POSTER 101

ABSENCE OF DYSTROPHIN ALTERS THE PASSIVE PROPERTIES OF THE EXTENSOR DIGITORUM LONGUS MUSCLE IN MICE

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Dystrophin is a cytoskeletal protein not directly participating the myosin-actin contractile apparatus in muscle. The loss of dystrophin leads to Duchenne muscular dystrophy. It is well-established that contractility is reduced in dystrophin-null muscle. Surprisingly, little is known about the influences of dystrophin-deficiency on the passive properties of muscle. We hypothesize that the loss of dystrophin alters the passive properties of the skeletal muscle. To test this hypothesis, we examined the passive properties of the extensor digitorum longus (EDL) muscle from normal BL10 and dystrophin-null mdx mice. Consistently, the mdx EDL showed an increase in muscle stiffness along age (2, 6, 14 and 20-month-old) compared to BL10. Interestingly, the EDL muscle failed at the proximal muscle tendon junction (MTJ) in ≥14-month-old mdx while it failed within muscle fiber in BL10 and younger mdx. Previous ultrastructural studies in mdx indicated a reduction in the MTJ strength due to a decrease in the junctional membrane folding. Thus, we initially suspected that the weakness of the MTJ in older dystrophic muscle might have shifted of the failure site toward the MTJ. However, we did not detect a difference in the junctional membrane folding between mdx and BL10. Nevertheless, we observed degeneration in some myofiber at the MTJ in 14-month-old mdx. Our results suggest that the shift of the failure site was likely a consequence of the increased muscle stiffness although MTJ degeneration may have also contributed.