and clinically relevant mitochondrial mutations in the goal to define the phenotypic characteristics of the common and clinically relevant mitochondrial mutations in the MTTS1 and MTRNR1 genes. Verkerk et al correctly point out that our methodology did not include mitochondrial genomic sequencing in all subjects, which would limit detection of rare or novel mitochondrial variants for both nonsyndromic and syndromic hearing loss. Given the advances in next-generation sequencing technology, this would be an interesting follow-up project to pursue. Our analysis of existing repository clinical data did exclude syndromic causes, insofar as this is possible when examining a database that may contain only a single point in time at the ascertainment of the proband.

As we describe in the Discussion, both in existing literature as well as the current study, incomplete penetrance is well documented in both A1555G and A7445G mutations, with only around two-thirds of those carrying the mutation developing hearing loss. Lack of a maternal phenotype of hearing loss could be due to discrepant use of aminoglycosides, the cause of gene-environment interaction in the A1555G variant, or a lack of penetrance and variable phenotype. In general, the sporadic mitochondrial phenotype is restricted to lethal mitochondrial deletions. Many of our pedigrees also came from dense, deaf-by-deaf matings, and we did not have maternal DNA in several cases to confirm matrilineal inheritance. We agree that future research should examine the role of nuclear modifiers, additional mitochondrial or autosomal mutations, and the role of mitochondrial dysfunction in deafness.

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Reference


Possible Errors in the Article “Clinical Features of Recurrent or Persistent Benign Paroxysmal Positional Vertigo”

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I read with great interest the article written by Choi et al, “Clinical Features of Recurrent or Persistent Benign Paroxysmal Positional Vertigo.”¹ The authors demonstrated the incidence of persistent or recurrent benign paroxysmal positional vertigo (BPPV). However, there were potentially incorrect data in the article. In the Materials and Methods section of article, the authors stated, “Eventually, 120 patients were enrolled, and their follow-up was 6 months at minimum” in the last lines (lines 1-2) of the left column on page 920. In the Results section of the abstract, the authors described the following: “Among 120 patients with BPPV, 93 (77.5%) were typical, 15 (12.5%) were persistent, and 12 (10.0%) were recurrent.” The authors also showed the similar description: “Of 120 patients with BPPV, 93 (77.5%) were typical, 15 (12.5%) persistent, and 12 (10.0%) recurrent (Figure 1)” in the first paragraph of the Results section of text on page 921. However, the corresponding case numbers (percentages) accounting for 3 types of BPPV were shown as “Typical BPPV 98 (58.3%), Persistent BPPV 15 (8.9%), and Recurrent BPPV 12 (7.1%)” in “Figure 1. The incidence of persistent or recurrent benign paroxysmal positional vertigo” on page 921. It seems that the data in figure 1 is incorrect. Therefore, I recommend that the authors correct any possible errors in the article.