Long-term, low-level noise exposure: Unexpected plasticity in ear and brain
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Abstract:
Research indicates long-duration, low-level noise exposures can induce plasticity in the auditory cortex, which can impair auditory perception; however, it is unclear if these functional changes originate more peripherally in the auditory pathway. Using a systems approach, we investigated the physiological plasticity that occurs in the cochlea and auditory midbrain following long-term, low-level noise exposures. The unexpected results are interpreted and discussed in relationship to environment-induced impairment and implications for treatment of auditory disorders such as tinnitus and hyperacusis.

Rationale/Purpose:
Many individuals live or work in environments in which they are continuously exposed to low-level noise. Previous studies have shown long-term, low-level noise exposure that cause little or no hearing loss can nevertheless disrupt intensity coding, temporal processing and tonotopy in the auditory cortex; however, it is unclear if these functional changes originate in the auditory cortex or more peripheral locations in the ascending auditory pathway. The purpose of this study was to investigate plasticity occurring along the auditory pathway following long-term, low-level noise exposure through use of a systems approach (measurements from cochlea and auditory midbrain). Further understanding of low-level noise induced plasticity can lead to the modification of current noise standards and be leveraged for treatment of auditory disorders such as tinnitus and hyperacusis.

Methods:
Rats, all housed in their home cage in the vivarium, were divided into two groups: (1) Control group and (2) Long-term, low-level noise (10-20 kHz, ~70 dB SPL, 24 h/d for 6 weeks). Cochlear function was assessed by measuring the compound action potential (CAP), summating potential (SP) and distortion product otoacoustic emissions (DPOAE). Local field potentials (LFP) and multi-unit firing rates were recorded from the central nucleus of the inferior colliculus (CIC) using linear 16-channel microelectrodes. Following physiological assessment, cochlear tissue was processed and stained for morphological assessment of structural damage and IHC ribbon synapses. The methodology used allows for comprehensive analysis of physiological functioning along the ascending auditory pathway, from the cochlea to the auditory midbrain.

Results & Conclusions:
The long-term, low-level exposure caused: (1) a very mild (~8-10 dB) shift in CAP threshold between 10-20 kHz, but had no effect on thresholds below or above the exposure frequency band. (2) Surprisingly, CAP suprathreshold amplitudes were reduced roughly 50% at frequencies within and above the exposure band. These results indicate that low-level noise exposure can greatly reduce the neural output of the cochlea in a frequency specific manner. (3) Paradoxically, sound evoked neural activity (multi-unit local field potentials and spike discharge rates) in the CIC was enhanced at test frequencies from 10 to 20 kHz. These results suggest that the central auditory system increases its gain in the 10-20 kHz region to compensate for the reduced cochlear output at these frequencies and (4) there was no evidence of hair cell loss and no detectable changes in Ctbp2 ribeye immunolabeling for ribbon synapses in the inner hair cell region. Our results indicate that low-level, long-term noise exposures, that would normally be considered “safe”, cause very mild CAP threshold shifts, but greatly reduce the neural output of the cochlea at frequencies within and above the exposure band. Despite a large reduction in cochlear output, sound evoked responses in the CIC are greatly enhanced within the exposure band. This knowledge opens a new arena for the modification of noise exposure standards as well as the possibility of leveraging noise-induced plasticity for the treatment of auditory disorders such as tinnitus and hyperacusis.

Learning Objective:
Upon completion, participants will be able to classify the physiological impact that low-level noise has on the ascending auditory pathway, from the cochlea to the auditory midbrain.