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Titel des Beitrags:
MyD88-dependent immune response contributes to hearing loss in experimental pneumococcal meningitis.

Abstract:
Hearing loss is one of the most common sequelae in survivors of pneumococcal meningitis, affecting up to 26% of them. Here, we established the first mouse model of meningitis-associated hearing loss and investigated the role played by the Toll-like receptor-associated adapter molecule MyD88. C57BL/6 mice were infected intracisternally by Streptococcus pneumoniae. By use of audiometry and histological analysis, cochleae were assessed in uninfected control mice during the acute stage and after recovery. MyD88-deficient mice were analyzed 24 h after infection. Wild-type mice lost hearing capacity to a significant degree, which was accompanied by a granulocytic cochlear inflammation. After recovery, hearing loss was still evident, and spiral ganglion neuronal loss, hair cell damage, and fibrocytic occlusion of the cochlea were observed. In contrast, mice lacking MyD88 developed significantly less hearing loss and had diminished cochlear inflammation. Our results strongly suggest a proinflammatory role for MyD88 in the initiation of the inflammatory response during pneumococcal meningitis-associated labyrinthitis.

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