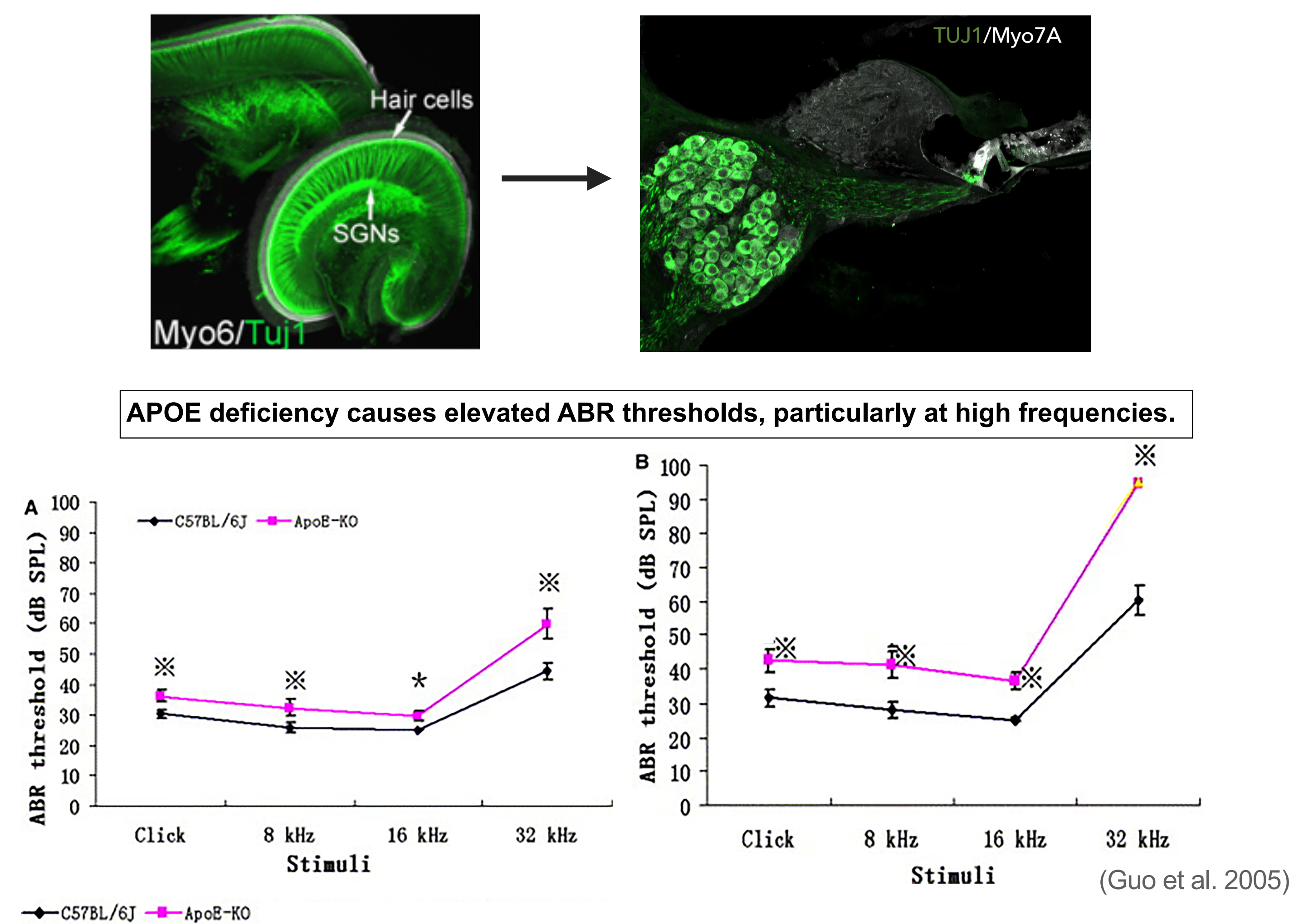


From Alzheimer's Disease to hearing loss: the role of APOE in SGN-hair cell connectivity

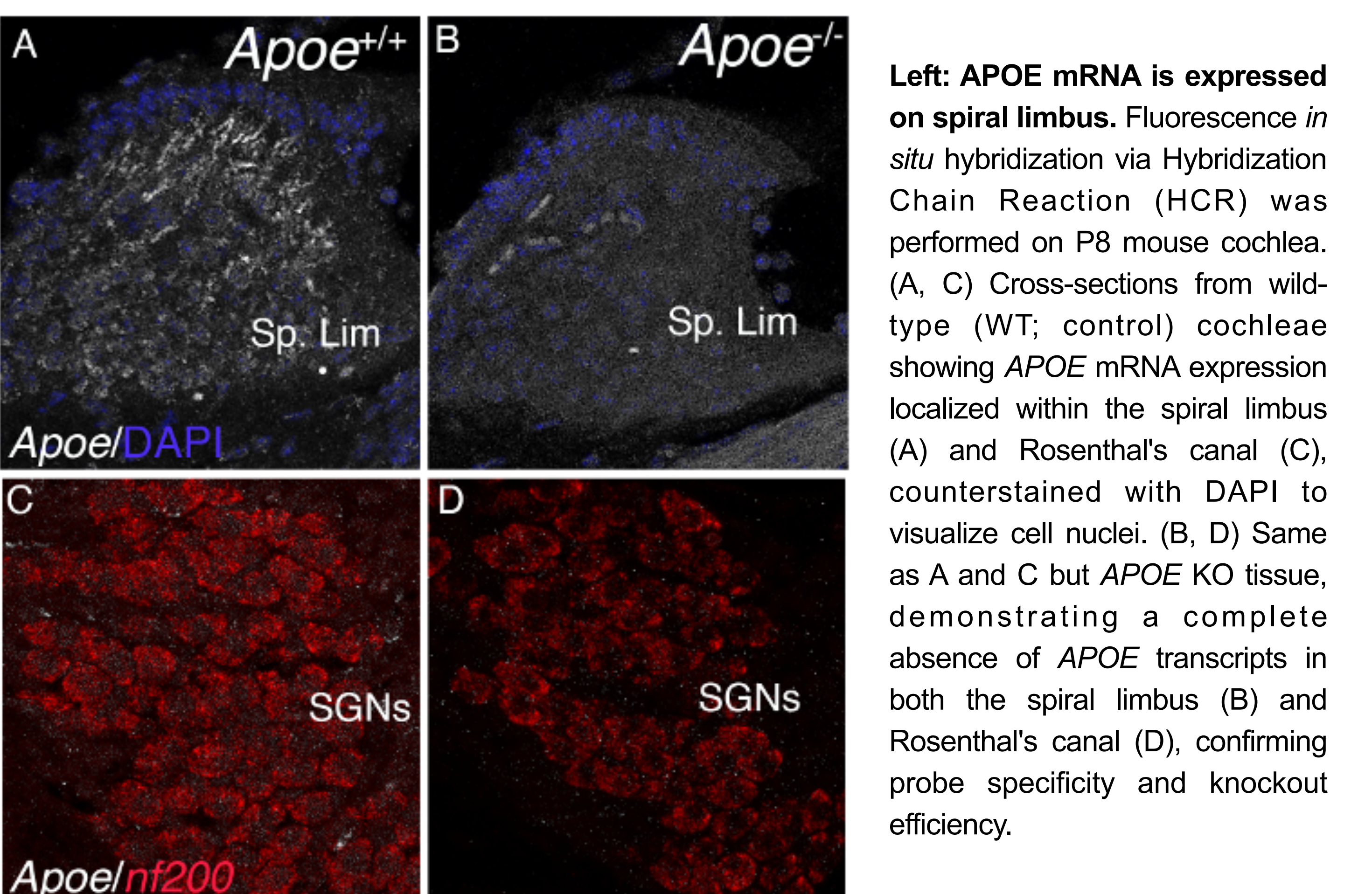
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Panel 1: Introduction

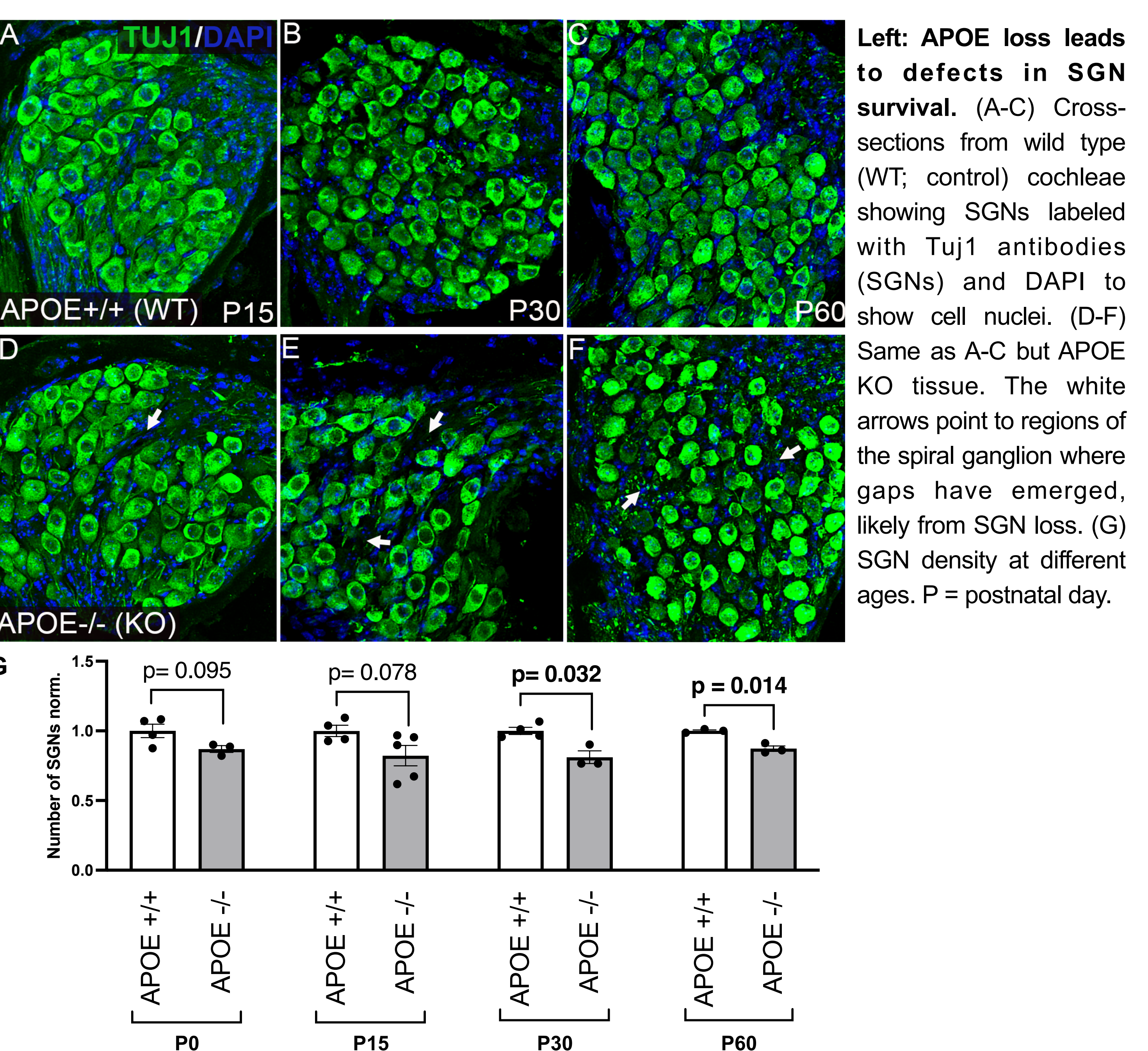


Panel 2: APOE mRNA expression on developing cochlea

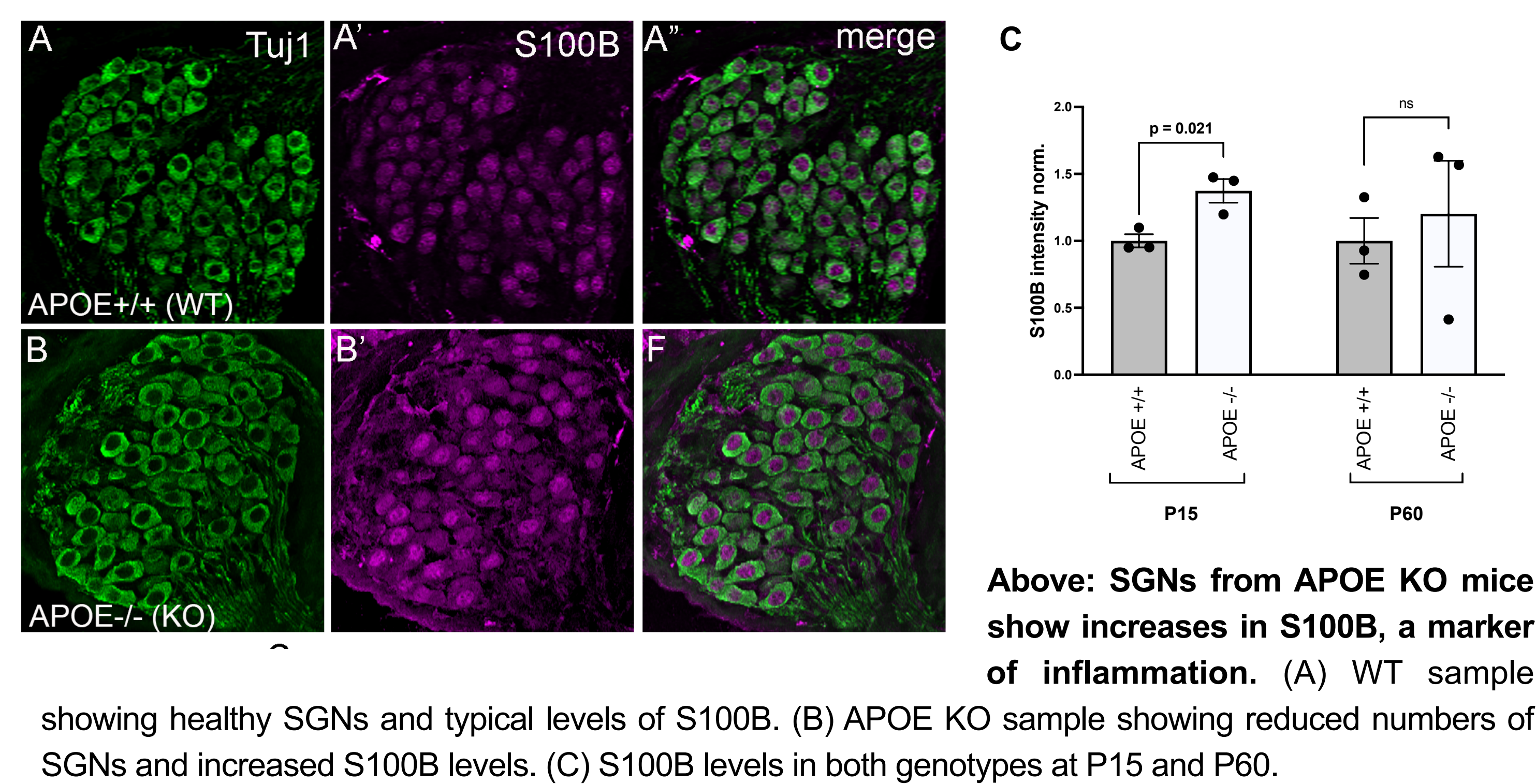


Representative confocal images demonstrate that APOE mRNA transcripts (shown in white) are robustly localized within the spiral limbus of the cochlea and also some were seen on Rosenthal's canala.

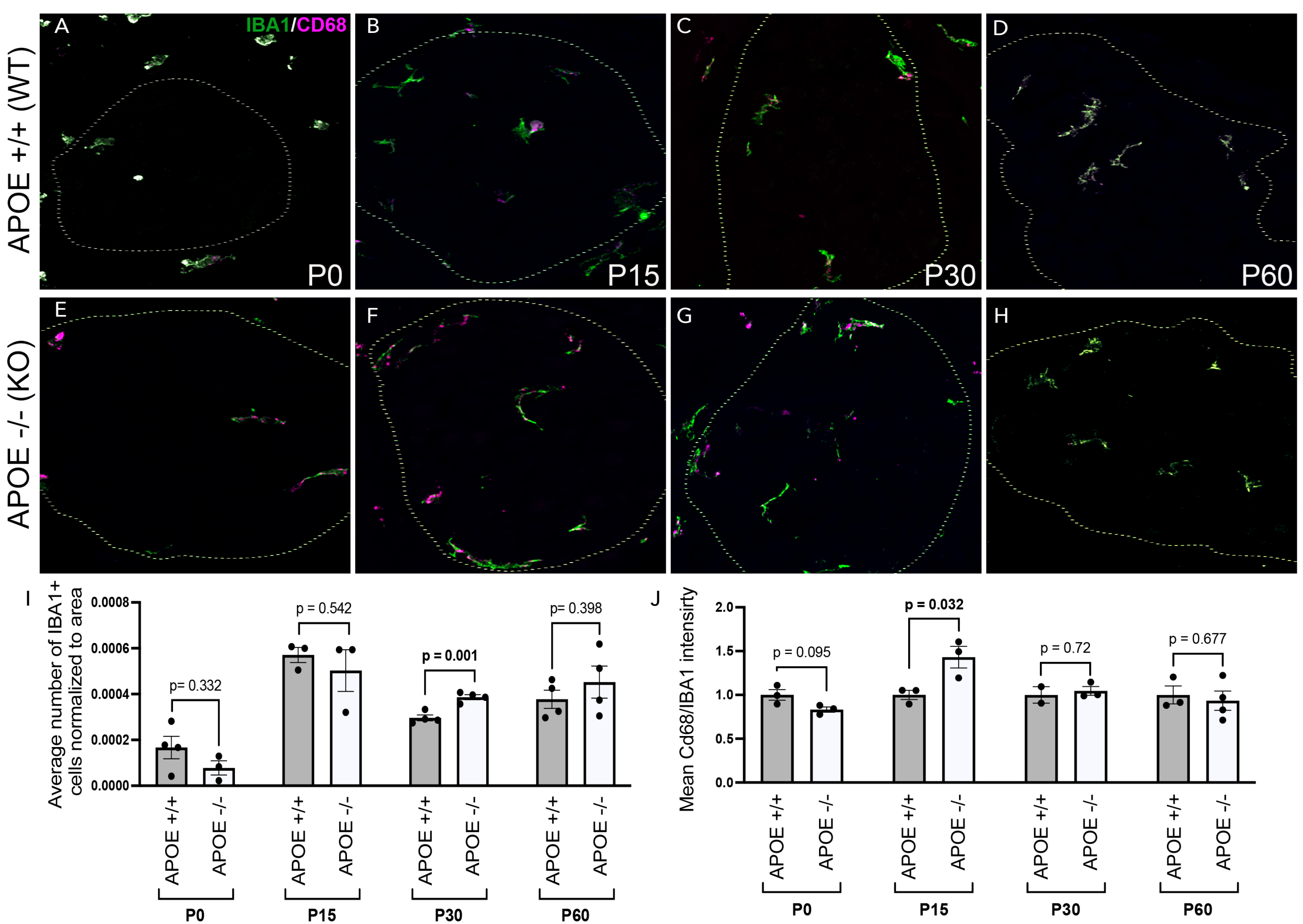
Panel 3: APOE is necessary for SGN survival



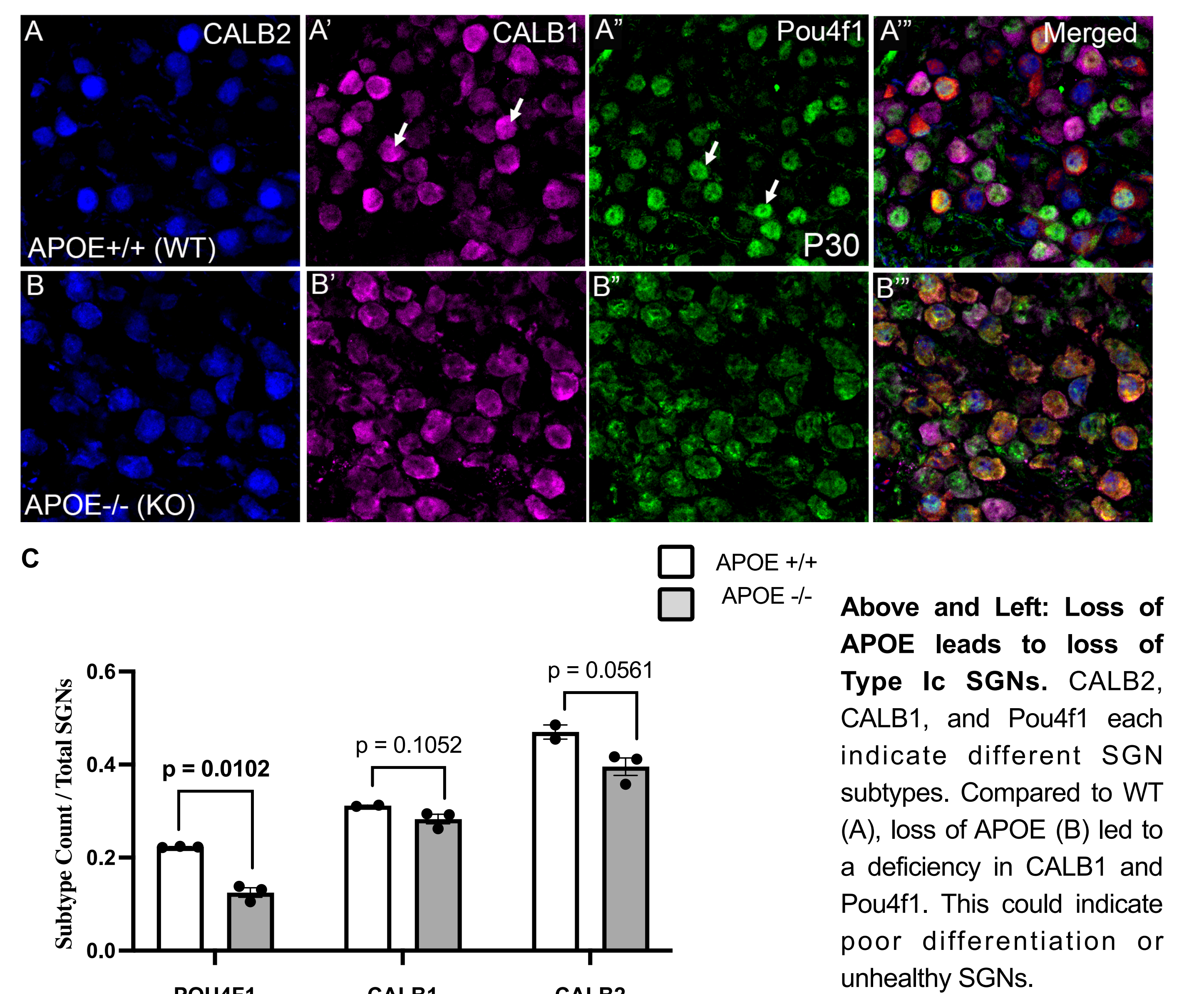
Panel 4: SGNs from APOE KO mice expresses heightened inflammation marker S100B



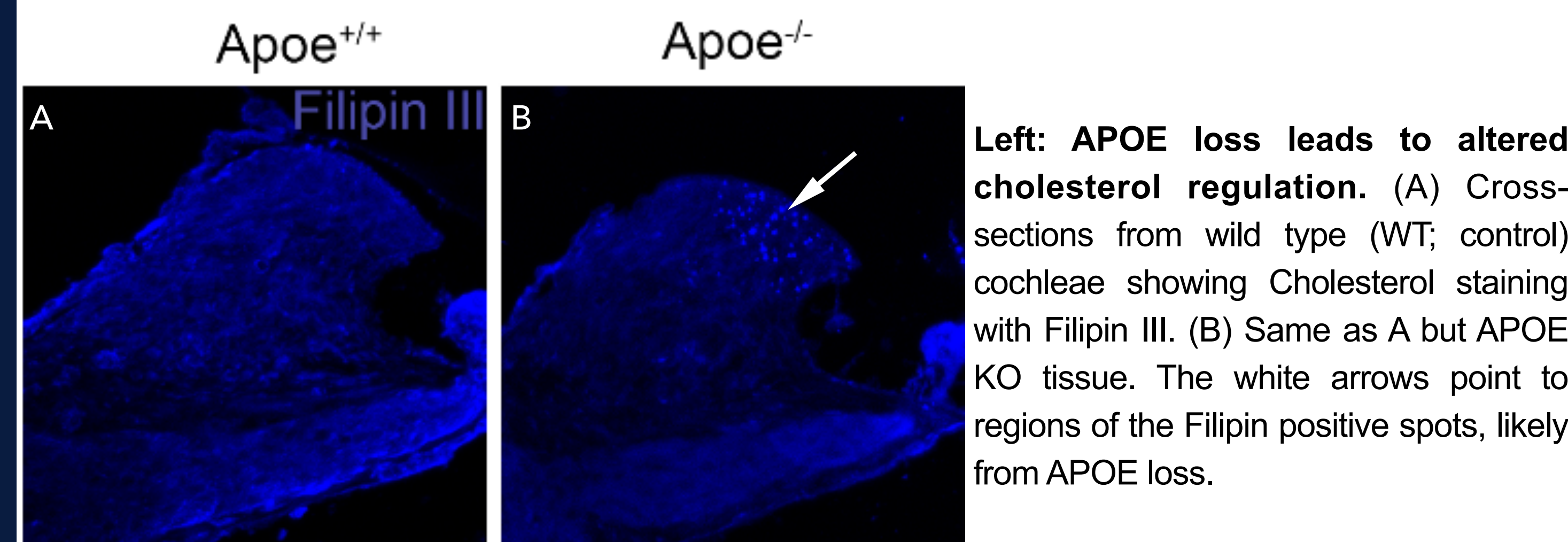
Panel 5: APOE Deficiency Enhances CD68 Signal and increases Number of IBA+ cells



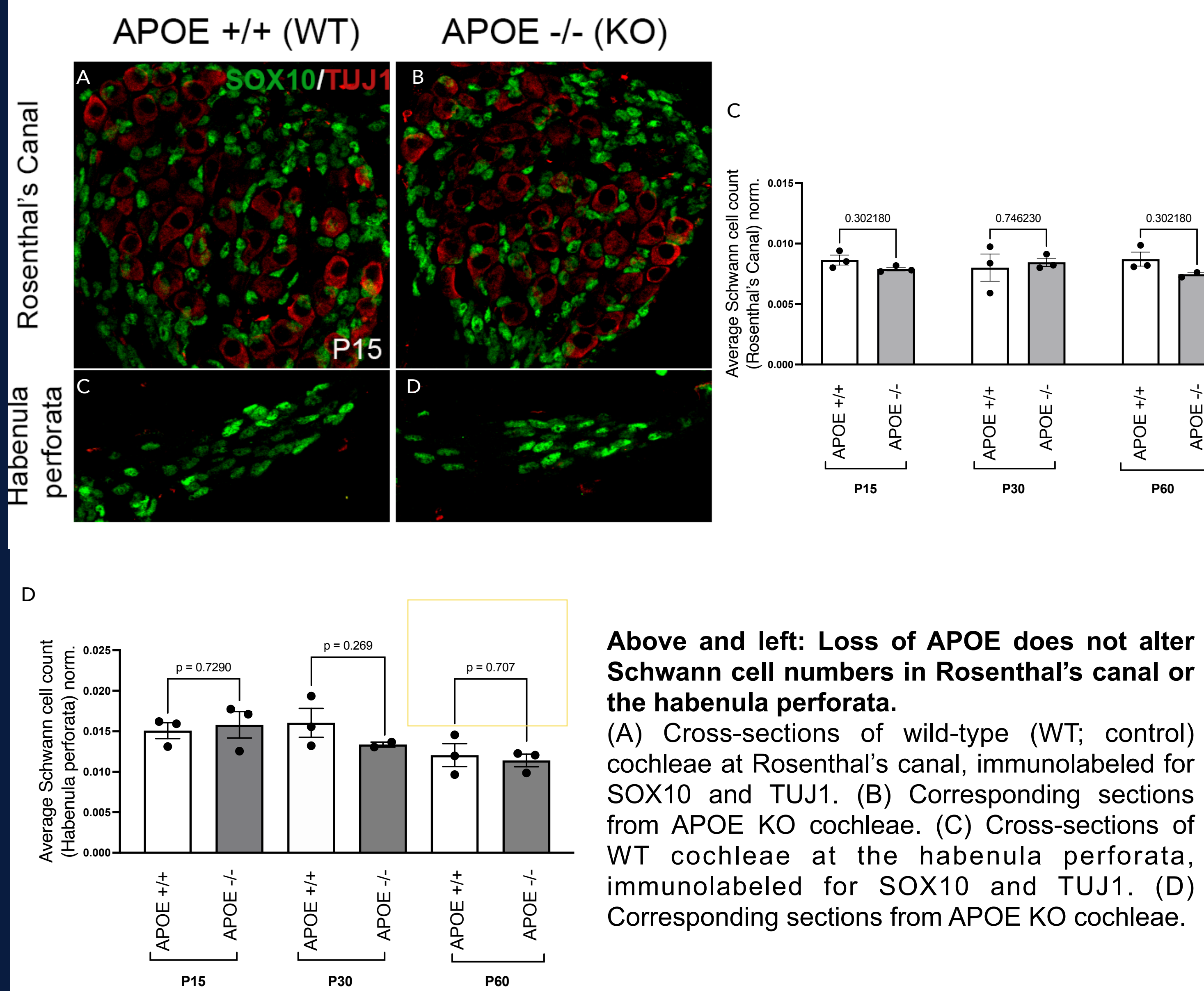
Panel 6: Type Ic SGNs are the most vulnerable



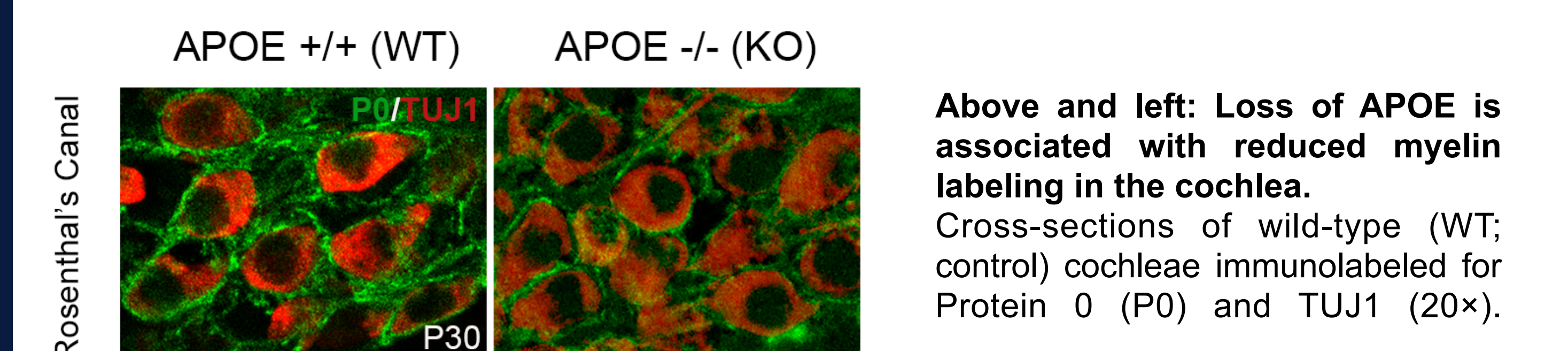
Panel 7: APOE KO mice shows altered cholesterol regulation in developing cochlea



Panel 8: APOE Deficiency doesn't effect Schwann cells numbers



Panel 9: APOEKO SGNs shows decreased myelination



Panel 8: Conclusion and future directions

APOE is a critical regulator of cochlear glial lipid and immune homeostasis that supports SGN survival. Disruption of APOE signaling promotes SGN vulnerability, providing a shared mechanistic link between hearing loss and Alzheimer's disease-related pathology.

APOE deficiency in cochlea → impaired lipid handling & immune regulation → SGN degeneration → Hearing loss

Future Directions

- **Noise Susceptibility:** Perform hearing assessments (ABR/DPOAE) in APOE KO mice noise exposure to evaluate vulnerability to acoustic trauma.
- **Myelination Dynamics:** Analyze cochlear glial myelination timepoints during development.
- **Therapeutic Targets:** Determine if **immune modulation** can prevent SGN degeneration and preserve survival.

References

- Guo, Y., Zhang, C., Du, X., Nair, U., & Yoo, T. J. (2005). Morphological and functional alterations of the cochlea in apolipoprotein E gene deficient mice. *Hearing research*, 208(1-2), 54-67.