Smad-interacting protein 1 (Sip1/Zfhx1b) is a transcription factor that has been previously implicated in TGFβ/BMP signalling and in the etiology of the Mowat-Wilson syndrome. The expression of Sip1 was documented during mouse corticogenesis. The cortex-specific ablation of Sip1 resulted in the lack of hippocampus and dentate gyrus and in severe cortical lamination defects. Sip1 mutant mice exhibited cell death of differentiated cells and decreased proliferation in the region of the prospective hippocampus and dentate gyrus. In Sip1 mutants, the expression of the Wnt antagonist Sfrp1 was ectopically activated in the developing hippocampus and strongly up-regulated in post-mitotic cortical areas. The activity of the non-canonical Wnt effector, JNK, was inhibited in the prospective hippocampus of Sip1 mutants. The dentate gyrus defect was partially rescued by introducing in the Sip1−/− background, a stabilized form of the canonical Wnt mediator β-catenin. Sip1 is therefore essential to the development of the hippocampus and dentate gyrus, and regulates both canonical and non-canonical Wnt signalling via modulating the levels of Sfrp1. In the Sip1 mutant neocortex, upper layers were expanded at the expense of the deeper layers. The cell specification defects were accompanied by ectopic proliferation, increased apoptotic cell death and premature gliogenesis. As Sfrp1 was strongly up-regulated in the mutant neocortical areas, a role of Sip1 in the modulation of Wnt signalling in the neocortex is suggested.