LEADING ARTICLE

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Detraining: Loss of Training-Induced Physiological and Performance Adaptations. Part II

Long Term Insufficient Training Stimulus

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Abstract

This part II discusses detraining following an insufficient training stimulus period longer than 4 weeks, as well as several strategies that may be useful to avoid its negative impact. The maximal oxygen uptake ($\dot{V}O_{2max}$) of athletes declines markedly but remains above control values during long term detraining, whereas recently acquired $\dot{V}O_{2max}$ gains are completely lost. This is partly due to reduced blood volume, cardiac dimensions and ventilatory efficiency, resulting in lower stroke volume and cardiac output, despite increased heart rates. Endurance performance is accordingly impaired. Resting muscle glycogen levels return to baseline, carbohydrate utilisation increases and the lactate threshold is lowered, although it remains above untrained values in the highly trained. At the muscle level, capillarisation, arterial-venous oxygen difference and oxidative enzyme activities decline in athletes and are completely reversed in recently trained individuals, contributing significantly to the long term loss in $\dot{V}O_{2max}$. Oxidative fibre proportion is decreased in endurance athletes, whereas it increases in strength athletes, whose fibre areas are significantly reduced. Force production declines slowly, and usually remains above control values for very long periods. All these negative effects can be avoided or limited by reduced training strategies, as long as training intensity is maintained and frequency reduced only moderately. On the other hand, training volume can be markedly reduced. Cross-training may also be effective in maintaining training-induced adaptations. Athletes should use similar-mode exercise, but moderately trained individuals could also benefit from dissimilar-mode cross-training. Finally, the existence of a cross-transfer effect between ipsilateral and contralateral limbs should be considered in order to limit detraining during periods of unilateral immobilisation.

In part I of this brief review, published in the preceding issue of *Sports Medicine*,[1] the nomenclature related to the detraining literature was defined and the short term (less than 4 weeks of insufficient training stimulus) characteristics of detraining were analysed. This part II focuses on long term detraining (more than 4 weeks of insufficient training stimulus). Maintaining the same structure of the previous part, attention is paid to the cardiorespiratory, metabolic, muscular and hormonal effects of training stoppage, both in highly trained athletes and in recently trained individuals. In addition, the avail-

able literature on the retention of training-induced adaptations is briefly reviewed, and several strategies to limit the negative impact of an insufficient training stimulus are discussed.

1. Long Term Cardiorespiratory Detraining

1.1 Maximal Oxygen Uptake

Highly trained athletes from a variety of sports have been shown to decrease their maximal oxygen uptake ($\dot{V}O_{2max}$) by 6 to 20% during long term training cessation.[2-9] $\dot{V}O_{2max}$ declines progressively and proportionally to the trained-state $\dot{V}O_{2max}$ during the initial 8 weeks, but stabilises thereafter at a level higher than[2,4,5] or equal to[3] that of sedentary control individuals.

Most studies on recently trained individuals indicate a complete reversal of $\dot{V}O_{2max}$ to pretraining values after long term inactivity,[10-15] but various degrees of retention of training-induced gains have also been reported.[16,17]

1.2 Blood Volume

The effects of long term training cessation on the blood volume of trained athletes have not been reported but, as indicated in part I of this review,[1] a few days of training cessation are enough to induce a reduction in total blood and plasma volumes.[18-21]

In young sedentary individuals, 8 weeks of limited physical activity resulted in 4 and 3.1% reductions in total blood and plasma volumes, respectively.[22]

1.3 Heart Rate

Maximal heart rate has been reported to increase by 5% after 84 days of training cessation in endurance-trained athletes.[4] Also, submaximal intensity exercise elicits a higher heart rate response following 5 to 23 weeks of inactivity,[2,3,5,6,23,24] and recovery heart rate after a standardised exercise task increases progressively with time of inactivity.[23] Increased submaximal heart rates, along with a diminished length of the isovolumetric contraction phase time at rest, suggest an increased sympatho-adrenergic tone in the detrained state.[2]

The maximal heart rate of recently trained individuals does not seem to be affected by long term training cessation.[10,13] On the other hand, partially or completely reversed effects have been reported on resting[10,15] and submaximal exercise heart rates.[15,25,26]

1.4 Stroke Volume

According to Pavlik et al.,[7] long term cessation of regular training results in increased resting stroke volume index (ml/beat/m^2) and ejection fraction in road cyclists. On the other hand, upright exercise stroke volume has been reported to decline progressively by 14 to 17% during 8 to 12 weeks of inactivity.[4,6]

The only available report on moderately trained individuals indicates a 3.9% reduction in stroke volume after 6 months of inactivity consecutive to 15 weeks of training.[11]

1.5 Cardiac Output

It has been shown that the cardiac index (L/m^2/min) of detrained cyclists increases at rest as a result of the increased stroke volume index.[7] During upright exercise, maximal cardiac output does not fall beyond the 8% decline recorded in the initial 3 weeks of inactivity,[4] but submaximal values represent increasingly higher percentages of the maximal during 84 days without training.[5] Submaximal supine exercise elicits slightly but significantly higher cardiac outputs after 8 weeks of training cessation.[6]

Exercise cardiac output has also been shown to decline by 6.9% during long term training stoppage in recently trained individuals.[11]

1.6 Cardiac Dimensions

Martin et al.[6] reported that the left ventricular end-diastolic dimension of trained individuals during upright exercise declined in parallel with stroke volume during 8 weeks of training cessation. During the same period, left ventricular posterior wall

Table I. Studies of cardiorespiratory characteristics of long term detraining

Detraining characteristic	Highly trained individuals (references)	Recently trained individuals (references)
↓ Maximal oxygen uptake	2-9	10-15
↓ Blood volume	18-21	22
↑ Maximal heart rate	4	
↑ Submaximal heart rate	2,3,5,6,23,24	15,25,26
↑ Recovery heart rate	23	
↓ Stroke volume during exercise	4,6	11
↓ Maximal cardiac output	4	11
↓ Ventricular dimension/mass	6,27	
↑ Mean blood pressure	6,24	15
↓ Maximal ventilatory volume	2,3,9	10,11,15
↑ Submaximal ventilatory volume	3,5,23	25
↓ Oxygen pulse	3	10
↑ Ventilatory equivalent	3,5,23	10,11
↓ Endurance performance	2,3,5,28	10,11,14,15

↓ indicates decreased; ↑ indicates increased.

thickness decreased progressively by 25%, but left ventricular mass did not change after the initial (3 weeks) decline. On the other hand, Giannattasio et al.[27] indicated a decreased left ventricular mass index to sedentary levels in former athletes after 5 years without training, and Pavlik et al.[7] observed no change in wall thickness nor in internal ventricular diameters during 60 days of inactivity. Mean and systolic blood pressures increase along with total peripheral resistance during 9 to 12 weeks without training,[6,24] but unchanged blood pressure values have also been reported.[3]

Training-induced blood pressure reduction is reversed within 12 weeks without training in moderately trained individuals.[15]

1.7 Ventilatory Function

Ventilatory function is impaired in highly trained individuals after long term training cessation. Indeed, maximal ventilatory volume declines by about 10 to 14%,[2,3,9] and ventilatory volume and ventilatory equivalent increase markedly during standardised submaximal exercise,[3,5,23] whereas the O_2 pulse decreases.[3] In addition, the ventilatory response to hypercapnia has been shown to increase after 2 years without training.[9]

Similar effects of training cessation on maximal[10,11,15] and submaximal[25] ventilatory volumes, ventilatory equivalent[10,11] and O_2 pulse[10] have been observed in recently trained individuals.

1.8 Endurance Performance

Swimming performance has been shown to decline during the inactivity period between 2 training seasons.[28] Also, exercise time to exhaustion has been shown to decline by 24% in 5 weeks of training cessation in soccer players,[2] and the oxygen uptake of endurance-trained athletes is significantly increased during submaximal exercise.[3,5]

Significant or complete reversal of training-induced performance gains are also evident in recently-trained individuals during long term inactivity.[10,11,14,15]

A summary of the cardiorespiratory changes which characterise long term detraining can be seen in table I.

2. Long Term Metabolic Detraining

2.1 Substrate Availability and Utilisation

The only available data on the metabolic response to long term training stoppage in highly trained athletes showed higher respiratory exchange ratio values during exercise, indicating an increased utilisation of carbohydrate.[3,5]

Smith and Stransky[25] reported unchanged exercise respiratory exchange ratios during a 14-week training-detraining paradigm in young women. On the other hand, Fournier et al.[13] observed significant declines during 3 months of training, and a partial reversal during subsequent 6 months of inactivity in adolescent males. Moreover, training-induced effects on epinephrine stimulated lipolysis[17] and high-density lipoprotein[26] are completely reversed in 7 to 12 weeks without training.

2.2 Blood Lactate Kinetics

Increased blood lactate levels during submaximal exercise at the same absolute and relative intensities have been reported in football players, endurance runners and cyclists after 9 to 12 weeks without training.[5,24] Moreover, the lactate threshold occurred at a lower percentage of $\dot{V}O_{2max}$ in the detrained state,[5,7] but this was still higher than that of untrained control individuals.[5]

Similar results have been observed in recently trained individuals.[16,26]

2.3 Muscle Glycogen

Although no specific data on muscle glycogen concentration in trained individuals during long term inactivity are available in the literature, short term detraining studies have shown a rapid return of muscle glycogen to control values.[29,30]

Table II shows a compilation of the metabolic characteristics of long term detraining.

3. Long Term Muscular Detraining

3.1 Muscle Capillarisation

Coyle et al.[4] reported unchanged capillarisation in endurance-trained athletes after 12 weeks of training cessation. In addition, values remained 50% higher than in control individuals. However, these results are in contrast with those of Houston et al.[31] and Bangsbo and Mizuno,[32] indicating a decreased capillarisation after only 2 to 3 weeks without training.

As indicated in part I,[1] recently gained capillary density returns to baseline values in as little as 4 weeks of training cessation.[12]

3.2 Arterial-Venous Oxygen Difference

An 8.4% decrease in arterial-venous oxygen difference appeared to be responsible for the 9% reduction in $\dot{V}O_{2max}$ observed between 3 and 12 weeks of training cessation in endurance athletes.[4]

3.3 Myoglobin Level

As during short term inactivity, long term inactivity does not seem to affect the myoglobin level of the gastrocnemius muscle in trained individuals.[4]

3.4 Enzymatic Activities

Oxidative enzyme activities, including citrate synthase, β-hydroxyacil-CoA dehydrogenase, malate dehydrogenase and succinate dehydrogenase, have been shown to decline between 25 and 40% after 4 to 12 weeks of training stoppage in highly

Table II. Studies of metabolic characteristics of long term detraining

Detraining characteristic	Highly trained individuals (references)	Recently trained individuals (references)
↑ Respiratory exchange ratio	3,5	13
↓ Adrenaline (epinephrine) stimulated lipolysis		17
↓ High density lipoprotein-cholesterol		26
↑ Submaximal blood lactate	5,24	16,26
↓ Lactate threshold	5,7	16,26
↓ Muscle glycogen level	29,30	

↓ indicates decreased; ↑ indicates increased.

trained endurance runners and cyclists,[4,5,33] rugby players[8] and adolescent soccer players,[34] but to remain stable significantly above sedentary values thereafter.[4,33] Interestingly, whereas mitochondrial enzyme levels decreased almost to untrained levels in slow twitch (ST) fibres, they remained 50 to 80% higher in fast twitch (FT) fibres.[33] These reductions in mitochondrial enzymes have been suggested to be associated with the observed long term reductions in $\dot{V}O_{2max}$ and arterial-venous oxygen difference.[4,8]

Long term athletic inactivity has been shown to result in variable and sometimes antagonistic changes in the activity levels of several non-mitochondrial enzymes.[5,8,33,34]

Whereas oxidative enzyme activities in recently trained individuals have been repeatedly shown to return to baseline values after long term inactivity,[12-14] glycolytic enzyme changes are more heterogeneous.[12-14,35]

3.5 Mitochondrial ATP Production

The effects of long term training cessation on mitochondrial ATP production have not been reported in the literature. However, considering the short term decline[36] reported in part I,[1] and the abovementioned decrease in mitochondrial enzyme activities, it could be assumed that a marked reduction in mitochondrial ATP production would take place during long term training cessation.

3.6 Muscle Fibre Characteristics

Long term detraining has been shown to increase oxidative fibre population to the detriment of FT fibres in an elite power-lifter[37] and a bodybuilder,[38] to decrease the ST proportion by 15% in oarsmen,[39] and not to change fibre distribution in dancers[40] and adolescent soccer players.[34] A large shift from FTa to FTb fibres has also been reported in endurance runners and cyclists.[5] Moreover, FT and ST fibre cross-sectional areas,[8,34,37,39,41,42] the FT : ST area ratio[8,38,41] and muscle mass[8,37,38,41] have been shown to decline in strength-trained and team athletes, but increased fibre areas have been reported in female dancers.[40]

In recently trained individuals, a shift from ST to FT fibres,[12] reduced cross-sectional fibre areas[12,35,43] and lean body mass losses[25] have also been observed after long term training cessation.

3.7 Strength Performance

The force production of strength-trained athletes has been shown to decline by only 7 to 12% during inactivity periods ranging from 8 to 12 weeks.[41,42,44] This force decline appears to be related to a decreased electromyogram (EMG) activity,[42,44] in addition to the abovementioned reductions in fibre areas and muscle mass.

High percentages of recently acquired strength gains are also maintained for at least 12 weeks despite training stoppage.[35,43,45-53] Interestingly, it has been suggested that performing eccentric muscle actions during training is essential to promote greater and more longer lasting neural adaptations to training,[50] and that speed-strength is better maintained during training stoppage if the previous training method focuses on developing explosive strength.[47]

Long term muscular detraining characteristics in highly trained and recently trained individuals are summarised in table III.

4. Long Term Hormonal Detraining

Although sympatho-adrenal activity has been suggested to be unaffected by 5 weeks of post-injury inactivity,[54] a less efficient catecholamine response is suggested by increased epinephrine and norepinephrine concentrations during submaximal exercise of the same absolute intensity after 12 weeks of inactivity, coupled with decreased concentrations at the same relative intensity.[5]

5. Retention of Training-Induced Adaptations

Several factors, such as overuse or other type of injuries, illness, travel for competition, long-lasting competition or end of season vacation can keep athletes from performing their habitual exercises

Table III. Studies of muscular characteristics of long term detraining

Detraining characteristic	Highly trained individuals (references)	Recently trained individuals (references)
↓ Capillary density	31,32	12
↓ Arterial-venous oxygen difference	4	
↓ Oxidative enzyme activities	4,5,8,33,34	12-14
Altered fibre distribution	5,37-39	12
↓ Mean fibre cross-sectional area	8,34,37,39,41,42	12,35,43
↓ FT : ST area ratio	8,38,41	
↓ Muscle mass	8,37,38,41	25
↓ EMG activity	42,44	43
↓ Strength/power performance	41,42,44	35,43,45,53

EMG = electromyogram; **FT** = fast twitch; **ST** = slow twitch; ↓ indicates decreased;

and/or from maintaining their habitual intensity level. In view of the negative effects on physiological characteristics and performance criteria induced by short term and long term insufficient training stimulus, it would seem worthwhile for the injured or less active athlete to perform either a reduced training programme or an alternative form of training (i.e. to cross-train), in an attempt to avoid or reduce detraining.

5.1 Reduced Training

From a cardiorespiratory viewpoint, reduced training (as defined in part I of this review[1]) has been shown to be a valuable strategy to retain many of the training-induced adaptations for at least 4 weeks in highly trained athletes,[55-61] and even longer in moderately trained individuals.[62-65] Indeed, unchanged $\dot{V}O_{2max}$,[30,55-58,60,62,63,65] resting,[59,63] maximal[30,56,65] and submaximal[56,57,59] heart rates, ventilatory volume,[30,56,59] left ventricular mass,[63] and exercise time to exhaustion[56,57,59,60,62,65] values have been reported during periods of reduced training. However, specific athletic performance can decline rapidly in highly trained athletes despite reduced training strategies.[30,55,59]

The respiratory exchange ratio has been shown to increase slightly during periods of reduced training.[57,59] Unchanged[56,57,63] and increased[55,59] blood lactate levels, and unchanged insulin action and GLUT-4 levels,[65] have also been reported as a result of reduced training.

Oxidative enzyme activities,[57,65] lean body mass[56-59,65,66] and muscular strength[51,55,57,60] can be readily maintained by means of reduced training programmes.

Unchanged testosterone, cortisol and testosterone : cortisol ratio have been reported in trained distance runners during 3 weeks of reduced training.[58]

Interestingly, it has been recognised that some amount of detraining might also occur during the competitive season in multicomponent sports such as football, in which the inseason maintenance programme might not be enough to fully retain the physical fitness levels attained by the end of the preseason training programme.[67] Several studies have indicated that the maintenance of training intensity during periods of reduced training and taper (as defined in part I[1]) is of paramount importance in order to keep training-induced physiological and performance adaptations.[59,64,68-74] On the other hand, training volume can be reduced to a great extent without falling into detraining. This reduction can reach 60 to 90% of previous weekly volume, depending on the duration of the reduced training period, both in highly trained athletes and recently trained individuals.[28,29,57-60,63,69-81] Finally, reports from the literature indicate that training-induced adaptations are readily maintained for several weeks during periods of reduced training frequencies, but the reductions should be more moderate in athletes (no more than 20 to 30%) than

in recently and moderately trained individuals (up to 50 to 70%).[55,56,62,65,66,69,70,72,73,82]

Readers can find additional data on the physiological and performance consequences of periods of reduced training stimulus (i.e. reduced training and taper) elsewhere.[69,70,72,73]

5.2 Cross-Training

Cross-training, defined here as the participation in an alternative training mode exclusive to the one normally used,[83] has been suggested as a possible means to avoid or limit detraining, especially during recovery from a sport-specific injury and during vacation periods between 2 training seasons. The scarce available literature on the effects of cross-training as opposed to training cessation indicates that moderately trained individuals may maintain fitness and delay deconditioning by performing dissimilar training modes.[84,85] However, it has been suggested that similar-mode cross-training would be necessary in more highly trained individuals.[83]

The possible benefits and practical use of cross-training in sport have been recently reviewed by Loy and co-workers.[83]

5.3 Cross-Transfer Effect

Strength training-induced neural (increased motor unit synchronisation and activation) and muscular (hypertrophy, increased content of creatine phosphate and glycogen) adaptations[42] may be at risk during prolonged periods of inactivity. However, a cross-transfer effect of training-induced strength gains between ipsilateral (i.e. trained limb) and contralateral (i.e. untrained limb) limbs, also referred to as cross-education and cross-training, has been repeatedly described in the literature.[35,43,46,53,86-92] This phenomenon has obvious implications to limit muscular detraining during periods of unilateral casting, rehabilitation from injuries, or following joint surgery.

6. Bed Rest

Confinement to bed is a common treatment for patients experiencing disease or injury. The bed rest-induced horizontal position implies a reduced hydrostatic pressure gradient within the cardiovascular system, greatly reduced longitudinal compression on the spine and long bones of the lower extremity, reduced application of muscular force on bones in general, and reduced energy utilisation. This condition leads by itself to physiological adaptations that are independent from illness or injury, as shown by bed rest studies performed on healthy individuals.[93,94]

Excellent comprehensive reviews of the literature on the cardiovascular[95,96] and musculoskeletal[97] consequences of bed rest are available elsewhere. Intervention guidelines to limit the negative impact of bed rest have also been previously reported.[93,94,97-100]

7. Conclusion

When physical training is markedly reduced or stopped for a period longer than 4 weeks, the $\dot{V}O_{2max}$ of highly trained athletes declines by 6 to 20%, but usually remains above sedentary values. In contrast, $\dot{V}O_{2max}$ gains in recently trained individuals are most often completely reversed. These declines are partially due to reduced total blood and plasma volumes, which result in higher maximal, submaximal and recovery heart rates, and decreased stroke volume and cardiac output during upright exercise. In addition, long term inactivity may promote a decline in cardiac dimensions and ventilatory efficiency, affecting both $\dot{V}O_{2max}$ and endurance performance of athletes and moderately trained individuals as well.

Long term metabolic detraining is characterised by increased utilisation of carbohydrate and higher blood lactate concentration during submaximal exercise, resulting in an accelerated appearance of the lactate threshold which, in athletes, remains nevertheless above control values. Meanwhile, resting muscle glycogen levels return to baseline values.

From a muscular perspective, long term insufficient training often results in a decreased muscle capillarity, a reduction in arterial-venous oxygen difference, and a large decline in oxidative enzyme activities, the latter 2 being directly related to the

long term reduction in $\dot{V}O_{2max}$. Whereas all the above muscular characteristics remain above sedentary values in the detrained athlete, training-induced muscular adaptations of recently trained individuals return to pretraining values. In endurance athletes, there may also be a decreased proportion of ST fibres and a large shift from FTa to FTb fibres, but strength-trained athletes may show an increased oxidative fibre population and a general decline in fibre areas. Force production declines slowly and in relation to decreased EMG activity. All these changes are also evident in the recently trained.

Long term detraining could also imply a less efficient catecholamine response during exercise.

Reduced training strategies have been shown to delay the onset of cardiorespiratory, metabolic, muscular and hormonal detraining. Maintaining training intensity seems to be the key factor for the retention of training-induced physiological and performance adaptations, whereas training volume can be reduced by 60 to 90%. On the other hand, training frequency reductions should be more moderate (no more than 20 to 30% in athletes, and up to 50% in less well trained individuals).

Performing alternative training modes exclusive to the one normally used (i.e. cross-training) may delay detraining in athletes if similar-mode exercises are performed, but even dissimilar-mode cross-training may be beneficial to the moderately trained individual.

Finally, because of the often observed cross-transfer effect between ipsilateral and contralateral limbs, exercising the healthy limb should be recommended during periods of unilateral casting, rehabilitation from injuries, or following joint surgery.

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