were found to have the mutation of $tRNA^{leu(UUR)}$, suggesting that the mitochondrial mutation is frequent among the patients with diabetes and auditory disturbance. However, there have been no reports, to our knowledge, about the real frequency of this mutation in diabetic patients with auditory disturbance.

To investigate whether sensorioneural auditory disturbance could be a good index to detect the mitochondrial mutation, we examined the mutation in Japanese type 2 diabetic patients with auditory disturbance. Subjects included 35 Japanese patients (21 men and 14 women; mean [±SD] age, 61.6 ± 12.8 years) with type 2 diabetes (n = 33) or impaired glucose tolerance (n = 2), according to World Health Organization criteria, presenting with hearing loss, which was confirmed to be sensorioneural auditory disturbance with audiometry by one of the investigators (H.S.). Genomic DNA was extracted from peripheral leukocytes of the patients and submitted to the detection of an A-to-G transition at the 3243 nucleotide of $tRNA^{leu(UUR)}$, using polymerase chain reaction and restriction fragment length polymorphisms methods with restriction endonuclease *Apa* I. The mutation of the 3243 nucleotide of $tRNA^{leu(UUR)}$ was found only in one female patent with type 2 diabetes (2.9%). There was no statistically significant difference (χ^2 test) between this frequency and the reported ones of the mutation among Japanese patients with type 2 diabetes: 0.9% (5) or 2.0% (6).

A previous report (6) showed that 3 out of 5 cases (60%) of Japanese diabetic patients with hearing loss were found to have the mitochondrial DNA mutation. However, all of those patients also had a family history of the association of diabetes and hearing loss. Our result suggests that auditory disturbance is often associated with NIDDM and that mitochondrial DNA mutation is infrequent in Japanese diabetic patients with auditory disturbance.

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Response to Fukunaga et al.

Fukunaga et al. found that the mutation at np 3243 in the mitochondrial $tRNA^{LEU(UUR)}$ gene was rare in Japanese nonselected type 2 diabetic patients with hearing loss. Although this point mutation is commonly present in patients with the syndrome of mitochondrial myopathy, encephalopathy, lacticacidosis, and stroke-like episodes (1,2), the presence of the 3243 mutation in diabetic patients with maternal inheritance and deafness was noted in a group of Dutch and Japanese populations (3,4). It is clear that no obvious relationship between the 3243 mutation and the onset or the type of diabetes is evident, as postulated by Maassen and Kadowaki (5). The low prevalence of the $tRNA^{LEU(UUR)}$ mutation is in keeping with some studies (6–8) where the mutation is likely to account for <1% of type 2 diabetes. As concerns the effect of the $tRNA^{LEU(UUR)}$ gene mutation on the nature of diabetes (i.e., type 2 diabetes), the situation is still less clear.

Although in our study our type 1 diabetic patients were not tested for the $tRNA^{LEU(UUR)}$ gene mutation, they underwent an audiological examination to exclude the presence of hearing loss before performing the evoked otoacoustic emissions. Thus, the impairment of cochlear receptor function found in some of our patients may indicate an early damage of peripheral nervous system that does not necessarily lead to deafness. Moreover, the finding of mitochondrial DNA mutation by using polymerase chain reaction techniques in peripheral leukocytes may not accurately reflect the number of mutated mitochondria in β-cells, which play an important role in pathogenesis of type 1 diabetes.

An interesting observation is that we showed that no mother of our type 1 diabetic patients with cochlear dysfunction was affected by diabetes. Two of our patients had diabetic fathers (type 1 diabetes in one case and type 2 in the other). We conclude that the $tRNA^{LEU(UUR)}$ mutation does not have a prominent place in the association with type 1 diabetes.

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Erratum

Tuomilehto J, Borch-Johnsen K, Molarius A, Jormanainen V, Lounamaa R, Grönhagen-Riska C, Reunanen A, Sarti C: The unchanging incidence of hospitalization for diabetic nephropathy in a population-based cohort of IDDM patients in Finland. *Diabetes Care* 20:1081–1086, 1997

In the above article, the wrong figure was printed as Fig. 3. The correct Fig. 3 and legend are provided below.

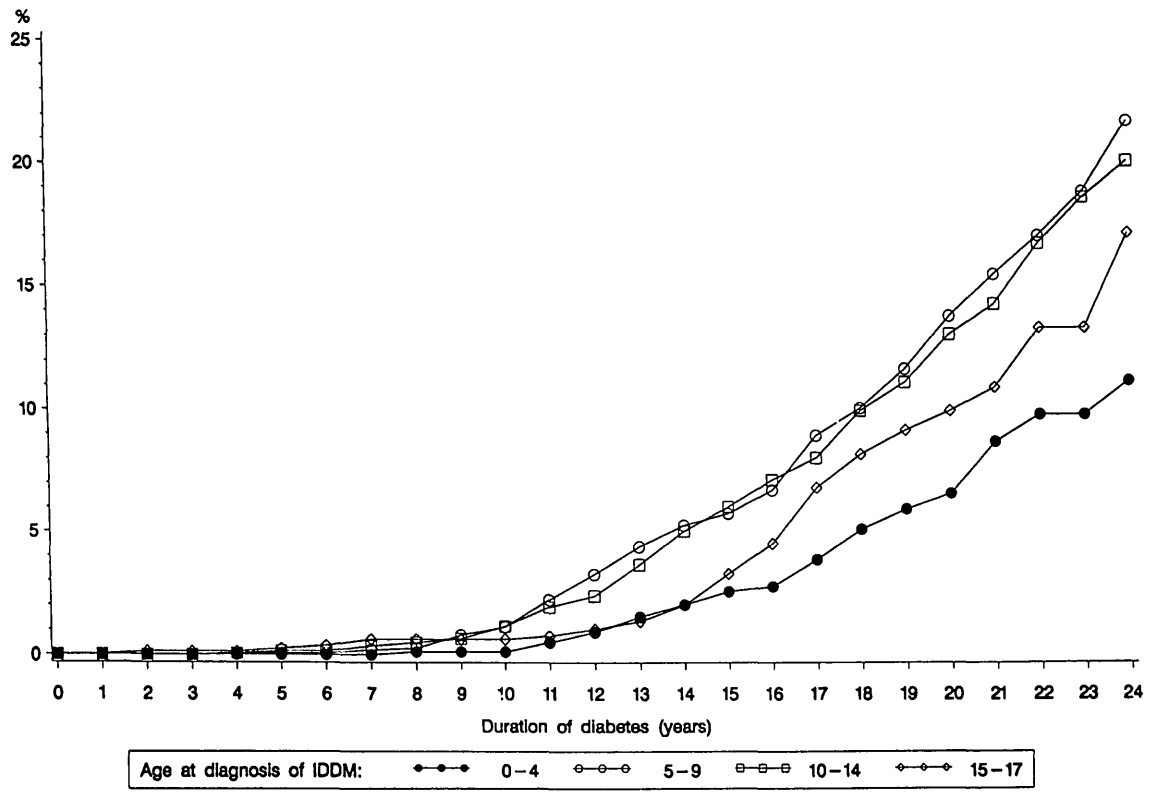

Figure 3—*Cumulative incidence of hospitalization for diabetic nephropathy according to age at onset of IDDM: ages 0–4, 5–9, 10–14, and 15–17 years.*