Genetic deletion or inactivation of Pten via reactive oxygen species can produce enhanced neural stem cell production. In the developing mouse fetus, maternal inflammation activates NADPH oxidase, which then activates reactive oxygen species, producing enlarged brains and many behavioral phenotypes similar to those found in autistic individuals. Surprisingly, we find that many of these behaviors can be temporarily reversed by pharmacologic treatment.



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Le Belle JE, Orozco NM, Paucar AA, Saxe JP, Mottahedeh J, Pyle AD, Wu H, Kornblum HI (2011) Proliferative Neural Stem Cells Have High Endogenous ROS Levels that Regulate Self-Renewal and Neurogenesis in a PI3K/Akt-Dependant Manner, Cell Stem Cell, Jan 7;8(1):59-71.PMC3018289

Le Belle JE, Sperry J, Ngo A, Laks DR, Kornblum HI (2014) Maternal inflammation contributes to brain overgrowth and autism-associated behavioral abnormalities through altered redox signaling in stem and progenitor cells. Stem Cell Reports, Nov 11;3(5):725-34. PMC4235743

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