Dlx5 and Dlx6 regulate the development of parvalbumin-expressing cortical interneurons.

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Public Summary:
Dlx5 and Dlx6 homeobox genes are expressed in developing and mature cortical interneurons. Simultaneous deletion of Dlx5 and 6 results in exencephaly of the anterior brain; despite this defect, prenatal basal ganglia differentiation appeared largely intact, while tangential migration of Lhx6(+) and Mafb(+) interneurons to the cortex was reduced and disordered. The migration deficits were associated with reduced CXCR4 expression. Transplantation of mutant immature interneurons into a wild-type brain demonstrated that loss of either Dlx5 or Dlx5&6 preferentially reduced the number of mature parvalbumin(+) interneurons; those parvalbumin(+) interneurons that were present had increased dendritic branching. Dlx5/6(+/-) mice, which appear normal histologically, show spontaneous electrographic seizures and reduced power of gamma oscillations. Thus, Dlx5&6 appeared to be required for development and function of somal innervating (parvalbumin(+)) neocortical interneurons. This contrasts with Dlx1, whose function is required for dendrite innervating (calretinin(+), somatostatin(+), and neuropeptide Y(+)) interneurons (Cobos et al., 2005).

Scientific Abstract:
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