Physiological effects of tapering in highly trained athletes

B. SHEPLEY, J. D. MACDOUGALL, N. CIPRIANO, J. R. SUTTON, M. A. TARNOPOLSKY, AND G. COATES
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Shepley, B., J. D. MacDougall, N. Cipriano, J. R. Sutton, M. A. Tarnopolsky, and G. Coates. Physiological effects of tapering in highly trained athletes. J. Appl. Physiol. 72(2): 706-711, 1992.—This study examined some of the physiological and performance effects of three different tapers in highly trained athletes. After 8 wk of training, nine male middle-distance runners were randomly assigned to one of three different 7-day tapers: a high-intensity low-volume taper (HIT), a low-intensity moderate-volume taper (LIT), or a rest-only taper (ROT). After the first taper, subjects resumed training for 4 wk and performed a second taper and then resumed training for 4 wk and completed the remaining taper, so that each subject underwent all three tapers. Performance was measured before and after each taper by a treadmill run to fatigue at a velocity equivalent to each subject’s best 1,500-m time. Voluntary isometric strength and evoked contractile properties of the quadriceps were measured before and after each taper, as were muscle glycogen concentration and citrate synthase activity (from needle biopsies) and total blood and red cell volume by ¹²⁵I and ⁵¹Cr tagging. Maximal O₂ consumption was unaffected by all three tapers, but running time to fatigue increased significantly after HIT (+22%). It was unaffected by LIT (+6%) and ROT (−3%) procedure. Citrate synthase activity increased significantly with HIT and decreased significantly with ROT. Muscle glycogen concentration increased significantly after ROT and HIT, and strength increased after all three tapers. Total blood volume increased significantly after HIT and decreased after ROT. Red cell volume also increased significantly after HIT. We conclude that highly trained middle-distance runners can improve their performance as a result of a taper where intensity is maintained and volume greatly reduced. The improvement may be due to increased oxidative enzyme activity and/or increases in blood and red cell volume.

METHODS

Subjects. Nine highly trained cross-country and middle-distance runners served as subjects. They were all members of the McMaster University cross-country and/or track teams and competed in both sports. Data were collected over the 5-mo period between the end of the fall cross-country season and the beginning of the summer track season. All subjects were fully informed of the purposes of the study and the associated risks as required by the Human Ethics Committee of McMaster University. Subjects ranged in age from 21 to 24 yr and in maximal O₂ consumption (V̇O₂max) from 66 to 71 ml·kg⁻¹·min⁻¹.

Design. Subjects trained intensively six times per week for 8 wk. During the final 2 wk of this training period, their running volume was 80 km/wk. Approximately one-third of this distance was comprised of high-inten-
sity (95–100% \( \dot{V}O_2_{\text{max}} \)) interval running and the remainder by continuous running at \( \sim 73\% \dot{V}O_2_{\text{max}} \). Intervals consisted of distances of 800–1,200 m followed by \( \sim 3 \) min of recovery and were performed two or three times per week. Subjects were then randomly assigned to one of three different 7-day taper procedures. The study employed a repeated-measures design so that each subject performed each of the three tapers, separated by 4 wk of training. Thus, after the first taper, subjects resumed training for 4 wk and performed a second taper and then resumed training for 4 wk and performed the remaining taper. The 4-wk training schedules were identical to that of the 4 wk preceding the first taper.

The taper procedures included a high-intensity (low-volume) taper, a low-intensity taper, and a rest-only taper. Subjects did not train on day 1 or 6 of the two exercise tapers. The high-intensity taper (HIT) consisted of a series of intense 500-m intervals over 5 days. Training on each day was preceded by an 800-m jog at \( \sim 50\% \dot{V}O_2_{\text{max}} \) and standard stretching exercises. Each 500-m interval was completed in 70–76 s (equivalent to 115–120% \( \dot{V}O_2_{\text{max}} \)) and separated by a 6- to 7-min walking recovery. Five repeats were performed on the 1st day, four on the 2nd day, three on the 3rd day, and so on for a total 5-day running volume of 7.5 km (not including the distance covered in warm up).

The low-intensity taper (LIT) consisted of continuous running at a pace equivalent to 57–60% \( \dot{V}O_2_{\text{max}} \) during each of the 5 days of the taper. On the 1st day subjects ran 10 km, and on each successive day they reduced this distance by 20% (2 km), for a total 5-day running volume of 30 km (not including warm-up). For the rest-only taper (ROT), subjects did no running over the 6-day period. All training and tapering sessions were directly supervised by the university cross-country coach, a co-investigator for the study.

The measurement sequence for each taper period was as follows. 1) After a normal training day, subjects refrained from training for 1 day, during which measurements of voluntary and evoked strength and an estimate of total blood volume were made. 2) On the following day, a needle biopsy was taken from the vastus lateralis using the Bergström technique (3) and subjects performed a treadmill run to exhaustion. 3) Over the next 5 days, they then participated in one of the three designated taper procedures. 4) On day 6, no exercise was performed and strength and blood volume measures were repeated as above. 5) On day 7, a post-taper needle biopsy sample was taken and the treadmill run to exhaustion was repeated. All pre- and posttaper measurements were made at the same time of day (±30 min). Subjects were instructed to maintain the same diets and energy intakes over all three tapers.

Measurements. The criterion running performance test was a timed run to exhaustion on a level treadmill at a constant velocity equivalent to each athlete’s average running velocity for his best 1,500-m race of that year. On the basis of the prediction equation of McMiken and Daniels (20), this velocity represented an energy requirement equivalent to \( \sim 115\% \dot{V}O_2_{\text{max}} \). Before the test, subjects performed a standard warm-up on the treadmill (12 min at 6 mph) and 3 min of stretching. They then stepped onto the treadmill, which was rotating at the preset velocity; timing began when they released their grasp of the handrail and terminated when they regrasped the handrail or began to stumble. Heart rate was continuously recorded throughout the test, as was \( \dot{O}_2 \) uptake (\( \dot{V}O_2 \)) by means of a computerized open-circuit system that calculated \( \dot{V}O_2 \) on-line every 20 s. Subjects received verbal encouragement throughout the run but were not allowed to view a clock or to receive any feedback regarding their performance times until completion of the entire study. Five minutes after termination of the test, a blood sample was taken by venipuncture for determination of plasma lactate concentration. Before the first taper, all subjects had performed the test on at least four occasions and were thus thoroughly familiar with the procedure.

Total blood volume was assessed on the nonexercise day before and after each taper by radioactive (\( ^{125}\)I) albumin tagging and red cell volume by \( ^{51}\)Cr tagging. Hemoglobin and hematocrit were also determined before and after each taper.

The needle biopsy samples taken before and after each taper were directly frozen in liquid nitrogen and subsequently assayed for glycogen concentration (2) and citrate synthase activity (19). One subject refused to undergo the biopsy procedure on his final taper but completed all other aspects of the study. Muscle data are thus confined to eight subjects.

Maximum voluntary isometric strength and percutaneously nerve-stimulated evoked contractile properties of the right knee extensors were measured on a custom-made dynamometer as previously described (22). Evoked contractile measurements included peak twitch torque, time to peak torque, and half-relaxation time. In addition, the extent of voluntary motor unit activation was measured by the interpolated twitch method (1). For this method, a supramaximal stimulus is delivered during a maximum voluntary contraction and the magnitude of the interpolated twitch, compared with that evoked at rest, is used to calculate motor unit activation.

Statistical significance was determined by analysis of variance with a repeated-measures design. When a significant main effect was detected, data were further analyzed with a Tukey post hoc analysis with \( P < 0.05 \) set as the acceptable level of significance.

RESULTS

The effects of the three different taper procedures on running performance and on maximum voluntary strength are summarized in Fig. 1. Pretaper running times to fatigue and knee extensor strength were remarkably consistent on all three occasions. After ROT time to fatigue decreased slightly (~3%), and after LIT it increased slightly (~6%), but these changes were not statistically significant. After HIT, subjects ran significantly longer (\( P < 0.05 \)) before fatiguing. This represented an improvement of ~22% compared with the pretaper performance. Maximum voluntary strength increased significantly and to a similar extent after all three taper procedures.

\( \dot{V}O_2_{\text{max}} \) (as indicated by peak \( \dot{V}O_2 \) during the run) and postrun plasma lactate concentrations (Table 1) were
not affected by any of the taper procedures. Peak twitch torque was \( \sim 10\% \) of the quadriceps before and after taper procedures. Peak twitch torque was \( \sim 10,19, \) and \( 13\% \) higher and motor unit activation \( \sim 0.5, 1.5, \) and \( 0.5\% \) higher after ROT, LIT, and HIT, respectively, but these changes were not statistically significant.

Muscle glycogen concentration and citrate synthase activity were similar in all pretaper situations (Fig. 2). After ROT muscle glycogen increased by \( \sim 8\% \) over pretaper values \( (P < 0.05) \), and after HIT it increased by \( \sim 15\% \) compared with pretaper concentrations. It was unaffected by LIT. Citrate synthase activity decreased significantly by \( \sim 13\% \) as a result of ROT and increased significantly by \( \sim 18\% \) as a result of HIT. Again, there was no change as a result of LIT.

Changes in total blood volume and red cell volume are illustrated in Fig. 3. Total blood volume showed a slight but significant decrease as a result of ROT, a slight but significant increase after HIT, and no change after LIT. Red cell volume showed a similar trend, but only the change after IIIIT was statistically significant \( (P < 0.05) \). Hematocrit increased from \( 42.8 \pm 0.2 \) to \( 43.9 \pm 0.2 \) with HIT, but the change was not statistically significant.

Subjects reported that they consumed a consistent diet of the same foods over all three tapers but that total energy intake tended to decrease during ROT. Food records kept by the subjects were not considered accurate enough for presentation in any more detail.

**DISCUSSION**

Although the intervals during HIT were performed at a slightly higher intensity than those during normal training, because of the extremely low training volume during HIT \( (<10\% \text{ of normal}) \) we think it unlikely that this would represent an additional training stimulus. We therefore interpret our data as indicating that trained middle-distance runners can significantly improve their performance on an exhaustive treadmill run by sharply reducing training volume while maintaining \( \text{(or slightly increasing) training intensity for a period of 7 days.} \)

This method is also superior to either a taper where both training intensity and volume are reduced or a period where no training is performed over 7 days.

On the basis of our finding that the group performance before all tapers was extremely consistent \((P = 0.05)\), we consider the timed treadmill run to be a reliable measure. It was selected over a 1,500-m race on the track because it would not be affected by such factors as weather or track conditions or the performance of other runners. Whether one can extrapolate from the performance on this test to race performance during an actual competition is, of course, open to debate, but it is probable that the same factors which cause fatigue during the treadmill run would also cause fatigue on the track.

Although a number of factors are probably involved in the development of fatigue during supramaximal exercise of this intensity \( (11, 18) \), we think it reasonable to assume that a major factor would be the elevation in intracellular \([H^+]\), which manifests itself by inhibiting glycogenolytic flux rate and possibly reducing the effectiveness of the contractile processes. In the present study, none of the manipulations affected \( \text{VO}_{2\text{max}} \), but time to fatigue was significantly prolonged as a result of HIT. Improvements in exercise endurance time independent of increases in \( \text{VO}_{2\text{max}} \) are not unusual \((5, 16) \) and are commonly interpreted as being due to adaptations at

**TABLE 1. \( \text{VO}_{2\text{max}} \) postexercise plasma lactate concentration, hematocrit, and evoked contractile properties of the quadriceps before and after taper**

<table>
<thead>
<tr>
<th></th>
<th>ROT</th>
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<th>LIT</th>
<th></th>
<th>HIT</th>
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<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>Pre</td>
<td>Post</td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td>( \text{VO}_{2\text{max}} ), ml\cdot kg(^{-1})\cdot min(^{-1})</td>
<td>67.2±1.7</td>
<td>67.0±1.2</td>
<td>66.9±1.1</td>
<td>66.7±1.1</td>
<td>67.2±1.4</td>
<td>67.3±1.4</td>
</tr>
<tr>
<td>Postexercise plasma lactate, mmol/l</td>
<td>13.1±1.9</td>
<td>12.9±2.1</td>
<td>12.3±1.7</td>
<td>13.5±1.1</td>
<td>12.5±2.0</td>
<td>13.4±1.7</td>
</tr>
<tr>
<td>Hematocrit, %</td>
<td>43.0±0.2</td>
<td>42.7±0.2</td>
<td>43.1±0.3</td>
<td>43.8±0.2</td>
<td>42.8±0.2</td>
<td>43.9±0.2</td>
</tr>
<tr>
<td>Peak twitch torque, N\cdot m</td>
<td>37.6±7.0</td>
<td>41.3±7.5</td>
<td>36.3±7.4</td>
<td>43.1±7.0</td>
<td>37.5±7.2</td>
<td>42.9±5.5</td>
</tr>
<tr>
<td>Motor unit activation, %</td>
<td>97.6±1.7</td>
<td>98.1±1.3</td>
<td>96.7±2.3</td>
<td>98.2±1.5</td>
<td>98.1±1.8</td>
<td>98.6±1.0</td>
</tr>
</tbody>
</table>

Values are means ± SD; \( n = 9 \). ROT, rest-only taper; LIT, low-intensity taper; HIT, high-intensity taper. \( \text{VO}_{2\text{max}} \) was assumed to be the peak value reached during the supramaximal treadmill run. Lactate concentrations refer to 5 min postexercise.
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A similar decrease in citrate synthase activity (9) and muscle respiratory capacity (6) has been noted in highly trained subjects as a result of 7 days of detraining. The mechanisms that promoted an increase in citrate synthase activity over the same time period with HIT are not known but are apparently related to the stimulus caused by an increase in training intensity and/or an increase in the recovery time which may be necessary for optimal mitochondrial protein synthesis to occur. LIT apparently provided enough training stimulus to prevent detraining but was not of sufficient intensity to promote a further increase in mitochondrial enzyme activity.

Although the duration of the treadmill run was probably too brief to have been affected by changes in resting muscle glycogen concentration, such changes could be of considerable significance for events of longer duration. The significant increase in muscle glycogen concentration after 1 wk of inactivity was a surprising finding and is in contrast to that of Costill et al. (6) that glycogen concentration in deltoid muscle decreased in a group of swimmers who detrained for 7 days. A possible interpretation is that, during their pretaper training, the athletes in the present study may have had slightly depressed glycogen concentrations due to chronic heavy daily training. The cessation of training in combination with a high carbohydrate intake (subjects attempted to maintain the same diets and energy intakes with each taper) may have stimulated an overshoot in glycogen synthesis (21). Such an interpretation does not, however, explain our finding that a similar overshoot was not detected after LIT, where training volume was reduced to <40% of the skeletal muscle level rather than improvements in \( O_2 \) delivery.

Adaptations at the muscle level that could improve endurance time at an exercise intensity that exceeds \( V_{O_2\max} \) would include 1) an elevation in mitochondrial capacity to extend the duration during which oxidative energy production can be maintained at maximum substrate flux, 2) an increased muscle buffering capacity, and/or 3) an increase in the rate at which \( H^+ \) effluxes from muscle. In the present study we have no direct measure of possible changes in mitochondrial integrity but the 18% increase in citrate synthase activity (Fig. 2) after HIT may be indicative of an increased capacity to maintain a high rate of oxidative energy production despite the potentially damaging or inhibitory effect of increasing intracellular temperature, \([H^+]\), lactate (independent of changes in \( pH \)), and perhaps even superoxide free radicals (10). Such an adaptation might also be considered beneficial through its potential to both forestall lactate production and to increase its removal by active muscle fibers.

Theoretically, an increase in muscle buffering or lactate and \( H^+ \) efflux rate might be reflected by elevations in postexercise plasma lactate concentration. Although plasma lactate concentration did not increase with the increased running time after HIT, any change may have been obscured by the increase in total blood volume that accompanied that condition. In addition, lactate removal kinetics may have been altered as a result of the taper procedure.

Our data indicate that citrate synthase activity was significantly reduced by ROT, significantly elevated by H1T', and unaffected by LIT (Fig. 2). A similar decrease in citrate synthase activity (9) and muscle respiratory capacity (6) has been noted in highly trained subjects as a result of 7 days of detraining. The mechanisms that promoted an increase in citrate synthase activity over the same time period with HIT are not known but are apparently related to the stimulus caused by an increase in training intensity and/or an increase in the recovery time which may be necessary for optimal mitochondrial protein synthesis to occur. LIT apparently provided enough training stimulus to prevent detraining but was not of sufficient intensity to promote a further increase in mitochondrial enzyme activity.

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FIG. 2. Resting muscle glycogen concentration (top) and citrate synthase activity (bottom) for vastus lateralis before and after each taper procedure (n = 8). Values are means ± SD and are expressed per unit dry weight. * Significant pre- to posttaper differences, \( P < 0.05 \).

FIG. 3. Total blood volume (top) and red cell volume (bottom) before and after each taper procedure (n = 9). Values are means ± SD. * Significant pre- to posttaper differences, \( P < 0.05 \).
that in the previous week. The overshoot in glycogen synthesis after HIT may relate to its depletion pattern during the higher intensity training as well as to the provision of adequate time for glycogen resynthesis afforded by the reduced training volume.

Pretraining measurements of voluntary strength were extremely consistent after each training cycle (Fig. 2). Our finding that this measure increased significantly as a result of all three taper procedures (and especially after ROT) suggests that voluntary strength of the quadriceps is suppressed by chronic intensive running training but recovers when training is reduced or discontinued. A similar finding has been noted in a group of swimmers who increased their power output on a biokinetic swim bench after 14 days of reduced swimming training (7). Although in the present study neither achieved statistical significance, both twitch torque and maximum motor unit activation showed a tendency to increase as a result of all three tapers. We interpret this as indicating that the suppressed strength found during periods of intense training may be a result of both alterations at the muscle level and inhibition at the level of the central nervous system. In the present study, it is doubtful whether the changes in voluntary strength influenced running performance, since strength increased after all three tapers while performance was significantly improved only after HIT. Such changes could, however, have implications for shorter distance events or team sports.

Increases in total blood volume similar to those after HIT have been observed after as little as 3 days of training (12) but were not accompanied by the increases in red cell volume found in the present study. This change in red cell volume was somewhat surprising, since the 6 days over which it occurred represent what is considered to be the minimal time course for maturation of erythrocytes after initiation of hematopoiesis (4). One interpretation of our data is that, during periods of heavy training, both red cell production rate and red cell destruction rate may be elevated (13). The sudden reduction in training volume might decrease the incidence of intravascular hemolysis and improve the reticuloendothelial system’s capacity for reuptake of dying erythrocytes (13), resulting in a transient period during which there is a net increase in erythrocytes. Such an explanation leads, of course, to the question as to why red cell volume did not increase as a result of the other two tapers. Perhaps the training volume in HIT was sufficient to offset this mechanism and perhaps an increased red cell volume would have been detected after ROT had we examined blood characteristics over a shorter time period (e.g., 2–3 days). The changes in blood and red cell volume did not affect $\text{Vo}_2\text{max}$ but may have influenced treadmill performance by altering blood buffering capacity.

In summary, our data indicate that highly trained middle-distance runners can improve their performance as a result of a taper period during which training volume is greatly reduced but training intensity remains high. The improvement may be the result of an increase in muscle oxidative enzyme activity and/or an increase in blood and red cell volume. In addition, a reduction in training volume results in an increase in muscle strength and glycogen concentration. The reduced training volume apparently allows sufficient recovery and adaptation (supercompensation) to occur, whereas the brief high-intensity training provides enough stimulus to prevent detraining. The time course, volumes, and intensities for the training tapers used in the present study were arbitrarily selected, and further investigations are warranted to determine the optimal combinations that would result in the greatest improvement in performance.

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