Reply to Corbeil *et al.*: Deletion of the transmembrane protein Prom1b in zebrafish disrupts outer-segment morphogenesis and causes photoreceptor degeneration

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We thank Corbeil *et al.* (1) for their interest in our work.

In our study, the gene ID of *prom1a* is 322857, and the reference sequence is NP_001108615.2. The gene ID of *prom1b* is 378834, and the reference sequence is NP_932337.1. The target sequence for *prom1a* knockout (see our paper (2), Fig. 1) exists in all known splice variants.

We too were intrigued by the lack of a phenotype in the prom1a knockout zebrafish. The simplest explanation is that the role of prom1a was compensated for by its homologue prom1b. However, we have no direct evidence to support this theory.

We agree that several valid points have been raised, which deserve future investigation, such as the subcellular localization of prom1b in photoreceptors. However, we agree even more with the view that it is more important for our paper to show "the functional relevance of prom1b deficiency on the visual system." That, in fact, is what we have shown in our paper (*i.e.* deletion of prom1b in zebrafish disrupts outer-segment morphogenesis and causes photoreceptor degeneration). In addition, deletion of prom1b prevents oligomerization and causes mislocalization of Prph2, which is an important protein for outer-segment morphogenesis.

The authors declare that they have no conflicts of interest with the contents of this article.

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References

1. Corbeil, D., Fargeas, C. A., and Jászai, J. (2019) Deciphering the roles of prominins in the visual system. *J. Biol. Chem.* **294,** 17166–17166 CrossRef
2. Lu, Z., Hu, X., Reilly, J., Jia, D., Liu, F., Yu, S., Liu, X., Xie, S., Qu, Z., Qin, Y., Huang, Y., Lv, Y., Li, J., Gao, P., Wong, F., Shu, X., Tang, Z., and Liu, M. (2019) Deletion of the transmembrane protein Prom1b in zebrafish disrupts outer-segment morphogenesis and causes photoreceptor degeneration. *J. Biol. Chem.* **294,** 13953–13963 CrossRef Medline