University of North Carolina at Chapel Hill

The Department of Radiology and the Biomedical Research Imaging Center (BRIC) at the University of North Carolina at Chapel Hill invite applications for an Assistant Professor (tenure track) level faculty position in the area of brain functional imaging, particularly in the area of brain network analysis. The State of North Carolina has made significant investment to establish the Biomedical Research Imaging Center, an institution center dedicated to imaging research. The BRIC houses a comprehensive collection of human and animal imaging devices. Of particularly relevant to this position are the 3T whole body MR, hybrid PET/MR scanner, and whole body 7T MR scanner. The BRIC is an institution center dedicated to imaging research. In addition, a cyclotron and radiochemistry facility has recently been established in the BRIC, further augmenting the imaging capability at UNC. The BRIC currently has 32 faculty with a wide array of imaging expertise, including imaging acquisition (MR, PET, SPECT, CT, ultrasound, and optical imaging), image analysis, imaging statistics and informatics, imaging hardware (MR, CT, and optical imaging), and applications. Qualified candidates should have a doctoral degree in Biomedical Engineering or equivalent areas and are expected to have an outstanding record in functional MR technique. Specifically, candidates should have experience on resting functional connectivity studies, particularly in statistical modeling and data analysis. The successful candidate is expected to establish an independent imaging research program and to effectively contribute to the missions of the BRIC as well as secure extramural grant funding. Interested applicants should submit an online application including your curriculum vitae and two letters of recommendation. Apply online at http://unc.peopleadmin.com/postings/77080. The University of North Carolina at Chapel Hill is an equal opportunity, affirmative action employer and welcomes all to apply regardless of race, color, gender, national origin, age, religion, genetic information, sexual orientation, gender identity or gender expression. We also encourage protected veterans and individuals with disabilities to apply.

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Functions of the Motor Cortical-Thalamic Circuit in Non-Human Primates

A Postdoctoral position is available at the University of Pittsburgh to study functions of the circuits that connect cerebellar- and basal ganglia-recipient regions of thalamus and the motor cortex. The project will compare the activities of identified cell types and circuits using multi-electrode single unit recording and electrophysiologic and optogenetic techniques in behaving monkeys.

Candidates with experience in motor control research and non-human primate neurophysiology are encouraged to apply. Additional experience in electrophysiology, behavioral testing, and statistical analysis of neuronal data analysis is preferred. Competitive salary with benefits and travel funds.

To apply, please send a statement of research interests, CV and names of two references to: Robert S. Turner; Dept. of Neurobiology, University of Pittsburgh; rturner@pitt.edu.

NIH *Eunice Kennedy Shriver* National Institute of Child Health and Human Development

Section on Molecular Neurobiology

Post-doctoral Fellow Position

A position is available for an electrophysiologist to investigate how the Neuregulin (NRG)\ErbB and dopamine signaling pathways, both genetically implicated with risks for schizophrenia and ADHD, regulate synaptic transmission and neuronal networks that underlie cognitive processes altered in these psychiatric disorders. Using gene targeting, neurochemistry, behavioral and electrophysiological approaches, our laboratory recently identified critical functional interactions between the NRG/ErbB4 tyrosine kinase and dopamine GPCR signaling pathways that converge on fast spiking GABAergic interneurons to regulate synaptic plasticity and gamma oscillations, as well as numerous behaviors in rodents designed to model traits associated with psychiatric disorders.

We are recruiting for a motivated electrophysiologist, with prior experience in whole cells recordings from acute brain slices and/or *in vivo*, to investigate how cortical excitatory/inhibitory balance is altered in different genetically modified mouse lines targeting the NRG\ErbB4 and dopamine pathways, and that exhibit a number of altered behaviors. The aim of this research is to identify and characterize the neuronal microcircuits and molecular pathways that contribute to complex behavioral processes associated with cognition.

Interested candidates who earned their PhD less than four years ago should send applications including a curriculum vitae, a statement of research interests and long-term career goals, and the name, email and mailing addresses of three references by July 9, 2015 to: Dr. Andres Buonanno, Section on Molecular Neurobiology, Porter Neuroscience Research Center, Building 35 Rm 2C-1000, Bethesda, MD 20892. Email: buonanno@mail.nih.gov

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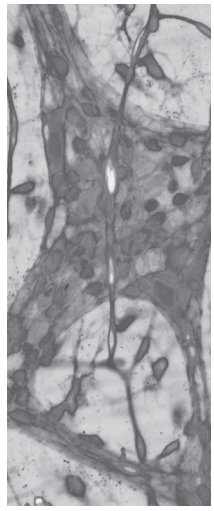

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This Week in The Journal

Efferent Input to Inner Hair Cells Reappears during Age-Related Hearing Loss

Stephen Paul Zachary and Paul Albert Fuchs

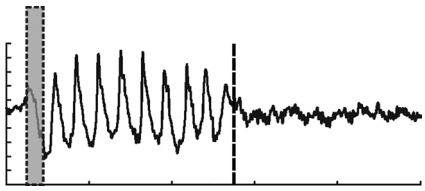
(see pages 9701–9706)

Before the onset of hearing in rodent pups, efferent cholinergic axons transiently innervate cochlear inner hair cells (IHCs). During the first 2 postnatal weeks, activation of nicotinic acetylcholine receptors (nAChRs) on IHCs inhibits the cells, and this is thought to help shape developing auditory circuits. Although efferent innervation of IHCs subsequently disappears, electron micrographic studies have noted the presence of presynaptic terminals abutting IHCs in old C57 mice—a strain that exhibits age-dependent hearing loss. Whether these contacts represent functional synapses, and if so, how the synapses affect IHCs, has been unclear. Zachary and Fuchs now answer these questions.

Whole-cell recordings from excised portions of the apical cochlear epithelium indicated that postsynaptic currents were present in IHCs from 1-week-old, but not 1-month-old C57 mice. Postsynaptic currents were again detected in IHCs after 9 months, and the proportion of IHCs exhibiting such currents increased to ~50% by 12 months. The reappearance of postsynaptic currents in IHCs coincided with increases in auditory thresholds, loss of ribbon synapses between IHCs and spiral ganglion dendrites, and loss of outer hair cells.

Like the postsynaptic currents measured in IHCs of newborn mice, those in aged IHCs were induced by acetylcholine and required nAChRs containing the $\alpha 9$ subunit. Furthermore, the currents were inhibitory and mediated by small-conductance calcium-activated potassium channels. These data indicate that cholinergic efferent inhibition similar to that present in immature IHCs re-emerges during age-dependent hearing loss in C57 mice.

It should be noted that C57 mice harbor a genetic mutation that alters a component of the tip links required for sound transduction by hair cells. Therefore, future experiments should determine whether efferent inhibition re-emerges in age-related hearing loss occurring in the absence of genetic predisposition. In addition, whether the re-emergence of efferent inhibition exacerbates hearing loss or attenuates it—for example by minimizing excitotoxic damage—is an important question for future research.



Light cues associated with reward sometimes elicit oscillations in V1 that persist from the time of cue presentation (shaded area) until the median reward delivery time (dashed vertical line). See Zold and Hussain Shuler for details.

V1 Activity Reflects Reward Rate and Timing

Camila L. Zold and Marshall G. Hussain Shuler

(see pages 9603–9614)

Experience-dependent plasticity of the visual system has been a rich vein of research for decades. Much work has focused on the effects of visual experience during development, particularly how this plasticity shapes feature-detection circuits in primary visual cortex (V1). But visual experience also alters V1 responses in adults. For example, repeated presentation of the same stimulus increases the amplitude of stimulus-evoked potentials recorded in V1. Recent studies have also found that rodent V1 plasticity involves more than simply enhancing representation of the physical attributes of visual stimuli (reviewed in Gavornik and Bear, 2014, Learn Mem 21:527). For example, after a visual stimulus has repeatedly been paired with a reward, V1 activity persists after the visual stimulus disappears and continues until the time reward is expected. Furthermore, if a given sequence of visual stimuli is presented repeatedly and one of the stimuli is then omitted, V1 responds as if that stimulus were still presented.

Zold and Hussain Shuler provide evidence that rodent V1 also encodes information about the recent history of reward associated with visual stimuli. They found that after light cues were repeatedly paired with delayed rewards delivered on 50% of trials, the cues began to evoke 6–9 Hz oscillations on some trials. Like in previous studies, the timing of oscillations—if they occurred—changed over the course of training. Initially, the duration reflected the intensity of the light stimulus, but this relationship was gradually lost and the duration instead began to reflect the expected time of reward. The probability of evoking an oscillation also varied over the course of training. Initially, the probability was determined primarily by the intensity of the light cue. But after the task and the reward timing were learned, the probability of evoking an oscillation was additionally influenced by the recent reward rate.

These results clearly indicate that V1 does not simply encode the physical attributes of a stimulus. What information visually evoked oscillations encode remains unknown, but Zold and Hussain Shuler suggest they reflect the behavioral relevance of visual cues. Thus, oscillations are evoked more often as the rat learns that the cues signal reward and less often as the rat becomes sated.

This Week in The Journal is written by Teresa Esch, Ph.D.

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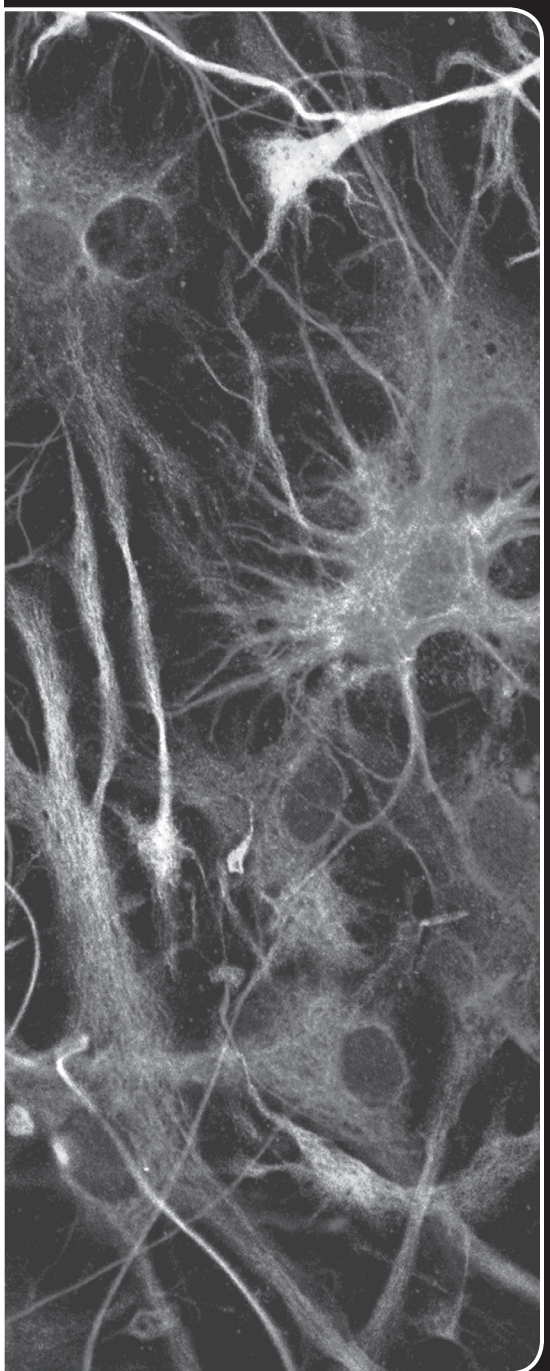

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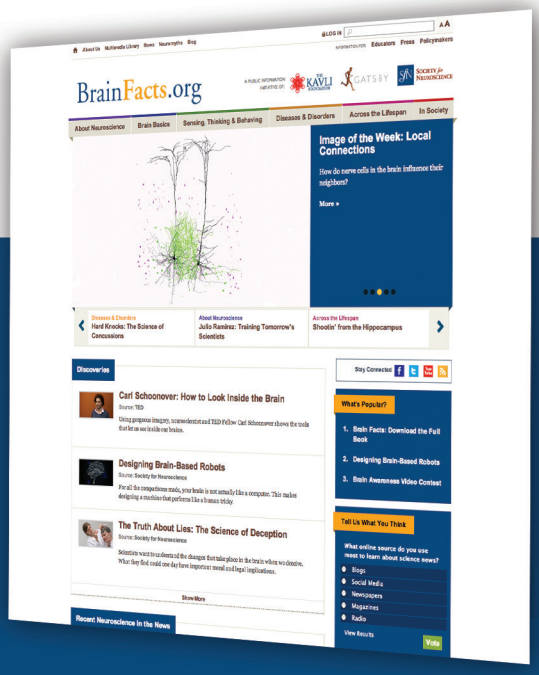

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