

KCC2 (a brain-specific potassium-chloride cotransporter) affects development of the cerebral cortex, including aspects of neuronal migration and cellular maturation and differentiation. KCC2 also modulates chloride homeostasis by influencing the switch of GABA from depolarizing in young neurons to hyperpolarizing in mature neurons. We describe the expression pattern, regional distribution, and cellular colocalization of KCC2 in the ferret cortex in normal kits and those treated with methylazoxymethanol acetate (MAM). We earlier developed a model of impaired cortical development by injecting MAM during mid-cortical gestation, which briefly interferes with neuronal production and additionally results in increased levels of KCC2 at P0. Using immunohistochemistry, we show a shift in KCC2 expression during development, being high in the subplate at P0, repositioning into a subtle laminar pattern in the neocortex at P7–P14, and becoming homogeneous at P35. KCC2 colocalizes with neuronal markers in the developing and mature cerebral cortex of normal ferrets and those treated with MAM, but shows a differential pattern of expression at different ages and locates in distinct cellular compartments during development. Subcellular localization shows that KCC2 predominantly situates in the membrane fraction of neocortical samples. These findings reveal that KCC2 colocalizes differentially with neurons and its expression pattern alters during development