1. The SCT gene was specifically knocked out in Purkinje cells. 2. General motor functions and normal anxiety levels were intact in SCT knockout mice. 3. SCT facilitated pre-synaptic GABA release in Purkinje cells. 4. Pur-Sct−/− juveniles might have delayed cerebellar development and lead to early onset motor neuron degeneration. 5. SCT facilitated pre-synaptic GABA release in Purkinje cells. 6. SCT deficiency did not exert any overt abnormalities of the global cerebellar anatomy but exhibited reduced EGL thickness during postnatal period. 7. The reduced EGL thickness in Sct−/− mice was due to the decrease in the number of granular cell progenitors and post-mitotic granular cells. 8. The decrease in progenitors and post-mitotic cells was secondary to 1) a premature exit of the cell cycle and migration into IGL and 2) Increased apoptosis of granular cells. 9. Purkinje cell density was reduced since postnatal days.