

Figure S1. OCT data from sham and injured $M-HDAC3^{-/-}$ and $HDAC3^{fl/fl}$ mice. Optical coherence tomography (OCT) performed on anesthetized mice at 7 (**A, B**) and 14 days (**C, D**) post-ONC shows no improvement in ganglion cell complex (GCC) layer thickness in $M-HDAC3^{-/-}$ retinas compared to control $HDAC3^{fl/fl}$. n= 9-10 per group.

Uninjured sham groups

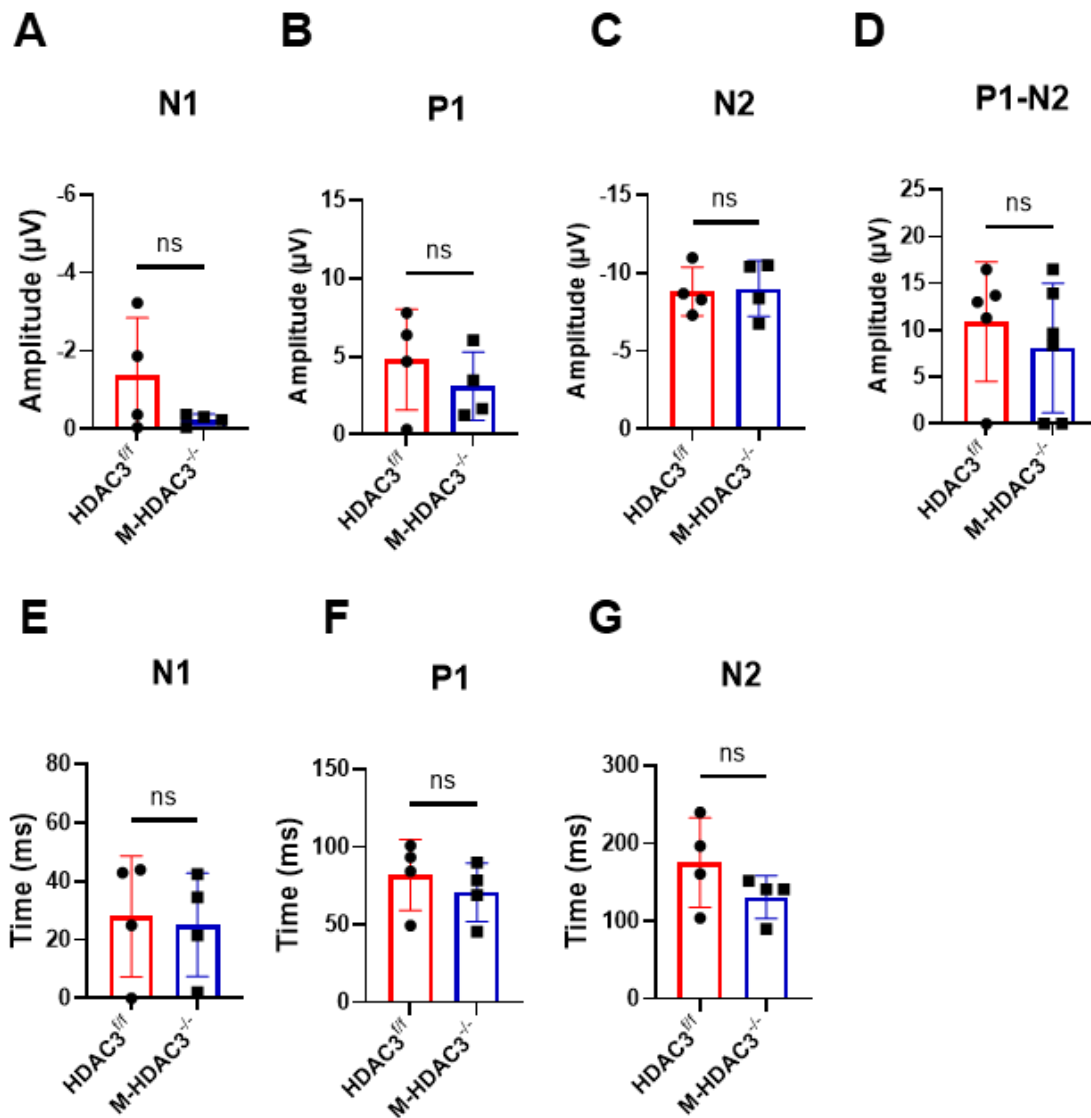


Figure S2. PERG data from M-HDAC3^{-/-} and HDAC3^{ff} sham mice. Quantification of N1, P1, and N2 waveforms from PERG conducted on HDAC3^{ff} and M-HDAC3^{-/-} sham mice demonstrates no significant impact of myeloid HDAC3 deletion on the amplitude of N1, P1, and N2 (A-D), nor on their latencies (E-G).

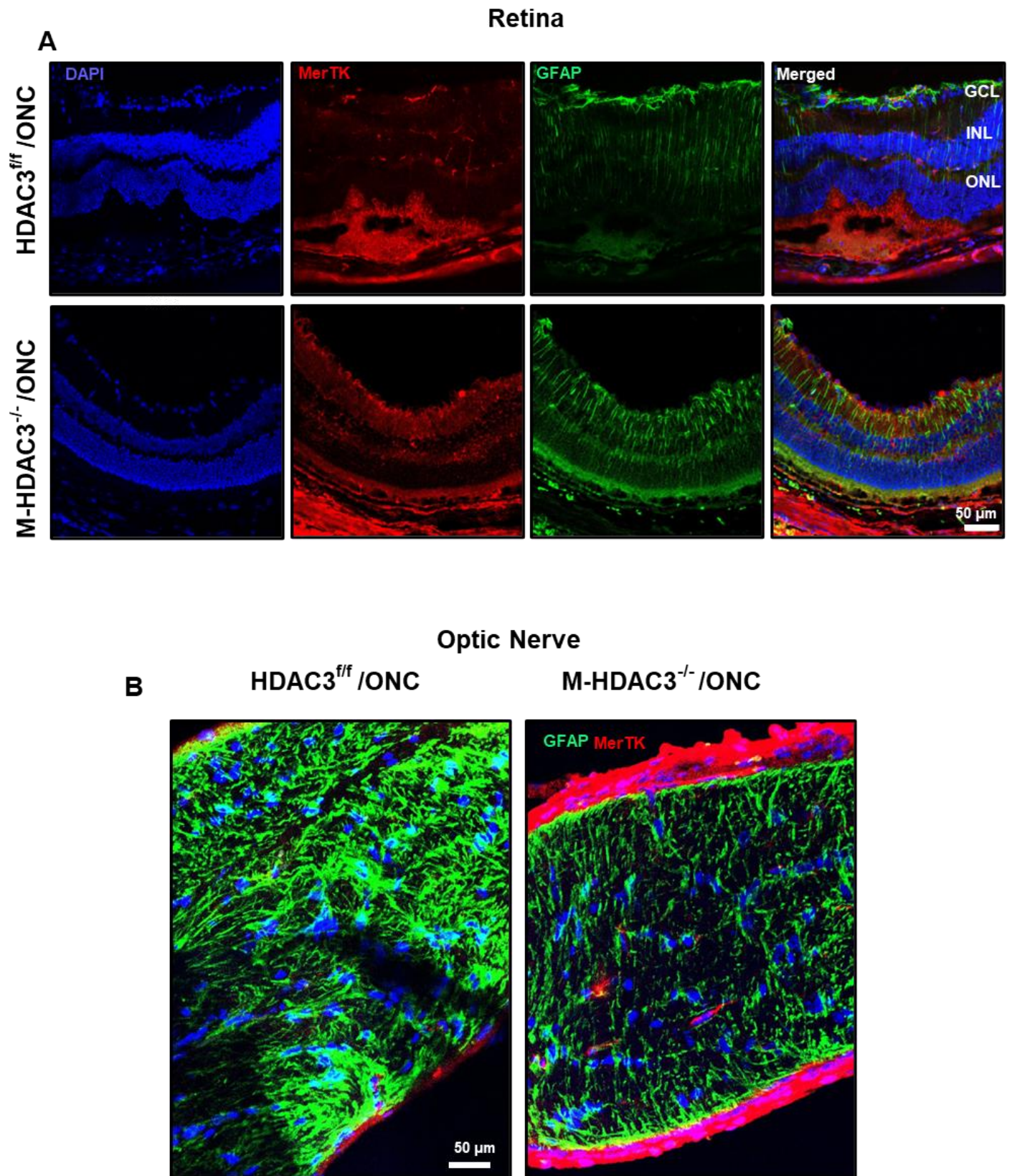


Figure S3. MerTK expression in glial cells following ONC injury. (A) Representative confocal images of glial cells expressing GFAP (green) in the retinas of HDAC3^{f/f} and M-HDAC3^{-/-} mice with minimal colocalization with

MerTK (red) at day 5 post-ONC. **(B)** Similarly, in injured optic nerve sections, GFAP and MerTK colocalization were minimal in M-HDAC3^{-/-} and HDAC3^{fl/fl} control mice at day 5 post-ONC.

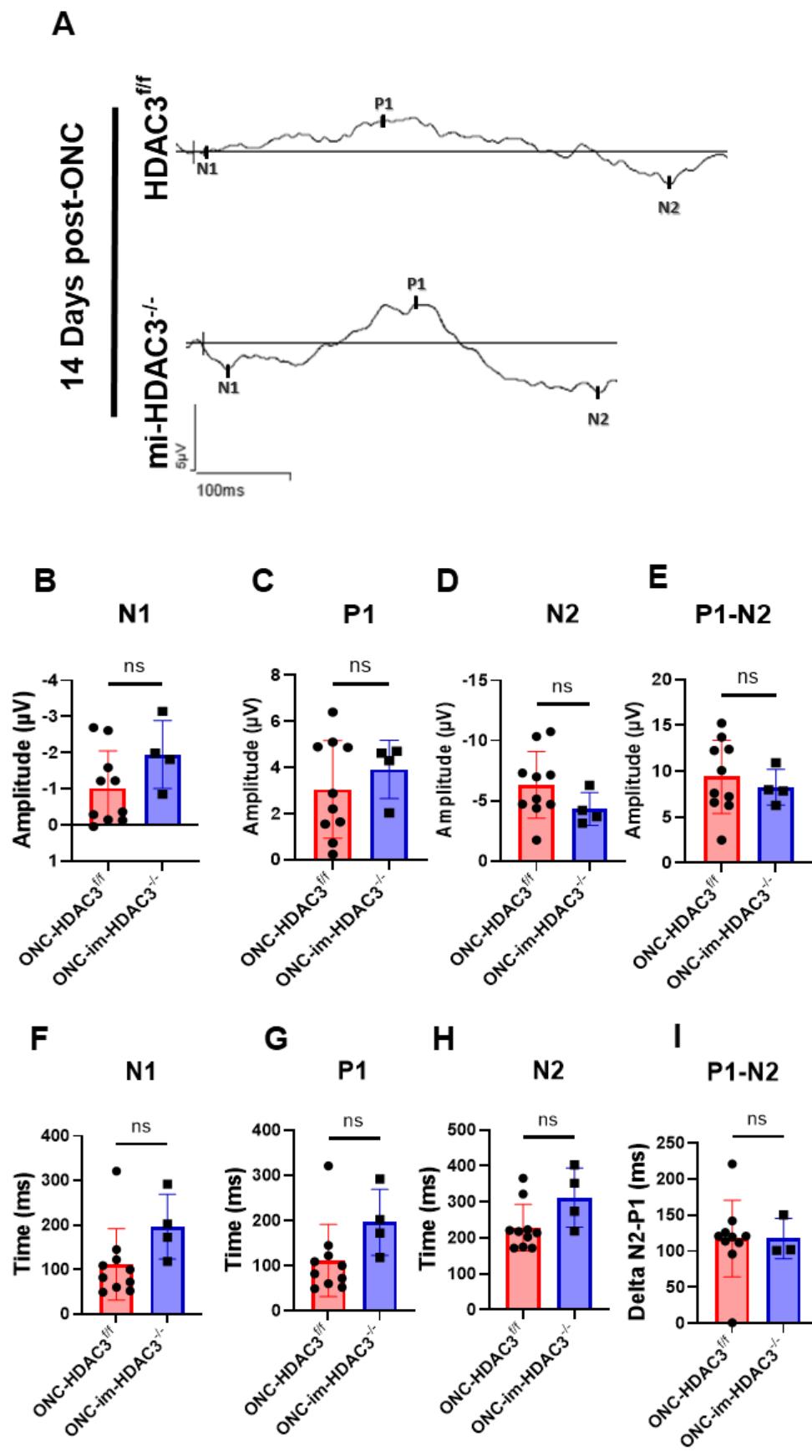


Figure S4. PERG data from sham and injured im-HDAC3^{-/-} and HDAC3^{fl/fl} mice. (A) Representative of N1, P1, and N2 waveforms in the retinas of microglia-specific HDAC3 KO (im-HDAC3^{-/-}) and HDAC3^{fl/fl} mice 14 days post-ONC. Quantification of N1, P1, and N2 waveforms demonstrates no significant impact of microglia-only HDAC3 deletion on the amplitude of N1, P1, and N2 **(B-E)**, nor their latencies **(F-I)**.

Uncropped Western blots

F6

