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The Reeler mutation was so named because of the alterations in gait that characterize homozygous mice. Several decades after the description of the Reeler phenotype, the mutated protein was discovered and named Reelin (Reln). Reln controls a number of fundamental steps in embryonic and postnatal brain development. A prominent embryonic function is the control of radial neuronal migration. As a consequence, homozygous Reeler mutants show disrupted cell layering in cortical brain structures. Reln also promotes postnatal neuronal maturation. Heterozygous mutants exhibit defects in dendrite extension and synapse formation, correlating with behavioral and cognitive deficits that are detectable at adult ages. The Reln-encoding gene is highly conserved between mice and humans. In humans, homozygous RELN mutations cause lissencephaly with cerebellar hypoplasia, a severe neuronal migration disorder that is reminiscent of the Reeler phenotype. In addition, RELN deficiency or dysfunction is also correlated with psychiatric and cognitive disorders, such as schizophrenia, bipolar disorder and autism, as well as some forms of epilepsy and Alzheimer's disease. Despite the wealth of anatomical studies of the Reeler mouse brain, and the molecular dissection of Reln signaling mechanisms, the consequences of Reln deficiency on the development and function of the human brain are not yet completely understood. This Research Topic include reviews that summarize our current knowledge of the molecular aspects of Reln

function, original articles that advance our understanding of its expression and function in different brain regions, and reviews that critically assess the potential role of Reln in human psychiatric and cognitive disorders.