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Abstract
Laminin-α2 related Congenital Muscular Dystrophy (LAMA2-CMD) is a fatal muscle disease caused by mutations in the LAMA2 gene. Laminin-α2 is critical for the formation of Laminin-211 and -221 heterotrimers in the muscle basal lamina. LAMA2-CMD patients exhibit hypotonia from birth and progressive muscle loss that results in developmental delay, confinement to a wheelchair, respiratory insufficiency and premature death. There is currently no cure or effective treatment for LAMA2-CMD. Several studies have shown Laminin-111 can serve as an protein replacement therapy for LAMA2-CMD. Studies have demonstrated early treatment with Laminin-111 protein results in an increase in life expectancy and improvements in muscle pathology and function. Since LAMA2-CMD patients are often diagnosed after advanced disease, it is unclear if Laminin-111 protein therapy at an advanced stage of the disease can have beneficial outcomes. In this study, we tested the efficacy of Laminin-111 protein therapy after disease onset in a mouse model of LAMA2-CMD. Our results showed Laminin-111 treatment after muscle disease onset increased life expectancy, promoted muscle growth, and increased muscle stiffness. Together these studies indicate Laminin-111 protein therapy either early or late in the disease process could serve as an effective protein replacement therapy for LAMA2-CMD.

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