392 Inflammatory Cells in the Mouse Cochlea After Acoustic Trauma

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After acoustic trauma, the cochlea sustains dramatic cellular injury and undergoes repair over subsequent days to weeks. The role of inflammation in this repair process has not been systematically evaluated. CBA/CaJ mice were exposed to octave-band noise at 106, 112 or 120 dB SPL for 2 hours and evaluated at 1, 3, 7 and 14 days after noise. Auditory brainstem responses were used to determine thresholds. Immunohistochemistry was performed with CD45, a cell-surface marker present on all leukocytes, to identify these cells within the cochleas of control and noise exposed mice. CD45 positive cells were counted at various locations within the cochlea including the organ of Corti, spiral ligament, stria vascularis, spiral ganglion, scala tympani and scala vestibuli. All exposure groups sustained permanent threshold shift, A small population of resident monocytes were found in the control cochleas in the inferior spiral ligament that closely resemble microglia of the brain, both histologically and by immunophenotype. At all sound pressure levels, there was a significant increase in CD45 positive cells which peaked at 7 days post-exposure. After 120 dB noise, CD45 positive cells returned to baseline levels by 14 days. Immunostaining for CD3, CD68, IBA and F4/80 suggest that the majority of these cells are either macrophages or microglia. Inflammatory cells may play a critical role in the repair process after acoustic trauma by both removing cellular debris and altering the local environment through elaboration of chemical mediators. Damage to non-sensory structures may be a result of bystander injury from activation of microglia and macrophages as opposed to direct injury from acoustic injury.

393 Hair-Cell-Membrane Changes in the Cochlea Following Noise

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The morphological correlates of temporary threshold shift (TTS) remain controversial. Mulroy et al ('98) provided indirect evidence that alligator lizards with noise-induced TTS have microlesions in hair-cell plasma membranes. To test this hypothesis directly, chinchillas were exposed for 24 hrs to a 4-kHz OBN at 92 dB SPL. ABR & DPOAE testing were carried out pre- & post-exposure. Three hrs post-exposure, 3 chinchillas had EP measured & carbon particles injected into the endolymphatic space before fixation. Two chinchillas recovered for 1 or 2 wks before EP measurement, carbon injection & cochlear fixation. Two controls underwent EP measurement (74 & 89 mV), carbon injection & cochlear fixation. Cochleas were embedded in plastic, dissected as flat preparations & missing cells counted. Cytocochleograms were prepared with functional data overlaid according to the chinchilla frequency-place map. Thin sections of the OC were cut in the region of maximum threshold shift (TS) for TEM. Immediately post-exposure, all animals had an ABR TS of 20-60 dB over a frequency range of 1-16 kHz & a DPOAE level shift (LS) of 10-50 dB over 2-16 kHz; EP was 21, 49 & 79 mV in the three 0-d-recovery animals. Most hair cells were present & had normal shapes by phase contrast microscopy; by TEM, OHC stereocilia were slightly disarrayed but the plasma membranes were intact. The 1-wk-recovery animal had an ABR TS of 10-20 dB for 1-16 kHz, a DPOAE LS of 12-30 dB for 3-12 kHz & an EP of 83 mV. The 2-wk-recovery animal had an ABR TS of 10-20 dB for 3-6 kHz, a DPOAE LS of 12-23 dB for 3-12 kHz & an EP of 80 mV. In the 1-wk-recovery animal, one region of the OC contained about 20% degenerating OHCs. By TEM, the apical membranes of some degenerating hair cells contained microlesions. We conclude that hair cells do not develop microlesions in noise-exposed mammalian cochleas unless the cells are degenerating.

394 Comparison of Noise-Induced DPOAE Temporary Level Shift with Detailed Histopathology

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DPOAE temporary level shift (TLS), ABR temporary threshold shift (TTS), and detailed histopathology were determined in 3 groups of chinchillas exposed to an octave band of noise (OBN) centered at 4 kHz at either 80, 86 or 92 dB SPL for 24 hours (n=3, 4, 6). DPOAEs at 39 frequencies from f_1=0.3 to 16 kHz (f_2/f_1=1.23; L_2 & L_1=55, 65 & 75 dB, = & ≠) and ABR thresholds at 13 frequencies from 0.5 to 20 kHz were collected pre- and post-exposure. The functional data were converted to pre- minus post-exposure shift and overlaid upon the cytocochleogram of cochlear damage using the frequency-place map for the chinchilla. The magnitude and frequency-specific location of components in the $2f_1$-f_2 TLS patterns were determined and group averages for each OBN and L_1, L_2 combination were calculated. The f_2-f_1 TLS was also examined in ears with focal lesions ≥ 0.4 mm. The $2f_1$-f_2 TLS (plotted at f_1) and TTS aligned with the extent and location of damaged supporting cells. The TLS patterns had two features which were unexpected; a local minimum at about a half octave above the center of the OBN with a local maximum above and below it, and a local minimum (often with negative shift) at the apical boundary of the supporting-cell damage. The magnitudes of the TLS and TTS generally increased with increasing exposure SPL. The peaks of the TLS and TTS, as well as the local TLS pattern components moved apically as the OBN was increased. However, there was no consistent pattern-relation with L_1, L_2 combinations. In addition, neither the $2f_1$-f_2 nor f_2-f_1 TLS for any L_1, L_2 combination consistently detected focal lesions (100% OHC loss) from 0.4 to 1.2 mm in length. Often at focal lesions, the TLS went in the opposite direction from what would be expected. Thus, TLS is sensitive to and reflects a differing mechanism for noise-induced temporary hearing loss than for permanent hearing loss.

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