Cardiorespiratory and metabolic characteristics of detraining in humans

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ABSTRACT

MUJIKA I., and S. PADILLA. Cardiorespiratory and metabolic characteristics of detraining in humans. *Med. Sci. Sports Exerc.,* Vol. 33, No. 3, 2001, pp. 413–421. Detraining can be defined as the partial or complete loss of training-induced adaptations, in response to an insufficient training stimulus. Detraining is characterized, among other changes, by marked alterations in the cardiorespiratory system and the metabolic patterns during exercise. In highly trained athletes, insufficient training induces a rapid decline in $\dot{V}O_{2max}$, but it remains above control values. Exercise heart rate increases insufficiently to counterbalance the decreased stroke volume resulting from a rapid blood volume loss, and maximal cardiac output is thus reduced. Cardiac dimensions are also reduced, as well as ventilatory efficiency. Consequently, endurance performance is also markedly impaired. These changes are more moderate in recently trained subjects in the short-term, but recently acquired $\dot{V}O_{2max}$ gains are completely lost after training stoppage periods longer than 4 wk. From a metabolic viewpoint, even short-term inactivity implies an increased reliance on carbohydrate metabolism during exercise, as shown by a higher exercise respiratory exchange ratio. This may result from a reduced insulin sensitivity and GLUT-4 transporter protein content, coupled with a lowered muscle lipoprotein lipase activity. These metabolic changes may take place within 10 d of training cessation. Resting muscle glycogen concentration returns to baseline within a few weeks without training, and trained athletes' lactate threshold is also lowered, but still remains above untrained values. **Key Words:** TRAINING CESSATION, OXYGEN UPTAKE, ENDURANCE PERFORMANCE, RESPIRATORY EXCHANGE RATIO, INSULIN SENSITIVITY

According to the principle of training reversibility, regular exercise training results in various physiological adaptations that enhance athletic performance, but the stoppage or marked reduction of training leads to a partial or complete reversal of these adaptations, thus compromising athletic performance. The principle of training reversibility is therefore the principle of detraining (23). Detraining has been recently redefined as the partial or complete loss of training-induced anatomical, physiological, and performance adaptations, as a consequence of training reduction or cessation (42). The need to redefine the concept of detraining rises from the failure of the existing exercise science literature in discriminating between the process through which a trained individual loses some or all of their training-induced adaptations (e.g., reduced training, training cessation, bed rest confinement) and the lost adaptations themselves, which are the outcome of that process. In addition, there has been some confusion between the terms "detraining," as defined above, and "detraining syndrome" (also known as "relaxation syndrome"), which is a clinical entity arising when athletes with a long endurance-training history suddenly abandon their regular physical activity (42), characterized by a tendency to dizziness and fainting, nonsystematic precordial disturbances, sensations of cardiac arrhythmia, extrasystolia and palpitation, headaches, loss of appetite, gastric disturbances, profuse sweating, insomnia, anxiety, and depression (28,46,56).

Several factors, such as illness, injury, travel or vacation may often interfere with an athlete's training process, forcing him to alter his physical activity pattern by reducing or even interrupting training. It is therefore necessary to identify the physiological and performance consequences of such interferences, as well as the possible mechanisms responsible. Moreover, it has often been reported that a training stimulus insufficient to maintain training-induced adaptations has a marked impact on the cardiovascular and respiratory systems, and results in altered metabolic patterns (6,7,15,22,30,42,43,55,61).

This brief review deals with the cardiorespiratory and metabolic characteristics of detraining. Given that highly trained athletes approaching their higher limits of adaptation, and moderately or recently trained subjects may show different detraining characteristics (6,7,15,22,30,40,44,61), the available data on these two types of population will be reviewed separately.

CARDIORESPIRATORY DETRAINING

Maximal oxygen uptake. Training cessation for a period shorter than 4 wk has been reported to induce a rapid reduction in maximal oxygen uptake ($\dot{V}O_{2max}$) in highly trained individuals with a large aerobic power (>62 mL·kg^{-1}·min^{-1}) and an extensive training background (40). Indeed, Houston et al. (27) showed that 15 d of inactivity (7 d of leg casting followed by 8 d without training) led to 4% reductions in $\dot{V}O_{2max}$ in well-trained endurance runners. $\dot{V}O_{2max}$ also decreased by 4.7% in a group of endurance-trained

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runners after 14 d of training cessation (24,25). Coyle et al. (8) studied eight endurance-trained subjects who refrained from training for 2 or 4 wk, and reported 6% $\dot{V}O_{2max}$ declines during upright exercise. Even more pronounced results were observed by Martin et al. (33), who indicated that 3–8 wk of physical deconditioning in highly trained subjects brought about a 20% reduction of $\dot{V}O_{2max}$. The same has been shown to be true for team sport athletes. As a matter of fact, male basketball players not training for 4 wk after their competitive season showed a 13.8% $\dot{V}O_{2max}$ reduction (18), and 11 college soccer players decreased their $\dot{V}O_{2max}$ by 6.9% during 5 wk of training cessation (14).

Conflicting results, however, have also been published. Indeed, 15 distance runners were shown to maintain $\dot{V}O_{2max}$ after 10 d of training cessation (11). The same was true for a group of female college swimmers not training for a similar period of time (4), and a group of soccer players after 3 wk of training stoppage (3). These conflicting results could be partly explained by the variable amount of physical activity performed by the athletes during the period of training stoppage.

The $\dot{V}O_{2max}$ loss during training cessation seems to be dependent on time and initial fitness level. Indeed, seven endurance-trained subjects refrained from training for 84 d, and their $\dot{V}O_{2max}$ declined by 7% in 21 d, and by 16% in 56 d, then stabilized at that level, which was still 17.3% higher than that of sedentary control subjects. A correlation of 0.93 was observed between trained $\dot{V}O_{2max}$ and percent decline of $\dot{V}O_{2max}$ with inactivity (10). Also, a linear decrease in $\dot{V}O_{2max}$ was observed until the 45th d of training withdrawal in a group of highly trained cyclists and endurance runners during 60 d of training cessation (46). Very long-term (2 yr) training stoppage has also been shown to be characterized by $\dot{V}O_{2max}$ declines of 6.3% in college badminton players (38).

These and other similar data (1,9,13,33) indicate that $\dot{V}O_{2max}$ of highly trained athletes decreases progressively and proportionally to the initial $\dot{V}O_{2max}$ during the first 8 wk of training cessation. This decline ranges between 4 and 20%. Most studies, however, indicate that $\dot{V}O_{2max}$ ceases to decline thereafter and remains higher than that of untrained counterparts (9,10,14), although there has been one report of a return to sedentary values (13).

In protocols with high training/detraining time ratios, it has been shown that short-term inactivity (3 wk) may not produce a decline in $\dot{V}O_{2max}$ in subjects with a lower post-training aerobic power (<56 mL·kg^{-1}·min^{-1}) and a limited (<1 yr) prior training history (40). Houmard et al. (26) observed that recently trained middle-aged individuals retained their training-induced gains in $\dot{V}O_{2max}$ (12.0%) during 2 wk of either 50% reduced training frequency or complete training cessation. However, the $\dot{V}O_{2max}$ of recently trained individuals (4–8 wk of training) has also been shown to decline by 3.6–6% during 2–4 wk of training cessation. Indeed, 4 wk of inactivity resulted in reduced $\dot{V}O_{2max}$ (3.6%) in subjects who had previously been endurance trained and heat acclimated for 4 wk (48). Reductions of 6% in $\dot{V}O_{2max}$ have been reported in subjects not training for 3 wk after a 6-wk endurance training program that induced a 9.6% increase in $\dot{V}O_{2max}$ (60). It has also been shown that only 24% of the gains in $\dot{V}O_{2max}$ induced by 6 wk of interval training were retained during 2 wk of training cessation in young, healthy females (50).

Longer periods of inactivity most often result in a complete reversal of $\dot{V}O_{2max}$ to pretraining values in the recently trained. Eight weeks of training cessation have been shown to decrease $\dot{V}O_{2max}$ toward pretraining values (29). Also, the effects of 8 wk of endurance training on $\dot{V}O_{2max}$ completely disappeared after 4–12 wk of deconditioning in a group of eight previously sedentary women (62). Middle-aged (35–54 yr) men who stopped training for 6 months following a 15-wk walking training program have been reported to decrease their $\dot{V}O_{2max}$ by 7.2%, returning to baseline values (39). Simoneau et al. (53) observed an 8.4% reduction in $\dot{V}O_{2max}$ after 7 wk without training following a 15-wk intermittent training program. Forty-four college women lost the gains in $\dot{V}O_{2max}$ attained during 10 wk of endurance training within 10 wk of consequent inactivity (17). Fournier et al. (16) trained 12 adolescent boys with either endurance or sprint exercise for 3 months, then stopped training them for a further 6 months. The latter resulted in a return of $\dot{V}O_{2max}$ to pretraining levels (8.0% and 2.3% reductions in endurance-trained and sprint-trained subjects, respectively).

However, a certain degree of retention of training-induced gains in $\dot{V}O_{2max}$ has also been reported. Ready and Quinney (51) observed decreased $\dot{V}O_{2max}$ values after 9 wk of training cessation in young subjects who had previously trained for 9 wk, but $\dot{V}O_{2max}$ remained above pretraining values. A similar result was reported by Després et al. (12) after a 20-wk/7-wk training/detraining paradigm in young (23.4 yr) males and females.

Blood volume. A decline in blood volume, which may be apparent within the first 2 d of inactivity (11,58), appears to be largely responsible for the observed reduction in cardiovascular function during short periods of training cessation. Houmard et al. (24) attributed part of the reduction in $\dot{V}O_{2max}$ observed in a group of distance runners after 14 d without training to a 5.1% reduction in estimated resting plasma volume. Identical 5% decreases in plasma volume have been observed by other authors in distance runners who ceased training for 10 d (11,58). Moreover, eight endurance athletes who refrained from training for 2 or 4 wk were reported to suffer 9% and 12% declines in blood and plasma volumes during upright exercise, respectively (8). The effects of longer periods of training cessation on the blood volume of highly trained athletes have not been reported in the exercise science literature.

As in the case of athletes, recently trained individuals' blood volume decreases with short-term inactivity. After 4 wk of endurance training and heat acclimation, 16 young males underwent 4 wk of training stoppage, which resulted in reduced $\dot{V}O_{2max}$ (3.6%) and blood volume (4.7%). Red cell volume decreased by 98 mL, plasma volume by 248 mL, total plasma protein by 16 g, and plasma albumin by 12 g. In addition, plasma volume changes were directly

related to plasma protein dynamics, as loss of protein appeared to be responsible for 97% of the reduction in plasma volume (48). Moreover, 6 d of training cessation have been shown to fully reverse the effects of short-term training (6 d) on resting plasma volume and concentrations of aldosterone and epinephrine during submaximal intensity exercise, but not those of arginine vasopressin, suggesting the implication of plasma volume as a significant variable in modifying the exercise response of fluid and electrolyte hormones (52). In contrast, a reduction of daily aerobic activity (active deconditioning) for 8 wk resulted in a significant (7%) $\dot{V}O_{2max}$ decrease in 8 women and 11 men, all untrained subjects. However, decreases in total blood volume (4.0%) and plasma volume (3.1%) were nonsignificant, and the baseline hemodynamic variables analyzed by the investigators were unaltered by the deconditioning period. These data suggest that some of the changes in $\dot{V}O_{2max}$ accompanying physical deconditioning may be independent of changes in total blood volume and plasma volume (49).

Heart rate. Whereas resting heart rate has been reported not to change in athletes following short-term (10 d) training cessation (11), exercise heart rate at both submaximal and maximal intensities increases by approximately 5–10%, as a consequence of the above-mentioned reduction in blood volume. Coyle et al.(8), studying endurance athletes, observed an 11% higher heart rate during submaximal exercise after 2–4 wk without training. They also reported that plasma volume expansion in the detrained state reversed these effects, suggesting that the reduction in blood volume during training cessation limits ventricular filling during upright exercise and is largely responsible for the inactivity-induced decline in cardiovascular function. Exercise heart rate at submaximal intensities (75 and 90% of $\dot{V}O_{2max}$) increased by 11 beats·min^{-1} in a group of endurance runners after 14 d without training, and maximal heart rate also increased by 9 beats·min^{-1} (24). Madsen et al. (32) also observed 6–7 beats·min^{-1} higher heart rates at submaximal exercise intensities, but unchanged maximal heart rates after 4 wk of insufficient training stimulus in nine endurance athletes. In contrast, Cullinane et al. (11) measured a 5% increase in maximal heart rate in 15 runners after 10 d of training cessation.

The effects of longer periods without training on athletes' heart rate have also been studied. Seven endurance-trained subjects exercised at the same absolute submaximal intensity in a trained state and during 84 d of training cessation. Heart rate increased, respectively, from 84% to 93% of maximal values during 56 d of inactivity, but stabilized thereafter. Exercise of the same relative submaximal intensity elicited almost identical percentages of maximal heart rate (9). The same subjects' maximal heart rate increased by 4–5% after the initial 12–21 d of training cessation, but it did not change thereafter (10). Eleven college soccer players showed increased heart rate at submaximal exercise intensities during 5 wk without training (14). This result, along with a shortened length of the cardiac isovolumetric contraction phase at rest, led this author to suggest an increased sympathoadrenergic tone as a result of inactivity. Drinkwater and Horvath (13) and Michael et al. (35) observed increased submaximal heart rates in adolescent (15–18 yr) female athletes as a result of 12 and 23 wk of training cessation following the track season, respectively. In addition, the latter investigators also observed a progressive 16% increase in postexercise recovery heart rate, but stated that both submaximal and recovery heart rate increases seemed to level off after the initial 7 wk of training stoppage. In line with the above results, six college football players showed increased heart rate during submaximal exercise after 9 wk of training cessation following their competitive season. Their values, nevertheless, remained lower than those of sedentary counterparts (47).

According to the scarce literature available, resting and maximal heart rates return to untrained values following short-term inactivity in recently trained subjects, whereas heart rate during submaximal exercise is not affected. Indeed, the effects of 8 wk of endurance training on resting heart rate and maximal heart rate were reversed within 4 wk of inactivity in a group of eight previously sedentary women (62). In a study on nine previously sedentary young (20 yr) subjects, 3 wk of training cessation did not reverse the reductions in submaximal heart rate produced by 6 wk of endurance training (60).

Longer term detraining studies indicate that recently trained individuals' maximal heart rate is not affected by training cessation. Forty-four college women retained to some degree the change in maximal heart rate resulting from 10 wk of endurance training during 10 wk of consequent inactivity (17). A similar result was observed in a group of adolescent boys (16–17 yr) taking part in a 12-wk training/24-wk detraining protocol (16). Resting heart rate, on the other hand, increased from 70 beats·min^{-1} to 74 beats·min^{-1} (17) and from 60 beats·min^{-1} to 74 beats·min^{-1} (62) in young women, indicative of a complete reversal of the training effects. Also, heart rates during submaximal exercise have been shown to return toward baseline values after 7 wk of training stoppage consecutive to 7 wk of training in young females (57), and after 12 wk of inactivity consecutive to 12 wk of brisk walking training in middle-aged women (20).

Stroke volume. It has been stated that the reduced maximal aerobic capacity observed in highly trained subjects after short periods of training cessation is a consequence of the decline in stroke volume resulting from the reduced blood volume that characterizes detraining (10). Coyle et al. (8) observed a 12% lower stroke volume during upright exercise in a group of endurance athletes who stopped training for 2–4 wk. This effect was reversed after plasma volume expansion, but this intervention did not restore $\dot{V}O_{2max}$, which remained 3% lower than in the trained state. In seven endurance-trained subjects who refrained from training for 84 d, stroke volume declined by 10% after the initial 12 d without training, and averaged 10–14% below trained levels during 12–84 d of inactivity. Values were not different from control. The authors indicated that the decline in $\dot{V}O_{2max}$ during the initial 21 d of training cessation was associated with a decreased stroke volume (10). Another study by the same group showed that

3–8 wk of physical deconditioning in highly trained subjects, which brought about a 20% reduction of $\dot{V}O_{2max}$, resulted in a significant 17.2% reduction in stroke volume during upright exercise (33). On the other hand, it has been reported that resting stroke volume index ($mL \cdot beats^{-1} \cdot m^{-2}$) and ejection fraction rose in a group of highly trained road cyclists and endurance runners during 60 d of training cessation. These changes were paralleled by a linear decrease in $\dot{V}O_{2max}$ until the 45th d of training withdrawal (46).

The only available report on moderately trained subjects' stroke volume during long-term training cessation indicates that 11 middle-aged (43.7 yr) men who stopped training for 6 months after a 15-wk walking training program showed a 3.9% decline in stroke volume. This decline could have been in part responsible for the 7.2% lower $\dot{V}O_{2max}$ observed after the deconditioning period (39).

Cardiac output. The magnitude of the above-mentioned increase in exercise heart rate values characterizing cardiovascular detraining is insufficient in highly trained athletes to counterbalance the decline in stroke volume, and results in a decreased cardiac output. Coyle et al. (10) reported that estimated maximal cardiac output stabilized at 8% below trained values after 21 d of training cessation in endurance athletes, not falling beyond that level between the 21st and 84th d without training. The same group of investigators informed of a progressive shift from 84% to 94% of maximal cardiac output between the trained and detrained (84 d without training) states when subjects exercised at the same absolute submaximal intensity, whereas exercise of the same relative submaximal intensity elicited almost identical percentages of cardiac output (9). Submaximal supine exercise also elicited slightly but significantly higher cardiac outputs following 8 wk of training cessation in highly trained subjects (33). Concerning resting cardiac output, the only data available in the literature indicate that detrained cyclists' and runners' cardiac index ($L \cdot m^{-2} \cdot min^{-1}$) increases at rest as a result of the increased stroke volume index (46).

Miyashita et al. (39) also observed a 6.9% decline in maximal cardiac output after long-term (6 months) training cessation in previously sedentary men who had been training for 15 wk.

Cardiac dimensions. Altered cardiac dimensions have been observed as a result of short-term training cessation in highly trained athletes. As a matter of fact, Martin et al. (33) reported significant reductions in left ventricular end-diastolic dimension (11.8%) and left ventricular wall thickness (25.0%), and an increased mean blood pressure during upright exercise in athletes who refrained from physical training for 3 wk. These investigators attributed these changes to a reduction in left ventricular mass (19.5%). The increased mean blood pressure during upright exercise measured by these and other authors (8) could indeed be because of a reduced left ventricular mass, coupled with a higher total peripheral resistance (TPR), which increased by 8% after 2–4 wk of training cessation (8). However, Cullinane et al. (11) did not observe any change in the cardiac dimensions and the blood pressure of 15 distance runners following 10 d of training stoppage.

During longer periods of training cessation (8 wk), the left ventricular end-diastolic dimension of highly trained athletes declined in parallel with stroke volume while performing upright exercise (33). Meanwhile, left ventricular posterior wall thickness decreased progressively by 25%, but left ventricular mass was unaltered after the decline observed following the initial 3 wk of deconditioning. Giannattasio et al. (19), on the other hand, observed that former professional sprint runners and hammer throwers had left ventricular end-diastolic diameter and mass index similar to those of sedentary subjects after 4–5 yr of training cessation. They also indicated that the impairment of the cardiopulmonary reflex that they observed in athletes is largely reversible with training cessation-induced regression of cardiac hypertrophy. In line with these results, Pavlik et al. (46) reported unchanged wall thickness and internal ventricular diameters in road cyclists and endurance runners during 60 d of inactivity. On the basis of their observations, these authors assumed that the cardiovascular regulation undergoes a peculiar shift in the period of training stoppage, in that the persisting cardiac enlargement is associated with a temporarily unstable autonomous control caused by the relatively high level of both sympathetic and parasympathetic activity, and that this imbalance may also lead to a hyperkinesis-like syndrome in athletes after an abrupt cessation of endurance training.

Mean and systolic blood pressures have been shown to increase along with TPR during 9–12 wk without training in six endurance cyclists and runners (33), and in six college football players (47); nevertheless, the values of the latter remained lower than those of sedentary counterparts. However, there has also been one report of unchanged blood pressure in seven female track athletes, aged 14–17 yr, following 12 wk of postcompetitive-season training cessation (13).

To the best of the authors' knowledge, there have been no reports on the effects of training cessation on cardiac dimensions in recently trained individuals, but the effects of 8 wk of endurance training on systolic and diastolic blood pressures have been shown to be reversed within 4 wk of detraining in a group of eight previously sedentary women (62).

Ventilatory function. Ventilatory function has been shown to suffer a rapid deterioration when highly trained athletes stop exercising. Maximal ventilation decreased in six runners after 15 d without training (27). Also, male basketball players not training for 4 wk after their competitive season showed a deterioration of cardiorespiratory efficiency, as shown by reduced maximal ventilation (9.3%), O_2 pulse (12.7%), and increased ventilatory equivalent (3.9%) (18). In the above-mentioned investigations, maximal ventilatory volume decreased in parallel with $\dot{V}O_{2max}$. Although Cullinane et al. (11) did not observe a decreased maximal ventilatory volume in male long-distance runners after 10 d of inactivity, they did observe a significantly lower maximal O_2 pulse.

Maximal ventilatory volume has also been shown to be negatively affected by longer periods of training cessation. Indeed, it decreased by 10% in 11 college soccer players in 5 wk (14), by 10.3% in young female track athletes in 12 wk (13), and by 14.5% in 5 badminton players in 2 yr (38). In addition, the latter athletes were shown to significantly increase their hypercapnic ventilatory responsiveness. Also, ventilatory volume shifted from 53% to 71% of the maximal in 56 d without training in endurance athletes (9). Drinkwater and Horvath (13) and Michael et al. (35) also reported markedly increased submaximal ventilatory volume and ventilatory equivalent in female athletes during long-term training cessation.

An impairment of ventilatory function as a result of training cessation has also been observed in recently trained individuals. Fringer and Stull (17) reported a decline in maximal ventilatory volume from 96.4 $L \cdot min^{-1}$ to 87.1 $L \cdot min^{-1}$, and an increased maximal ventilatory equivalent from 3.6 $L \cdot dlO_2^{-1}$ to 4.2 $L \cdot dlO_2^{-1}$ after 4 wk without training in college women. Miyashita et al. (39) reported a 13.9% lower maximal ventilatory volume and a 7.1% higher maximal ventilatory equivalent in recently trained males who stopped training for 6 months. A complete reversal of training-induced improvements in maximal ventilatory volume has also been shown in young females participating in an 8-wk training/12-wk detraining protocol (62). Also, submaximal ventilatory volume and ventilatory equivalent deteriorated to pretraining levels during 7 wk of inactivity consecutive to 7 wk of training in young females (57).

Endurance performance. It has been repeatedly shown that highly trained athletes' endurance performance suffers a rapid deterioration when the training stimulus disappears or is insufficient to maintain training-induced adaptations. Female competitive swimmers were 2.6% slower in a 366-m swim after only 10 d without training (4). Exercise time to exhaustion has also been reported to be reduced during training cessation, by 9.2% (24,25) and 25% (27) in 2 wk, and by 7.6% (8) and 21% (32) in 4 wk. The latter investigators stated that altered substrate utilization and/or altered Mg^{2+} transport from the extracellular to the intracellular area, which could inhibit Ca^{2+} release from the sarcoplasmic reticulum, could have contributed to the reduced endurance performance after training stoppage. It is worth noticing that Houmard et al. (24) did not observe a decline in running economy at submaximal exercise intensities (75 and 90% of $\dot{V}O_{2max}$) in their 12 distance runners, which suggests that the short-term training cessation-induced performance impairment was primarily because of the loss in cardiorespiratory fitness suffered by the athletes.

One hundred- and 200-m swimming performance has also been shown to decline by 3–13% in national and international level swimmers during the longer-term inactivity period in between two training seasons (41). Also, exercise time to exhaustion has been reported to decline by 23.8% in 5 wk of training cessation in 11 college soccer players (14). In addition, endurance-trained male and female runners' oxygen uptake during a standardized submaximal exercise task increased significantly by about 3–8% following 12 wk of training cessation (13). In line with these results, Coyle et al. (9) observed that a submaximal exercise bout requiring a $\dot{V}O_2$ of 3.11 ± 0.23 $L \cdot min^{-1}$ (74 ± 2% of $\dot{V}O_{2max}$) when their athletes were trained, elicited a $\dot{V}O_2$ of 3.20 ± 0.25 $L \cdot min^{-1}$ (90 ± 3% of $\dot{V}O_{2max}$) after 84 d without training.

Recently trained individuals' endurance performance does not seem to be significantly affected by short-term training cessation. Indeed, Houmard et al. (26), studying a group of previously sedentary middle-aged males and females, reported that the 19.4% gain in time to exhaustion achieved during 12 wk of training was retained during the following 2 wk of inactivity. Similar results were observed by Ready et al. (50) during a 6-wk training/2-wk detraining protocol in young females. Long-term inactivity, on the other hand, appears to result in significant or complete reversal of the performance gains achieved through training. This has been proven true in young (25 yr) sedentary males and females who stopped training for 7 wk after 15 wk of training, and whose work output during a 90-s effort fell by 8.3% (53); in college women participating in a 10-wk training/10-wk detraining protocol, and whose total work performed during a maximal exercise task decreased by 22% (17); in young females training for 8 wk and not training for the following 12 wk, as a result of which time to exhaustion declined by approximately 37%, reverting to pretraining values (62); and in middle-aged males training for 15 wk and not training for 6 months, and whose time to exhaustion declined by 13.5% (39).

METABOLIC DETRAINING

Substrate availability and utilization. An increased respiratory exchange ratio (RER) at submaximal and maximal exercise intensities is one of the metabolic consequences of a short period of insufficient training stimulus in athletes. Moore et al. (40) demonstrated in an athletic population that 3 wk of training stoppage result in an increased RER from 0.89 to 0.95 at submaximal intensity exercise (60% of $\dot{V}O_{2max}$). In another investigation, seven endurance-trained athletes exercised at the same absolute submaximal intensity in a trained state and during 84 d of training cessation, and RER increased from 0.93 to 1.00. Exercise of the same relative submaximal intensity elicited almost identical percentages of maximal heart rate, ventilation, and cardiac output, but RER increased from 0.93 to 0.96 (9). Also, nine highly trained endurance athletes' RER shifted from 0.89 to 0.91 when cycling at 75% of $\dot{V}O_{2max}$ after 4 wk of insufficient training (32). In a group of endurance-trained runners, maximal RER also increased from 1.03 to 1.06 after 14 d without training (24). Finally, Drinkwater and Horvath (13) showed higher RER values during both submaximal and maximal exercise in female athletes 12 wk after the end of the track season. Taken as a whole, these results are a clear indication of a shift toward an increased reliance on carbohydrate as an energy substrate for exercising muscles, in concomitance with a decreased contribution from lipid metabolism.

In addition, there have been several reports of a rapid decline in sensitivity for insulin-mediated whole-body glucose uptake during short-term inactivity. Five days without training were enough in endurance athletes to decrease insulin sensitivity to levels found in untrained subjects (36). The same authors, using the sequential hyperglycemic clamp technique, reported that glucose-induced β-cell secretion increased slightly toward untrained levels during the same period, indicating that β-cells are subjected to an adaptation during training (37). Hardman et al. (21) observed that the postprandial serum insulin response was 15.8% higher in endurance athletes after 6.5 d without exercise. In a similar short-term inactivity study (6 d) in endurance runners, it was shown that training cessation reduced glucose disposal rates after insulin infusion by 14.2–29.5%, despite 10.0–21.9% higher plasma insulin. Insulin clearance and muscle GLUT-4 transporter protein were also reduced by 8–29% and 17.5%, respectively. These results indicated that short-term inactivity decreases insulin action in endurance runners, suggesting that a reduction in muscle GLUT-4 transporter level may play a role in the decrease in glucose disposal rates (59). McCoy et al. (34) reported that 10 d of training stoppage in trained triathletes resulted in a 43.3% increase in the area under the insulin response curve during an oral glucose tolerance test, but still remained 24.3% below values of untrained control subjects. No change was observed following inactivity in the glucose response to the test, but GLUT-4 protein levels decreased by 33.2%, in parallel with a 28.6% reduction in citrate synthase activity, suggesting that glucose transport and oxidation are regulated by the muscle activity level. In agreement with the previous findings, the area under the glucose and insulin curves increased by 65% and 73%, respectively, during 7–10 d without training in endurance-trained athletes. Resting metabolic rate (RMR) fell by 4%, and the RER during the oral glucose tolerance test increased by the same amount. However, no change was observed in the calf and forearm blood flow. These data indicated a deterioration in glucose tolerance and energy metabolism, but these changes did not seem to be mediated by limb blood flow (2). Following 14 d of training cessation, Houmard et al. (25) observed an increased area under the glucose curve (14.8%) in 12 endurance runners, as well as in the insulin curve in both endurance (30.3%) and strength (23.3%) athletes during an oral glucose tolerance test. Insulin sensitivity index decreased by 23.7% in the former, and 16.0% in the latter. However, they observed no change in GLUT-4 content in endurance runners or in weight lifters. This lack of change was evident despite a 25.3% decrease in citrate synthase activity in the endurance runners. The authors concluded that the decrement in insulin sensitivity with training cessation was not associated with a decrease in GLUT-4 protein content, and that muscle oxidative capacity does not necessarily change in tandem with GLUT-4 protein content.

On the other hand, short-term (2 wk) training cessation from endurance running has been shown to yield a condition that favors the storage of adipose tissue, as there is a marked increase in adipose tissue lipoprotein lipase activity, coupled with a marked decrease in muscle lipoprotein lipase activity (54). Moreover, endurance-trained subjects in the fasted state have been reported to increase their concentrations of triacylglycerol (47%), as well as their very-low-density lipoprotein cholesterol (28.2%) and the ratio of total cholesterol to high-density lipoprotein cholesterol (7.5%) during 6.5 d without exercise. Concentrations of nonesterified fatty acids, on the other hand, decreased during the same period by 43.5%. Because of a higher rate of triacylglycerol removal, probably related to their high lipoprotein lipase activity attributable to their large and well-vascularized skeletal muscle mass, endurance-trained subjects usually exhibit low levels of postprandial lipemia. However, 6.5 d without training increased postprandial lipemia by 42.2%, as shown by the increased area under the plasma triacylglycerol versus time curve, indicating that frequent exercise is necessary to maintain a low level of postprandial lipemia in endurance-trained subjects (21). Ten days of training cessation also resulted in a 15% reduction in high-density lipoprotein cholesterol and a 10% increase in low-density lipoprotein cholesterol in endurance-trained athletes (58).

Altered metabolic patterns have also been observed as a result of short-term training stoppage in recently trained individuals. Moore et al. (40) reported that RER while exercising at 60% of $\dot{V}O_{2max}$ shifted from 0.87 to 0.96 after 3 wk without training consecutive to a 7-wk endurance training program. Fournier et al. (16) observed significant declines in RER values during 3 months of training, followed by a partial reversal during the subsequent 6 months of inactivity in 12 adolescent (16–17 yr) males. Smith and Stransky (57), on the other hand, were the only authors to report unchanged exercise RER values during a 14-wk training/detraining protocol in 16 young (21.2 yr) women. Houmard et al. (26) observed a return of the insulin sensitivity index and the GLUT-4 transporter protein concentration to initial values after a 12-wk training/2-wk detraining paradigm in previously sedentary middle-aged males and females. Moreover, Després et al. (12) showed that 50 d of physical inactivity completely reversed the training-induced increase in epinephrine-stimulated lipolysis attained by 12 young (23.4 yr) men during 20 wk of aerobic training. Additionally, subjects regained their initial body fatness level after the detraining period. Finally, the improvements on serum concentrations of high-density lipoprotein cholesterol achieved during 12 wk of brisk walking training were reversed in previously sedentary women aged 47 yr during the following 12 wk without training (20).

Blood lactate kinetics. Highly trained athletes have been shown to respond to submaximal exercise of the same absolute intensity with higher blood lactate concentrations after only a few days of training cessation. Competitive swimmers' skeletal muscle metabolic characteristics have been reported to suffer dramatic changes affecting blood lactate kinetics in as little as 1–4 wk without training. Muscle respiratory capacity decreased by 50% after 1 wk of inactivity in a group of eight swimmers. When subjects performed a standardized 183-m submaximal swim,

postswim blood lactate was 2.3 times higher, pH significantly lower (7.183 vs 7.259), bicarbonate concentration 22.7% lower, and base deficit twice as high (5). A group of 24 college swimmers not training for 4 wk after 5 months of competitive training showed a 5.5 $mmol \cdot L^{-1}$ increase in blood lactate concentration following a standardized 183-m submaximal swim, which was also indicative of a reduction in the muscle oxidative capacity, and/or a change in the swimmers' mechanical efficiency (45). Similar results were observed by Claude and Sharp (4) in seven female swimmers who refrained from training for 10 d. Endurance runners and cyclists performing an exercise task of the same relative submaximal intensity before and after 84 d of training stoppage showed a shift in blood lactate concentration from 1.9 $mmol \cdot L^{-1}$ to 3.2 $mmol \cdot L^{-1}$, along with a marked decline in the muscle respiratory capacity (9). An increased blood lactate concentration at submaximal exercise intensity has also been reported in six college football players after 9 wk of postseason break (47). Moreover, the lactate threshold has been shown to decline with 84 d of inactivity from 79.3% to 74.7% of $\dot{V}O_{2max}$, but to remain above sedentary control values of 62.2% (9). That there is a decline in the lactate threshold with training cessation has been confirmed by a recent meta-analysis study (31).

Recently trained subjects' exercise blood lactate concentration does not seem to be as affected by short-term training cessation. As a matter of fact, Wibom et al. (60) indicated that 3 wk without training did not reverse the reductions in submaximal blood lactate concentration produced by a 6-wk endurance training program in nine young (20 yr) males, and Ready et al. (50) reported a 40% retention of the gains in peak blood lactate concentration induced by 6 wk of interval training during 2 wk without training in young (23.9 yr) healthy females. In a longer term training/detraining study, Hardman and Hudson (20) reported an incomplete reversal of submaximal and maximal exercise blood lactate concentration values toward baseline after 12 wk without training, subsequent to 12 wk of brisk walking training. Ready and Quinney (51) observed a significantly lower ventilatory threshold after 9 wk of training cessation in 21 male subjects who had previously trained for 9 wk, but it remained above pretraining values. In light of the similar time course and magnitude of changes in $\dot{V}O_{2max}$ and ventilatory threshold, these authors suggested that the two parameters are not entirely independent measures.

Muscle glycogen. Muscle glycogen concentration experiences a rapid decline with training cessation in highly trained athletes, in relation with an also rapid decline in glucose-to-glycogen conversion and glycogen synthase activity. Indeed, eight competitive swimmers' muscle glycogen concentration declined by 20% in the first wk of training cessation after the competitive season, and by 8–10% per week without training thereafter (5). Preexercise muscle glycogen has also been shown to be reduced by 20% in a group of triathletes, cyclists, and runners undergoing 4 wk of insufficient training (32). In addition, short-term (5 d) inactivity has been shown to be enough in seven endurance-trained athletes to decrease glucose-to-glycogen conversion and glycogen synthase activity toward sedentary values (36). As far as the authors know, the effects of training cessation on recently trained individuals' muscle glycogen concentration have not been reported in the literature.

CONCLUSION

Detraining, defined as the partial or complete loss of training-induced adaptations in response to an insufficient training stimulus, may take place within short periods of training cessation or marked reduction in habitual physical activity level. Short-term cardiorespiratory detraining is characterized in highly trained athletes by a rapid $\dot{V}O_{2max}$ decline, but it usually remains above sedentary values. $\dot{V}O_{2max}$ decreases to a lesser extent in recently trained subjects in the short run, but training-induced gains are most often completely reversed when training is stopped for a period longer than 4 wk. The $\dot{V}O_{2max}$ loss is the outcome of an immediate reduction in total blood and plasma volumes, the latter being caused by a reduced plasma protein content. Even though exercise heart rate increases at both maximal and submaximal intensities, this is not sufficient to counterbalance the reduced stroke volume, and maximal cardiac output declines. Cardiac dimensions often decrease, blood pressure increases, and ventilatory efficiency is most usually impaired after periods of training cessation. This general loss in cardiorespiratory fitness results in a rapid decline in the trained athletes' endurance performance. Recently acquired endurance performance gains, on the other hand, can be readily maintained for at least 2 wk without training.

From a metabolic perspective, even short-term detraining is characterized by a higher reliance on carbohydrate as a fuel for exercising muscles, as indicated by an increased respiratory exchange ratio. Whole-body glucose uptake is reduced, because of a decline in insulin sensitivity and a reduced muscle GLUT-4 transporter protein content, both in athletes and in recently trained individuals. In addition, muscle lipoprotein lipase activity decreases. Exercise blood lactate concentration increases at submaximal intensities, and the lactate threshold is apparent at a lower percentage of $\dot{V}O_{2max}$. These changes, coupled with a base deficit, result in a higher postexercise acidosis. Finally, muscle glycogen concentration suffers a rapid decline, reverting to sedentary values within a few wk of training cessation.

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