

Short communication

Normal audiogram but poor sensitivity to brief sounds in mice with

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tremors, and dystonic postures. Consequently, they dragged their hind limbs as they moved about (Sprunger et al., 1999).

The medJ mice and the phenotypically normal mice tested for comparison, were produced by crossing two congenic lines, C57BL/6J-med<sup>J</sup>/+ and C3HeB/FeJ-med<sup>J</sup>/+. They had one copy of the resistant Scnm allele, which they received from the C3HeB/FeJ parent (Buchner et al., 2003). Thus, the medJ mice were med<sup>J</sup>/med<sup>J</sup>, while the phenotypically normal mice (hereafter referred to as control mice) were either heterozygous for the med<sup>J</sup> mutation (med<sup>J</sup>/+), or homozygous wild types (+/+). In other words, all of the mice were from the same homogeneous F1 background and differed only with respect to the med<sup>J</sup> mutation. Three mice (animals A, B, and C) were homozygous medJ. Their detection thresholds for brief sounds were compared to those of two control mice (animals D and E) of the same F1 genetic background. All subjects were experienced in auditory tests, and the determination of thresholds for short-duration tones reported here was conducted immediately following their audiograms for longer duration sounds. Thus, these mice were young adults at the time of testing. The procedures were approved by the Animal Care and Use Committee of the University of Toledo.

## 2.2. Acoustic apparatus

Pure tones at 16 kHz, the frequency of best hearing for mice,

#### **4. Discussion**

- Psychoacoustics. Birkhauser, Basel, pp. 73–87.
- Heffner, H.E., Koay, G., Heffner, R.S., 2006. Behavioral assessment of hearing in mice – conditioned suppression. In: Crawley, J.N., Gerfen, C.R., Rogawski, M.A., Sibley, D.R., Skolnick, P., Wray, S. (Eds.), *Current Protocols in Neuroscience*, Suppl. 34. Wiley & Sons, New York, pp. 8.21D.1 - 8.21D.15.
- Kearney, J.A., Buchner, D.A., De Haan, G., Adamska, M., Levin, S.I., Furay, A.R., Albin, R.L., Jones, J.M., Montal, M., Stevens, M.J., Sprunger, L.K., Meisler, M.H., 2002. Molecular and pathological effects of a modifier gene on deficiency of the sodium channel *Scn8a* (Na(v)1.6). *Hum. Mol. Genet.* 11 (22), 2765–2775.