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Deficiency of the miR-29a/b-1 cluster leads to ataxic features and cerebellar alterations in mice

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Titre	Deficiency of the miR-29a/b-1 cluster leads to ataxic features and cerebellar alterations in mice
Type de publication	Article de revue
Auteur	Papadopoulou, Aikaterini S [1], Serneels, Lutgarde [2], Achsel, Tilmann [3], Mandemakers, Wim [4], Callaerts-Vegh, Zsuzsanna [5], Dooley, James [6], Lau, Pierre [7], Ayoubi, Torik [8], Radaelli, Enrico [9], Spinazzi, Marco [10], Neumann, Melanie [11], Hébert, Sébastien S [12], Silahtaroglu, Asli [13], Liston, Adrian [14], D'Hooge, Rudi [15], Glatzel, Markus [16], De Strooper, Bart [17]
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Résumé en anglais	miR-29 is expressed strongly in the brain and alterations in expression have been linked to several neurological disorders. To further explore the function of this miRNA in the brain, we generated miR-29a/b-1 knockout animals. Knockout mice develop a progressive disorder characterized by locomotor impairment and ataxia. The different members of the miR-29 family are strongly expressed in neurons of the olfactory bulb, the hippocampus and in the Purkinje cells of the cerebellum. Morphological analysis showed that Purkinje cells are smaller and display less dendritic arborisation compared to their wildtype littermates. In addition, a decreased number of parallel fibers form synapses on the Purkinje cells. We identified several mRNAs significantly up-regulated in the absence of the miR-29a/b-1 cluster. At the protein level, however, the voltage-gated potassium channel Kcnc3 (Kv3.3) was significantly up-regulated in the cerebella of the miR-29a/b knockout mice. Dysregulation of KCNC3 expression may contribute to the ataxic phenotype.
URL de la notice	http://okina.univ-angers.fr/publications/ua19356 [29]
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Liens

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