

Media Contact: Sandy Van
Telephone: 1-800-880-2397
E-mail: sandy@prpacific.com

CEDARS-SINAI RESEARCHERS PRESENT NEW FINDINGS AT NEUROSCIENCE MEETINGS
The Society for Neuroscience's 38th annual meeting will be Nov. 15 - 19 in Washington, D.C.

LOS ANGELES (Nov. 19, 2008) – Researchers from Cedars-Sinai Medical Center will present recent findings during the 38th annual meeting of the Society for Neuroscience. Neuroscience 2008 will be held at the Walter E. Washington Convention Center in Washington, D.C., Nov. 15 through 19, and is expected to draw more than 30,000 attendees from around the world.

Cedars-Sinai scientists from the Department of Neurosurgery, the Maxine Dunitz Neurosurgical Institute, the Board of Governors Gene Therapeutics Research Institute, and Biomedical Sciences are among collaborators or presenters of these presentations:

Saturday, Nov. 15, 3 to 4 p.m., poster presentation: **“CD20 and CD40L microclusters segregate three-dimensionally in vivo at B Cell-T Cell immunological synapses after viral immunity in primate brain.”** Long after viral infections in the brain have been cleared, T cells remain active at the original infection site, even if there is no evidence of viral proteins remaining. Using 3D imaging of animal brains, researchers describe immunological interactions between B cells and T cells that form microclusters of proteins that may keep T cells activated. These data have important implications related to the functioning of the brain's immune system during viral infections, inflammation or autoimmunity.

Saturday, Nov. 15, 4 to 5 p.m., poster presentation: **“The stumpy gene is required for mammalian ciliogenesis and hindbrain development.”** In an animal study, researchers found that this gene is essential for the genesis of cilia, hair-like projects on cells, and appears to be involved in the development of some human congenital malformations such as hydrocephalus.

Monday, Nov. 17, 10 to 11 a.m., poster presentation: **“Adenovirus-mediated expression of GAD65 into the trigeminal ganglion produces long-lasting analgesia.”** Gamma-aminobutyric acid (GABA) is a neurotransmitter in the central nervous system that modulates the sensation of pain. It depends on glutamic acid decarboxylase (GAD) for synthesis. GAD activity and GABA synthesis do not occur naturally in the peripheral nervous system but the researchers in a study of laboratory rats report that transferring a GAD gene into the trigeminal ganglion produced GABA and reduced orofacial neuropathic pain.

Monday, Nov. 17, 10 to 11 a.m., poster presentation: **“Primary cilia regulate hippocampal neurogenesis by mediating sonic hedgehog signaling.”** Hair-like projections called cilia exist on nerve cells and structural cells of the brain, but their function has been largely unknown. In a study using mutant laboratory mice, researchers determined that cilia serve as “antennae” in a molecular signaling pathway that regulates the development and behavior of certain neural stem cells responsible for populating the hippocampal regions of the brain.

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Tuesday, Nov. 18, 10 to 11 a.m., poster presentation: **“Overexpression of S100B exacerbates cerebral amyloidosis and gliosis in the Tg2576 mouse model of Alzheimer’s disease.”** The accumulation of protein fragments (amyloid plaque) between neurons in the brain is a hallmark of Alzheimer’s disease. Reactive astrocytes (cells that respond to injury in the brain) produce neuron-damaging chemicals, including the molecule S100B, which is found in high levels near plaque deposits in Alzheimer’s disease. This study provides genetic evidence that S100B exacerbates Alzheimer’s-like disease in laboratory mice and suggests that targeting astrocyte activation and blocking S100B may offer a therapeutic strategy against Alzheimer’s disease.

Tuesday, Nov. 18, 2 to 3 p.m., poster presentation: **“Ex-vivo adjuvant treatment of dendritic cells with Toll-like receptor ligands may improve anti-glioma dendritic cell based vaccination therapy.”** Clinical trials of a dendritic cell-based vaccination therapy against aggressive brain tumors called glioblastoma multiforme have provided a limited increase in length of survival. Now, in experiments conducted in laboratory mice, researchers boosted immune response by stimulating Toll-like receptors – components of the innate immune system that can induce an adaptive immune response. Results suggest that this approach may improve the effectiveness of the vaccine.

Wednesday, Nov. 19, 8 to 9 a.m., poster presentation: **“Dendritic-cell based vaccination with a weak agonist of myelin-derived peptide attenuates Alzheimer’s-like pathology in APP/PS1 transgenic mice.”** In a study using a mouse model of Alzheimer’s disease, researchers administered a vaccine to boost recruitment of blood-borne immune cells and halt progression of the disease. The vaccine caused a reduction in the amyloid plaques and toxic proteins associated with Alzheimer’s disease, arrested cognitive deterioration, and increased repair and renewal of damaged neuronal tissue. The vaccine appears to restore the brain’s ability to fight off the disease by recruiting monocytes affecting the behavior of resident immune cells of the brain – changing them from destructive to supportive cells. This study – a collaborative effort of Cedars-Sinai’s Department of Neurosurgery and the Weizmann Institute of Science in Rehovot, Israel – supports our pioneering studies over the past decade demonstrating that cellular components of the immune system have a pivotal function in brain maintenance, function, renewal and repair.

Wednesday, Nov. 19, 9:45 to 10 a.m., slide presentation: **“Nerve injection of recombinant adenovirus expressing an shRNA targeting mutant SOD1 slows progression of amyotrophic lateral sclerosis in mice.”** Inherited neurodegenerative disease, including amyotrophic lateral sclerosis, are caused by mutant genes that harm cells in the nervous system. Direct injection of genetically modified viruses to block expression of defective genes has shown promise in other neurodegenerative diseases, but ALS affects wide areas of the central nervous system. Injection into muscles is not practical, but this study in mice suggests that injection into nerves can be used to administer viral vectors that deliver RNA interference that blocks gene expression, eliciting a significant slowdown of the progression of ALS.

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