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Histology and gene expression of thymus and muscle in the myogenin and myf5 knockout mouse models

Autor: Bo Hu
Institut / Klinik: Pathologisches Institut
Doktorvater: Prof. Dr. A. Marx

The function of thymus in myasthenia gravis (MG) has been a very popular and interesting research. However, the mechanisms still remains largely unknown. The role of thymic myoid cells (TMCs) in MG has gradually been revealed, and TMCs may play a critical role in the pathogenesis of MG. In our study, we established myogenin and myf5 knockout mouse models. Myogenin homozygous mice can't survive, but myf5 homozygous mice can survive and continue to breed. The HE staining of muscles showed that the muscle fibers in the myogenin homozygous embryos are abnormally thin, and myofibers are obviously decreased. However muscles of myf5 homozygous mice were nearly the same as WT mice. HE staining of thymuses showed the same structure among myogenin homozygous, myf5 homozygous and WT mice. We also compared them by quantitative analysis of some related genes. Knocking down myogenin and myf5 will led to a decrease in the expression of titin and desmin compared to the WT mice. The IHC result also showed that myogenin and desmin positive cells are disappeared in the thymus of myogenin homozygous mice. Taken together, myogenin and myf5 knockout mice can cause some skeletal muscle gene decrease, and might lead to the lack of TMCs in thymuses. Furthermore, the nude mice transplanted with myogenin and WT murine thymuses can export T cells. Therefore, MRFs homozygous thymus transplantation into nude mice may hopefully unravel a new pathological mechanism.