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every breath counts

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PA2301

Small airways in in sedentary and endurance-trained dystrophic (mdx) mice

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The effects of mild endurance exercise training on the small airways in mdx mice are unknown. We compared epithelial thickness and turnover, apoptosis, and stress marker expression in small airways of mdx mice and wild-type (WT) controls, at rest and during exercise training. Mdx and WT mice were randomly assigned to sedentary (mdx-S, n=17; WT-S, n=19) or trained (mdx-EX, n=14; WT-EX, n=16) groups. Low-intensity endurance training (running on a wheel) was done 5 d/wk for 6 wk at progressively increasing speed (rpm from 16 to 24) and time (15 min to 1 h). Lungs were processed for light microscopy and periodic acid Schiff (PAS) staining. Hsp60 and PCNA were quantified by immunohistochemistry. Apoptosis was assessed by TUNEL. Bronchial epithelial thickness decreased over time in WT mice irrespective of training (linear regression for time trends: WT-S: R^2=0.43, r= -0.65; WT-EX: R^2=0.68, r= -0.82, p<0.0005 for both); conversely, no significant change occurred in mdx mice. The number of PAS+ goblet cells was much lower in the bronchiolar epithelium of mdx compared to WT mice in all conditions. At 30 days, PCNA positivity was higher in EX than S animals in both groups; however, at 45 days it sharply decreased in mdx-S and -EX, but not in WT mice. The percentage of TUNEL+ cells was higher in mdx-EX than WT-EX mice at 45 days. In mdx mice, expression of Hsp60 progressively decreased (p<0.01), and was inversely related to the percentage of TUNEL+ cells (R^2=0.44, r=-0.66, p=0.01). In conclusion, bronchiolar epithelium in mdx mice is poor of goblet cells, and progressively deteriorates over time possibly because of loss of stress-related protective mechanism. Mild training did not cause any additional damage.