Fibroblast growth factors (FGFs) are critical in many aspects of embryonic development. FGF8a, specifically, is known to initiate retinal ganglion cell (RGC) differentiation along with FGF3 at 28 hours post fertilization hpf in zebrafish (Martinez-Morales et al., 2005b). Here we show mRNA expression of fgf8a in the presumptive RGCs of 2 day-old zebrafish, past the time of RGC differentiation (28-48 hours) (Schmitt and Dowling, 1996). In addition, mRNA expression of putative receptor, FGFR1b, was localized outside the retina on the presumptive vasculature. Acerebellar (ace) mutants lacking FGF8a show mispatterned retinal vasculature and a lack of blood flow through the eye at 48 hpf. We found a reduction in the size of ace mutant eyes and also a reduction in total cell numbers in the retina starting at 48 (hpf) suggesting a role for fgf8a in neurovascular signaling. The cause of the small eye phenotype was found to be due to a lack of proliferating cells. We questioned if this phenotype was a result of a lack of blood flow to the retina. To investigate the role that blood flow plays on the developing retina we utilized a silent heart mutant (sih), which develop without a beating heart. Retina cell counts show a decreased eye diameter and a loss in total retina cell numbers due to lack of proliferation, phenocopying ace mutants. sih mutants also show a mispattering of their retinal vasculature with ectopic vessel branches. After morpholino knock down of the receptor, fgfr1b, we see mispatterend vasculature that phenocopies what we see in ace mutants. These finding led us to hypothesize that FGF8a, secreted by the RGCs, signals through its receptor, FGFR1b, on the retinal vasculature to promote proper patterning. Further, the retinal vasculature subsequently responds by secreting an unknown factor to support the proliferation and maintenance of the RGCs.