

6月5日(金) 12:30~13:30 第6会場(ホールD7) 【英語 Animal Experiments】

0-0106

Prevention of gamma motoneuronal loss in Otsuka Long-Evans Tokushima Fatty rats by exercise training

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key words Diabetic polyneuropathy • motoneuron • exercise training

【Purpose】

We recently reported that there is a significant ($P < 0.01$) decrease in the absolute number of gamma motoneurons in type 1 and type 2 diabetic rats. However, the effect of exercise training on gamma motoneuronal loss is unknown. The purpose of this study was to investigate the effect of 20 weeks of exercise training on motoneuronal loss in type 2 diabetic rats, i.e., the Otsuka Long-Evans Tokushima Fatty (OLETF) rats.

【Methods】

OLETF rats were divided into the exercise training group (Ex-OLETF, $n = 6$) and the sedentary group (Sed-OLETF, $n = 6$), whereas the Long-Evans Tokushima Otsuka (LETO) rats were used as the control group (Cont-LETO, $n = 6$). Ex-OLETF rats were trained on a treadmill (20 m/min, 6 times/week, starting at 25 weeks). Alterations in the number and size of medial gastrocnemius (MG) motoneurons were studied in the MG motor nucleus for each group at 45 weeks. The intraperitoneal glucose tolerance test (IPGTT) was conducted to assess glucose metabolism in the rats.

【Results】

IPGTT indicated that exercise training improved glucose tolerance in the Ex-OLETF group. The Sed-OLETF group had significantly fewer motoneurons as compared with the Cont-LETO group ($P < 0.01$) ; however, there was no difference between the Ex-OLETF and Cont-LETO groups. The distribution of average soma area in the MG motoneurons of all the groups was bimodal ; cells with larger areas were presumed to be alpha-motoneurons, and those with smaller areas were presumed to be gamma-motoneurons. As compared with the Cont-LETO group, the number of gamma motoneurons was reduced by almost half in the Sed-OLETF group but was preserved in the Ex-OLETF group.

【Discussion】

Our results indicate that exercise training not only improves glucose tolerance but also prevents gamma motoneuronal loss in type 2 diabetic rats.