

Dr. Radic, *et al* reply

To the Editor:

We thank Conway and colleagues for their interest in our article¹. The major thrust of our report was not to debate whether biological therapy is good or bad, beneficial or not, for autoimmune sensorineural hearing loss (AISNHL) treatment. Our purpose was to present a very interesting case report of successful use of adalimumab for treating rheumatoid arthritis (RA) with AISNHL. The classic description of AISNHL by McCabe includes responsiveness to immunosuppression². Use of other immunomodulators to maintain improvements in steroid-responsive patients is advocated to reduce the side effects of longterm corticosteroids. The most researched nonsteroidal agents in animal models of AISNHL are inhibitors of TNF- α , mostly etanercept^{3,4,5}. Animal models with keyhole limpet hemocyanin-induced labyrinthitis treated with etanercept found decreased cochlear inflammation with reduction of hearing loss. Nevertheless, human clinical studies have given contradictory results on AISNHL treatment with etanercept, mostly negative^{6,7}. One pilot study described the efficacy of intratympanic application of infliximab for treatment of AISNHL by allowing steroid taper and facilitating hearing improvement⁸.

In contrast to our results, Conway, *et al* described 2 cases of AISNHL related to adalimumab therapy. It is very hard to distinguish whether this is a side effect of adalimumab therapy or AISNHL in association with autoimmune disease such as RA. The pathophysiology of AISNHL is largely unknown and possible treatment with nonsteroidal medications has yet to be identified. We agree that inhibitors of TNF- α should be used with great caution for AISNHL treatment. Appropriate treatment of AISNHL may provide reversal of sensorineural hearing loss and may best be served by exploring new treatment modalities, focusing on intratympanic delivery.

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