

Supplementary Figures

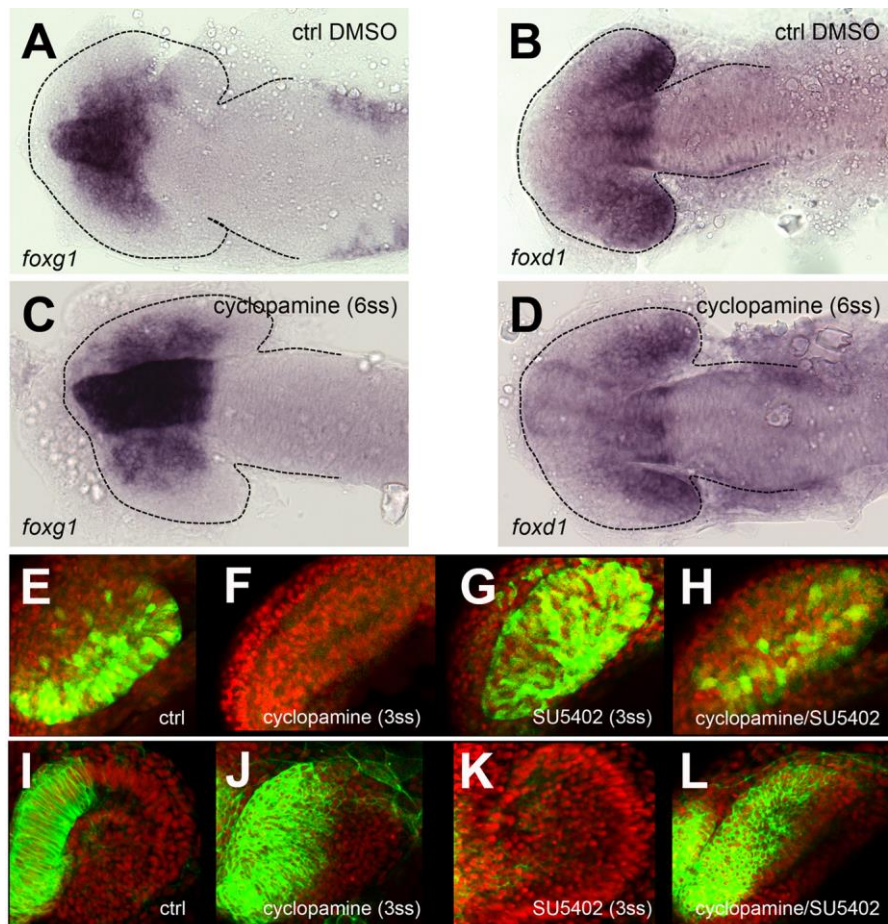


Figure S1: Hh is required for temporal specification only before 6ss.

foxg1 (A,C) and *foxd1* (B,D) expression is normal in embryos treated with cycloamine from 6ss. (E-L) Images showing effects of cycloamine and SU5402 upon transgenes expressed in nasal (*Tg{CldnB::GFP}*) and temporal (*Tg{HGn42a::GFP}*) domains. (A-D) are dorsal views with anterior to the left; (E-L) are lateral views of the eye with anterior to the left.

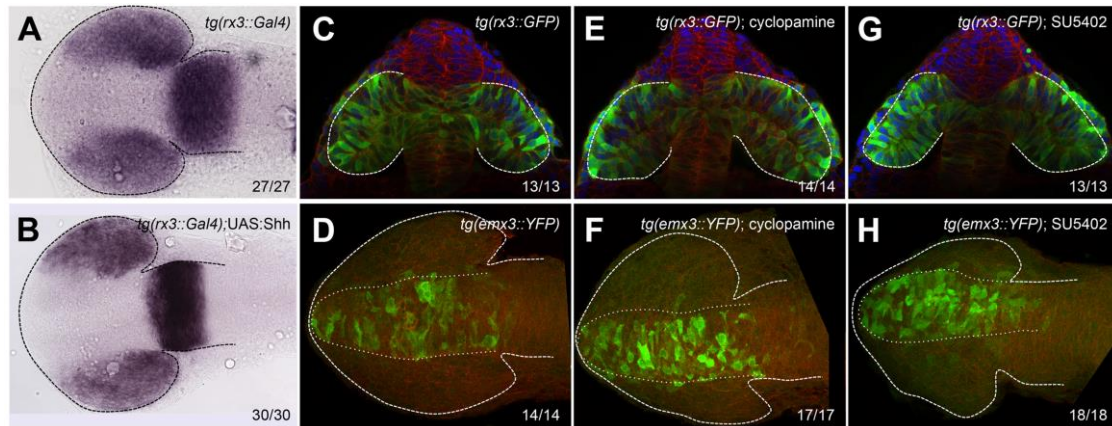


Figure S2: Primary forebrain subdivision is unaffected upon manipulation of Hh and Fgf signals in the eye field.

(A-B) dorsal views with anterior to the left showing *mab21/2* expression in *Tg{rx3::Gal4}* (A) and *Tg{rx3::Gal4};UAS:Shh* (B) eyes and brains. (C-H) Frontal (C,E,G) and dorsal (D,F,H) views of brains and eyes following drug treatments in *Tg{rx3::GFP}* and *Tg{emx3::YFP}*. In all conditions there are no changes in primary forebrain subdivisions. All embryos are 12ss.

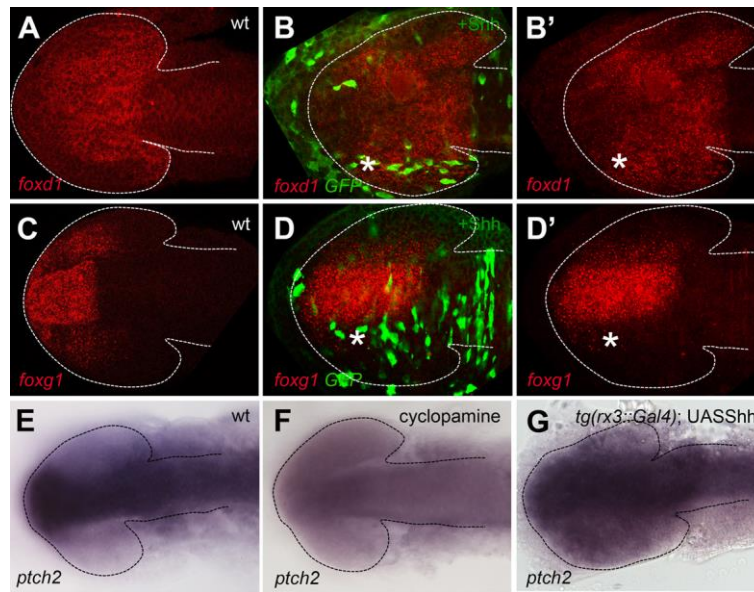


Figure S3: Mosaic overexpression of Hh activity in the optic vesicle promotes temporal fate.

(A-D') Mosaic overexpression of Shh reproduces the phenotypes observed by using the Gal4/UAS system (compare to main figure 2). (E-G) Images of brains and eyes showing expression of the Shh target *ptch2* is lost in a cyclopamine treated embryo (H) and overexpressed in a *tg(rx3::Gal4);UAS:shh* embryo, consistent with the expected alterations to Hh pathway activity under these conditions.

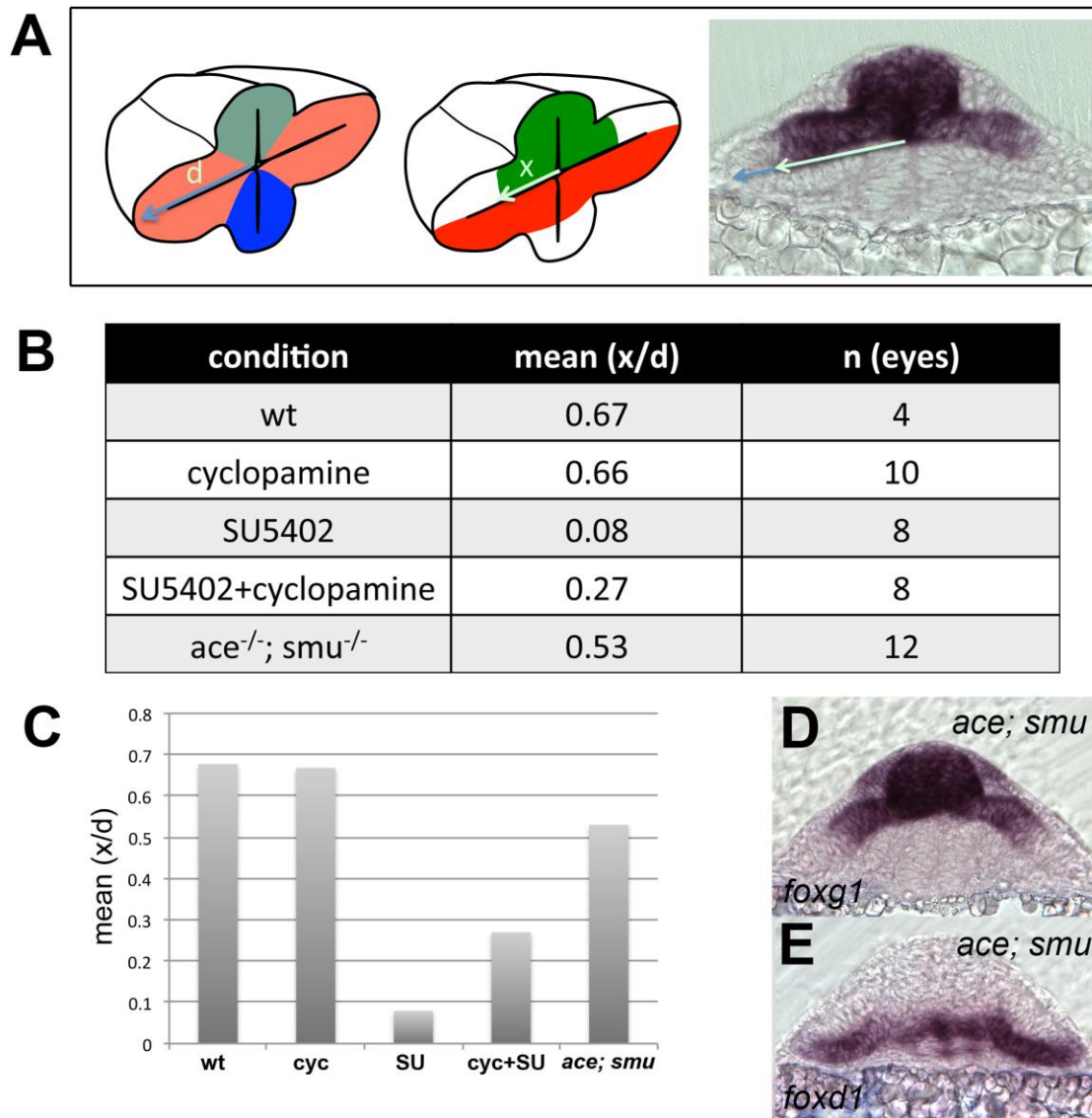


Figure S4: Quantification of extension of *foxg1* expression in all the experimental conditions presented in the study.

(A) Rationale of the quantification strategy. Dividing x by d normalises the extension of *foxg1* expression to the total length of the optic vesicle. (B) Table showing the mean of x/d for the number of eyes (n) quantified. (C) Graph representing the results from (B).