Table S1: Overexpression of EGFR-Ras and PI3K-Akt pathways in Drosophila glia

Gene (UAS construct)	repo-Gal4 glial overexpression phenotypes
$dEGFR^{\lambda}$	glial hyperplasia, CNS morphogenesis defects, lethal
$dEGFR^{Elp}$	
$dEGFR^{wild-type}$	
$dRas85D^{V12}$	
$dRaf^{gof}$	
$dp110^{CAAX}$	normal glial cell numbers-slight glial hyperplasia,
$dp110^{wild-type}$	viable
$dPTEN^{dsRNA}$	
dAkt	
$dEGFR^{\lambda}$; $dp110^{CAAX}$	glial neoplasia, neuronal loss, behavior defects,
$dEGFR^{\lambda}$; $dAkt$	CNS morphogenesis defects including enlarged brain
$dRas85D^{V12}$; $dp110^{CAAX}$	lobes and elongated ventral nerve cord, early lethality
$dRas85D^{V12}$; $dPTEN^{dsRNA}$	or prolonged larval phase before pupal lethality
$dRas85D^{V12}$; $dPTEN^{+/-}$	
$dRas85D^{V12}$; $dAkt$	
$dRaf^{sof}$; $dp110^{CAAX}$	
$dEGFR^{\lambda}$; $dp110^{CAAX}$; $dRas85D^{NI7}$	Suppression of neoplasia and other phenotypes,
	sometimes viable
$dEGFR^{\lambda}$; $dp110^{CAAX}$; $dPTEN^{wild-type}$	Suppression of neoplasia and other phenotypes, lethal

 $^{3^{\}rm rd}$ instar larval brains for each genotype were scored for glial cell numbers, morphology, and position, when possible. $1^{\rm st}$ and $2^{\rm nd}$ instar brains were scored for genotypes that showed earlier lethality, including $dEGFR^{E/p}$ and $dRas85D^{V12}$; dAkt.