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Lack of dcf1 causes myelin development disorder

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Multiple sclerosis is a demyelinating disease of the central nervous system. The myelin of patients with multiple sclerosis is accompanied by a pathological change, resulting in abnormal signal transduction, and then the patients will be onset with some symptoms, like muscle weakness, coordination and balance ability impaired, restless, cognitive memory disorders. Currently there is no effective treatment for multiple sclerosis, the main reason lies in the mechanism of demyelinating and myelin regeneration is still not clear. In this study, we reported that dcf1 is closely related to the development of myelin for the first time which put forward a new thought on the mechanism of demyelination. According to the proteomics analysis, we showed the knockout of dcf1 led to the decrease of MBP by 24.31 times. Then our research found that in the central nervous system of dcf1 knockout mice, in addition to MBP, the expression of MAG, MOG and CNPase were also lower significantly. We observed that the myelin was obvious thinner; uncompact by Transmission electron microscope (TEM). Furthermore, behavioral experiments found that the grip strength of dcf1 knockout mice was reduced and the coordination and balance ability was damage. In order to further explore the effect of dcf1 on myelin development, we had a rescue operation on dcf1 knockout mice and the results indicated that when dcf1 is expressed again, the myelin could almost return to that like the morphology of wild type mice. Moreover, an increase in axon diameter was seen in the ultrastructure of dcf1 knockout mice, so we had a preliminary exploration to expound the molecular mechanism. Western blotting showed that expression of NRG1 was lower, thus we speculated that the knockout of dcf1 induced demyelination via axonal signal NRG1, it laid the foundation for the further study of myelin dysplasia and demyelinating disease.

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