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Methylcobalamin (methyl-B12) promotes regeneration of motor nerve terminals degenerating in anterior gracile muscle of gracile axonal dystrophy (GAD) mutant mouse.

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Abstract

We examined the effects of methylcobalamin (methyl-B12, mecobalamin) on degeneration of motor nerve terminals in the anterior gracile muscle of gracile axonal dystrophy (GAD) mutant mice. GAD mice received orally methyl-B12 (1 mg/kg body wt/day) from the 40th day after birth for 25 days. In the distal endplate zone of the muscle, although most terminals were degenerated in both the untreated and methyl-B12-treated GAD mice, sprouts were more frequently observed in the latter. In the proximal endplate zone, where few degenerated terminals were seen in both groups of the mice, the perimeter of the terminals was increased and the area of the terminals was decreased significantly in the methyl-B12-treated GAD mice. These findings indicate that methyl-B12 promotes regeneration of degenerating nerve terminals in GAD mice.

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