Impaired Jumping Performance Arising from Sedentary Growth is Recovered through Exercise in Adulthood

Matthew Q. Salzano[1], Suzanne M. Cox[2], Stephen J. Piazza[2], and Jonas Rubenson[1,2]

[1]Integrative and Biomedical Physiology, The Pennsylvania State University, University Park, PA, USA
[2] Kinesiology, The Pennsylvania State University, University Park, PA, USA
email: salzano@psu.edu, web: http://locomotionlab.net/

INTRODUCTION

Physical inactivity is known to contribute to a broad array of health problems throughout the lifespan, but biomechanical factors that limit physical activity are poorly understood. The musculoskeletal system is known to adapt to its activity-dependent loading, and is especially plastic during growth [1]. Studies of animal models have revealed differences in bone shape and joint structure between groups with different levels of physical activity during growth [2]. We have recently demonstrated using an avian, bipedal animal model that optimal fascicle lengths in hip and ankle muscles decrease substantially during growth (by ~15%) in sedentary compared to running-trained animals [3]. The shorter muscle lengths in the sedentary group may limit force generating capacity during dynamic movements due to force-length-velocity constraints [5]. Furthermore, preliminary data on ankle moment arms between these groups support the hypothesis that torque- and power-generating capacity will be compromised [5].

These morphological alterations over the growth span may reduce locomotor performance and increase effort when inactive animals with suboptimal musculoskeletal structure attempt to achieve a locomotor goal. The purpose of this study was to determine (1) the effects of growth-period inactivity upon locomotor performance immediately post-growth and (2) the extent to which these adaptations to inactivity can be reversed after a period of increased activity in adulthood.

This goal was addressed using an experimental design in which guinea fowl were grouped into 1) a control exercise group (EXE) where normal activity, including opportunities for running and jumping, was provided over the growth span and adulthood, 2) a disuse group (DIS) that was subject to limited movement and muscle disuse (using botulinum toxin) during growth and subsequently provided the same access to activity as the EXE group during adulthood.

METHODS

One-day old guinea fowl (*Numida meleagris*) were obtained from a regional breeder (Guineafarm, OH). At 4 weeks of age, guinea fowl were divided into EXE (n = 16) and DIS (n = 16) groups. EXE birds were housed in a large, circular pen that allowed ample room for locomotion and objects for jumping/perching. The DIS group were reared in small, square pens with low ceilings to restrict movement and jumping. Furthermore, the DIS groups was subjected to focal muscle disuse via injection of botulinum toxin-A (BTX-A) into the lateral (LG) and medial gastrocnemius (MG). This protocol created an enhanced disuse signal that was reversible.

Each animal received an initial BTX-A injection (4 units (LD50)/kg) at 7-8 weeks of age under general anesthesia (1.5% isoflurane). The animals received an additional injection every five weeks for a total of 4 injections over 20 weeks, by which time the animals had reached skeletal maturity. The EXE groups received sham injections of saline.

At 27-28 weeks of age (~6 months), a maximal jumping task was used to assess power capacity. Birds were placed on dual-force plates (AMTI 6x6; 20 lb. capacity each) that were surrounded by a vertical box (Fig 1). Birds were given 5 minutes to jump at least 3 times voluntarily or with encouragement. This was repeated across two days. Forces were recorded at 100 Hz and filtered using a

low-pass Butterworth filter with a cutoff frequency of 50 Hz. Peak vertical forces from the top three jumps were averaged and normalized to body weight (BW).

Following the 6 month testing, half of the animals from each group were sacrificed (pentobarbital >1.6 mg/kg) for morphological analyses (to be performed in a companion study). The remaining animals from the DIS group were housed in the same condition as those of the EXE group throughout adulthood. Adult animals were retested at 15 months of age.

Student t-tests were performed to assess group differences at an alpha level $p < 0.05$.

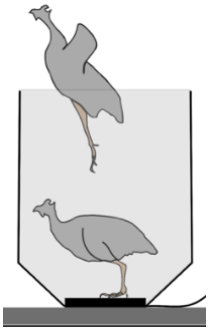

Figure 1. Schematic illustration of jumping task.

RESULTS AND DISCUSSION

The average maximum weight-normalized vertical force at the 6-month post-growth time point was ~6 N/BW in the DIS group vs. ~8 N/BW in the EXE group ($p < 0.05$). Upon retesting at the 15 month time point both the DIS and EXE groups average maximum weight-normalized vertical force was ~7 N/BW. The jumping force data are summarized in Fig. 2.

Not surprisingly, the jumping performance of DIS animals was markedly poorer than EXE birds after a growth period marked by both general and localized muscle disuse. Nevertheless, the magnitude of peak vertical force in the DIS group was remarkably high, reaching nearly 6x body weight. This value is comparable to the peak forces reported previously from cage-reared guinea fowl [6]. This suggests that localized muscle paralysis during growth is compensated for, possibly either through growing new muscle, or by increased growth and use of use of synergists. It also suggests that the difference in force observed between groups at 6 months is due primarily to the availability of exercise as opposed to a BTX-A effect.

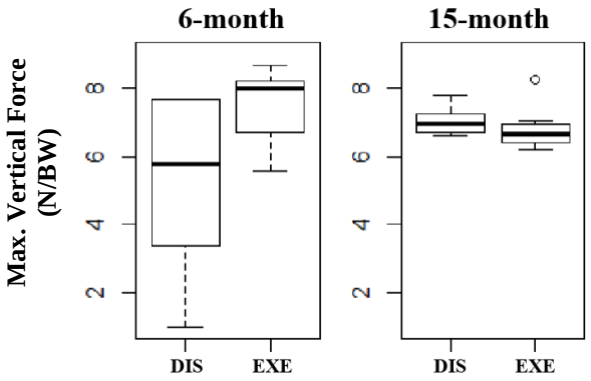

Figure 2. Box-and-whisker plots of max normalized vertical force at 6- (left) and 15-months (right).

To our knowledge, this is the first study to systematically assess whether movement impairment due to disuse during growth can be reversed. Our results suggest that maximal locomotor performance (jumping) can indeed be recovered after even severe locomotor and muscle disuse during growth. This finding has encouraging implications for exercise interventions for restoring function. How these findings correlate with morphological adaptations is currently being explored. The logical subsequent steps are to establish the exercise dose necessary to restore normal function and its timeframe, and if these findings relate generally across modes of movement and species.

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