Plyometric Exercise Increases Serum Indices of Muscle Damage and Collagen Breakdown

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Abstract

The aim of the present study was to examine the effect of acute plyometric exercise on indices of muscle damage and collagen breakdown. Nine untrained men performed an intense bout of plyometric jumping exercises (experimental group) and nine men remained at rest (control group). Seven days before and 24, 48, and 72 hours after plyometric exercise or rest, several physiological and biochemical indices of muscle damage and two biochemical indices of collagen damage were determined. No significant changes in concentric and eccentric peak torque of knee extensors and flexors or flexion and extension range of motion were found after the plyometric exercise. Delayed-onset muscle soreness increased 48 hours after exercise. Creatine kinase increased 48 and 72 hours post exercise, whereas lactate dehydrogenase increased 24, 48, and 72 hours post exercise. Serum hydroxyproline increased 24 hours post exercise, peaked at 48 hours, and remained elevated up to 72 hours post exercise. Hydroxylysine (which was measured only before exercise and at 48 hours) was found increased 48 hours post exercise. No differences were found in any physiological or biochemical index in the control group. Intense plyometric exercise increased muscle damage, delayed-onset muscle soreness, and serum indices of collagen breakdown without a concomitant decrease in the functional capacity of muscles. Hydroxyproline and hydroxylysine levels in serum seem promising measures for describing exercise-induced collagen degradation. Coaches need to keep in mind that by using plyometric activities, despite the increased muscle damage and collagen turnover that follow, it is not necessarily accompanied by decreases in skeletal muscle capacity.

Key Words eccentric, hydroxyproline, hydroxylysine, stretch-shortening cycle

Introduction

Plyometric exercise is a widely used training mode to enhance the ability of skeletal muscles to generate power. It is characterized by rapid deceleration of the body, followed by rapid acceleration in the opposite direction (2). This combination of eccentric and concentric contractions is a natural type of muscle function called stretch-shortening cycle (2). The stretch-shortening cycle provides a physiological advantage in that the muscular force developed during the concentric phase is potentiated by the preceding eccentric contraction (2).

Plyometric training has been shown to improve jumping ability (10), running economy (28), and overall athletic performance (23). It is well documented that eccentric muscle contractions (such as during plyometric exercise) induce muscle damage and soreness, particularly when the muscles are unaccustomed to this type of exercise (8). Eccentric contractions induce long-lasting decreases in muscle force generation and range of motion (ROM), muscle swelling, and soreness, as well as increased levels of muscle damage indices in the blood (7,14,20). Beside damage to muscle fibers, eccentric exercise may also disrupt connective tissue (25). Thus, measurement of biochemical indices of collagen breakdown may be used to identify the effect of exercise on connective tissue. Hydroxyproline and hydroxylysine are amino acids characteristic of, but not exclusive to, collagen; therefore, increased levels of these amino acids in blood and urine have been used as an index of collagen breakdown in several exercise studies (1,3,4,13,30,31).

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22(2)/490–496

Journal of Strength and Conditioning Research

The available evidence regarding the effects of exercise on these collagen indices in humans produced conflicting results. Two studies have found increased levels (1,3), and four studies reported no differences (4,13,30,31) in hydroxyproline and/or hydroxylysine concentration after exercise. Differences in age and sex of the participants, exercise mode (e.g., concentric vs. eccentric), exercise intensity, and type of tissue sample collected (blood serum or urine) can all account for the discrepancies among studies.

Although many studies have examined the effects of exercise on indirect indices of collagen degradation in humans (1,3,4,13,30,31), there is no consensus. Additionally, there are limited data regarding the effects of a real-life exercise mode on collagen breakdown indices, because all but one (13) of the relevant studies have used non-physiological exercise protocols, such as isokinetic contractions or downhill running (1,3,4,30,31). Considering the relatively intensive nature of plyometric exercise, it seems important to examine the effects of a usual plyometric training session on indices of collagen status. Therefore, the aim of the present investigation was to examine the effects of an acute bout of plyometric exercise on indirect indices of muscle damage and collagen breakdown in untrained male subjects. We hypothesized that a single session of plyometric exercise would induce muscle damage and increase collagen breakdown.

Methods

Experimental Approach to the Problem

To examine the effects of an acute bout of plyometric exercise on muscle and collagen breakdown, several indices of muscle and collagen damage were assessed using a repeated-measures design, including a control group to account for the day-to-day variation of the physiological and biochemical assays employed. To this end, we monitored changes in concentric and eccentric peak torque of knee extensors and knee flexors, range of motion (ROM), delayed-onset muscle soreness (DOMS), creatine kinase (CK), and lactate dehydrogenase (LDH) as indices of muscle damage, as well as hydroxyproline and hydroxylysine as indices of collagen damage.

Subjects

Eighteen untrained healthy men were divided into a control (age 21.3 ± 1.3 years, weight 74.6 ± 4.4 kg, height 178.1 ± 4.2 cm) and an experimental group (age 22.1 ± 1.8 years, weight 76.5 ± 7.7 kg, height 175.5 ± 6.0 cm). Subjects had no previous history of participation in a structured exercise program involving plyometric exercises, and they were asked to refrain from strenuous physical activities and from consumption of foods rich in gelatin (e.g., gelatin desserts, gummy bears, or aspic) 72 hours before and during the experimental period. All subjects were informed about the nature of the study and the associated risks and benefits, and they signed an informed consent form. Procedures were in accordance with the Helsinki Declaration of 1975, and institutional review board approval was received for this study.

Plyometric Exercise

The experimental group performed an intense bout of plyometric exercises on a wrestling-type mat. Before exercise, subjects performed a warm-up consisting of 5 minutes of low-intensity running and stretching. Plyometric exercise consisted of 96 jumps over 50-cm hurdles (eight sets of 12 repetitions) and 96 jumps onto a 50-cm plyometric box (eight sets of 12 repetitions). A 90-second resting interval was allowed between sets, and there was a 3-minute resting interval between the two series of jumps. Exhaustion was usually reached after 100–400 repetitions, corresponding to 2–5 minutes of intensive series of jumps (12,19).

Muscle Performance and Muscle Damage Indicators

Seven days before and 24, 48, and 72 hours after plyometric exercise–or at four consecutive days for the control group–-each subject's maximal voluntary concentric peak torque of knee extensors (CPTE), concentric peak torque of knee flexors (CPTF), eccentric peak torque of knee extensors (EPTE), and eccentric peak torque of knee flexors (EPTF) was determined. Peak torque was measured according to Paschalis et al. (21) on a computer-controlled isokinetic dynamometer (Cybex Norm Lumex, Ronkonkoma, NY). Before the assessment of peak torque, volunteers warmed up for 8 minutes on a Monark cycle ergometer (Monark, Vansbro, Sweden) at 50 W and 70 rpm, followed by 3 minutes of stretching exercises. All subjects were familiarized with the isokinetic dynamometer and the testing procedures by performing eight consecutive submaximal (<50% max) concentric and eccentric warm-up repetitions. Visual feedback and verbal encouragement were given during the trials. The knee moved through the whole range of motion, from 10° to 110° (0° = straight leg) at an angular velocity of 1.05 $rad \cdot s^{-1}$. The reliability coefficient for repetitive measurements in CPTE, CPTF, EPTE, and EPTF was 0.98, 0.96, 0.97, and 0.97, respectively.

ROM was determined using the goniometer of the isokinetic dynamometer. Knee flexion ROM was assessed with the subject lying prone on the reclining chair of the isokinetic dynamometer with his knees fully extended. From this position, passive flexion was performed by T.T. at a very low angular velocity of 0.35 $rad \cdot s^{-1}$ while the position in which the subject felt any discomfort was taken as the pain-free flexion ROM. ROM for knee extension was assessed while the subject was sitting on the reclining chair of the isokinetic dynamometer with his knees fully flexed. From this position, the same passive extension was performed by the same investigator, and the position in which the subject felt any discomfort was taken to indicate the pain-free extension ROM. ROM was evaluated before and 24, 48, and 72 hours after the plyometric exercise. Reliability coefficient for repetitive measurements was 0.99.

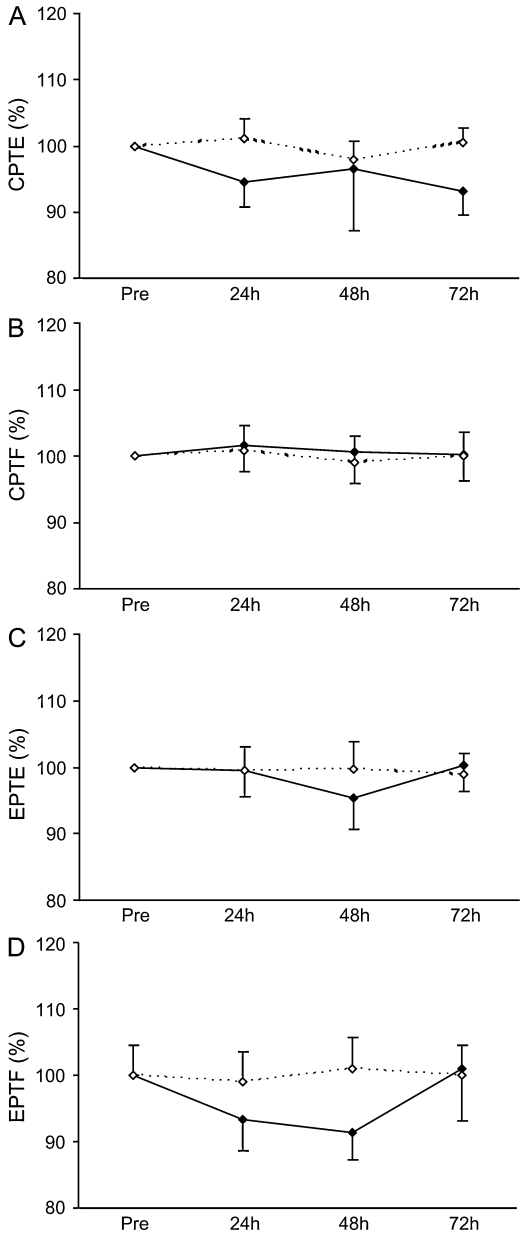

Figure 1. Normalized changes in concentric peak torque of knee extensors (CPTE) (A), concentric peak torque of knee flexors (CPTF) (B), eccentric peak torque of knee extensors (EPTE) (C), and eccentric peak torque of knee flexors (EPTF) (D) from the pre-exercise level (100%) after plyometric exercises. Data are presented as mean (*SEM*).

DOMS was assessed using an established questionnaire (5). Subjects rated their soreness during active movements of knee flexors and extensors ranging from 1 (normal) to 10 (very, very sore). Muscle soreness was evaluated before and 24, 48, and 72 hours after the plyometric exercise. Reliability coefficient for repetitive measurements in DOMS was 0.98.

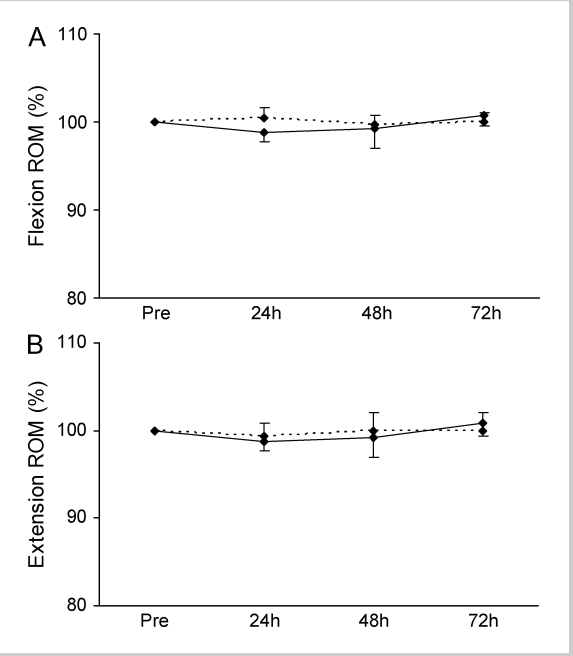

Figure 2. Normalized changes in flexion (A) and extension (B) range of motion (ROM) from the pre-exercise level (100%) after plyometric exercises. Data are presented as mean (*SEM*).

Blood Collection and Biochemical Assays

Blood samples (10 mL) were drawn from the antecubital vein into plain evacuated test tubes before and 24, 48, and 72 hours post exercise. The blood was allowed to clot at room temperature for 30 minutes and centrifuged at 1500*g* for 10 minutes. The serum layer was removed and frozen at –20°C in multiple aliquots for analysis of CK, LDH, hydroxyproline,

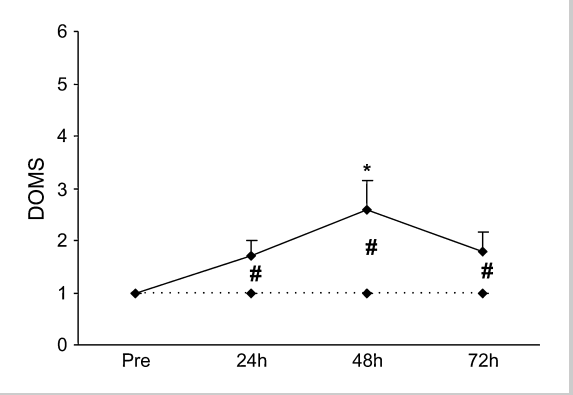

Figure 3. Normalized changes in delayed onset muscle soreness (DOMS) from the pre-exercise level (100%) after plyometric exercises. Data are presented as mean (*SEM*). *Significantly different from the pre-exercise value in the experimental group. #Significantly different between the control and experimental groups at the same time point.

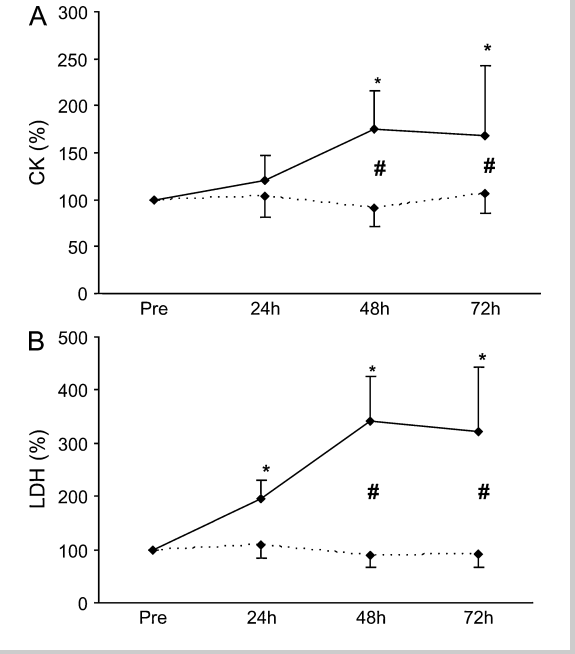

Figure 4. Normalized changes in creatine kinase (CK) (a) and lactate dehydrogenase (LDH) (b) from the pre-exercise level (100%) after plyometric exercises. Data are presented as mean (*SEM*). *Significantly different from the pre-exercise value in the experimental group. #Significantly different between the control and experimental groups at the same time point.

and hydroxylysine. Hydroxylysine was measured only before and 48 hours after the plyometric exercise because of accidental loss of part of the serum.

CK was assayed spectrophotometrically (Spectronic 401, Milton Roy, NY) using an enzymatic method based on the rate of NADPH formation that absorbs at 340 nm (11) with a kit from Megalab (Athens, Greece). LDH was determined in the same spectrophotometer using an enzymatic method based on oxidation rate of NADH that absorbs at 340 nm (29) using commercially available kits from Randox (Antrim, UK). The normal reference ranges for men using these kits is 24–195 IU·L^{-1} for CK and 230–460 IU·L^{-1} for LDH. The intra-assay coefficients of variation were 8.8% for CK and 4.6% for LDH.

Serum total hydroxyproline was measured as described previously (22). In brief, this assay is based on alkaline hydrolysis of test samples in 2 N final concentration of sodium hydroxide by autoclaving at 120°C for 20 minutes and subsequent quantitation of the free hydroxyproline in hydrolyzates. Chloramine-T was used to oxidize the free hydroxyproline for the production of a pyrrole. The oxidation was allowed to proceed for 25 minutes at room temperature. The addition of Ehrlich's reagent resulted in the formation of a chromophore, which developed by incubating the samples at 65°C for 20 minutes. Absorbance of each sample was read at 550 nm using a spectrophotometer. The calibration curve was initially established using standards (0–20 μg) of hydroxyproline. Hydroxylysine was measured in serum after hydrolysis (26) according to Euli et al. (9). The intra-assay coefficient of variation was 3.8% for hydroxyproline and 3.3% for hydroxylysine.

Fingertip capillary blood samples were taken before exercise and 3 minutes post exercise to determine spectrophotometrically (LP 20, Dr Lange, Berlin, Germany) blood lactate concentration.

Statistical Analyses

Data are presented as mean ± *SEM*. The distribution of all dependent variables was examined by the Kolmogorov-Smirnov test and was found not to differ significantly from normal. Data were analyzed through two-way (group × time) ANOVA with planned contrasts on different time points. A paired sample *t*-test was used to analyze hydroxylysine and lactate levels. The test-retest reliability of position sense and joint reaction angle to release was determined by performing the intraclass reliability test. The level of statistical significance was set at $\alpha = 0.05$. SPSS version 13.0 was used for all analyses (SPSS Inc., NC).

Results

The statistical power of the present study to detect an average change of 20% for all dependent parameters between rest

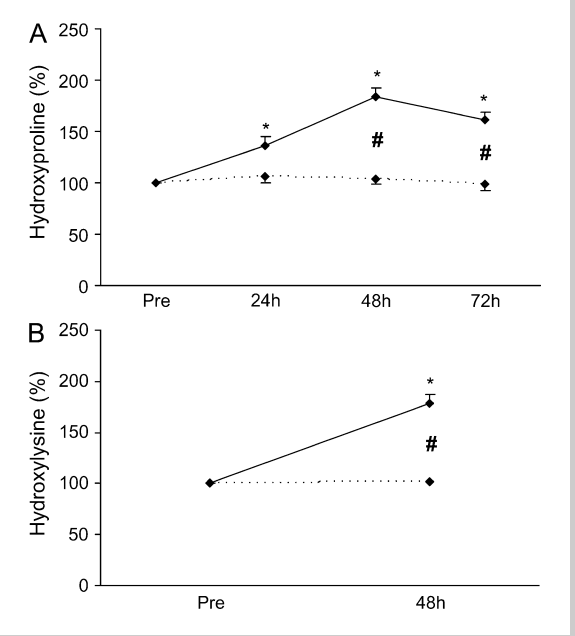

Figure 5. Normalized changes in hydroxyproline (a) and hydroxylysine (b) from the pre-exercise level (100%) after plyometric exercises. Data are presented as mean (*SEM*). *Significantly different from the pre-exercise value in the experimental group. #Significantly different between the control and experimental groups at the same time point.

and post exercise was 0.76, slightly less than the recommended power of 0.8 at $\alpha = 0.05$.

Muscle Performance

No significant changes in CPTE, CPTF, EPTE, EPTF (Figure 1), or flexion and extension ROM (Figure 2) were found after plyometric exercise in the control and experimental groups.

Muscle Damage Indices

Compared with baseline data, DOMS increased ($P < 0.05$) 48 hours after exercise (Figure 3). CK and LDH levels before exercise averaged 193 ± 80 IU·L^{-1} and 310 ± 59 IU·L^{-1}, respectively. CK increased ($P < 0.01$) 48 and 72 hours post exercise (Figure 4), whereas LDH increased ($P < 0.05$) 24, 48, and 72 hours post exercise (Figure 4). No differences were detected in muscle damage indices in the control group.

Collagen Damage Indices

Hydroxyproline and hydroxylysine levels before exercise averaged 6.6 ± 0.3 mg·L^{-1} and 0.64 ± 0.08 mg·L^{-1}, respectively. Compared with baseline data, serum hydroxyproline increased ($P < 0.001$) 24 hours post exercise, peaked at 48 hours ($P < 0.05$), and remained elevated up to 72 hours ($P < 0.001$) post exercise (Figure 5). Hydroxylysine was increased 48 hours post exercise compared with resting values ($P < 0.01$) (Figure 5). No differences were detected in collagen damage indices in the control group.

Lactate Assay

Plyometric exercise increased significantly ($P < 0.001$) in the blood lactate concentration, from 1.3 mM at rest to 6.1 mM immediately post exercise.

Discussion

In the present study, we decided, as a first step, to examine whether one bout of plyometric exercise would have any effect on collagen degradation of untrained individuals. Additionally, because different levels of plyometric training experience would potentially differentially affect the levels of muscle and collagen damage, it would be difficult to reliably standardize the volunteers based on their previous experience in plyometric training. Therefore, the rationale of this study was to investigate whether one bout of plyometric exercise could affect muscle damage and collagen degradation of untrained individuals.

Although several studies have examined the effects of plyometric exercise on indirect indices of muscle damage (e.g., 18), to our knowledge, this is the first attempt to examine the effects of plyometric exercise on indices of collagen degradation. This work showed that an intense acute bout of plyometric exercise increased muscle damage, DOMS, and serum indices of collagen breakdown without a concomitant decrease in functional capacity of muscles (as evidenced by the absence of significant changes in muscle strength and ROM).

Previous research has shown that plyometric exercise increases the risk of muscle damage because of the forces generated during ground impact and after intense eccentric contraction (14,18). CK and LDH activity in the blood are the most frequently used indices of muscle damage, and both were significantly increased after the plyometric exercise, which is in accordance with data from relevant studies (e.g., 11). Unexpectedly, however, indices of muscle performance (peak torque and ROM) did not change in our study. Perhaps the extent of muscle damage was not great enough to decrease muscle performance. It is also possible that the jumping exercises employed did decrease muscle performance without this decrement being detectable by the evaluation on the isokinetic dynamometer (because of different kinematic characteristics of leg extensions compared with jumps).

Supporting the supposition of non-extensive muscle damage after the jumping exercises used, DOMS increased only 48 hours post exercise. This increase could be considered moderate because the reported soreness values of 4 or 5 in the present study (on a scale of 1 to 10) indicate limited muscle damage (6). Consequently, these low soreness values are also in agreement with the absence of differences in muscle force and ROM. This lack of difference in muscle force and ROM may be associated with the execution of the plyometric exercises on a soft surface (wrestling-type mat) because it is known that performing plyometrics on a soft surface induces less muscle damage than a firm surface (18,24).

In the present study, we monitored changes in two indices of collagen breakdown after an acute bout of plyometric exercise. The observed large increases in hydroxyproline and hydroxylysine are most likely a result of connective tissue injuries provoked by the high forces of the plyometric exercise used. Additionally, evidence suggests that there is a rapid increase in collagen turnover within the connective tissue of muscle and tendon after strenuous exercise (15,17). Hence, the increases in hydroxyproline and hydroxylysine found in the present study may be the result of exercise-induced connective tissue injuries and increased collagen turnover as an adaptive response of connective tissue to muscle loading.

Hydroxyproline concentration peaked at 48 hours, and hydroxylysine was considerably higher at 48 hours compared with baseline. Both increases may relate to the well-described exercise-induced inflammatory response, which frequently takes place in muscle around the same time after a muscle-damaging exercise (16). Exercise-induced muscle injury has also been associated with inflammation in connective tissue, and inflammatory mediators may have promoted collagen breakdown via stimulation of collagenase production (27). Migration of neutrophils to the injury site and subsequent release of elastase, collagenases, and cytotoxic factors may also have induced the breakdown of surrounding connective tissue (3).

In the present study, we found an almost twofold increase in both hydroxyproline and hydroxylysine levels at 48 hours compared with baseline values after an acute bout of

plyometric exercise. On the contrary, the relevant studies have produced conflicting results, with some studies reporting no differences (4,13,30,31) and others increased levels (1,3) after exercise. The literature suggests that if the exercise employed was able to induce adequate muscle damage (as demonstrated by considerable changes in several physiological and biochemical muscle damage indices), then collagen damage had been induced (1,3). This observation is also supported by Brown et al. (4), who reported that concentric exercise that caused no muscle damage did not increase type I collagen concentration (another index of collagen breakdown), whereas eccentric muscle-damaging exercise increased the levels of type I collagen concentration.

Subjects in the present study were unaccustomed to plyometric exercise. It is well known that previous experience with muscle-damaging exercise has a prophylactic effect on muscle damage (7). Although the effect of acute muscle-damaging exercise on collagen breakdown of subjects accustomed to eccentric exercise is unknown (because all relevant studies used subjects unaccustomed to eccentric exercise), it seems rational to assume that repeated eccentric exercise bouts can lead to lesser collagen breakdown after a similar bout of muscle-damaging exercise performed later. Consequently, the present findings do not adequately apply to, for example, strength-trained individuals who are starting plyometric training.

Connective tissue remodelling may involve transient periods of mechanical weakness (32), and connective tissue could be damaged if insufficient time is allowed for adaptation (3). Therefore, the measurement of hydroxyproline and hydroxylysine levels in serum of competitive athletes (particularly those participating in sports involving many intense eccentric contractions) may have important practical value. Certainly, further studies should delineate the extent to which hydroxyproline and hydroxylysine concentrations reflect connective tissue breakdown and whether these indices are sensitive in describing exercise-induced effects on connective tissue status.

In conclusion, in this study, we showed that an intense bout of plyometric exercise increased muscle damage, DOMS, and increased serum indices of collagen breakdown without a concomitant decrease in functional capacity of muscles. Hydroxyproline and hydroxylysine levels in serum seem promising measures for describing exercise-induced collagen degradation, although these results should be applied with caution until further studies corroborate the present findings.

Practical Applications

An intense acute bout of plyometric exercise can increase muscle damage, DOMS, and serum indices of collagen breakdown without a concomitant decrease in the functional capacity of skeletal muscles. Therefore, coaches need to keep in mind that by using this type of activity (i.e., plyometric), despite the increased muscle damage that follows, it is not necessarily accompanied by decreases in skeletal muscle capacity. Furthermore, hydroxyproline and hydroxylysine levels in serum seem promising measures for describing exercise-induced collagen degradation. In the future, they can probably be used in the biochemical evaluation of strength athletes.

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