Section: BASIC EXPERIMENTAL
Abstract: BAS-02

Study of the effects of arabinogalactan on Muscular Dystrophy in the mdx mouse model

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INTRODUCTION: Duchenne Muscular Dystrophy (DMD) is a genetic disorder characterized by dystrophin deficiency, leading to progressive muscle degeneration and increased oxidative stress. The mdx mouse model mimics this pathophysiology, presenting muscle weakness, necrosis, and elevated creatine kinase levels. Arabinogalactan (ResistAid), a polysaccharide with antioxidant and immunomodulatory properties, emerges as a potential therapeutic agent to attenuate muscle damage. **METHODS:** Sixteen male mdx mice (8 weeks old) were divided into a control group (n=6), receiving saline solution, and a treated group (n=10), receiving ResistAid (200 mg/kg/day in 0.2 mL) via oral gavage for 8 weeks. Muscle strength was assessed biweekly using the Kondziela inverted screen test. Following treatment, animals were euthanized for blood collection (CPK, ALT, and AST) and tissue harvesting. Histological analysis (H&E) of the diaphragm was performed to evaluate centralized nuclei and Feret's diameter. The study was approved by the local Ethics Committee on Animal Use (CEUA #01/2025). **RESULTS**: The treated group showed significantly higher body weight gain (29.9%) compared to the control (19.2%). Functional improvement was observed in the treated group during the strength tests. Histological analysis of the diaphragm revealed a significant 14.91% reduction in the percentage of internalized nuclei (36.1% vs. 51.01%; p = 0.041). Serum levels of CPK, ALT, and AST remained elevated in both groups. Data collection has been completed; complementary morphological evaluations and further statistical analyses are currently underway. **DISCUSSION**: The reduction in internalized nuclei suggests a possible protective effect of arabinogalactan on sarcolemma integrity, thereby reducing the demand for muscle regeneration in the diaphragm. Weight gain and functional improvement suggest an attenuation of muscle impairment. The persistence of high enzyme levels reflects the underlying genetic nature of the disease. Further analyses are being conducted to deepen the morphological and functional characterization of the model. **CONCLUSION:** Preliminary findings suggest that arabinogalactan may contribute to the preservation of muscle morphology and function in the mdx model. Additional studies, currently in development, are necessary to confirm these effects.

KEYWORDS: Muscular Dystrophy, Duchenne; Arabinogalactans; Mice, inbred mdx; Diaphragm.

ABCS Health Sci. 2026;51(Suppl.):e0261002
https://doi.org/10.7322/2026comuabc51.bas02