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INTRODUCTION

Weightlessness has been shown to cause limb muscle wasting and a reduced peak force and power in the antigravity soleus muscle [1, 2]. Despite a reduced peak power, Caiozzo et al. [2] observed an increased maximal shortening velocity in the rat soleus muscle following a 14-day space flight. The major purpose of the present investigation was to determine if weightlessness induced an elevated velocity in the antigravity slow type I fibers of the rhesus monkey (*Macaca mulatta*), as well as to establish a cellular mechanism for the effect.

Spaceflight or models of weightlessness have been shown to increase glucose uptake [6], elevate muscle glycogen content, and increase fatigability of the soleus muscle [2]. The latter appears to be in part caused by a reduced ability of the slow oxidative fibers to oxidize fats [1]. A second goal of this study was to establish the extent to which weightlessness altered the substrate profile and glycolytic and oxidative enzyme capacity of individual slow- and fast-twitch fibers.

METHODS

The animal selection and biopsy procedures are described in the companion abstract [Fitts et al., p. 53]. Single fibers were isolated and the following parameters were measured: 1) peak force (P_o); 2) maximal unloaded shortening velocity (V_o); and 3) peak rate of tension redevelopment, k_{tr}. Following each experiment, the fiber was solubilized in SDS and the profile of the myosin heavy and light chain isozymes was determined. A 1 mm cross-section was cut from the soleus biopsy and fixed in 6.25% glutaraldehyde for determination of mitochondria and myofibril content. Single fiber biochemical assays were conducted on freeze-dried fibers by enzymatic cycling.

RESULTS

Maximal unloaded shortening velocity (V_o), and peak rate of tension redevelopment (k_{tr})

The maximal shortening velocity (V_o) of the slow type I soleus fiber decreased with growth (0.94 ± 0.04 vs 0.85 ± 0.03 FL/s), but it showed a slight increase (significant at $p < 0.1$) compared to the post-vivarium group following both flight (0.96 ± 0.04 FL/s) and the R+17 simulation (1.04 ± 0.04 FL/s). The increased velocity of the slow type I fiber was not caused by the expression of fast type proteins. This population of fibers contained no fast myosin heavy chain, and although the myosin light chain (MLC) 2s/total MLC ratio was increased, there was no change in the MLC3/MLC2 ratio. Growth decreased V_o in the type I (0.84 ± 0.03 vs 0.55 ± 0.03 FL/s) and II (5.82 ± 0.43 vs 4.37 ± 0.36 FL/s) gastrocnemius fibers, but in contrast to the soleus, flight had no effect on this variable in the gastrocnemius. In the type I soleus fiber, k_{tr} significantly declined in the vivarium group (2.48 ± .05 vs 2.07 ± .05 s-1), but was not altered in the R+17 or flight groups. Similar results were observed for the type I gastrocnemius fibers.

Morphology

We hypothesized that spaceflight might induce a selective loss of myofibrillar protein. However, the myofibrillar volume density (%) of the soleus fibers was unchanged by flight. The myofibrillar density of the 2 flight animals preflight was 71.8% and 69.8% compared to 74.0% and 69.7% postflight. The vivarium control and R+17 groups showed myofibrillar densities that were not significantly different from the flight animals.

Single fiber enzyme and substrates

Flight had no significant effect on any of the glycolytic (glycogen synthase [GS], phosphorylase [PP], hexokinase [HK], phosphofructokinase [PFK], lactate dehydrogenase [LDH]) or oxidative (β-hydroxyacyl-CoA dehydrogenase [βOAC] and citrate synthase

[CS]) enzymes assayed in either the slow- or fast-twitch fiber types. Additionally, the acetyl-CoA regulating enzyme carnitine acetyltransferase (CAT) was unaltered by flight. Further evidence that mitochondria are resistant to alteration with microgravity was the finding that mitochondrial volume density showed no significant change in any of the three groups. The substrates—glycogen, ATP, PCr—and lactates were also unaltered by flight.

CONCLUSIONS

The Flight and the R+17 groups showed a small increase in the type I fiber V_o compared to the 2nd vivarium controls ($p<0.1$). This increase could not be explained by an altered myosin heavy or light chain pattern. We hypothesized that the change might be caused by an increased myofilament lattice spacing. Although the myofibrillar volume density measurements do not support this idea, the possibility exists that the sensitivity of the morphometric analysis was insufficient to detect small but functionally significant increase in the myofilament lattice spacing. This change would explain the increased V_o and the reduced peak force (kN/m2) of the slow type I fiber. Spaceflight has been shown to reduce fatty acid oxidation in rat soleus muscle [1]. The present results demonstrate that the spaceflight-induced inhibition of fat oxidation cannot be explained by a reduced CAT or βOAC activity. Unlike bed rest, spaceflight did not cause glycogen loading. The failure of spaceflight to increase cell glycogen in the slow type I fiber may have resulted from insufficient caloric or carbohydrate intake inflight.

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