Ablation of the mTORC2 component rictor in brain or Purkinje cells affects size and neuron morphology

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The mammalian target of rapamycin (mTOR) assembles into two distinct multi-protein complexes called mTORC1 and mTORC2. Whereas mTORC1 is known to regulate cell and organismal growth, the role of mTORC2 is less understood. We describe two mouse lines that are devoid of the mTORC2 component rictor in the entire central nervous system or in Purkinje cells. In both lines neurons were smaller and their morphology and function were strongly affected. The phenotypes were accompanied by loss of activation of Akt, PKC, and SGK1 without effects on mTORC1 activity. The striking decrease in the activation and expression of several PKC isoforms, the subsequent loss of activation of GAP-43 and MARCKS, and the established role of PKCs in spinocerebellar ataxia and in shaping the actin cytoskeleton strongly suggest that the morphological deficits observed in rictor-deficient neurons are mediated by PKCs. Together our experiments show that mTORC2 has a particularly important role in the brain and that it affects size, morphology, and function of neurons.

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