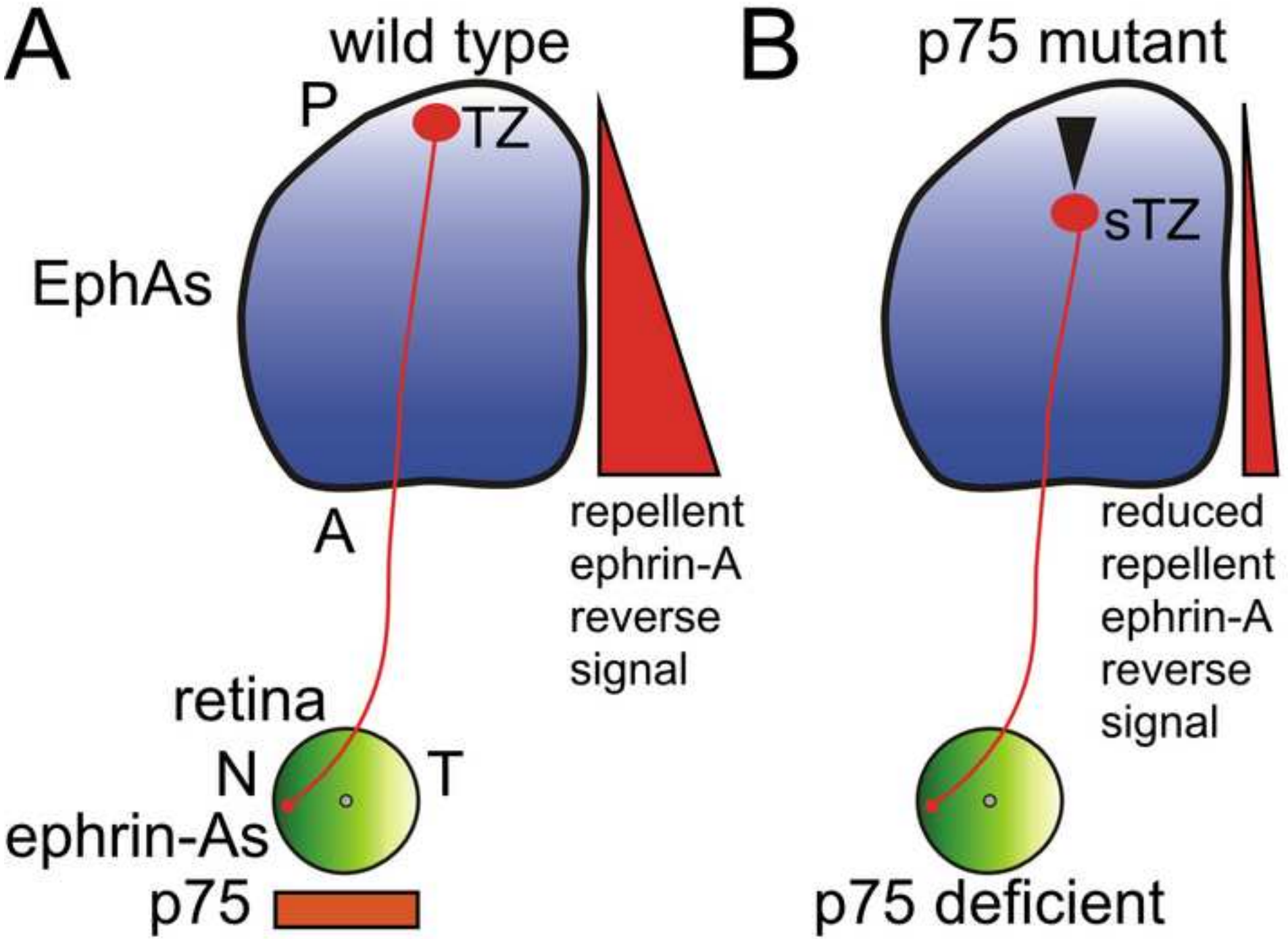
Supplemental Figure 1. Summary of the Retinocollicular Mapping Defects in p75 Mutant Mice.

(A) In wild type mice, ephrin-As expressed in the retina (green gradient) and along retinal ganglion cell (RGC) axons interact with EphAs (blue gradient) in the superior colliculus (SC). In addition, p75 is expressed in the retina (orange) and along RGC axons and acts as an ephrin-A signaling partner. Therefore, p75 complexes with ephrin-As along RGC axons and, upon binding EphAs in the SC, transduces a repellent ephrin-A reverse signal (red gradient) that parallels the anterior (A) - posterior (P) EphA gradient in the SC. Thus, nasal (N) RGC axons, expressing high levels of ephrin-As, form a termination zone (TZ) in posterior SC, which expresses low levels of EphAs. **(B)** In p75 mutant mice an ephrin-A signaling partner is lacking from the retina. Thus, the repellent ephrin-A reverse signal is reduced (diminished red gradient) allowing nasal RGC axons to form anteriorly shifted TZs (sTZ). The expression patterns of ephrin-As and EphAs are unchanged in p75 mutant mice. Therefore, the formation of sTZs in areas of high EphA expression is due to the loss of the ephrin-A signaling partner, p75, and the concomitant reduction in repellent ephrin-A reverse signal.


A
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