Supplementary Material

Ayumi Miyake and Nobuyuki Itoh doi: 10.1242/bio.20134226

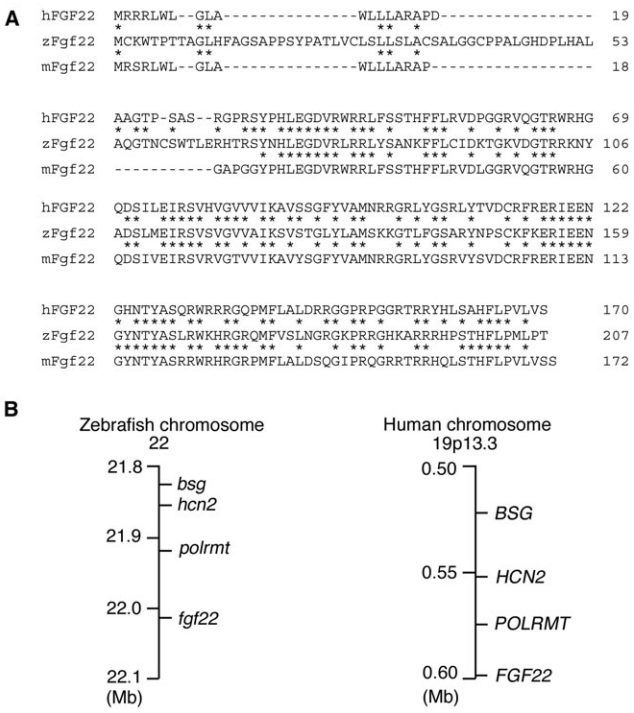

Fig. S1. Molecular analysis of zebrafish Fgf22. (**A**) The amino acid sequence of zebrafish Fgf22 (zFgf22) aligned with human FGF22 (hFGF22) and mouse Fgf22 (mFgf22). The numbers refer to the amino acid positions of zebrafish Fgf22, human FGF22, and mouse Fgf22. Asterisks indicate identical amino acid residues in the sequences. Dashes indicate introduced gaps. (**B**) Syntenic relationship between zebrafish chromosome 22 and human chromosome 19 at p13.3. Both the zebrafish *fgf22* and human *FGF22* genes are closely linked to the zebrafish and human *bsg*, *hcn2*, and *polrmt* genes. Mb, megabase.

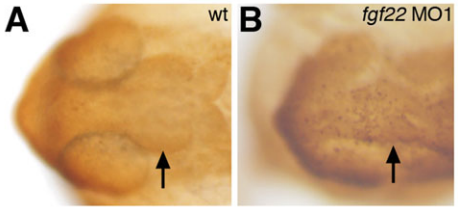

Fig. S2. Apoptosis in the midbrain of *fgf22* morphants. At 24 hpf, apoptotic cells in the midbrain of the wild-type (**A**) and *fgf22* MO1-injected (**B**) embryos were marked via TUNEL. Dorsal view with anterior to the left.

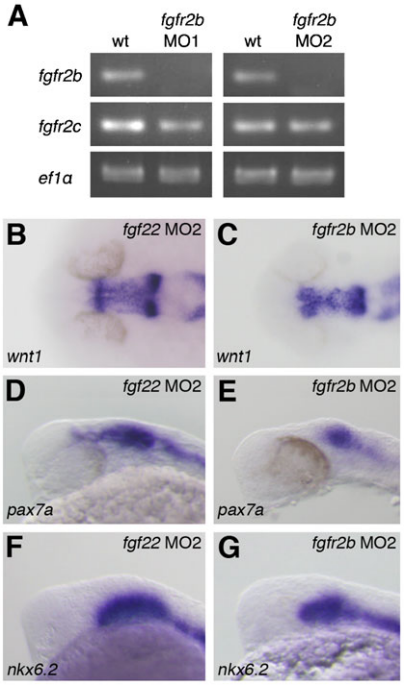

Fig. S3. Inhibition of *fgf22* and *fgfr2b* functions in zebrafish embryos. (**A**) The cDNA of *fgfr2b* and *fgfr2c* was amplified from wild-type or *Fgfr2b* MO-injected embryonic cDNA by RT-PCR using forward and reverse primers. *ef1α* cDNA was also amplified as a control. (**B–G**) The expression of *wnt1* (B,C), *pax7a* (D,E), and *nkx6.2* (F,G) at 24 hpf in *fgf22* MO2-injected (B,D,F), and *fgfr2b* MO2-injected (C,E,G) embryos.

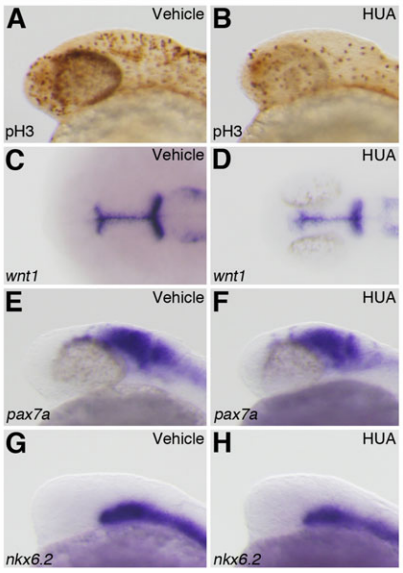

Fig. S4. Cellular proliferation does not contribute to the reduction of dorsal midbrain. (**A**,**B**) Wild-type embryos treated with 4% DMSO (A) or HUA (B) were stained using an anti-pH3 antibody. (**C–H**) The expression of *wnt1* (C,D), *pax7a* (E,F), and *nkx6.2* (G,H) at 24 hpf in wild-type embryos treated with 4% DMSO (C,E,G) or HUA (D,F,H).