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Differential effects of strength training leading to failure versus not to failure on hormonal responses, strength, and muscle power gains

Mikel Izquierdo,¹ Javier Ibañez,¹ Juan José González-Badillo,² Keijo Häkkinen,³ Nicholas A. Ratamess,⁴ William J. Kraemer,⁵ Duncan N. French,⁶ Jesus Eslava,¹ Aritz Altadill,¹ Xabier Asiain,¹ and Esteban M. Gorostiaga¹

¹Studies, Research and Sport Medicine Center, Government of Navarra and ²Olympic Center of Sport Studies, Spanish Olympic Committee, Madrid, Spain; ³Department of Biology of Physical Activity, University of Jyväskylä, Jyväskylä, Finland, ⁴Department of Health and Exercise Science, The College of New Jersey, Ewing, New Jersey; ⁵Department of Kinesiology, Human Performance Laboratory, University of Connecticut, Storrs, Connecticut; and ⁶Institute of Sport, Northumbria University, Newcastle, United Kingdom

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Izquierdo, Mikel, Javier Ibañez, Juan José González-Badillo, Keijo Häkkinen, Nicholas A. Ratamess, William J. Kraemer, Duncan N. French, Jesus Eslava, Aritz Altadill, Xabier Asiain, and Esteban M. Gorostiaga. Differential effects of strength training leading to failure versus not to failure on hormonal responses, strength, and muscle power gains. *J Appl Physiol* 100: 1647–1656, 2006. First published January 12, 2006; doi:10.1152/jappphysiol.01400.2005.—The purpose of this study was to examine the efficacy of 11 wk of resistance training to failure vs. nonfailure, followed by an identical 5-wk peaking period of maximal strength and power training for both groups as well as to examine the underlying physiological changes in basal circulating anabolic and catabolic hormones. Forty-two physically active men were matched and then randomly assigned to either a training to failure (RF; $n = 14$), nonfailure (NRF; $n = 15$), or control groups (C; $n = 13$). Muscular and power testing and blood draws to determine basal hormonal concentrations were conducted before the initiation of training (T0), after 6 wk of training (T1), after 11 wk of training (T2), and after 16 wk of training (T3). Both RF and NRF resulted in similar gains in 1-repetition maximum bench press (23 and 23%) and parallel squat (22 and 23%), muscle power output of the arm (27 and 28%) and leg extensor muscles (26 and 29%), and maximal number of repetitions performed during parallel squat (66 and 69%). RF group experienced larger gains in the maximal number of repetitions performed during the bench press. The peaking phase (T2 to T3) after NRF resulted in larger gains in muscle power output of the lower extremities, whereas after RF it resulted in larger gains in the maximal number of repetitions performed during the bench press. Strength training leading to RF resulted in reductions in resting concentrations of IGF-1 and elevations in IGFBP-3, whereas NRF resulted in reduced resting cortisol concentrations and an elevation in resting serum total testosterone concentration. This investigation demonstrated a potential beneficial stimulus of NRF for improving strength and power, especially during the subsequent peaking training period, whereas performing sets to failure resulted in greater gains in local muscular endurance. Elevation in IGFBP-3 after resistance training may have been compensatory to accommodate the reduction in IGF-1 to preserve IGF availability.

strength training; repetition to failure; insulin-like growth factor 1; insulin-like growth factor-binding protein-3; testosterone; cortisol

THE OPTIMAL MANIPULATION OF strength training variables to maximize performance and to understand the physiological mechanisms underlying training-induced gains in strength and

power is of interest to the strength and conditioning researcher. It appears that training intensity with loads corresponding to 80–100% of one-repetition maximum (1RM) (12) of sufficient training volume is most effective for increasing maximal dynamic strength (5, 8–10, 12, 16). In addition, training leading to repetition failure (inability to complete a repetition in a full range of motion due to fatigue) has been of interest within the strength and conditioning profession. The primary role of training leading to repetition failure has been related to increased motor unit activation (3, 32) and high mechanical stress with its associated gene expression and damage and repair muscle process (11). However, some studies conclude that training to failure may not be necessary for optimal strength gains, because fatigue reduces the force that a muscle can generate (6, 24, 33). It appears that the choice of the number of repetitions with a given load may impact the extent of muscle damage and subsequent decrements in velocity and force production (21). Thus the role of resistance training to failure vs. nonfailure to optimize strength and power gains is unclear.

The discrepancies between these studies may in part result from differences in the volume and intensity of training, dependent variable selection, the pretraining physical fitness status, and muscle groups tested. Thus it may not be feasible to compare training programs using isokinetic or isometric single-joint training and testing devices (6, 32) with programs using dynamic multiple-joint free weight exercises (e.g., squat or bench press) (3, 24, 33) or athletic movements (e.g., jumping performance or bench throw power) (3, 33). In addition, the majority of studies used untrained subjects (6, 32) with non-equated intensity and/or volume of training (24, 33) during short-term periods (e.g., 6–9 wk) (3, 6, 32–33). Therefore, we hypothesized that a training approach not leading to repetition failure that equates volume and intensity would lead to similar gains in maximal strength. To date, no studies have isolated the effects of training leading to failure in a multigroup experimental design while controlling other variables in a long-term training protocol in elite athletes.

Homeostatic hormonal changes in response to strength training have been thought to play an important role in protein accretion, increased neurotransmitter synthesis, and strength

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development (17, 26, 28). The primary hormones studied typically include testosterone (both total and free), human growth hormone, and cortisol (25–27). Although the acute response of these hormones has been investigated to a large extent, periods of substantially increased volume or intensity have been shown to elicit alterations (e.g., reduced total testosterone, elevated cortisol), thereby indicating these hormones to be useful markers of chronic strength training stress (9, 17, 26, 28). In addition, resting insulin-like growth-factor 1 (IGF-1) and its binding proteins (e.g., IGFBP-3) concentrations have been studied and used as markers of stress (2, 4). In light of these observations, we hypothesized that a training approach not leading to repetition failure would result in less stress and subsequently fewer basal anabolic and catabolic hormonal changes. Therefore, the purpose of this study was to examine the efficacy of 11 wk of resistance training to failure vs. nonfailure, followed by a 5-wk peaking period of maximal and power training for increasing strength and power of the upper and lower body musculature. A secondary purpose was to examine the underlying physiological changes in basal circulating anabolic and catabolic hormones.

METHODS

Experimental Design and Approach to the Problem

A longitudinal research design using two different resistance training programs (e.g., training leading to repetition failure vs. not to failure) was used to parcel out differential training adaptations in hormonal changes, as well as in strength and power gains of the upper and lower body musculature. To eliminate any possible effect of confounding factors, several variables such as maximal relative strength (% of 1RM), average intensity and frequency of training, type of exercise, and volume were controlled by equating their values among the treatment groups. This was critical to the study design because differences in overall training intensity and volume have been proposed to influence performance adaptations (5, 12). After baseline testing, subjects were matched according to physical characteristics and muscle strength-power indexes and then randomly assigned to either a training to failure (RF; $n = 14$) or training to nonfailure (NRF; $n = 15$) training groups. As a control, a third population of subjects (C; $n = 13$) did not follow a set strength training intervention but continued practicing specific Basque ball games and were tested before and after a 16-wk period to assess the reliability of the observations. Testing was conducted on four occasions: before the initiation of training (T0), after 6 wk of training (T1), after 11 wk of training (T2), and after 16 wk of training (T3).

Subjects. A group of 42 Basque ball players, with 12.5 ± 5 yr of regular training and competition experience in Basque ball, volunteered to participate in a 16-wk training study. Basque ball is a name for a variety of court sports played with the bare hand, a racket, a wooden bat, or a basket propulsor (e.g., jai alai), against a wall. Nowadays, this game is widely played in several regions of Spain (including the Basque Country), as well as in several other Europe and American countries (e.g., Cuba, Mexico, Argentina). Subjects' initial characteristics are shown in Table 1. All the subjects were members of the Spanish national team of Basque ball. The study was performed during the first competitive period (February to June) before commencement of the XIV World Basque Ball championship. Each subject was informed carefully of the experimental procedures and about possible risks and benefits of the study and subsequently signed an institutionally approved informed consent document. The experimental procedures were approved by the Institutional Review Committee of the Instituto Navarro de Deporte y Juventud, according to the declaration of Helsinki. During the 5 mo before the experimental period, subjects trained two times a week for Basque ball and twice a

Table 1. *Physical characteristics during the experimental period*

	RF ($n = 14$)	NRF ($n = 15$)	Control ($n = 13$)
Age, yr	24.8 (SD 2.9)	23.9 (SD 1.9)	24.4 (SD 2.1)
Height, m	1.80 (SD 0.01)	1.81 (SD 0.01)	1.80 (SD 0.02)
Body mass, kg			
T0	81.1 (SD 4.2)	80.5 (SD 7.4)	81.1 (SD 7.2)
T1	80.7 (SD 4.4)	80.3 (SD 7.5)	
T2	81.5 (SD 5.1)	80.9 (SD 7.6)	
T3	80.3 (SD 3.9)	80.1 (SD 7.2)	82.4 (SD 6.7)
Body fat, %			
T0	13.4 (SD 4.1)	11 (SD 3.4)	13 (SD 5.2)
T1	13.5 (SD 3.7)	10.8 (SD 3.2)	
T2	12.7 (SD 3.2)	11.1 (SD 3.4)	
T3	12.1 (SD 3.8)*‡	10.3 (SD 3.1)*†	13.1 (SD 5.2)
BMI			
T0	24.9 (SD 2.5)	24.6 (SD 1.9)	25.2 (SD 2.3)
T1	25 (SD 2.5)	24.5 (SD 1.8)	
T2	24.8 (SD 2.3)	24.6 (SD 1.9)	
T3	24.6 (SD 2.3)	24.4 (SD 1.8)	25.1 (SD 9.2)

Values are means and SD. NRF, non-repetition-to-failure group; RF, repetition to failure group; BMI, body mass index; T0, before initiation of training; T1, after 6 wk of training; T2, after 11 wk of training; T3, after 16 wk of training. * $P < 0.05$ from the corresponding time point T0. † $P < 0.05$ from the corresponding time point T1. ‡ $P < 0.05$ from the corresponding time point T2.

week for strength and endurance training, and they played in one official Basque ball game per week. Basque ball practice sessions lasted 60–90 min and usually consisted of various skill activities at different intensities and 45 min of continuous play with only brief interruptions by the coach. The strength training program required each subject to perform a combination of free weight and fixed machine weight exercises in each session, mainly consisting of three sets of six to eight repetitions, with a relative intensity of 70–80% of 1RM. The exercises completed in each weight training session were the supine bench press, shoulder press, lateral pull-down, parallel squat, knee flexion, standing leg curl, abdominal crunch, and another exercise for the trunk extensors. The total duration of each strength training session was 35–40 min. The running endurance program consisted of one training session per week and lasted 20–30 min at a self-adjusted intensity. The subjects were not taking exogenous anabolic-androgenic steroids or other drugs (excluded by specific tests) expected to affect physical performance or hormonal balance before or during this study.

Testing procedures. Subjects completed a 2-day experimental protocol separated by 2 days. All players were tested on the same day, and the tests were performed in the same order. During the first testing session, each subject was assessed using two countermovement jump (CMJ) protocols, performed on a contact platform 1) using body-weight and 2) with an external load of 30% of body mass (CMJ 30%), administered using a weighted barbell positioned across the shoulders. In addition, each subject was tested for 1RM and power output using a relative load 60% of their 1RM in bench press and parallel squat exercises.

During the second testing session, each subject performed maximal repetitions to failure with a submaximal load of 75% of 1RM for the bench press and parallel squat. All of the subjects were familiar with the testing protocol, as they had been previously tested on several occasions during the season with the same testing procedures. The test-retest intraclass correlation coefficients for all strength and power variables were greater than 0.91, and the coefficients of variation ranged from 0.9 to 2.3%. Training was integrated into the test week schedules. Body mass and percent body fat (estimated from the thickness of seven skinfold sites) were taken at the beginning of the second testing session (22).

Jumping test. Subjects were asked to perform a maximal vertical CMJ on a contact platform (Newtest OY, Oulu, Finland) without any load and with an extra load of 30% of body mass loaded on a barbell kept on the shoulders throughout. Using a preparatory countermovement, subjects initiated the jump from an extended leg position, descended to 90° knee flexion, and immediately performed an explosive concentric action for maximal height. The jumping height was calculated from the flight time. Two maximal jumps were recorded interspersed with ~10 s of rest, and the peak value was used for further analysis.

Bench press and parallel squat muscular performance. A detailed description of the maximal strength and muscle power testing procedures can be found elsewhere (20). In brief, lower and upper body maximal strength was assessed using 1RM bench press (1RM_{BP}) and parallel squat (1RM_{HS}) actions. In the 1RM_{BP} protocol, the test began with the subject lowering the bar from a fully extended arm position above the chest until the bar was positioned 1 cm above the subject's chest. From that position (supported by the bottom stops of the measurement device), the subject was instructed to perform a purely concentric action (as fast as possible) maintaining a shoulder position of 90° abduction position. This completed a successful repetition. No bouncing or arching of the back was allowed.

The 1RM_{HS} began with the bar on the shoulders with the knees and hips in the extended position. The subjects descended to the parallel to the floor thigh position. On the verbal command "up," the subject ascended (as fast as possible) to a full knee extension of 180°. All tests were performed using a Smith machine in which the barbell was attached at both ends with linear bearings allowing only vertical movements.

A warm-up for both half squat and bench press consisted of a set of five repetitions at loads of 40–60% of the perceived maximum. Thereafter, four to five separate single attempts were performed until 1RM was attained. The rest between maximal attempts was always 2 min.

Power output of the leg and arm extensor muscles was measured in the concentric portion of the parallel-squat and bench-press actions by using a relative load 60% of 1RM. The subject was instructed to lift the bar as fast as possible. Two testing trials were recorded and the best trial was taken for further analyses.

During the parallel-squat test, bar displacement, average velocity (m/s) and mean power (W) were recorded by linking a rotary encoder to the end of the bar. The rotary encoder (Computer Optical Products, Chatsworth, CA) recorded the position and direction of the bar within an accuracy of 0.2 mm and timed events with an accuracy of 1 ms. Customized software (JLML I+D, Madrid, Spain) was used to calculate the power output and average velocity for each repetition.

Parallel squat and bench press endurance test. Upper and lower body muscular endurance was assessed at pretraining by measuring the maximal number of repetitions until failure with 75% of 1RM for both the bench press and parallel squat exercises, respectively. During training (e.g., at T1, T2, and T3), muscle endurance tests were performed with the same absolute load (75% of 1RM) used at pretraining. The subjects were asked to move the bar as fast as possible during the concentric phase of each repetition until failure. Failure was defined as the time point when the bar ceased to move, if the subject paused more than 1 s when the leg or arms were in the extended position, or if the subject was unable to complete each repetition in a full range of motion. During the first repetitions the cadence was controlled with a metronome at a frequency of 19 Hz. As fatigue increased and performance of repetitions became progressively more difficult, a self-selected cadence under 19 Hz was allowed with the time of rest between the repetitions remaining constant (1 s).

Assessment of resting hormone concentrations. Resting blood samples were collected between 0800–0900 on the first testing day after a 12-h overnight fast and abstinence from strenuous exercise for 36–48 h. In all cases blood samples were obtained via venipuncture from an antecubital forearm vein by using a 20-gauge needle and

Vacutainers. Whole blood was centrifuged at 3,000 rpm (4°C) for 15 min, and the resultant serum was then removed and stored at –20°C until subsequent analysis. Circulating concentrations of total testosterone and cortisol were determined using commercially available enzyme immunoassay kits (Diagnostic Systems Laboratories, Webster, TX). Plasma growth hormone (GH) concentrations were determined by ¹²⁵I liquid-phase immunoradiometric assay (Nichols Institute Diagnostics, San Juan Capistrano, CA). IGF-1 and IGFBP-3 concentrations were established by enzyme-linked immunosorbent assay (ELISA) kits (Diagnostic Systems Laboratories, Webster, TX) according to the manufacturer's procedures. All samples were assayed in duplicate and were decoded only after analyses were completed (i.e., blinded analysis procedure). The minimum enzyme immunoassay detection limits for total testosterone and cortisol were 0.14 and 2.76 nmol/l, respectively. Immunoradiometric assay detection limits for GH were 0.04 ng/ml. Minimum ELISA detection limits for IGF-1 and IGFBP-3 were 0.0013 and 0.0014 nmol/l, respectively. The coefficient of intra-assay variation was 4.4% for total testosterone, 5.1% for cortisol, and 6.0 and 6.4% for IGF-1 and IGFBP-3, respectively. All samples were analyzed in the same assay for each analyte. For all procedures, samples were only thawed once before the analysis.

Training programs. All training sessions started with a general warm-up and included cool-down periods of 5–10 min of low-intensity aerobic and stretching exercises. A trained researcher supervised each workout session carefully and recorded the compliance and individual workout data during each training session so that exercise prescriptions were properly administered during each training session (e.g., number of repetitions, rest, and velocity of movement). Compliance with the study was 100% of the programmed sessions.

Both treatment groups were asked to train two times per week for 16 wk to perform dynamic resistance exercise from 45 to 60 min per session. A minimum of 2 days elapsed between two consecutive training sessions. Resistance exercise choice and order were identical for the two treatment groups. During the whole training period, the core exercises were the parallel squat and bench press, in addition to supplementary strengthening exercises for selected muscle groups (shoulder press, lateral pull-down, abdominal crunch, trunk extension, and standing leg curl). In addition, the training program included ballistic exercises (e.g., countermovement vertical jumps, loaded vertical jumps, sprints, and various throwing exercises with a 1-kg ball) during the last 5 peaking wk of explosive strength training (from T2 to T3). Subjects performed all free-weight bench press and squat training using a standard 20-kg barbell.

Both groups performed a 16-wk periodized resistance training program divided into three periods of 5–6 wk. One group performed high-fatigue strength training exercise to failure (RF), whereas the other performed the same volume and intensity but did not complete sets leading to failure (NRF). The assigned training intensities were gradually increased during the course of the 16-wk training period on the basis of the athletes' 10- (10RM) and 6-repetition maximum (6RM) testing, using a repetition maximum approach. In the RF training group, the load was reduced and the training continued immediately when the load was paused for more than 1 s or if the subject was unable to reach the full extension position of the arms or leg. The training load was reduced three to four times in a training session during RF training, whereas load remained constant in the NRF group.

During the first 6 wk of training (from T0 to T1), the RF group trained with three sets of 10RM for the bench press and 80% of 10RM for the parallel squat, whereas the NRF group performed six sets of five repetitions at a similar intensity (10RM). During the middle 5 wk of training (from T1 to T2), subjects in the RF group trained at 6RM and 80% of 6RM and performed three sets for the bench press and parallel squat, respectively, whereas the NRF group performed six sets of three repetitions at a similar intensity. During the final 5 wk of training (from T2 to T3), both groups trained at 85–90% of 1RM

(~5RM), two to four repetitions per set, and performed three sets for both upper and lower extremity exercises and performed the ballistic training program (e.g., vertical countermovement jumps, loaded vertical jumps, sprint runs, and various throwing exercises with a ball of 1 kg). In addition, the subjects performed bench press sets with loads ranging from 40 to 45% of 1RM. During this phase, subjects performed three to four repetitions per set and three to five sets of each exercise in a ballistic manner. Approximately 2-min rest periods were allowed between each set and each exercise. This ballistic strength training was included because it has been shown the most effective way to enhance explosive strength and speed (5). During the first 11 wk (from T0 to T2), these different trainings protocols (RF vs. NRF) enabled comparison of two equal volume and relative training intensity programs on hormonal responses and muscle power- and strength training-induced changes of the upper and lower extremity muscles. In addition, the last 5 peaking weeks (from T2 to T3) were used to produce a similar "rebound effect" for all groups and to avoid overreaching (8–10, 12).

During the squat lifts and bench press exercises, the subjects were instructed carefully to perform all the concentric actions at the highest possible speed. The eccentric actions were performed at low velocity during the "lowering" phase of the movement. Apart from the formal requirements of this study, both groups performed similar whole body strength training programs (60–70% of 1RM, 8–10 repetitions) involving selected muscle groups (shoulder press and lateral pull-down for the upper body abdominal crunch and another exercise for the trunk extensors, and the standing leg curl).

Statistical Analyses

Standard statistical methods were used for the calculation of means and SD. One-way ANOVA was used to determine any differences among the three groups' initial strength, power, and hormonal profile. The training-related effects were assessed by a two-way ANOVA with repeated measures (groups \times time). When a significant *F*-value was achieved, Scheffé's post hoc procedures were performed to locate the pairwise differences between the means. Selected absolute changes were analyzed via one-way ANOVA. Statistical power calculations for this study ranged from 0.75 to 0.80. The $P \leq 0.05$ criterion was used for establishing statistical significance.

RESULTS

Body Composition

At the beginning of the training program, no significant differences were observed between the groups in age, height, body mass, or percent body fat. A significant decrease in percent body fat was observed at T3 for NRF compared with T0 and T1, as well as at T3 for RF compared with T0 and T2. A significant decrease in body mass was observed at T3 for RF, whereas no significant differences in body mass were observed for NRF at any point (Table 1).

Maximal Muscle Strength and Power

The maximal strength results are presented in Fig. 1. No significant differences were observed between the groups in 1RM_{HS} and 1RM_{BP} at T0. Significant increases took place in 1RM_{BP} for the RF and NRF groups at T1 and T2. No significant differences were observed in the magnitude of the increase in 1RM_{BP} between RF and NRF at T1 (9 and 8%) and T2 (20 and 20%), respectively (Fig. 1A). During the peaking phase (from T2 to T3), no significant changes occurred in 1RM_{BP} in either RF or NRF. Significant increases in 1RM_{HS} were observed in the RF and NRF groups at T1 and T2. No significant differences were observed in the magnitude of the

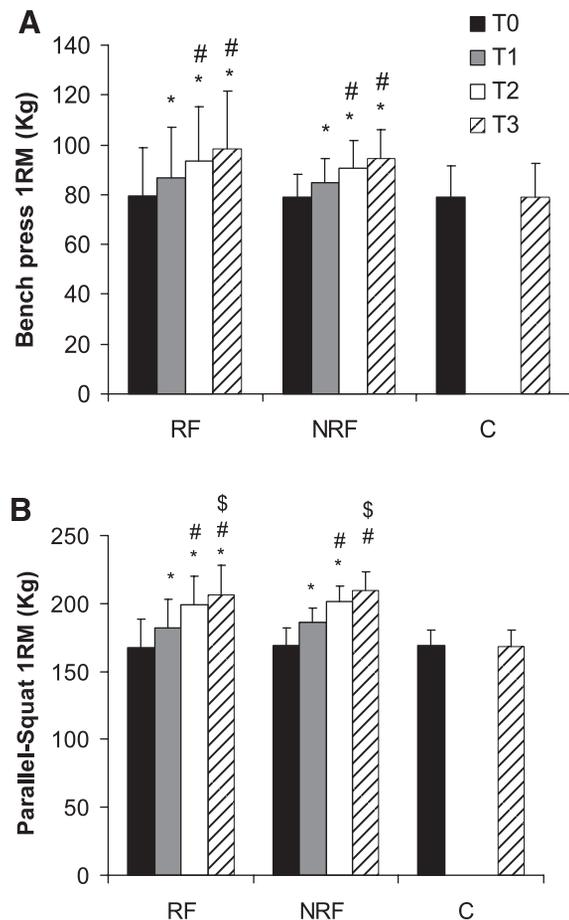


Fig. 1. Maximal bench press (A) and parallel squat strength (B) during the experimental period. * $P < 0.05$ from the corresponding time point T0 (before training). # $P < 0.05$ from the corresponding time point T1 (after 6 wk of training). \$ $P < 0.05$ from the corresponding time point T2 (after 11 wk). T3, time point after 16 wk of training. 1RM, 1-repetition maximum; NRF, no repetition to failure group; RF, repetition to failure group; C, control group. Data are means and SD.

increase in 1RM_{HS} between RF and NRF at T1 (9 and 10%) and T2 (19 and 20%) compared with T0, as well as at T3 (3 and 3%) compared with T2, respectively (Fig. 1B). No significant differences for any variable were observed in the C group over the training period.

No significant differences were observed between the groups in muscle power output of either the lower or upper extremity at T0. Significant increases were observed in muscle power output for 60% of 1RM_{BP} for the RF and NRF groups at T2. No significant differences were observed in the magnitude of the increase between RF and NRF at T2 (20 and 23%) compared with T0, respectively (Fig. 2A). During the peaking phase (from T2 to T3), no significant changes occurred in muscle power output for 60% of 1RM_{BP} either in RF or NRF.

Significant increases were observed in muscle power output at the 60% of 1RM_{HS} for the RF and NRF groups at T1 and T2 compared with T0 and T1, respectively. No significant differences were observed in the magnitude of the increase in muscle power output at the 60% of 1RM_{HS} between RF and NRF at T2 (26 and 29%) compared with T0 (Fig. 2B). However, only NRF showed a significant increase at T3 compared with T2.

Significant increases were observed in the height of CMJ and CMJ30% for the RF and NRF groups at T2 compared with

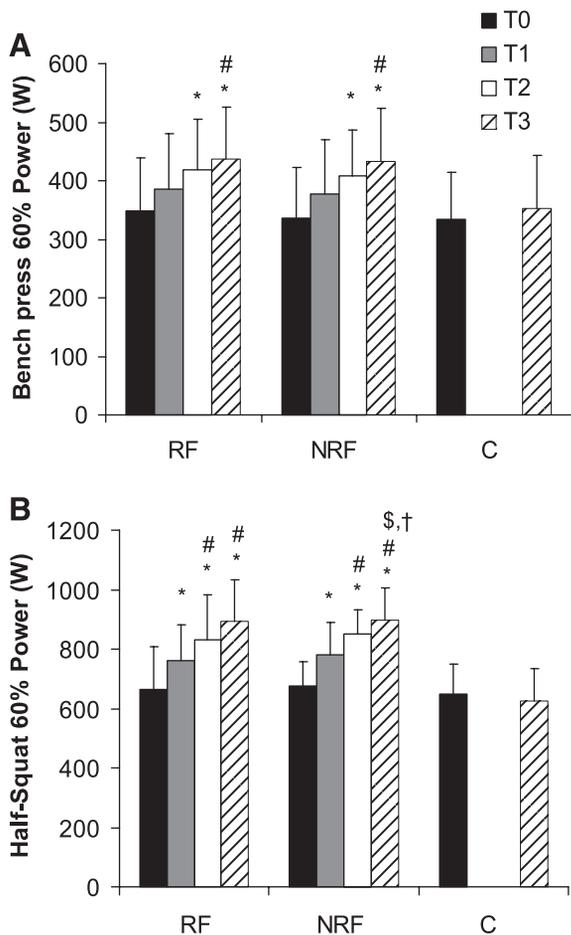


Fig. 2. Bench press (A) and parallel squat (B) muscle power output during the experimental period. * $P < 0.05$ from the corresponding time point T0. # $P < 0.05$ from the corresponding time point T1. \$ $P < 0.05$ from the corresponding time point T2. † $P < 0.05$ from relative change at point time T1 between the groups. Data are means and SD.

T0. No significant differences for any lower or upper body maximal muscle power variables were observed in the C group over the training period (Fig. 3).

The number of repetitions performed with 75% of 1RM_{BP} increased significantly at T1 and T2 in both RF and NRF. Significant group \times time interaction was observed for the number of repetitions performed with 75% of 1RM_{BP}, with a significantly larger ($P < 0.01$) magnitude of increase for RF at T1 (46%) and T2 (85%) compared with T0 than that recorded in NRF (28 and 69%, respectively) (Fig. 4A). Only RF showed a significant increase at T3 compared with T2.

The number of repetitions performed with 75% of 1RM for the parallel squat increased significantly at T2 compared with T0 in both RF and NRF. No significant differences were observed in the magnitude of increase between RF and NRF at T2 (66 and 69%, respectively), compared with T0, as well as at T3 compared with T0 (Fig. 4B).

Hormonal Data

Resting serum hormonal data are presented in Figs. 5–8. No significant differences were observed between the groups in hormonal data at T0. Serum total testosterone concentrations were significantly elevated in NRF at T2 compared with T0

and T1. There was a significant group \times time interaction, with a significantly larger ($P < 0.05$) mean improvement in the serum total testosterone concentration for NRF at T2 compared with T0 and T1 (6 and 12%; $P < 0.05$, respectively) than that recorded in RF (0 and -1%) (Fig. 5). In addition, there was a significant group \times time interaction with a larger ($P < 0.05$) magnitude of reductions in serum total testosterone concentration for NRF at T3 (-11%) compared with T2 than that recorded in RF (2%). Serum cortisol concentrations were significantly reduced in NRF at T2 compared with T0, whereas in RF a tendency ($P = 0.07$) for elevation was observed at T2 and T3 compared with T0. In addition, there was a significant group \times time interaction with a larger ($P < 0.05$ –0.01) magnitude of reductions in NRF at T2 and T3 compared with T0 than that recorded in RF (Fig. 6). No significant changes were observed at any point in the testosterone-to-cortisol ratio. Serum IGF-1 concentrations decreased significantly in RF at T2 and T3 compared with T0, T1, and T2 respectively (Fig. 7). Serum IGFBP-3 concentrations were elevated significantly in RF at T1, T2, and T3 compared with T0, whereas NRF only showed a significant elevation at T3 (Fig. 8). No significant differences in serum GH were observed in either training group at any time point. In addition, no significant hormonal changes were observed in the C group at any time point.

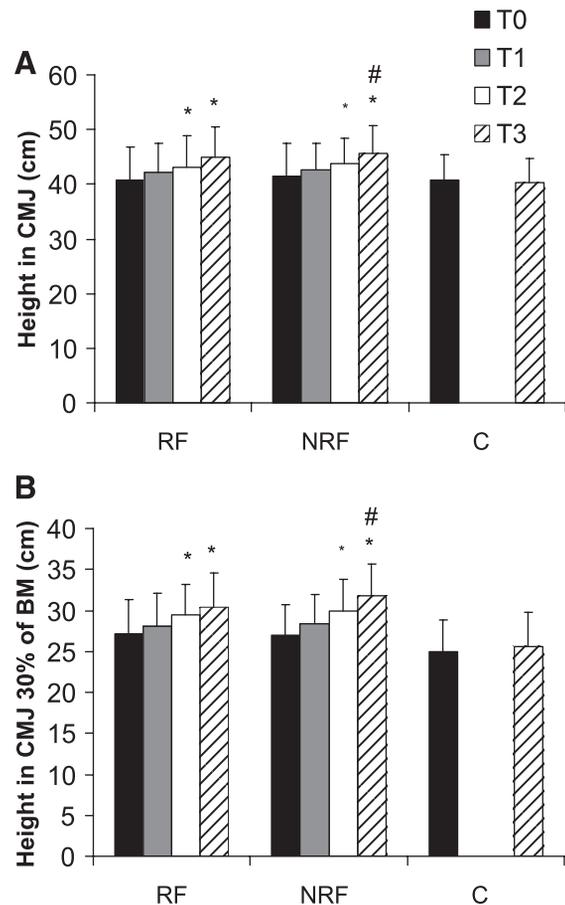


Fig. 3. Height in the countermovement jump (CMJ) without load (A) and with extra load of 30% of body mass (BM) (B) during the experimental period. * $P < 0.05$ from the corresponding time point T0. # $P < 0.05$ from the corresponding time point T1. Data are means and SD.

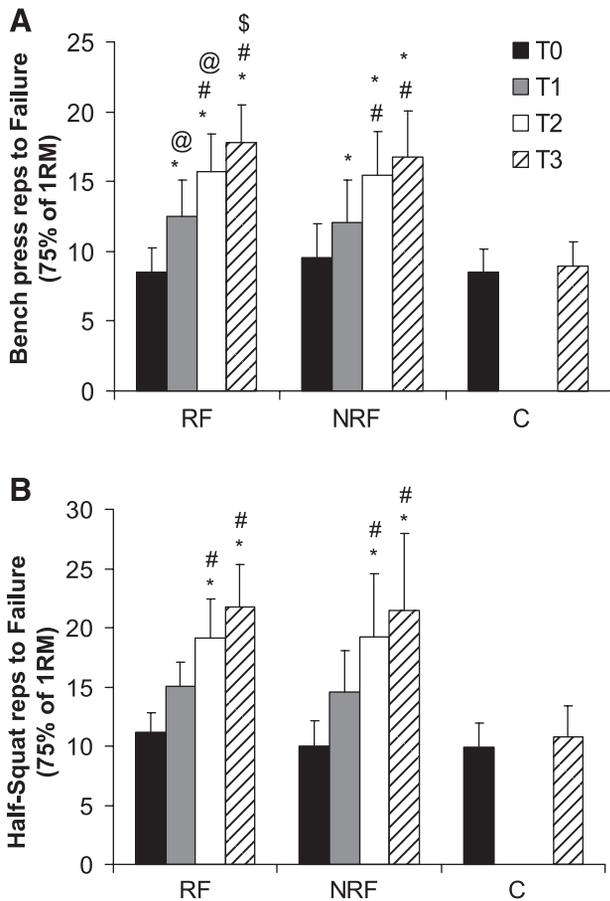


Fig. 4. Maximal number of repetitions until failure with 75% of 1RM for both the bench press (A) and parallel squat (B) exercises during the experimental period. * $P < 0.05$ from the corresponding time point T0. # $P < 0.05$ from the corresponding time point T1. \$ $P < 0.05$ from the corresponding time point T2. @ $P < 0.05$ from relative change at time point T0 between the groups. Data are means and SD.

DISCUSSION

The major findings of this study were that, after the 11-wk training period (from T0 to T2), 1) similar gains in bench press 1RM, parallel squat 1RM, muscle power output of the arm and

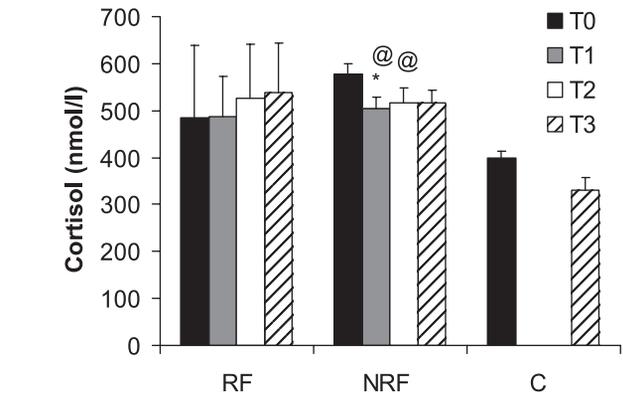


Fig. 6. Resting serum cortisol concentrations during the experimental period. * $P < 0.05$ from the corresponding time point T0. @ $P < 0.05$ from relative change at time point T0 between the groups. Data are means and SD.

leg extensor muscles, and maximal number of repetitions performed during parallel squat were observed between RF and NRF; and 2) the RF group experienced larger gains in the maximal number of repetitions performed during the bench press. During the peaking phase (from T2 to T3), 3) larger gains in muscle power output of the lower extremity were observed after the NRF training approach, and 4) larger gains were found in the maximal number of repetitions performed during the bench press after RF training approach. In addition, long-term strength training leading to repetition to failure resulted in reductions in resting serum IGF-1 concentrations and elevations in IGFBP-3, whereas the NRF group experienced an elevation in resting serum testosterone concentrations and reductions in cortisol. These data indicated that performing repetitions not to failure provided favorable conditions for improving muscle power whereas performing sets to failure resulted in greater gains in local muscular endurance.

Few studies have isolated the effects of training leading to repetition failure using a multigroup experimental design, while controlling other variables, in resistance-trained individuals (3, 6, 24, 32, 33). These studies have shown that short-term training (<9 wk) leading to repetition failure produces greater improvements in strength (3, 32) or may not be necessary for optimal strength gains (6, 32–33) when compared with

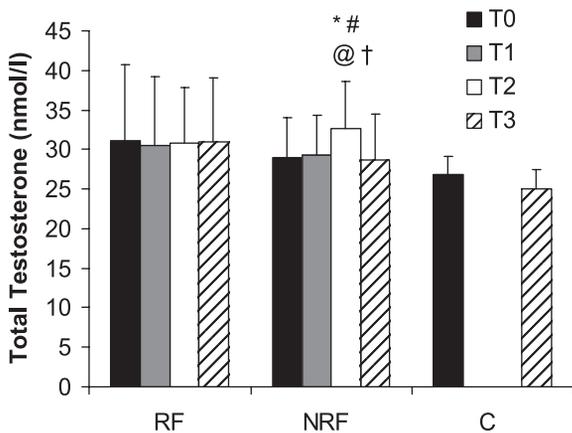


Fig. 5. Resting serum total testosterone concentrations during the experimental period. * $P < 0.05$ from the corresponding time point T0. # $P < 0.05$ from the corresponding time point T1. @ $P < 0.05$ from relative change at time point T0 between the groups. † $P < 0.05$ from relative change at point time T1 between the groups. Data are means and SD.

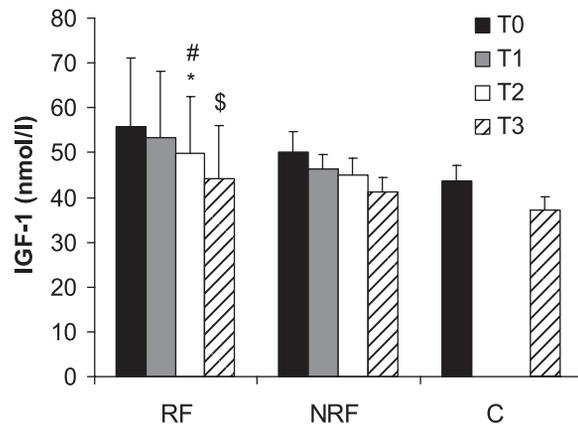


Fig. 7. Resting serum insulin-like growth factor 1 (IGF-1) concentrations during the experimental period. * $P < 0.05$ from the corresponding time point T0. # $P < 0.05$ from the corresponding time point T1. \$ $P < 0.05$ from the corresponding time point T2. Data are means and SD.

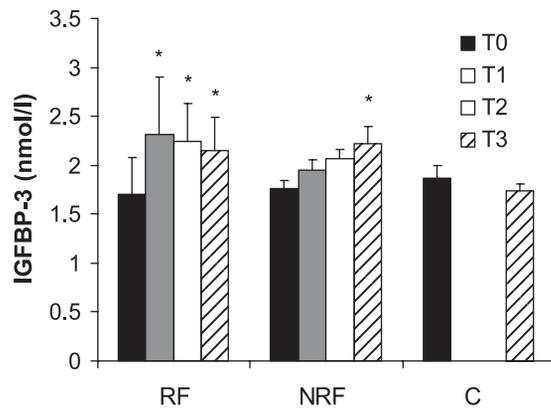


Fig. 8. Resting serum IGF binding protein-3 (IGFBP-3) concentrations during the experimental period. * $P < 0.05$ from the corresponding time point T0. Data are means and SD.

a nontraining to repetition failure approach. The discrepancies between the results of these studies may in part result from differences with respect to the volume and intensity of training, dependent variable selection, the pretraining physical fitness status, and muscle groups tested. Thus it may not be feasible to compare training programs using isokinetic or isometric single-joint training or testing devices (6–32) with training programs using dynamic multijoint free weight (e.g., squat or bench press) (3, 32–33) or power (e.g., vertical jumps, ballistic bench press) (3, 33) exercises. In addition, the majority of studies have used untrained subjects (6, 32) and involved a nonequated high intensity and/or volume of training (24, 33) during short-term periods (e.g., 6–9 wk) (3, 6, 32–33). Our results support previous studies showing that training to failure did not result in greater gains in strength in resistance-trained men. Therefore, the role of performing sets to failure (and how many) requires further research in the strength-trained population.

Strength training consisting of repetitions performed to failure has been shown to lead to greater strength gains in some studies. Rooney et al. (32) showed that untrained subjects who performed repetitions to failure without rest intervals between repetitions attained significantly greater (56%) mean increases in dynamic strength of the elbow flexors than subjects training without assistance but permitted resting for 30 s between repetitions (41%) during a short-term training program (6 wk). Both training groups performed the same number of lifts at the same relative intensity. Likewise, Drinkwater and coworkers (3) reported greater bench press strength and power gains after 6 wk of training consisting of training to failure with four sets of six repetitions every 260 s (9.5 and 10.6%, respectively) compared with training with eight sets of three repetitions every 113 s (5 and 6.8%, respectively). Both studies suggested that training to failure was a critical strength training stimulus. The authors hypothesized that greater accumulation of metabolites (with no rest in between repetitions) and recruiting additional motor units to maintain force output as fatigue increases (3, 21, 32) could potentially maximize strength gains, which in theory would support training to muscular failure. However, it is unclear whether motor unit activity is enhanced, and the impact of accumulative fatigue potentially resulting in overtraining needs to be considered. Thus evidence does support training to failure but it is unclear how to optimally include it into program design (e.g., number of sets per work-

out or number of repetitions performed to failure). On the contrary, other studies have shown that training to failure may not be necessary for optimal training gains (6, 24, 33). However, the experimental groups were not carefully equated and controlled for volume and intensity.

The use of the upper and lower body maximal strength training followed by a 5-wk peaking period of maximal and power training in the present study is unique and provides meaningful data for program design of strength and/or power athletes. Performing each set not to muscular failure led to larger gains in muscle power output of the lower extremity. These data indicate that during the peaking phase (from T2 to T3), following a training period, leading repetition to failure does not provide a better stimulus for improving muscle power and may lead to reduced power output during long-term strength training. Maximum effort to each repetition is critical to power training. Many programs targeting maximal power (e.g., weightlifting) perform few explosive repetitions per set. The rationale is to minimize fatigue so that maximal effort (and power output) can be applied to each repetition (21). Our results indirectly support this training philosophy because greater gains in power were observed when training was not performed to muscular failure.

A different pattern of strength and power gains was observed when performing repetitions to failure between the upper and leg extremity muscles. Similar to previous studies (13), these differences may have been explained by a difference in the initial level of conditioning between knee extensors and upper body muscles and may be related to differences in the pattern and/or intensity of daily physical use in normal life (13), as well as in practicing Basque ball training. The quadriceps muscle, owing to its weight-bearing role during habitual physical activity, would be more likely to be at a higher initial level of conditioning than the upper body muscles, which have been shown to be used less frequently. From our results, it appears that the use of training not to failure might be more efficient for training the upper and specially the lower extensor muscles. In addition, one may hypothesize that the aerobic nature of the Basque ball training and games in addition to repetition to failure approach might interfere with optimal gains in maximal and explosive strength in the legs (13), especially during prolonged training periods.

The results of the present study demonstrated that training to failure did provide an advantage, for upper body musculature during maximal number of repetitions performed for the bench press, for improving local muscular endurance. In this case, training for enhanced muscle endurance implies that the athlete needs to train in a semifatigued to fatigued state. Thus training to failure for upper body musculature may provide a novel stimulus as the greater fatigue encountered may further enhance muscle endurance. It is unclear why similar results were not observed during lower body muscle endurance assessment (e.g., maximal parallel squat repetitions). It may be argued that the lower extremities in these elite athletes were highly conditioned. Thus the added effects of training to failure were not evident based on the level of conditioning. However, further research is warranted to examine the role of training to failure in different muscle groups.

Examination of resting concentrations of anabolic and catabolic hormones may provide insight as to the physiological mechanisms involved for higher levels of muscular perfor-

mance and subsequent adaptations. Large increases in the volume and/or intensity of resistance training may overstress the neuroendocrine system, leading to altered circulating hormonal concentrations (9). However, less is known concerning the effects of resistance training to failure vs. not resistance training to failure on resting hormonal concentrations. It may be hypothesized that performing each set to failure may increase the risk of overtraining over long-term periods. Thus a change in hormonal profile may ensue.

No significant differences in resting serum GH concentrations were observed in either training group in the present study. This was not surprising considering that resting GH concentrations typically do not change during traditional strength training (25), despite the enhanced adaptation ability for tissue remodeling to acute exercise-induced response after resistance exercise (27). Our data support previous investigations demonstrating a lack of change in resting GH concentrations. Although overnight pulsatility profiles and other molecular weight GH variants were not measured in the present investigation, these have been shown to be altered by high-volume strength training (29) and require further investigation.

The response of IGF-1 to chronic resistance training is less clear. Short-term strength training studies have reported no change in resting concentration of IGF-1 (25–27), whereas long-term studies in men and women have reported significant elevations in resting IGF-1 (2, 23, 28). Acute overreaching, resulting from a dramatic increase in volume and/or intensity of training, has been shown to reduce IGF-1 concentrations by 11% (30) but return them to baseline when normal training resumed over the next cycle (30). In the present study, resting serum IGF-1 concentrations were significantly reduced in RF at T2 and T3 compared with T0, T1, and T2, respectively, whereas no significant reductions were observed in the NRF group. Collectively, our results indicate that chronic IGF-1 adaptations after resistance training may be mediated, in part, by volume and intensity manipulation (26, 30).

IGF-1 concentrations are highly regulated by GH secretion. Although the mechanisms of GH-activated IGF-1 gene expression remain unclear, the GH superfamily stimulates IGF-1 secretion by the liver and other tissues. Although no resting GH changes were observed in the present study, it is possible that nonmeasured alterations in GH pulsatility (e.g., overnight or at a time of day not examined in the present study) or other non-22-kDa molecular weight GH variants could have occurred. Nindl et al. (29) showed reduced GH pulsatility overnight after high-volume resistance exercise presumably due to the high level of stress. In addition, delayed secretion of IGF-1, e.g., 3–9 h, after GH-stimulated messenger RNA synthesis has been shown (26). Therefore, the IGF-1 concentrations observed in the present study may have reflected a delayed response in conjunction with GH alterations (e.g., reduced pulsatility), which could explain a reduction in IGF-1 independent of GH changes considering that each hormone was sampled at the same time point. Nevertheless, the higher stress of training may have led to reduced IGF-1 concentrations in the RF group.

Few studies have examined chronic circulating concentrations of IGF-1 after long-term resistance training. The reduction in resting IGF-1 observed in RF occurred parallel to elevations in serum IGF-1 concentrations with only small elevations observed at T3 in the NRF group in the present

study. Interestingly, Elloumi et al. (4) have proposed that a reduction in resting IGF-1 may be used as a marker of overtraining. Borst et al. (2) demonstrated a reduction in IGF-1 concentrations that paralleled a concomitant elevation in IGF-1 concentrations after a 25-wk training period. On the basis of our data, it may be hypothesized that the elevation in IGF-1 may have been compensatory to accommodate the reduction in IGF-1 to preserve IGF availability (26). However, further research is required to examine the impact of resistance training intensity and volume manipulation on training-related changes in IGF-1.

Circulating testosterone and cortisol have been proposed as physiological markers to evaluate the tissue-remodeling process during a strength training period (17, 26). In the present study, an elevation in total testosterone was observed at T2 in NRF whereas no differences were observed in RF. It is unclear why an elevation was only observed at T2 in NRF. However, changes in resting total testosterone concentrations have shown variable responses such that no apparent consistent patterns have been observed. Rather, it appears that resting concentrations reflect the current state of muscle tissue (or perhaps the response to the previous workout performed before blood sampling) such that elevations or reductions may occur at various stages depending on the volume and intensity of the training stimulus (17). Elevated resting serum concentrations of testosterone (25, 28) have been reported during resistance training studies, whereas several studies have shown reductions (1). In contrast, several studies have shown no change in testosterone (15, 19). In addition, resting total testosterone concentrations have been shown moderately to correlate with strength performance (31), thereby suggesting that the acute response may play a more prominent role. Therefore, the lack of change observed in RF was not surprising; however, the elevation at T2 in NRF may have reflected this inconsistency as a transient response to the previous workouts. Lastly, the free testosterone response was not measured in the present study. Therefore, any potential changes in the biologically active form of testosterone were not identified.

Significant reductions were observed at T2 in NRF in resting serum cortisol concentrations whereas no changes were observed in RF. In addition, there were no changes observed in the testosterone-cortisol ratio. Elevations (18), reductions (15, 28), and no changes (16–17) in resting cortisol concentrations have been reported during resistance training. Thus the cortisol response may also show great interindividual variability. It may be hypothesized that not training to failure may reduce the overall stress of resistance training; consequently the cortisol response may be attenuated, and, therefore, the anabolic status of skeletal muscle enhanced. However, although it is attractive the use of the testosterone-to-cortisol ratio as a common marker to indicate a potential anabolic or catabolic state, positively related with performance improvements, it appears to be an oversimplification. In fact, it has been suggested that a transient drop in the testosterone-to-cortisol ratio below 45% cannot be interpreted as a sign of overstrain or neuroendocrine dysfunction and may not be associated with decreased performance (9, 14, 26). Indeed, in some circumstances it may be related to a temporary positive stress stimulus and may even be expressed in a beneficial effect on performance (14). Thus some authors have shown a decrease in the testosterone-to-cortisol ratio or the free testosterone-to-cortisol ratio to be

associated with an increase (8–9, 34) or no change in performance (16). Our data support this hypothesis to a certain extent. Although a cortisol reduction was observed in NRF, no changes were observed in the testosterone-to-cortisol ratio. Thus the use of the testosterone-to-cortisol ratio remains questionable.

In conclusion, both training to failure and training not to failure resulted in similar gains in 1RM strength, muscle power output of the arm and leg extensor muscles, and maximal number of repetitions performed during the parallel squat. However, during the peaking phase (from T2 to T3) larger gains in muscle power output of the lower extremity were observed after the preceding NRF training approach. Training to failure resulted in larger gains in the number of repetitions performed in the bench press. Strength training leading to repetition to failure resulted in reductions in resting concentrations of IGF-1 and elevations in IGFBP-3, whereas not training to failure resulted in reduced resting cortisol concentrations, an elevation in resting serum total concentration at T2, and an elevation IGFBP-3. Briefly, this investigation demonstrated a potential beneficial stimulus of resistance training not leading to failure for improving strength and power, especially during the subsequent peaking training period. However, training leading to repetition failure seemed to more beneficial for enhancing upper body local muscular endurance.

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