Supplementary Figure 1

(a) Dissociated cells from embryonic E14 cortex or ganglionic eminence (GE) were infected with viral vectors indicated at the x-axis at the day of plating and their progeny analyzed 7 days later\(^2\). This is a well-established in vitro system for the analysis of telencephalic precursor fate, where the effect of Pax6 is particularly well characterized\(^25,26\). Previous work had shown that Pax6 overexpression promotes neurogenesis and reduces clone size, i.e. the number of descendants generated by a single precursor cell, while the loss of functional Pax6 in Small Eye mutant mice (Pax6\(^{16flox/flox}\)) resulted in a significant reduction of GFAP– and DCX–negative supposedly transit–amplifying precursors (GFP: 22%, \(n = 183\), 4 hemispheres, 3 animals; Olig2\(^bHLH\): 8%, \(n = 90\), 3 hemispheres, 3 animals; \(P < 0.02\) compared to control). Thus, the Pax6\(^{engrailed}\) is as sufficient as the loss of functional Pax6 in vivo by other means than the dominant–negative construct, Pax6\(^{engrailed}\) containing virus resulted also in a decrease of neurogenesis that was comparable to the effect observed in a significant reduction of GFAP– and DCX–negative supposedly transit–amplifying precursors (GFP: 22%, \(n = 183\), 4 hemispheres, 3 animals; Olig2\(^bHLH\): 8%, \(n = 90\), 3 hemispheres, 3 animals; \(P < 0.02\) compared to control, Olig2\(^{2VP16}\): 2.5%, \(n = 148\), 3 hemispheres, 3 animals, \(P < 0.05\) compared to control) comparable to the effect observed with the dominant–negative Olig2\(^{2VP16}\) viral construct.