Hearing Loss and Protection

Inner Ear and Auditory Cortex Responses to Blast Shockwave Exposure

Hearing loss is one of the most common disabilities in military personnel exposed to blast shockwaves. To better understand the pathological processes underlying the injury, investigators at Walter Reed Army Institute of Research (Silver Spring, MD) evaluated changes in ear structures, gene activity in the cochlea (the structure that senses and converts sound waves into electrical signals), and neuronal structures in the auditory region of the brain (auditory cortex) using a rodent model of blast injury. Anesthetized mice and rats were exposed to blast overpressure using an Advanced Blast Simulator resulting in auditory deficits observed across the entire acoustic frequency spectrum. Perforation of the eardrum and hemorrhage in the middle and inner ear were observed at one- and seven-days post-injury. Most were healed at 28 days after injury.

In the acute phase post-injury (one day), over 1000 genes displayed different activity in the cochlea. In the chronic phase post-injury (one month), activity only differed in 48 genes. These genes play a role in several biological processes including nervous system development and cell signaling. Genes with altered activity in the chronic phase were primarily related to immune system activity.

Changes in the signaling surfaces between neurons (synapses) were also evaluated. Changes in neuronal structure indicative of stronger connections between neurons (increased dendrite density) were observed on auditory cortex neurons in the acute phase after blast exposure. The shape and number of dendrites correlated with the strength of the signal transmission.

Taken together, these findings reveal the impact of blast shockwave injury to the ear and hearing-related neural activities that affect auditory function and govern regenerative processes. Further study of altered gene activity may provide insights into new methods for treating or preventing blast-induced auditory deficits.

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