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Original article

Improving glucose tolerance by muscle-damaging exercise

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ABSTRACT

Tissue damage is regarded as an unwanted medical condition to be avoided. However, introducing tolerable tissue damages has been used as a therapeutic intervention in traditional and complementary medicine to cure discomfort and illness. Eccentric exercise is known to cause significant necrosis and insulin resistance of skeletal muscle. The purpose of this study was to determine the magnitude of muscle damage and blood glucose responses during an oral glucose tolerance test (OGTT) after eccentric training in 21 young participants. They were challenged by 5 times of 100-meter downhill sprinting and 20 times of squats training at 30 pounds weight load for 3 days, which resulted in a wide spectrum of muscle creatine kinase (CK) surges in plasma, 48 h after the last bout of exercise. Participants were then divided into two groups according the magnitude of CK increases (low CK: +48% ± 0.3; high CK: +137% ± 0.5, $P < 0.05$). Both groups show comparable decreases in blood glucose levels in OGTT, suggesting that this muscle-damaging exercise does not appear to decrease but rather improve glycemic control in men. Conclusion: The result of the study rejects the hypothesis that eccentric exercise decreases glucose tolerance. Improved glucose tolerance with CK increase implicates a beneficial effect of replacing metabolically weaker muscle fibers by eccentric exercise in Darwinian natural selection fashion.

1. Introduction

Moderate traumatic physical challenge has been frequently used as a therapeutic modality in traditional Chinese medicine,[1] which leads to an anticipation of beneficial health outcome following muscle-damaging exercise.[2] We have previously reported an increased necrosis and white blood cell infiltration in exercised muscle after downhill running, an exercise regimen containing eccentric muscle contraction.[3] However, decreased insulin sensitivity in glucose transport and impaired glycogen storage of skeletal muscle after eccentric muscle contraction has been reported in animals and humans.[4,5]

Skeletal muscle, accounted for 40% of body weight, is the major tissue for post-meal glucose uptake. Approximately 85% of post-prandial glucose in circulation is disposed into muscle tissues.[2] The whole-body insulin sensitivity, assessed by hyperinsulinemic euglycemic clamp technique, suggests that insulin resistance occurs during the first 48 h after eccentric exercise.[6,7] Decreased muscle insulin sensitivity is expected to produce a negative consequence in the whole-body glycemic regulation. Based on these findings, we hypothesized that muscle-damaging exercise training will decrease glucose tolerance in men. Blood creatine kinase (CK) level has been commonly used to measure the levels of exercise-induced muscle damage. Histological analysis suggests that blood level of muscle CK should be regarded as a biomarker for post-exercise muscle regeneration.[2,8] In this study, we assessed oral glucose tolerance together with muscle CK in plasma, two days following the last bout of exercise training containing eccentric muscle contraction. We tested the hypothesis that 1) muscle-damaging exercise will decrease the whole-body glucose tolerance; 2) The degree of CK increase is associated with attenuated glucose tolerance in men.

2. Methods

2.1. Participants

Twenty-one healthy junior athletes (16.3 ± 0.5 years of age) with no history of musculoskeletal disorders of the lower limbs

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were enrolled in this study. All participants were requested to avoid physical training and medication during the experimental period. Participants provided informed consent prior to participation in the study. This study was conducted in accordance with the guidelines in the Declaration of Helsinki.

2.2. Experimental procedure

Participants performed barbell front squat and downhill run for 3 consecutive days then allowed to rest for another 2 days (Fig. 1). This physical challenge produced a wide range of muscle damage among participants. For OGTT, blood was collected at Day 1 before exercise challenge and again at Day 4, 12 h after the last bout of training. To verify the result of muscle damage, blood sample was collected at Day 1 (24 h) and day 2 (48 h) for CK analysis after the last exercise bout.

2.3. Muscle-damaging exercise

2.3.1. Barbell front squat

Participants were instructed to perform 5 sets of barbell front squat for 20 repetitions at 30 pounds a day for 3 consecutive days and allowed to rest for another 3 consecutive days.

2.3.2. Downhill sprinting

The 100-meter downhill sprinting consisted of 5 repetitions a day for 3 consecutive days and allowed to rest for another 3 consecutive days.

2.4. OGTT

Under a 12-h overnight fasted condition, a 500-ml solution containing 75 g of glucose was orally delivered, and blood was taken from the finger at 0 (before the solution load), 30, 60 and 90 min afterward. Blood glucose level was determined by an automated glucose analyzer (LifeScan, Inc., Milpitas, California 95035, USA).

2.5. Plasma creatine kinase (CK) analysis

CK (CKMB isoform) in plasma is an indicator of muscle regeneration after exercise-induced muscle damage. Plasma sample was taken from fingertip to measure muscle CK activity. Plasma was obtained by centrifuging the blood at 4 °C for 10 min at 3000 rpm and was stored at −80 °C until analysis. CK activity was measured using a DT-60 automated hematology analyzer (Kodak Co., Rochester, NY, USA).

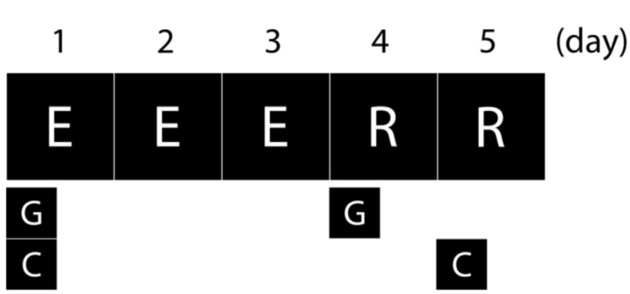

Fig. 1. Time table of experimental procedure. Participants were scheduled to be challenged by eccentric exercise for 3 consecutive days from Day 1 to Day 3, then allowed to recover for another 2 days (from Day 4 and Day 5). Prior to the exercise, oral glucose tolerance test (OGTT) and creatine kinase (CK) measurements were carried out to obtain basal values. OGTT was conducted again 24 h after the last exercise at day 3 and CK was measured again at the end of day 5. E, eccentric exercise; R, rest (no exercise); G, OGTT measurement; C: CK measurement.

2.6. Statistical analysis

Data were expressed as mean ± standard deviation (SD). Statistical differences between groups were calculated by analysis of variance (ANOVA). Student's t-test was used to compare the mean difference in each variable between the two groups (low CK group vs. high CK group). Statistical significance for type I error was set at $P < 0.05$ for all measures.

3. Results

The current exercise challenge protocol (Fig. 1) results in a wide range of muscle CK surge (Table 1). For those participants showing relatively lower CK increases (Fig. 2A), glucose levels during OGTT decreased significantly after exercise, indicating an improvement in the glucose tolerance. However, for those participants showing relatively high CK increase (Fig. 2B), similar magnitude of improvement on glucose level during OGTT was also reached. Data on area under curve (AUC) in OGTT show no difference in improvement of glycemic control between two groups (Fig. 2C).

4. Discussion

It has been shown that aerobic endurance exercise, as an exercise regimen producing insignificant muscle damage, can improve glucose tolerance.[9] On the other hand, muscle-damaging eccentric muscle contraction results in insulin resistance in glucose transport and glycogen storage of exercised skeletal muscle.[4,5] Based on these early findings, we hypothesized that muscle-damaging exercise will decrease glucose tolerance in humans. However, the result of the study rejects our hypothesis. In a contrary, our data suggest the benefit of improving glycemic control by a muscle-damaging exercise.

Skeletal muscle is the main storage site of postprandial glucose after a carbohydrate meal.[2] Therefore, insulin sensitivity of skeletal muscle should significantly influence the whole-body glucose tolerance. The previous report on onset of muscle insulin resistance after eccentric muscle contraction appears to be associated with

Table 1
Plasma levels of muscle creatine kinase.

Unit: U/L	PRE	Post	% Increase
A	292	313	7%
B	668	726	9%
C	479	618	29%
D	391	526	35%
E	379	521	37%
F	232	376	62%
G	283	476	68%
H	242	416	72%
I	240	416	73%
J	200	372	86%
K	229	427	86%
L	223	426	91%
M	222	432	95%
N	275	536	95%
O	182	364	100%
P	231	482	109%
Q	80	178	123%
R	117	326	179%
S	128	368	188%
T	90	275	206%
U	109	364	234%
Mean	252	426	94%