Impulse noises induce a combination of stereocilia damage and hidden hearing loss

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INTRODUCTION

Noise-induced hearing loss is a common cause of hearing impairments in industrialized countries. It is well-known that high intensity noise exposure damages sensory hair cells, leading to a permanent threshold shift. Recent studies have demonstrated that moderate exposure to a continuous noise can induce a temporary threshold shift (TTS) with a permanent degeneration of auditory nerve fibers, although hair cells remain intact. In this study, we tested the hypothesis that gunshot-like impulse noise exposure causing TTS could also produce cochlear synaptopathy with minimal hair cell loss.

METHODS

10–14-week-old male CBA/J mice were exposed to an impulse noise (peak pressure: 145 dB SPL, 1 pulse/second, for 700 seconds). Hearing was evaluated with ABR and DPOAE before, 30 minutes, 24 hours and 2 weeks after exposure. The morphology of the sensory hair cells and ribbon synapses was studied with electron and confocal microscopy.

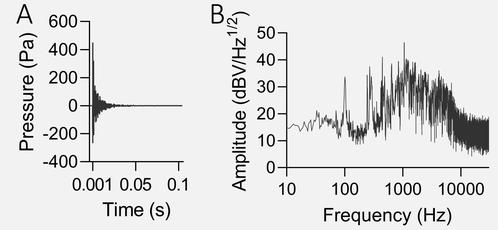

Figure 1 : Impulse noise
A. Impulse noise triggers sharp transient pressure wave, B. Energy distribution as function of frequencies

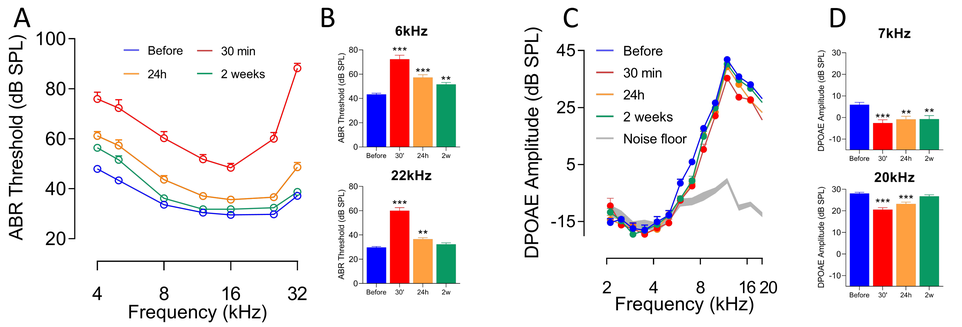

Figure 2: Impulse noise-induced reversible hearing loss
(A,D): ABR thresholds (A) and DPOAE amplitudes (C) recorded before (blue) and 30 min (red), 24 h (orange), and 2 weeks (green) after impulse-noise exposure. Note the complete recovery of ABR thresholds and DPOAE amplitudes in the higher-frequencies by 2 weeks after exposure. (B): Mean ABR thresholds at 6 and 22 kHz. (D): Mean DPOAE amplitudes at 7 and 20 kHz. All data are expressed as mean ± SEM (*n* = 22 cochleae per time point)

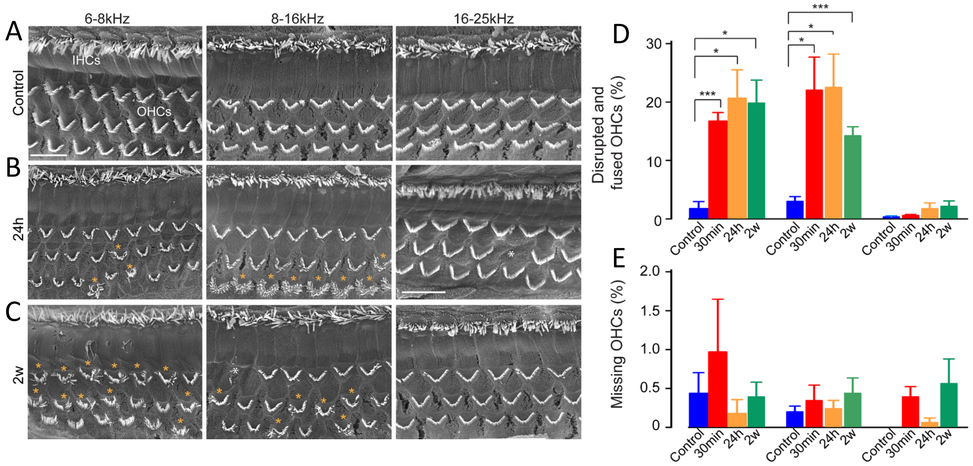

Figure 3 : Alteration of the OHC stereocilia in the mid-apical cochlear region
A, B and C: Scanning electron micrography from different coding regions of cochleae (6–8 kHz, 8–16 kHz, 16–25 kHz) from control (A), 24 h (B), and 2 weeks (C) after exposure. (D–E): Cytocochleograms representing the percentage of disrupted and fused OHCs (D), and missing OHCs (E) in different coding regions (6–8kHz, *left* ; 8–16kHz, *middle* ; 16–25kHz, *right*). Data are expressed as mean ± SEM (*n* = 6 to 12 cochleae per group)

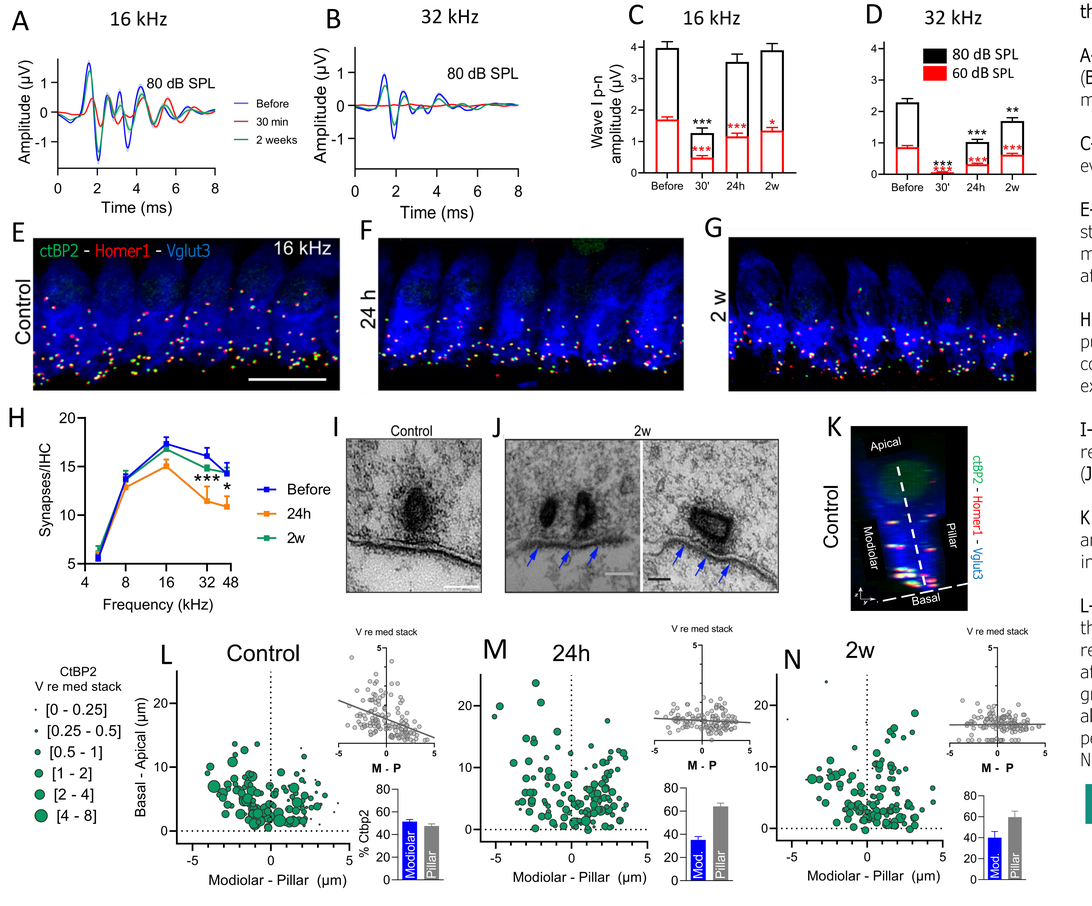

Figure 4: Impulse noise-induced hidden hearing loss in the basal half of the cochlea

A-B: Mean ABR waveforms evoked by 16 (A) or 32 kHz (B) tone-bursts at 80 dB SPL recorded before (blue), 30 min (red) and 2 weeks (green) after exposure.

C-D: ABR wave I p-n amplitude (µV) at 80 (black) or evoked by 16 (C) or 32 kHz (D) tone-bursts.

E-G: Maximum intensity projections of confocal z-stacks of IHC in the mid-cochlear region of control mice (E), noise-exposed mice at 24 h (F), and 2 weeks after (G). Scale bar = 10µm.

H: Quantifications of synapses (paired CtBP2-Homer1 puncta) per IHC along the tonotopic axis of the cochleae from control (blue), and impulse noise-exposed mice at 24 h (orange) and 2 weeks (green)

I-J: TEM images of ribbon synapses from the cochlear regions coding 16–25 kHz from control (I), and 2 weeks (J) after exposure. Scale bars = 200 nm.

K: Maximum zy projection used to quantify ribbon size and position, a vertical axis is placed to bisect the IHC into pillar and modiolar halves.

L-N: ctBP2 volume (re median stack) and position along the modiolar-pillar axis of IHC in the 16-25kHz regions, in control conditions (L), 24h (M) and 2 weeks after noise (N). Right upper panel shows the size gradient of ctBP2 (green dots) and Homer1 (red dots) along the modiolar-pillar axis. Lower panel shows the percentage of pillar and modiolar ctBP2 puncta.
N= 2 stacks, 8 IHC

CONCLUSION

These results clarify the pathology underlying impulse noise-induced sensory dysfunction, and links impulse noise injury to hidden hearing loss.

FINANCIAL SUPPORT

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