calization cues are readily available to animals with large heads, the effectiveness of either cue is diminished in animals with functionally close-set ears. In the case of Δt, the available time difference may be so small that the nervous system can detect only gross changes in sound direction. However, an animal with a small head always has a Δfi cue available, providing only that it is able to perceive frequencies which are high enough to be effectively shadowed by its head and pinnae. Therefore, given the ecological importance of an animal's localizing the sound of a stealthy intruder, animals with functionally close-set ears are subjected to more selective pressure to hear high frequencies than animals with more widely set ears.

Finally, the finding that the elephant is unable to hear significantly above 10 kHz has two immediate implications for ecological and evolutionary acoustics. (i) It suggests that when the selective pressure for high-frequency hearing is reduced as a consequence of evolving a large interaural distance, the upper limit of hearing is reduced to the point at which it does not greatly exceed that of nonmammalian vertebrates, such as birds, many of which hear up to 10 kHz. (ii) It appears that humans should no longer be considered aberrant among mammals for their lack of ability to hear above 20 kHz. Instead, restricted high-frequency hearing seems to be a consequence of a relatively large interaural distance and not the result of a special adaptation for the reception of speech sounds, as was once widely believed.

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6. Loudspeakers used were a 15-inch (38-cm) woofer in a 0.45-m³ sealed enclosure and a piezoelectric tweeter. Sound production and measurement were by techniques described elsewhere [R. Heffner, H. Heffner, B. Masterton, *J. Acoust. Soc. Am.* **49**, 1888 (1971)].
7. Varying the definition of threshold over a wide range has no significant effect on the main conclusions.
8. Maximum Δt in a given species is the maximum possible difference in the time of arrival of a sound at the two ears. The value for maximum Δt depends on the path which the sound travels from ear to ear as well as the velocity of sound in the particular medium, that is, the distance from ear to ear divided by the speed of sound in air (340 m/sec) around the head in terrestial mammals; and the distance from ear to ear through the head divided by the speed of sound in water and tissue (1500 m/sec) in most marine mammals.
9. For a list of the audiograms, see R. L. Francis [in *Sound Reception in Mammals*, R. J. Bench, A. Pye, J. D. Pye, Eds. (Academic Press, London, 1975), pp. 237-289], H. Heffner and B. Masterson (*J. Accoust. Soc. Am.*, in press) and the sources in (*2*).
10. We thank the Ralph Mitchell Zoo of Independence, Kans., for allowing us to study their elephant, and N. Stichman, for his help in training her. Supported by NSF grant BNS 58-07391.

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Cadmium Concentrations in Blood

In their report on in vivo cadmium measurements, Ellis *et al.* state that they found no significant difference in mean blood plasma or urine concentrations of smokers as compared with nonsmokers (*1*). The plasma and urine cadmium concentrations for nonexposed individuals reported in (*1*) are at least three to five times higher than those reported in the current literature (*2*).

Studies of cadmium partitioning in whole blood from laboratory animals exposed to cadmium indicate that the major portion of the metal is contained in the erythrocytes (*3*). Thus, the whole blood cadmium concentration might be a more appropriate parameter to measure. For nonoccupationally exposed nonsmokers over 36 years ($N = 27$), Pleban and Pearson found the mean blood cadmium concentration to be 1.00 ± 0.48 μg/liter (arithmetric mean ± standard deviation), whereas smokers over 36 years ($N = 18$) had a mean blood cadmium concentration of 2.21 ± 0.92 μg/liter (*4*). These means are significantly different (*t*-test, $P < .001$) and are in agreement with similar findings of others (*5*).

The normally low body fluid concentrations of cadmium require that extreme care be taken in sample collection and analysis procedures. This is particularly true for analyses in which extraction and wet ashing procedures are used. Sporadic cadmium contamination of 0.5 to 1.0 μg/liter, which is not as critical when measuring higher cadmium concentrations in organs of the body, can mask significant differences between populations with body fluid concentrations at the microgram per liter level.

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Pleban leaves the impression that our values are too high and due to contamination when in fact the blood cadmium concentrations for nonoccupationally exposed smokers and nonsmokers she cites are not statistically different from the plasma values we reported in (*1*). We agree that extreme care must be taken in the collection and analysis procedures, and the wide range of values reported in the literature renders interlaboratory comparisons difficult. Nevertheless, we concur with Pleban that cadmium concentrations in the blood of smokers are generally higher than those in the blood of nonsmokers. The *P* values (mean differences by the *t*-test) for our small sample population of smokers and nonsmokers were .07 and .06 for cadmium concentrations in the plasma and urine, respectively.

However, in our investigation we found no significant relationship between the plasma or urine data and the kidney or liver burden of cadmium. Recently, we have studied workers occupationally exposed to cadmium (*2*). Although blood and urine cadmium concentrations were marginally correlated on a group basis, they were not predictive of the cadmium burden in the kidney or liver of an individual.

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