

# DETRAINING AND TAPERING EFFECTS ON HORMONAL RESPONSES AND STRENGTH PERFORMANCE


MIKEL IZQUIERDO,<sup>1</sup> JAVIER IBAÑEZ,<sup>1</sup> JUAN JOSÉ GONZÁLEZ-BADILLO,<sup>2</sup> NICHOLAS A. RATAMESS,<sup>3</sup> WILLIAM J. KRAEMER,<sup>4</sup> KEIJO HÄKKINEN,<sup>5</sup> HENRI BONNABAU,<sup>1</sup> CRISTINA GRANADOS,<sup>1</sup> DUNCAN N. FRENCH,<sup>6</sup> AND ESTEBAN M. GOROSTIAGA<sup>1</sup>

<sup>1</sup>Studies, Research and Sport Medicine Center, Government of Navarra, Navarra, Spain; <sup>2</sup>Olympic Center of Sport Studies, Spanish Olympic Committee, Madrid, Spain; <sup>3</sup>Department of Health and Exercise Science, The College of New Jersey, Ewing, New Jersey 08628; <sup>4</sup>Department of Kinesiology, Human Performance Laboratory, University of Connecticut, Storrs, Connecticut 06269; <sup>5</sup>Department of Biology of Physical Activity, University of Jyväskylä, Jyväskylä, Finland; <sup>6</sup>Institute of Sport, Northumbria University, Northumbria, United Kingdom.

**ABSTRACT.** Izquierdo, M., J. Ibañez, J.J. González-Badillo, N.A. Ratamess, W.J. Kraemer, K. Häkkinen, H. Bonnabau, C. Granados, D.N. French, and E.M. Gorostiaga. Detraining and tapering effects on hormonal responses and strength performance. *J. Strength. Cond. Res.* 21(3):768–775. 2007.—This study examined the impact of 4 weeks of either complete cessation of training (DTR) or a tapering period (TAP; short-term reduction of the strength training volume, while the intensity is kept high), subsequent to 16 weeks of periodized heavy resistance training (PRT) on strength/power gains and the underlying physiologic changes in basal circulating anabolic/catabolic hormones in strength-trained athletes. Forty-six physically active men were matched and randomly assigned to a TAP ( $n = 11$ ), DTR ( $n = 14$ ), or control group (C;  $n = 21$ ), subsequent to a 16-week PRT program. Muscular and power testing and blood draws to determine basal hormonal concentrations were conducted before the initiation of training (T0), after 16 weeks of training (T1), and after 4 weeks of either DTR or TAP (T2). Short-term DTR (4 weeks) results in significant decreases in maximal strength (–6 to –9%) and muscle power output (–17 and –14%) of the arm and leg extensor muscles. However, DTR had a significant ( $p < 0.01$ ) larger effect on muscle power output more than on strength measurements of both upper and lower extremity muscles. Short-term (4 weeks) TAP reached further increases for leg (2%) and arm (2%) maximal strength, whereas no further changes were observed in both upper and lower muscle power output. Short-term DTR resulted in a tendency for elevation resting serum insulin-like growth factor (IGF)-1 concentrations, whereas the corresponding TAP experienced elevation in resting serum insulin-like binding protein-3 (IGFBP-3). These data indicated that DTR may induce larger declines in muscle power output than in maximal strength, whereas TAP may result in further strength enhancement (but not muscle power), mediated, in part, by training-related differences in IGF-1 and IGFBP-3 concentrations.

**KEY WORDS.** insulin-like growth factor-1, serum insulin-like binding protein-3, taper, training cessation, resistance training

## INTRODUCTION

 Optimal strength training results from the proper manipulation of program variables, including the intensity, frequency, and volume of exercise (9, 27). Periodized strength training typically incorporates a taper phase to reduce accumulated fatigue. It is believed that the taper enhances performance by allowing greater recovery (8, 27). Short-term reduction of the strength training volume while the intensity is kept high is a well-known coaching practice used to peak performance (9, 27). However, a marked reduction of training intensity and volume, or complete

training cessation, could bring about a partial or complete loss of recently acquired training-induced increases (26, 27). It is critical, therefore, to determine the role of a taper phase and/or complete training cessation to optimize strength and power gains.

Decreased strength performance (7–12%) has been shown after short-term periods (4–8 weeks) of training cessation or periods of reduced training (11–13). On the contrary, other studies have shown that previously untrained or recreationally trained athletes can maintain or suffer only a slightly decrement in their neuromuscular performance during short periods (i.e., 2–3 weeks) of training cessation (17, 22, 26). Recently, Andersen et al. (2) reported that 3 weeks of resistance training cessation led to increased velocity and power of maximal unloaded limb movement in previously untrained subjects but isokinetic maximal strength reverted to pretraining levels. After a period of tapering, moderately strength-trained subjects improved low velocity isokinetic strength performance of the elbow flexors for at least 8 days (8). González-Badillo et al. (9) examined the effect of 3 resistance training volumes and reported that short-term resistance training (10 weeks) using moderate volume tended to produce greater enhancements in strength performance compared with low and high training volumes of similar relative intensity in trained young weightlifters. However, to the best of the authors' knowledge, little is known concerning the impact of short-term (4 weeks) tapering subsequent to a periodized heavy and explosive training program on muscle power output in strength-trained athletes. In light of these observations, we hypothesized short-term (4 weeks) detraining after 16 weeks of resistance training in strength-trained athletes would lead to a complete loss of recently acquired maximal strength and power gains, whereas a taper phase would lead to further increases in muscle strength and power.

Manipulation of training variables (i.e., large increases in the intensity and volume of resistance training) may overstress the neuroendocrine system, leading to elevated catabolic and/or lowered anabolic state and limited strength development (6, 15, 16, 23). Both anabolic and catabolic hormonal changes in response to strength training have been proposed as physiologic markers to monitor the tissue-remodeling process and other training-related responses (i.e., increased neurotransmitter synthesis) involved in strength development (15, 23). In light of these observations, along with the fact that there is a lack of data with regard to the impact of training cessation or a

period of taper (i.e., progressive decrease in training volume and increased training intensity before competition), we hypothesized that an optimize anabolic environment and decreased catabolic process induced by either detraining and/or tapering period could enhance optimal strength and power gains.

Therefore, the purpose of this study was to examine the impact of 4 weeks of either cessation of training or a tapering period subsequent to 16 weeks of periodized explosive and heavy resistance training on strength and power gains in strength-trained athletes. A secondary purpose was to examine the underlying physiologic changes in basal circulating anabolic/catabolic hormones.

## METHODS

### Experimental Approach to the Problem

To address the primary hypotheses presented in this study, 4 weeks of either cessation of training or tapering was used subsequent to 16-week periodized resistance training program to examine hormonal changes and strength and power changes of the upper and lower body musculature. This study design enabled us to make comparisons between 2 types of peaking approaches. Subsequent to the 16-week strength training period, subjects were matched according to physical characteristics, as well as to maximal strength/muscle power during bench press and parallel squat performances, and randomly assigned to either a tapering (TAP;  $n = 11$ ) or detraining (DTR;  $n = 14$ ) group. As a control, a third population of subjects (C;  $n = 21$ ) did not follow a set strength-training intervention but continued with Basque ball practices and games and were tested before and after a 16-week period to assess the reliability of the observations. During the tapering period, the training volume was reduced, whereas the intensity was increased. The DTR group discontinued resistance training and did not perform any resistance or sprint exercise throughout the 4-week period but continued with Basque ball practices and games. Testing was conducted on 3 occasions: before the initiation of training (T0), after 16 weeks of training (T1), and after 4 weeks of either training cessation or tapering period (T2).

### Subjects

A group of 46 Basque ball players, with  $12.5 \pm 5$  years of regular training and competition experience in Basque ball, volunteered to participate in a 4-week period of either training cessation or tapering after a period of 16 weeks of resistance training. 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 states of Spain (including the Basque Country), as well as in several other Europe and American countries (e.g., Cuba, Mexico, and 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 starting the XIV World Basque Ball championship. Each subject was informed carefully of the experimental procedures and about the 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 months before the experimental period, subjects trained 2 times a week for Basque ball, twice a week for strength and endurance train-

**TABLE 1.** Physical characteristics of the tapering (TAP), detraining (DTR), and control groups.\*

	TAP ( $N = 11$ )	DTR ( $N = 14$ )	Control ( $N = 21$ )
Age (years)	$24.8 \pm 2.9$	$23.9 \pm 1.9$	$24.4 \pm 2.1$
Height (m)	$1.80 \pm 0.01$	$1.81 \pm 0.01$	$1.80 \pm 0.02$
Body mass (kg)			
T0	$83.3 \pm 4.5$	$78.9 \pm 6.6$	$80.8 \pm 7$
T1	$82.1 \pm 4.3^\dagger$	$77.6 \pm 6.6^\dagger$	
T2	$82.1 \pm 3.6^\dagger$	$77.9 \pm 6.8$	$82 \pm 6.5^\dagger$
Body fat (%)			
T0	$12.7 \pm 3.6$	$11.7 \pm 4.4$	$13.1 \pm 5$
T1	$11.4 \pm 2.6^\dagger$	$10.9 \pm 3.8^\dagger$	
T2	$11.1 \pm 2.4^\dagger$	$11 \pm 3.3$	$13.1 \pm 5$
BMI			
T0	$24.9 \pm 2.5$	$24.6 \pm 2.2$	$24.5 \pm 1.9$
T1	$24.5 \pm 2.1$	$24.4 \pm 2.1$	
T2	$24.5 \pm 2.1$	$24.3 \pm 2.2$	$24.9 \pm 1.7$

\* BMI = body mass index.

$^\dagger p < 0.05$  from the corresponding time point T0.

ing, and played in 1 official Basque ball game per week. Basque ball practice sessions lasted 60–90 minutes and usually consisted of various skill activities at different intensities and 45 minutes 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 exercises in each session, mainly consisting of 3 sets of 6–8 repetitions, with a relative intensity of 50–60% of 1 repetition maximum (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 trunk extension. The total duration of each strength training session was 35–40 minutes. The running endurance program consisted of 1 training session per week and lasted 20–30 minutes at a self-adjusted intensity. The subjects were not taking exogenous anabolic-androgenic steroids or other drugs 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 tested using a counter-movement jump (CMJ) performed on a force platform. In addition, each subject was tested for 1RM bench press and parallel squat and power output using a relative load of 60% of their 1RM for each exercise. Body mass and percent body fat (estimated from the thickness of 7 skinfold sites) were taken at the beginning of the testing session. All of the subjects were familiar with the testing protocol, because they had been previously tested on several occasions during the season with the same testing procedures. The test–retest intraclass correlations coefficients were  $>0.91$ , and the coefficients of variation (CVs) ranged from 0.9 to 2.3%. Training was integrated into the test week schedules.

### Bench Press and Parallel Squat Muscular Performance

A detailed description of the maximal strength and power testing procedures can be found elsewhere (18). In brief, lower and upper body maximal strength was assessed using 1RM bench press ( $1RM_{BP}$ ) and thigh parallel squat

(1RM<sub>HS</sub>) actions. In the 1RM<sub>BP</sub> 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. No bouncing or arching of the back was allowed.

The 1RM<sub>HS</sub> began with the bar on the shoulders with the knees and hips in the extended position. The subjects descended to the parallel thigh position. On the verbal command "up," the subject ascended (as fast as possible) to 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 consisted of a set of 5 repetitions at loads of 40–60% of the perceived maximum. Thereafter, 4–5 separate single attempts were performed until 1RM was attained. The rest between maximal attempts was always 2 minutes.

Power output of the leg and arm extensor muscles was measured concentrically in the parallel squat and bench presses using a relative load 60% of 1RM. The subject was instructed to lift with maximal bar velocity. Two testing trials were recorded, and the best trial was taken for further analyses.

During the parallel squat test, bar displacement, average velocity ( $\text{m}\cdot\text{s}^{-1}$ ) 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. The test–retest intraclass correlations coefficients of the testing procedure variables used in this study were  $>0.91$  and the CVs ranged from 0.9 to 7.3%.

### Jumping Test

Subjects were asked to perform a maximal vertical CMJ on a contact platform (Newtest OY, Oulu, Finland) without any load. Using a preparatory counter-movement, subjects initiated the jump from an extended leg position, descended to 90° knee flexion, and immediately performed an explosive concentric action for maximal height. In these jumping conditions without an extra load, the subjects were instructed to keep their hands on the hips throughout the entire jump and to minimize lateral and horizontal displacement during performance. The jumping height was calculated from the flight time. Two maximal jumps were recorded interspersed with  $\sim 10$  seconds of rest, and the peak value was used for further analysis.

### Assessment of Resting Hormone Concentrations

Resting blood samples were collected between 0800 and 0900 hours on the day of testing after a 12-hour overnight fast and abstinence from alcohol and strenuous exercise for 36–48 hours before. In all cases, blood samples were obtained through venipuncture from an antecubital forearm vein using a 20-gauge needle and vacutainers. Whole blood was centrifuged at 3,000 rpm (4°C) for 15 minutes, and the resultant serum and plasma were removed and stored at  $-20^{\circ}\text{C}$  until subsequent analysis. Circulating concentrations of total testosterone (TT), free testosterone (FT), and cortisol (C) were determined using commercial-

ly available enzyme immunoassay (EIA) kits (Diagnostic Systems Laboratories, Webster, TX). Plasma growth hormone (GH) concentrations were determined using  $^{125}\text{I}$  liquid-phase immunoradiometric assay (IRA; Nichols Institute Diagnostics, San Juan Capistrano, CA). Insulin-like growth factor-1 (IGF-1) and IGF binding protein-3 (IGFBP-3) concentrations were measured by enzyme-linked immunosorbent assay (ELISA) kits (Diagnostic Systems Laboratories) 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 EIA detection limits for TT, FT, and C were 0.14, 0.66, and 2.76  $\text{nmol}\cdot\text{L}^{-1}$ , respectively. IRA detection limits for GH were 0.04  $\text{ng}\cdot\text{ml}^{-1}$ . Minimum ELISA detection limits for IGF-1 and IGFBP-3 were 0.0013 and 0.0014  $\text{nmol}\cdot\text{L}^{-1}$ , respectively. The coefficient of intra-assay variation was 4.4 and 4.2% for TT and FT, 5.1% for C, and 6.0 and 6.4% for IGF-1 and IGFBP-3, respectively. All samples were analyzed in the same assay for each analyte according to the instructions. 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 minutes 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 groups trained 2 times per week using a similar 16-week (macrocycle) periodized resistance training program divided into 3 mesocycles of 5–6 weeks (from T0 to T1) (19). Subsequently, the subjects participated in a 4-week period of either detraining or tapering (from T1 to T2). During the course of the 16-week training period, the assigned training intensities were gradually increased on the basis of the athlete's 10RM and 6RM testing, using a repetition maximum approach. During the first 6 weeks of training, both groups trained with 3 sets of 10RM for the bench press and 80% of 10RM for the parallel squat. During the middle 5 weeks of training (from week 7 to 11), subjects trained at 6RM and 80% of 6RM and performed 3 sets for the bench press and parallel squat, respectively. During the final 5 weeks of training (from week 12 to 16), both groups trained at 85–90% of 1RM ( $\sim 5\text{RM}$ ), 2–4 repetitions per set, and performed 3 sets for both upper and lower extremity exercises and performed the ballistic training program (i.e., vertical CMJs, loaded vertical jumps, sprint runs, and various throwing exercises with a 1-kg ball). In addition, the subjects performed bench press sets with loads ranging from 40 to 45% of 1RM. During this phase, subjects performed 3–4 repetitions per set and 3–5 sets of each exercise in a ballistic manner. Approximately 2-minute rest periods were allowed between each set and each exercise. This ballistic strength training was included because it has been shown to be the most effective way to enhance explosive strength and speed. In addition, the last peaking mesocycle during the last 5 weeks was used to produce a similar "rebound effect" for all groups and to avoid overreaching (19).

During the entire training period, the core exercises were the parallel squat and bench press, in addition to

supplementary strengthening exercises for selected muscle groups. In addition, the training program included ballistic exercises during the last 5 peaking weeks of explosive strength training. Subjects performed all free-weight bench press and squat training using a standard 20-kg barbell.

After the 16-week resistance training period, subjects were assigned in a random, counterbalanced manner to 4 weeks of either DTR or TAP. No strength training was performed during DTR, whereas TAP consisted of a period of progressive lowering of training volume with increasing intensity. Specifically, during TAP, subjects trained at 90–95% of 1RM (3–4RM), 2–4 repetitions per set, and performed 2–3 sets for both upper and lower extremity exercises.

### Statistical Analyses

Standard statistical methods were used for the calculation of means and *SD*. One-way analysis of variance (ANOVA) was used to determine any differences among the 3 groups in initial strength, power, and hormonal profile. The training-related effects were assessed using a two-way ANOVA with repeated measures (groups  $\times$  time). When a significant *F*-value was achieved, Sheffé post hoc procedures were performed to locate the pairwise differences between the means. Selected absolute changes were analyzed by 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

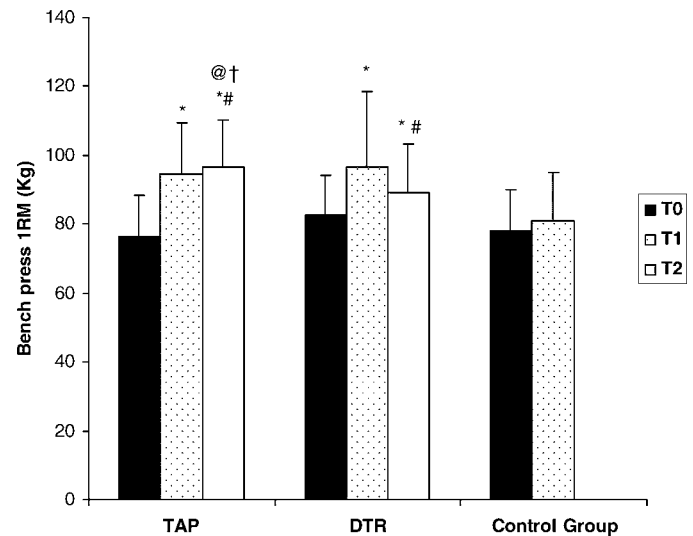
### Body Composition

At the beginning of the training program, no significant differences were observed among the groups in the pre-training age, height, body mass, or percent body fat. Significant decreases in body mass ( $-1.4$  and  $-1.3\%$ ,  $p < 0.05$ ) and body fat ( $-10$  and  $7\%$ ,  $p < 0.05$ ) were observed at T1 for TAP and DTR groups compared with T0. No significant changes were observed in body mass and body fat at T2 for TAP and DTR compared with T1. No changes were observed in the control group.

### Maximal Strength and Muscle Power Output

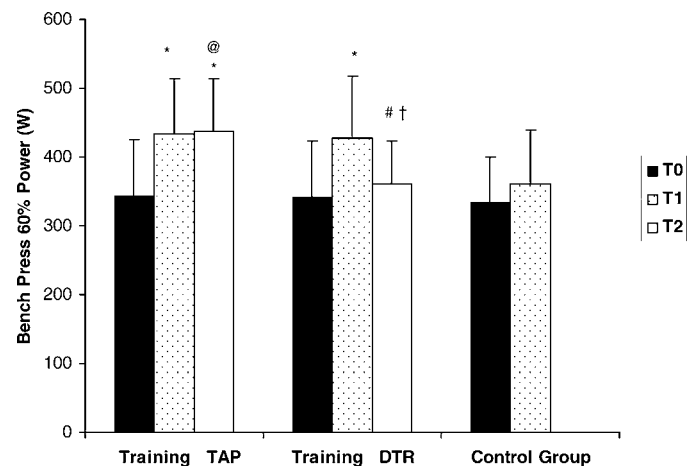
No significant differences were observed between groups at the beginning of the study for  $1RM_{HS}$  and  $1RM_{BP}$ . Nevertheless, significant increases were observed in  $1RM_{BP}$  for TAP and DTR groups at T1 (24 and 17%, respectively;  $p < 0.001$ ) compared with T0. After the tapering period,  $1RM_{BP}$  significantly increased (2%) from T1 to T2, whereas a significant decrease ( $-9\%$ ) was observed in the DTR group. The magnitude of change in  $1RM_{BP}$  values for the TAP group (2%) at T2 compared with T1 were significantly larger ( $p < 0.001$ ) than that recorded in the DTR group ( $-9\%$ ; Figure 1). No significant differences in  $1RM_{BP}$  were observed in the control group during the study.

No significant differences were observed between groups in muscle power output at 60% of  $1RM_{HS}$  and  $1RM_{BP}$  at the beginning of the study. Significant increases took place in muscle power output at 60% of  $1RM_{BP}$  for TAP (29%,  $p < 0.001$ ) and DTR (26%,  $p < 0.001$ ) at T1 compared with T0. After the tapering period (from T1 to T2), muscle power output in the bench press remained unaltered (1%) in the TAP group, whereas a significant decrease in muscle power output ( $-17\%$ ) was observed in DTR. After detraining, the magnitude of muscle power output decrement at 60% of  $1RM_{BP}$  was greater ( $p < 0.001$ ) than that recorded in TAP (Figure 2).

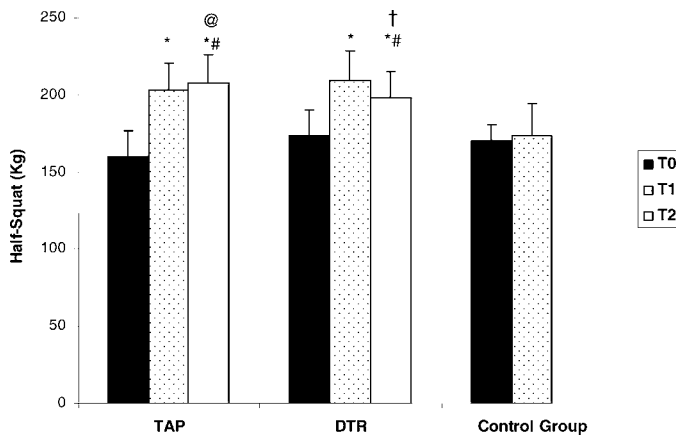


**FIGURE 1.** Maximal bench press strength at pretraining (T0), after 16-week periodized explosive and heavy resistance training program (T1), and after the subsequent 4 weeks (T2) of either cessation of training or a taper period. \* $p < 0.05$  from the corresponding time-point T0. # $p < 0.05$  from the corresponding time-point T1. @ $p < 0.05$  from relative change at time-point T0 between groups. † $p < 0.05$  from relative change at time-point T1 between groups. TAP = tapering group; DTR = detraining group and control group. Data presented are mean  $\pm$  *SD*.

Significant increases took place in maximum concentric  $1RM_{HS}$  strength for the TAP and DTR groups at T1 (27%,  $p < 0.001$  and 22%,  $p < 0.001$ , respectively) and T2 (30%,  $p < 0.001$  and 16%,  $p < 0.001$ , respectively) compared with T0. After the tapering period (from T1 to T2), a significant increase (3%,  $p < 0.001$ ) was observed in TAP, whereas a significant decrease ( $-6\%$ ,  $p < 0.001$ ) was observed in DTR (Figure 3). The magnitude of change in  $1RM_{HS}$  during the tapering period (2%,  $p < 0.001$ ) was significantly different to that observed in DTR ( $-6\%$ ,  $p < 0.001$ ).



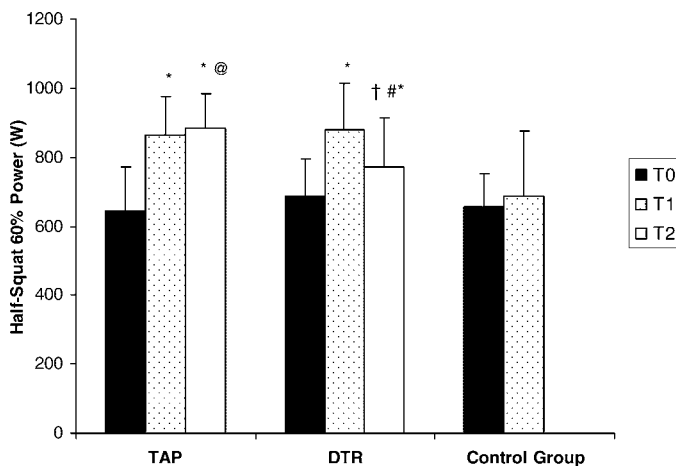
**FIGURE 2.** Bench press muscle power output at pretraining (T0), after 16-week periodized explosive and heavy resistance training program (T1), and after the subsequent 4 weeks (T2) of either cessation of training or a taper period. \* $p < 0.05$  from the corresponding time-point T0. # $p < 0.05$  from the corresponding time-point T1. @ $p < 0.05$  from relative change at time-point T0 between groups. † $p < 0.05$  from relative change at time-point T1 between groups. TAP = tapering group; DTR = detraining group and control group. Data presented are mean  $\pm$  *SD*.



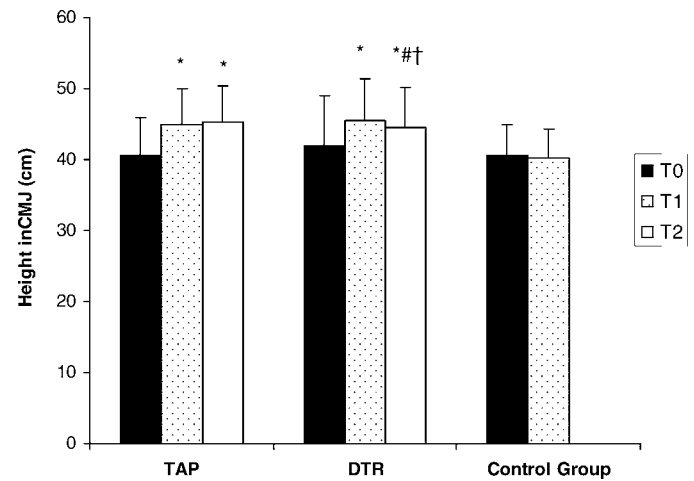
**FIGURE 3.** Maximal parallel squat strength at pretraining (T0), after 16-week periodized explosive and heavy resistance training program (T1), and after the subsequent 4 weeks (T2) of either cessation of training or a taper period. \*  $p < 0.05$  from the corresponding time-point T0. #  $p < 0.05$  from the corresponding time-point T1. @  $p < 0.05$  from relative change at time-point T0 between groups. †  $p < 0.05$  from relative change at time-point T1 between groups. TAP = tapering group; DTR = detraining group and control group. Data presented are mean  $\pm$  SD.

Significant increases were observed in muscle power output at 60% of  $1RM_{HS}$  for the TAP and DTR groups at T1 (37 and 29%, respectively,  $p < 0.001$ ) and T2 (48%,  $p < 0.001$  and 17%,  $p < 0.01$ , respectively) compared with T0. During the tapering period, no significant changes were observed for leg muscle power (3%), whereas a significant decrease was observed in DTR (-14%,  $p < 0.01$ ). The magnitude of changes in leg muscle power during TAP (3%) was significantly different ( $p < 0.001$ ) to that observed during DTR (Figure 4). No changes were observed in the control group.

Significant increments took place in the height in CMJ for TAP and DTR groups at T1 compared with T0.



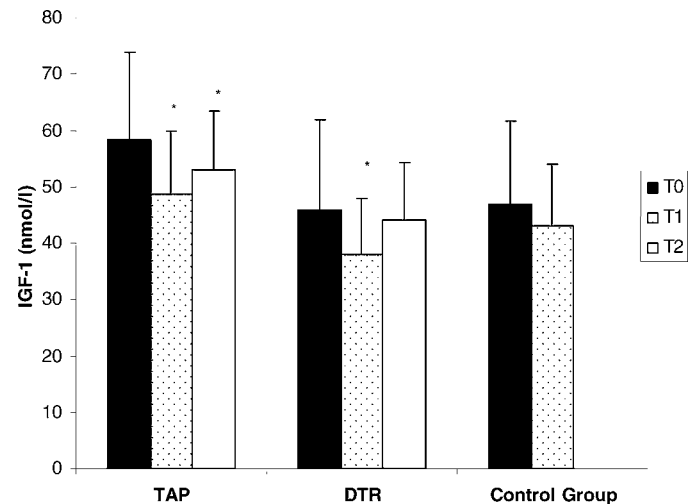
**FIGURE 4.** Parallel squat muscle power output at pretraining (T0), after 16-week periodized explosive and heavy resistance training program (T1), and after the subsequent 4 weeks (T2) of either cessation of training or a taper period. \*  $p < 0.05$  from the corresponding time-point T0. #  $p < 0.05$  from the corresponding time-point T1. @  $p < 0.05$  from relative change at time-point T0 between groups. †  $p < 0.05$  from relative change at time-point T1 between groups. TAP = tapering group; DTR = detraining group and control group. Data presented are mean  $\pm$  SD.



**FIGURE 5.** Height in the countermovement jump (CMJ) at pretraining (T0), after 16-week periodized explosive and heavy resistance training program (T1) and after the subsequent 4 weeks (T2) of either cessation of training or a taper 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 T1 between groups. TAP = tapering group; DTR = detraining group and control group. Data presented are mean  $\pm$  SD.

During TAP, the height in CMJ remained unaltered, whereas a significant decrease was observed in DTR (-3%,  $p < 0.001$ ). This difference was significantly different ( $p < 0.01$ ; Figure 5). No changes were observed in the control group.

No significant differences in serum C, TT, FT, and GH concentrations were observed in either group at any time. During the strength training period (from T0 to T1), no significant differences were observed in the magnitude of reduction in IGF-1 concentrations between TAP (-14%,  $p < 0.01$ ) and DTR (-11%,  $p < 0.01$ ). During DTR (from T1 to T2), a tendency ( $p = 0.07$ ) for elevation was observed in IGF-1, whereas no significant change was observed in TAP (Figure 6).



**FIGURE 6.** Resting serum insulin-like growth factor-1 (IGF-1) concentrations at pretraining (T0), after 16-week periodized explosive and heavy resistance training program (T1), and after the subsequent 4 weeks (T2) of either cessation of training or a taper period. \*  $p < 0.05$  from the corresponding time-point T0. TAP = tapering group; DTR = detraining group and control group. Data presented are mean  $\pm$  SD.

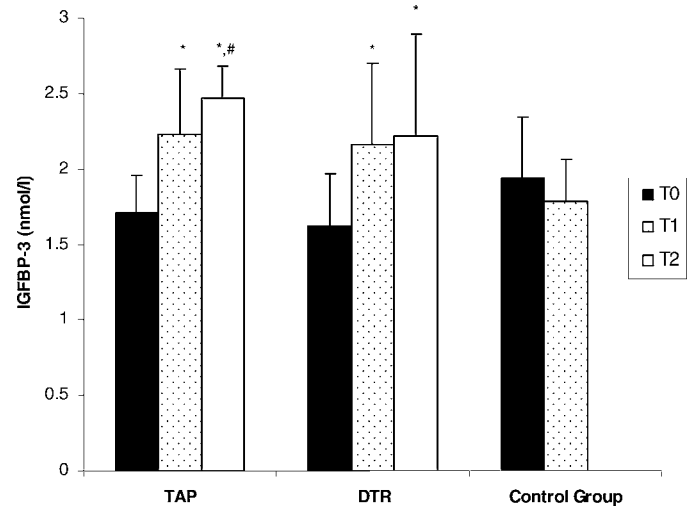
During the strength training period (from T0 to T1), no significant difference was observed in the magnitude of elevation in serum IGFBP-3 between TAP (34%,  $p < 0.01$ ) and DTR (37%,  $p < 0.01$ ). During TAP (from T1 to T2), the magnitude of elevation in serum IGFBP-3 concentration for TAP at T2 (15%;  $p < 0.01$ ) was larger than that recorded in DTR (4%; not significant). In addition, no significant hormonal differences were observed in the control group at any time-point (Figure 7).

## DISCUSSION

The major findings of this study were that, after a period of 4 weeks of detraining after 16 weeks of resistance training, (a) significant reductions were observed in bench press and parallel squat 1RM, muscle power output of the upper and lower extremities and (b) the detraining period had a larger effect on muscle power output than strength of the upper and lower extremities. In contrast, during tapering (progressively lowered volume, higher intensity), further increases in maximal strength were observed, whereas no further changes were observed in upper and lower body power output. In addition, a unique finding in this study was that the short-term detraining period resulted in a tendency for elevated resting serum IGF-1 concentrations, whereas the corresponding tapering period experienced an elevation in resting serum IGFBP-3. These data indicated that cessation of training may induce larger declines in muscle performance, whereas tapering resulted in further strength enhancement perhaps mediated, in part, by training related differences in endocrine responses.

Few studies have examined the effects of training cessation (4–8 weeks) subsequent to a periodized heavy and explosive resistance training program in resistance-trained individuals. These studies have shown that training cessation during periods of inactivity ranging from 4 to 12 weeks led to pronounced declines (7–12%) in strength performance (11–14). Häkkinen and Komi (12) reported a 10% decrease in 1RM squat after 4 weeks of training cessation. Results from the same research group have shown 11–12% decreases in 1RM squat and leg extension force after 8 weeks of training cessation (14). On the contrary, other studies have shown that untrained or recreationally trained athletes can maintain or suffer only a slightly reduction in neuromuscular performance during short periods (i.e., 2–3 weeks) of training cessation (17, 22, 26). It seems that highly trained athletes are more susceptible to performance decrements because of their high level of conditioning. Our results support previous studies showing that training cessation during a period of 4 weeks resulted in larger decreases in strength, presumably caused by the highly conditioned population examined.

A limited number of studies have examined the impact of short-term (4 weeks) detraining or tapering subsequent to a periodized heavy and explosive training on muscle power output in strength-trained athletes. Muscle power output of the upper and lower extremities was markedly reduced detraining. However, a major finding of this study was that detraining resulted in a larger reduction in muscle power than maximal strength in the upper and lower extremities. In contrast, other studies have reported that vertical jump performance (22), isometric fast force production (11–13), and power during unloaded knee extension (2) can be maintained or slightly increased over 3–6 weeks of detraining. The discrepancy between this study and previous studies may be related



**FIGURE 7.** Resting serum insulin-like binding protein-3 (IGFBP-3) concentrations at pretraining (T0), after 16-week periodized explosive and heavy resistance training program (T1), and after the subsequent 4 weeks (T2) of either cessation of training or a taper period. \*  $p < 0.05$  from the corresponding time-point T0. #  $p < 0.05$  from the corresponding time-point T1. TAP = tapering group; DTR = detraining group and control group. Data presented are mean  $\pm$  SD.

to the highest level of strength of our subjects in comparison with those of other studies (21), as well as the impact from prior power training history (21). Thus, power gains in strength-trained athletes seem to be lost at a greater rate than strength after detraining. To what extent preferential decreases in muscle power occur during the early phase of detraining (i.e., 3 weeks) may be related to preferential atrophy of type II muscle fibers (17, 31) and/or with reductions in neural drive (2, 11, 12, 14) remain to be elucidated.

An interesting finding of this study was that strength-trained athletes were able to significantly increase both lower and upper body maximal strength by tapering for 4 weeks. In contrast, no further changes were observed in upper and lower body power output after tapering. These data indicate that tapering induced long-lasting maximal strength gains but no further increases in muscle power. A few studies have examined the effects of tapering. Gibala et al. (8) reported a significant increase in low-velocity isokinetic strength performance of the elbow flexors, using similar intensity with reduced training volume in resistance-trained athletes. González-Badillo et al. (9) reported that short-term moderate volume resistance training tended to produce greater enhancements in strength performance compared with low and high training volumes of similar relative intensity in young weightlifters. Results from the same research group also showed that short-term resistance training using moderate volumes of high relative intensity (e.g., 80–90% of 1RM) tended to produce higher increases in weightlifting performance compared with low and high volumes of high relative training intensities of equal total volume in experienced trained young weightlifters (10). These data indicate that strength-trained athletes, after a 16-week (macrocycle) periodized resistance training program (including a peaking mesocycle of 5 weeks), can improve maximal strength and achieve higher performance levels as a result of a tapering for at least 4 weeks.

The results of this study indicate that, after periodized resistance training in previously strength-trained ath-

letes, strength values may approach one's maximal adaptation limits. Thus, proper manipulation of acute program variables is critical to progression beyond this point. Once a given volume and intensity is reached, a substantial increase in training intensity and volume could result in overtraining (6,7). It is critical, therefore, for the coach to develop strategies to optimally manipulate training volume and intensity to peak maximal strength and power while avoiding overtraining.

Both anabolic and catabolic hormones have been proposed as physiologic markers to evaluate the tissue remodeling process and other related mechanisms involved for higher levels of muscular performance and subsequent adaptations during a strength training period (16, 23). Manipulation of training variables (i.e., large increases in the intensity and volume of resistance training) may over-stress the neuroendocrine system, leading to altered circulating hormonal concentrations (6). In this hypothesis, an optimal anabolic environment (or reduced catabolic processes) induced by either detraining and/or tapering potentially could enhance performance. However, the effects of hormonal changes subsequent to both detraining and tapering have received little attention.

No significant differences were observed in resting serum concentrations of TT, C, FT, and GH after DTR and TAP. In agreement with this study, Häkkinen et al. (13) reported no significant changes in TT, C, or GH but did show a significant reduction in the T:C ratio after 12 weeks of training cessation. Kraemer et al. (14) reported no significant change in resting hormonal concentrations of GH, TT, and C after 6 weeks of detraining in recreationally trained men. However, after 2 weeks of detraining, Hortobagay et al. (17) reported significant elevations in GH, TT, and T:C ratio, with a concomitant reduction in C concentrations, thereby suggesting that short-term detraining might represent an enhanced stimulus for tissue remodeling and repair. Resting concentrations seem to 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 (15, 16). Ahtianen et al. (1) reported higher FT and TT concentrations during a 7-week high volume training phase compared with pre-training values, but return to baseline when volume was reduced, and intensity was increased over a subsequent 7-week training period. In addition, resting serum catabolic and anabolic hormonal concentrations have been shown to poorly correlate with strength performance, especially in strength-trained athletes who do not use anabolic steroids (30), thereby suggesting that the acute response may play a more prominent role. Therefore, in lieu with the absence of anabolic and catabolic hormonal level alterations during long-term resistance training in this study, the lack of change in resting serum concentration of TT, FT, C, and GH observed after the subsequent 4-week training cessation was not surprising and seemed to be a noncontributing factor to the changes in DTR and TAP between T1 and T2.

Few studies have examined chronic circulating concentrations of the IGF family after long-term resistance training and subsequent detraining or tapering. A unique finding in this study was that short-term detraining resulted in a tendency for elevated resting serum IGF-1 concentrations similar to pretraining levels, whereas tapering resulted in further elevation in resting serum IGFBP-3 concentrations. This finding is consistent with

a previous study involving strength-trained athletes, which showed that chronic IGF-1 concentrations may be mediated, in part, by volume and intensity manipulation (3, 19, 23, 25, 29). Elevated resting IGF-1 has been reported in long-term training studies (3, 20, 25), whereas in short-term training studies, IGF-1 concentration remain unaltered (21, 24). A dramatic increase in volume and/or intensity of training has been shown to dramatically reduce IGF-1 concentrations by 11–12% (4, 20, 29) but return to baseline when normal training resumed over the next cycle (4, 29). In this study, resting serum IGF-1 concentrations were significantly elevated after detraining to baseline values with no accompanying significant changes during TAP. Collectively, our results indicate that chronic IGF-1 adaptations in response to resistance training and subsequent DTR and TAP may be mediated, in part, by volume and intensity manipulation (23, 29). Thus, the reduced overall stress during cessation of training may have reduced IGF-1, but this was not observed after TAP.

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 this study, it is possible that nonmeasured alterations in GH pulsatility (i.e., overnight or at a time of day not examined in this study) or other non-22-kd molecular weight GH variants could have occurred. Nindl et al. (28) showed reduced GH pulsatility overnight after high-volume resistance exercise, presumably caused by the high level of stress. In addition, delayed secretion of IGF-1 (i.e., 3–9 hours) after GH-stimulated mRNA synthesis has been shown (23). Therefore, the IGF-1 concentrations observed in this 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 tapering group. Considering that no biopsy samples were obtained in this study, autocrine actions of the IGFs on muscle adaptations may be difficult to ascertain.

To the authors' knowledge, no studies have examined chronic circulating concentrations of IGFBP-3 after either training cessation or tapering. A unique finding in this study was that short-term tapering resulted in further elevation in resting serum IGFBP-3 concentrations occurring parallel to the reduced IGF-1 concentrations observed after resistance training. Interestingly, Elloumi et al. (5) have proposed that a reduction in resting IGFBP-3 may be used as a marker of overtraining. This finding is consistent with a previous study involving strength-trained athletes, which showed that the reduction in resting IGF-1 observed when training to repetition to failure occurred parallel to elevations in serum IGFBP-3 concentrations (19). Borst et al. (3) showed a reduction in IGFBP-3 concentrations that paralleled a concomitant elevation in IGF-1 concentrations after a 25-week training period. Based on our data, it may be hypothesized that the elevation in IGFBP-3 may have been compensatory to accommodate the reduction in IGF-1 to preserve IGF availability (19). However, further research is needed to examine the impact of resistance training intensity and volume manipulation on training-related changes in IGFs.

## PRACTICAL APPLICATIONS

Our results indicate that 4 weeks of detraining subsequent to a 16-week period of periodized heavy and explosive resistance training results in significant decreases in bench press 1RM, parallel squat 1RM, and muscle power output of the upper and lower body musculature. However, detraining had a larger effect on muscle power output more than on strength measurements of both upper and lower extremity muscles. In contrast, short-term (4 weeks) tapering resulted in further increases for upper and lower body maximal strength, whereas no further changes were observed in muscle power output. Short-term detraining resulted in a tendency for elevated resting serum IGF-1 concentrations, whereas the corresponding tapering period resulted in elevated resting serum IGFBP-3 concentrations. These data indicated that detraining may induce larger declines in muscle power output than in maximal strength, whereas tapering may result in further strength enhancement (but not muscle power), mediated, in part, by training-related differences in IGF-1 and IGFBP-3 concentrations.

## REFERENCES

- AHTIANEN, J.P., A. PAKARINEN, W.J. KRAEMER, AND K. HÄKKINEN. Acute hormonal and neuromuscular responses and recovery to forced vs maximum repetitions multiple resistance exercises. *Int. J. Sports Med.* 24: 410–418. 2003.
- ANDERSEN, L.L., J.L. ANDERSEN, S.P. MAGNUSSEN, C. SUETTA, J.L. MADSEN, J.R. CHRISTENSEN, AND P. AAGAARD. Changes in the human muscle force-velocity relationship in response to resistance training and subsequent detraining. *J. Appl. Physiol.* 99:87–94. 2005.
- BORST, S.E., D.V. DE HOYOS, L. GARZARELLA, K. VINCENT, B.H. POLLOCK, D.T. LOWENTHAL, AND M.L. POLLOCK. Effects of resistance training on insulin-like growth factor-I and IGF binding proteins. *Med. Sci. Sports Exerc.* 33:648–653. 2001.
- ELIAKIM, A., D. NEMET, S. BAR-SELA, Y. HIGER, AND B. FALK. Changes in circulating IGF-1 and their correlation with self assessment and fitness among elite athletes. *Int. J. Sports Med.* 23:600–603. 2002.
- ELLOUMI, M., N. EL ELJ, M. ZAOUALI, F. MASO, E. FILAIRE, Z. TABKA, AND G. LAC. IGFBP-3, a sensitive marker of physical training and overtraining. *Br. J. Sports Med.* 39:604–610. 2005.
- FRY, A.C., AND W.J. KRAEMER. Resistance exercise overtraining and overreaching: Neuroendocrine responses. *Sports Med.* 23:106–129. 1997.
- FRY, A.C., W.J. KRAEMER, F. VAN BORSELEN, J.M. LYNCH, J.L. MARSIT, E.P. ROY, N.T. TRIPLETT, AND H.G. KNUTTGEN. Performance decrements with high-intensity resistance exercise overtraining. *Med. Sci. Sports Exerc.* 26:1165–1173. 1994.
- GIBALA, M.J., J.D. MACDOUGALL, AND D.G. SALE. The effects of tapering on strength performance in trained athletes. *Int. J. Sports Med.* 15:492–497. 1994.
- GONZÁLEZ-BADILLO, J.J., E.M. GOROSTIAGA, R. ARELLANO, AND M. IZQUIERDO. Moderate resistance training volume produces more favorable strength gains than high or low volumes. *J. Strength Cond. Res.* 19:689–697. 2005.
- GONZÁLEZ-BADILLO, J.J., M. IZQUIERDO, AND E.M. GOROSTIAGA. Moderate volume of high relative training intensity produces greater strength gains compared with low and high volumes in competitive weightlifters. *J. Strength Cond. Res.* 20:73–81. 2006.
- HÄKKINEN, K., M. ALEN, AND P.V. KOMI. Changes in isometric force- and relaxation-time, electromyographic and muscle fibre characteristics of human skeletal muscle during strength training and detraining. *Acta Physiol. Scand.* 125:573–585. 1985.
- HÄKKINEN, K., AND P.V. KOMI. Electromyographic changes during strength training and detraining. *Med. Sci. Sport Exerc.* 15:455–460. 1983.
- HÄKKINEN, K., P.V. KOMI, AND M. ALEN. Effect of explosive type strength training on isometric force- and relaxation-time, electromyographic and muscle fibre characteristics of leg extensor muscles. *Acta Physiol. Scand.* 125:587–600. 1985.
- HÄKKINEN, K., P.V. KOMI, AND P.A. TESCH. Effect of combined concentric and eccentric strength training and detraining on force-time, muscle fiber and metabolic characteristics of leg extensor muscles. *Scand. J. Sports Sci.* 2:50–58. 1981.
- HÄKKINEN, K., A. PAKARINEN, M. ALEN, A. KAUKANEN, AND P.V. KOMI. Neuromuscular and hormonal adaptations in athletes to strength training in two years. *J. Appl. Physiol.* 65:2406–2412. 1988.
- HÄKKINEN, K., A. PAKARINEN, M. ALEN, AND P.V. KOMI. Serum hormones during prolonged training of neuromuscular performance. *Eur. J. Appl. Physiol.* 53:287–293. 1985.
- HORTOBAGY, T., J.A. HOUMARD, J.R. STEVENSON, D.D. FRASER, R.A. JOHNS, AND R.G. ISRAEL. The effects of detraining on power athletes. *Med. Sci. Sports Exerc.* 25:929–935. 1993.
- IZQUIERDO, M., K. HÄKKINEN, J.J. GONZÁLEZ-BADILLO, J. IBÁÑEZ, AND E.M. GOROSTIAGA. Effects of long-term training specificity on maximal strength and power of the upper and lower extremities in athletes from different sports. *Eur. J. Appl. Physiol.* 87:264–271. 2002.
- IZQUIERDO, M., J. IBÁÑEZ, J.J. GONZÁLEZ-BADILLO, K. HÄKKINEN, N.A. RATAMESS, W.J. KRAEMER, D.N. FRENCH, J. ESLAVA, A. ALTADILL, X. ASIAIN, AND E.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.
- KOZIRIS, L.P., R.C. HICKSON, R.T. CHATTERTON JR, R.T. CHATTERTON, R.T. GROSETH, J.M. CHRISTIE, D.G. GOLDFLIES, AND T.G. UNTERMAN. Serum levels of resistance training on insulin-like growth factor-I and IGFBP-3 are increased and maintained in long-term training. *J. Appl. Physiol.* 86:1436–1442. 1999.
- KRAEMER, W.J., K. HÄKKINEN, R.U. NEWTON, B.C. NINDL, J.S. VOLEK, M. MCCORMICK, L.A. GOTSHALK, S.E. GORDON, S.J. FLECK, W.W. CAMPBELL, M. PUTUKIAN, AND W.J. EVANS. Effects of heavy resistance training on hormonal responses patterns in younger vs. older men. *J. Appl. Physiol.* 87:982–992. 1999.
- KRAEMER, W.J., L.P. KOZIRIS, N.A. RATAMESS, K. HÄKKINEN, N. TRIPLETT-MCBRIDE, A.C. FRY, S.E. GORDON, J.S. VOLEK, D.N. FRENCH, M.R. RUBIN, A.L. GOMEZ, M.J. SHARMAN, J.M. LYNCH, M. IZQUIERDO, R.U. NEWTON, AND S.J. FLECK. Detraining produces minimal changes in physical performance and hormonal variables in recreationally strength-trained men. *J. Strength Cond. Res.* 16:373–382. 2002.
- KRAEMER, W.J., AND N.A. RATAMESS. Hormonal responses and adaptations to resistance exercise and training. *Sports Med.* 35:339–361. 2005.
- MACCALL, G.E., W.C. BYRNES, S.J. FLECK, A. DICKINSON, AND W.J. KRAEMER. Acute and chronic hormonal responses to resistance training designed to promote muscle hypertrophy. *Can. J. Appl. Physiol.* 24:96–107. 1999.
- MARX, J.O., N.A. RATAMESS, B.C. NINDL, L.A. GOTSHALK, J.S. VOLEK, K. DOHI, J.A. BUSH, A.L. GOMEZ, S.A. MAZZETTI, S.J. FLECK, K. HÄKKINEN, R.U. NEWTON, AND W.J. KRAEMER. Low-volume circuit versus high-volume periodized resistance training in women. *Med. Sci. Sports Exerc.* 33: 635–643. 2001.
- MUJKA, I., AND S. PADILLA. Muscular characteristics of detraining in humans. *Med. Sci. Sports Exerc.* 33:1297–1303. 2001.
- MUJKA, I., S. PADILLA, D. PYNE, AND T. BUSSO. Physiological changes associated with the pre-event taper in athletes. *Sports Med.* 13:891–927. 2004.
- NINDL, B.C., W.C. HYMER, D.R. DEEVER, AND W.J. KRAEMER. Growth hormone pulsatility profile characteristics following acute heavy resistance exercise. *J. Appl. Physiol.* 91:163–172. 2001.
- RAASTAD, T., T. GLOMSHELLER, T. BJRO, AND T. HALLEN. Recovery of skeletal muscle contractility and hormonal responses to strength exercises after two weeks of high-volume strength training. *Scand. J. Med. Sci. Sports* 13:159–168. 2003.
- RATAMESS, N.A., W.J. KRAEMER, J.S. VOLEK, C.M. MARESH, J.L. VAN-HEEST, M.S. SHARMAN, M.R. RUBIN, D.N. FRENCH, J.D. VESCOVI, R. SILVESTRE, D.L. HATFIELD, S.J. FLECK, AND M.R. DESCHENES. Effects of heavy resistance exercise volume on post-exercise androgen receptor content in resistance-trained men. *J. Steroid. Biochem. Mol. Biol.* 93:35–42. 2005.
- STARON, R.S., M.J. LEONARDI, S.L. KARAPONDO, E.S. MALICKY, J.E. FALKEL, F.C. HAGERMAN, AND R.S. HIKIDA. Strength and skeletal muscle adaptations in heavy-resistance-trained women after detraining and retraining. *J. Appl. Physiol.* 70:631–640. 1991.

## Acknowledgments

This study was supported in part by a grant from the Consejo Superior de Deportes. Ministerio de Educación, Cultura y Deporte, Spain (Grant 01/EPB10/03). The authors disclose professional relationships with companies or manufacturers who will benefit from the results of this study. The results of this study do not constitute endorsement of the product by the authors of the NSCA.

Address correspondence to Dr. Mikel Izquierdo, mikel.izquierdo@ceimd.org.