GALECTIN-1 IS HIGHLY EXPRESSED IN DYSTROPHIC SKELETAL MUSCLES

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Galectin-1 (Gal1), a 14kDa beta-galactoside binding lectin, plays crucial roles in processes such as cell adhesion, apoptosis, phagocitosis, inflammatory events, immune reactions, cell proliferation/differentiation. Recent studies showed that Gal1 increases skeletal muscle differentiation and regeneration, suggesting its potential involvement in neuromuscular diseases like Duchenne Muscular Dystrophy (DMD). There are no previous reports about Gal1 expression in dystrophic muscles. In this study, we compared Gal1 expression and location in the DMD models mdx mouse and GRMD dog. We studied gastrocnemius and diaphragm from mdx (3 to 6 weeks old, and after 5 weeks of compulsory activity), and femoral biceps from GRMD (3 and 12 months old). By western blot and immunohistochemical assays we observed an increase in Gal1 expression after the third week, with a peak in 4 weeks, and a decline in 6 weeks, returning to levels detected in normal mice. Physical activity intensified the dystrophinopathy and increased the Gal1 level. In dogs, whose phenotype is closer to human, we observed high Gal1 expression in the two ages. Our data showed that Gal1 levels are augmented in conditions of significant muscular injury, suggesting that it participates in the muscular degeneration.

Key words: galectin-1, mdx, GRMD, Duchenne Muscular Dystrophy

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