Dear Editor:

Hsu et al. divided 412 young noise-exposed male workers into two groups: those with and those without audiometric notches. Only 20% had bilateral notches; notch presence was unrelated to age or to noise exposure level or duration. After correcting for age and exposure, the men with notches had worse hearing at 3, 4, and 6 kHz, and slightly better hearing at 1 and 2 kHz, than the men who did not have notches. This appears to be a predictable consequence of the notch definition they used: any threshold at 3, 4, or 6 kHz that was at least 10 dB worse than the threshold at 1 or 2 kHz, and at least 10 dB worse than the threshold at 8 kHz.

As Schlauch and Carney have pointed out, normal test-retest variability can lead to spurious shallow notches that are seen on one test and absent on the next. A person whose “true” audiogram (defined as the average of several separate tests) is completely flat may appear to have a notch if, on a particular day, his threshold at 4 kHz is slightly higher, or his thresholds at 2 and 8 kHz are slightly lower, than their usual values. The fact that the men with notches had better thresholds at 1 and 2 kHz than the men without notches suggests that at least some of these notches were spurious. Hsu et al. do not report 8 kHz thresholds. I suggest that at this frequency, as for 1 and 2 kHz, the men with notches had better hearing than those without notches. This would strengthen the argument that many of the notches were spurious.

The authors were correct to note that factors other than age and occupational noise exposure must play a role in determining who will develop an audiometric notch. They did not identify any such factors, but genetic susceptibility and nonoccupational noise exposure would be among the logical candidates in addition to test-retest variance. However, they also state that after using the ISO-1999 model to correct for age, sex, and noise exposure, they “assumed that the variability in the shifts [deviations from the ISO-1999 medians] was determined by an individual’s vulnerability to noise.” That is equivalent to assuming that in the absence of noise exposure, all people of a given age and sex (e.g., 40-year-old men) would have identical audiograms. Contradicting that assumption, even highly screened populations (people who deny any noise exposure) have, at every age, marked variations in hearing thresholds (see Annex A of ISO-1999). Therefore, if one could identify the risk factors (such as genetic variants) that predicted either audiometric notches or increased thresholds at specific frequencies, those risk factors might be related to susceptibility to noise, but could also be related to audiometric variation present at birth or to susceptibility to age-related threshold shifts. Longitudinal studies in very young adults, early in a career with high levels of occupational noise exposure, could minimize age-related changes and would have the best chance of separating risk factors that are related to congenital variation (present at baseline) from those that are related to noise susceptibility (as demonstrated by longitudinal change).

A final comment. The authors refer to audiometric notches as “pathognomonic for [noise-induced hearing loss] NIHL.” A pathognomonic finding is one that is unique to a particular disorder, on the basis of which a firm diagnosis can be made. The authors note that notches occur also in cases of closed head injury, barotrauma, and labyrinthitis, so they clearly know that one cannot diagnose NIHL with certainty based simply on the presence of a notch. As noted above, many notches are spurious. In their own data, there was no correlation between audiometric notching and noise exposure, which suggests that many of the notches were related to factors other than noise exposure. Nondahl et al. also found a poor correlation between reported noise exposure and notches in the Beaver Dam cohort. I hope that the authors would agree that an audiometric notch is suggestive of NIHL but not pathognomonic.

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