

**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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# 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.

## References

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

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