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Title: Locomotor muscle susceptibility for eccentric exercise-induced muscle injury in cigarette smoke-exposed mice.

INTRODUCTION: Several studies show a strong dose and time-dependent correlation between tobacco cigarette usage and muscle injury, with limb muscle biopsies obtained from smokers displaying disruption of myofilament microstructure. Eccentric exercise-induced muscle damage (EEIMD) is the most common sports-related injury, and data comparing smokers and non-smokers indicate a greater prevalence of EEIMD with tobacco cigarette use. However, it is unknown whether chronic cigarette smoke (CS) exposure directly predisposes individuals to EEIMD.

PURPOSE: The aim was to investigate whether chronic CS exposure in mice changes locomotor muscle susceptibility to EEIMD.

METHODS: Adult wild-type male mice (48 total) were either exposed to Air or CS for two months and, at the end of the exposure period, were injected intraperitoneally with Evans Blue Dye (EBD) to track injured myofibers. The following day, one leg was subjected to a lengthening contractions protocol (LCP), and mice recovered for up to 3 days. In vivo dorsiflexion torque, extensor digitorum longus (EDL) ex vivo contractility, and tibialis anterior (TA) myofiber cross-sectional area and damage were measured.

RESULTS: Although no exposure and interaction eVects were found in the torque evoked by diVerent frequencies of pulse on Days 1 and 3 post-LCP, torque was higher at submaximal pulse frequencies in CS- vs Air-exposed mice immediately after LCP. In isolated EDL, force evoked by diVerent pulse frequencies between the two exposure groups showed an interaction eVect in the non-injured (control) leg. In LCP legs, force was significantly higher in CS vs Air-exposed mice on the Day 0 group. However, on Days 1 and 3 post-LCP, force was not diVerent between exposure groups. EDLs from CS-exposed mice had a slower contraction rate but similar relaxation rate to air-exposed mice. LCP reduced contraction and relaxation rates in both exposure groups on Days 1 and 3. Myofiber cross-sectional area did not diVer between exposure groups or legs on any recovery day. EBD-positive myofibers were not detected in any exposure groups and legs on Day 0, but were detected in the LCP-subjected legs on Days 1 and 3. There were no interaction and exposure eVects on the percentage of EBD-positive myofibers on Days 1 and 3.

CONCLUSION: Therefore, the data suggest that two months of cigarette smoke exposure in mice does not aVect the time course of muscle force decay and myofiber injury after a session of lengthening contractions.