LEADING ARTICLE

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

Short Term Insufficient Training Stimulus

Iñigo Mujika and *Sabino Padilla*

Department of Research and Development, Medical Services, Athletic Club of Bilbao, Basque Country, Spain

Abstract

Detraining is the partial or complete loss of training-induced adaptations, in response to an insufficient training stimulus. Detraining characteristics may be different depending on the duration of training cessation or insufficient training. Short term detraining (less than 4 weeks of insufficient training stimulus) is analysed in part I of this review, whereas part II will deal with long term detraining (more than 4 weeks of insufficient training stimulus). Short term cardiorespiratory detraining is characterised in highly trained athletes by a rapid decline in maximal oxygen uptake ($\dot{V}O_{2max}$) and blood volume. Exercise heart rate increases insufficiently to counterbalance the decreased stroke volume, and maximal cardiac output is thus reduced. Ventilatory efficiency and endurance performance are also impaired. These changes are more moderate in recently trained individuals. From a metabolic viewpoint, short term inactivity implies an increased reliance on carbohydrate metabolism during exercise, as shown by a higher exercise respiratory exchange ratio, and lowered lipase activity, GLUT-4 content, glycogen level and lactate threshold. At the muscle level, capillary density and oxidative enzyme activities are reduced. Training-induced changes in fibre cross-sectional area are reversed, but strength performance declines are limited. Hormonal changes include a reduced insulin sensitivity, a possible increase in testosterone and growth hormone levels in strength athletes, and a reversal of short term training-induced adaptations in fluid-electrolyte regulating hormones.

The principle of training reversibility states that whereas regular physical training results in several physiological adaptations that enhance athletic performance, stopping or markedly reducing training induces a partial or complete reversal of these adaptations, compromising athletic performance. In other words, the reversibility principle is the principle of detraining.[1] Athletes often experience interruptions to training process and competition programmes because of illness, injury, postseason break or other factors, which may result in a reduction or cessation of their habitual physical activity level. Therefore, it is extremely important to identify the effects and to understand the mechanisms of any associated changes in physiological capacities and athletic performance.

In the past, there has been some confusion in the exercise science literature concerning the terminol-

ogy used in detraining studies. This confusion arises primarily because the process through which a trained individual has lost some or all training-induced adaptations (e.g. reduced training, training cessation, bed rest confinement) has not been discriminated from the outcome of that process (i.e. the lost adaptations themselves). Published studies supposedly dealing with detraining have used reduced training and tapering strategies in their experimental procedures, leading to, instead of detraining, a reduction or elimination of the negative impact of training and a maintenance of the physiological adaptations achieved during previous training periods.[2] This lack of discrimination has led to some contradictory and confounding results. Moreover, the terms 'detraining' and 'detraining syndrome' are sometimes used as synonyms by some, although they represent quite distinct concepts. In order to avoid confusion, and to encourage the use of standard terminology in the international exercise science literature, these terms need to be defined.

Based on previously given definitions,[3,4] detraining will be redefined in this review as the partial or complete loss of training-induced anatomical, physiological and performance adaptations, as a consequence of training reduction or cessation.

Training cessation implies a temporary discontinuation or complete abandonment of a systematic programme of physical conditioning.

Reduced training is a nonprogressive standardised reduction in the quantity of training,[2] which may maintain or even improve many of the positive physiological and performance adaptations gained with training.[2,5-15] This procedure has also been referred to as 'step taper'.[2,16,17]

According to the literature,[2,16-28] taper could be defined as a progressive nonlinear reduction of the training load during a variable period of time, in an attempt to reduce the physiological and psychological stress of daily training and optimise sports performance. Excellent reviews on reduced training and taper have been previously published.[2,25,29,30]

Finally, the detraining syndrome (or relaxation syndrome) is a clinical entity arising when athletes with a long endurance-training history suddenly abandon their regular physical activity. This syndrome is characterised 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.[31-33]

This brief review will focus on the physiological and performance consequences of an insufficient training stimulus to maintain training-induced adaptations, i.e. detraining. Quantitative and qualitative losses of training-induced adaptations differ depending on the duration of the period of insufficient training stimulus.[3,30,34-47] Taking the usual 4-week postseason break of many highly trained athletes as a reference time length, study periods shorter and longer than 4 weeks will be referred to in this review as short term and long term, respectively. Short term detraining characteristics are treated in part I of this review, whereas part II will deal with long term detraining. In addition, research-based methods to avoid or limit physiological detraining and its negative impact on performance will also be discussed in part II.

Some detraining characteristics are not necessarily identical in highly trained athletes with a training background of several years aiming to improve their sports performance, and in recently trained but previously sedentary or moderately active individuals, who engage in a programme of physical activity usually with either health-related or research purposes.[3,30,41,44-48] In both parts of the review, detraining will therefore be analysed separately in these populations, placing the emphasis on the available data on athletic populations.

1. Short Term Cardiorespiratory Detraining

1.1 Maximal Oxygen Uptake

Maximal oxygen uptake ($\dot{V}O_{2max}$) has been shown to decline with short term (less than 4 weeks) training cessation in highly trained individuals with a large aerobic power and an extensive training background, the percentage loss ranging between

4 and 14%.[40,43,48-53] It has been suggested that the higher the trained-state $\dot{V}O_{2max}$, the bigger its decline during training stoppage.[40] However, some studies reported that $\dot{V}O_{2max}$ was maintained by trained athletes during periods of training cessation.[54-56] These differences could be related with the amount of physical activity performed by the athletes during the follow-up period.

The $\dot{V}O_{2max}$ of recently trained individuals has been shown to decline to a lesser extent (3.6 to 6%) during 2 to 4 weeks of training stoppage subsequent to 4 to 8 weeks of training,[38,57-59] and not to change with higher training/detraining time ratios.[15,48]

1.2 Blood Volume

The decline in cardiovascular function observed as a result of short term training cessation is largely due to a reduced blood volume.[50,52] Indeed, total blood volume and plasma volume have been shown to decline by 5 to 12% in endurance-trained athletes,[50,52,54,60] which appears to limit ventricular filling during upright exercise.[43,50] Estimated plasma volume may decline within the first 2 days of inactivity.[54,60]

Short term detraining in recently trained individuals is also characterised by a decreased blood volume, as a result of a loss in both red cell volume[58] and plasma volume,[58,61] the latter being induced by a loss in plasma protein content.[58]

1.3 Heart Rate

As a result of the above-mentioned reduction in blood volume in detrained athletes, exercise heart rate is increased at submaximal[50,52,62] and maximal[40,49,52,54] intensities by about 5 to 10%. These increased heart rate values are reversed when plasma volume is expanded.[50] Interestingly, maximal heart rate increase appears to stabilise after 2 to 3 weeks without training.[40] Resting heart rate has been reported not to change after 10 days of training cessation.[54]

Resting and maximal heart rate, which decrease with short term training, revert to untrained levels during short term inactivity in recently trained individuals,[63] but submaximal exercise heart rate is not affected by short term inactivity.[59]

1.4 Stroke Volume

A reduced stroke volume resulting from the reduced blood volume that characterises the short term detrained state is responsible for the reduced maximal aerobic capacity shown by athletes.[40] Stroke volume reductions of 10 to 17% have been reported after 12 to 21 days of training cessation,[40,43,50] along with a 12% reduction in the left ventricular end diastolic dimension.[43] Stroke volume was reestablished after plasma volume expansion, indicating its dependence on blood volume rather than on cardiac dimensions, but $\dot{V}O_{2max}$ remained 3% lower than in the trained state and exercise time to exhaustion fell 4.5% more after this intervention.[50]

1.5 Cardiac Output

The increased exercise heart rate values resulting from cardiovascular detraining do not seem to be sufficient to counterbalance the reduction in stroke volume. Indeed, estimated maximal cardiac output has been shown to be 8% lower after 21 days without training.[40] As well, submaximal cardiac output increased from 84 to 89% of the maximal at the same absolute exercise intensity.[42]

1.6 Cardiac Dimensions

Whereas Cullinane et al.[54] observed no change in cardiac dimensions and blood pressure of distance runners following 10 days of training cessation, Martin et al.[43] reported a 25% decrease in left ventricular wall thickness and a 19.5% reduction in left ventricular mass after 3 weeks of physical deconditioning. Reduced left ventricular mass and a higher total peripheral resistance could be responsible for the increased mean blood pressure measured during upright exercise.[43,50]

The effects of 8 weeks of training on systolic and diastolic blood pressures were reversed within 4 weeks of training stoppage in previously sedentary individuals.[63]

Table I. Studies of cardiorespiratory characteristics of short term detraining

Detraining characteristic	Highly trained individuals (references)	Recently trained individuals (references)
↓ Maximal oxygen uptake	40,43,48-53	38,57-59
↓ Blood volume	50,52,54,60	58,61
↑ Maximal heart rate	40,49,52,54	63
↑ Submaximal heart rate	50,52,62	
↓ Stroke volume during exercise	40,43,50	
↓ Maximal cardiac output	40	
↓ Ventricular mass	43	
↑ Mean blood pressure	43,50	63
↓ Maximal ventilatory volume	49,51	
↓ Oxygen pulse	51,54	
↑ Ventilatory equivalent	51,54	
↓ Endurance performance	49,50,52,56,58,62	

↓ indicates decreased; ↑ indicates increased.

1.7 Ventilatory Function

Although unchanged maximal ventilation values have been reported,[54] a rapid deterioration of ventilatory function characterises detraining in highly trained individuals, as shown by a decline in maximal ventilatory volume, which often declines in parallel with $\dot{V}O_{2max}$,[49,51] a reduced O_2 pulse and an increased ventilatory equivalent.[51,54]

1.8 Endurance Performance

The endurance performance of a trained athlete declines rapidly as a consequence of an insufficient training stimulus leading to detraining, as indicated by the reported 2.6% slower times in a 366m swim for competitive swimmers[56] or the 4 to 25% shorter exercise time to exhaustion found in endurance-trained athletes.[49,50,52,53,62] However, Houmard et al.[52] found no change in running economy at submaximal exercise intensities (75 and 90% of $\dot{V}O_{2max}$), suggesting that loss in cardiorespiratory fitness is largely responsible for the detraining-induced performance impairment in well-trained individuals.

On the other hand, 2 weeks of training cessation did not significantly reduce exercise time to exhaustion gains achieved during 6 to 12 weeks of training in previously sedentary individuals.[15,57]

Table I summarises the characteristics of cardiorespiratory detraining in both highly trained and recently trained individuals.

2. Short Term Metabolic Detraining

2.1 Substrate Availability and Utilisation

Short term detraining is characterised by an increased respiratory exchange ratio (RER) at both submaximal[40,48,62] and maximal[52] exercise intensities, indicative of a shift towards a higher reliance on carbohydrate as a substrate for exercising muscles at the expense of lipid metabolism. Sensitivity for insulin-mediated whole-body glucose uptake decreases rapidly with inactivity.[53,64-69] This decline may be associated with a reduced muscle GLUT-4 transporter protein content, which has been shown to decrease by 17 to 33% after 6 to 10 days without training.[67,68] On the other hand, inactivity induces a rapid decrease in muscle lipoprotein lipase activity,[70] along with a markedly increased postprandial lipemia,[66] decreased high-density lipoprotein cholesterol, and increased low-density lipoprotein cholesterol.[60]

A 10% increased RER,[48] and a return of the insulin sensitivity index and the GLUT-4 transporter protein concentration to initial values,[15] have been observed in previously sedentary individuals studied during a short term training-detraining paradigm.

2.2 Blood Lactate Kinetics

Competitive swimmers have been shown to respond to a standardised submaximal swim with

higher blood lactate levels after only a few days of training cessation.[8,18,56] Similar results have also been reported in endurance-trained runners and cyclists.[42] Increased submaximal blood lactate in swimmers was accompanied by a lowered bicarbonate level, resulting in a base deficit and a higher post-exercise acidosis.[18] Moreover, the lactate threshold appears at a lower percentage of $\dot{V}O_{2max}$.[42,71] These results are indicative of a reduction in the muscle's oxidative capacity, which may fall by as much as 50% in 1 week.[18]

In sedentary individuals, blood lactate levels elicited by maximal and submaximal exercise intensities did not change during 3 weeks of inactivity consecutive to 6 weeks of training.[57,59]

2.3 Muscle Glycogen

Muscle glycogen level is negatively affected by training cessation in as little as 1 week. Indeed, 20% reductions have been reported in this time period in competitive swimmers,[18] and in 4 weeks in triathletes, cyclists and runners.[62] This reduction is due to a rapid decline in glucose-to-glycogen conversion and glycogen synthase activity.[64]

A compilation of the metabolic consequences of a short term insufficient training stimulus can be found in table II.

3. Short Term Muscular Detraining

3.1 Muscle Capillarisation

Capillary density has been shown to decline[49,55] or not change[40] as a result of short term training stoppage in athletes. Interestingly, capillarisation in this population appears to remain about 50% higher than in sedentary controls.[40]

In recently trained individuals, capillaries per mm^2, capillaries per fibre, and capillaries around all 3 fibre types have been shown to decline, but to remain above pretraining values, with 4 weeks of inactivity.[38]

3.2 Arterial-Venous Oxygen Difference

The only available data on arterial-venous oxygen difference during short term training cessation in highly trained individuals indicated no change in 21 days, lending support to the suggestion that rapid $\dot{V}O_{2max}$ loss is due to a decreased stroke volume, as stated above.[40]

3.3 Myoglobin Level

Short term training stoppage did not affect myoglobin level of the gastrocnemius muscle in trained runners and cyclists, which, in addition, was similar in trained and sedentary individuals.[40]

Table II. Studies of metabolic characteristics of short term detraining

Detraining characteristic	Highly trained individuals (references)	Recently trained individuals (references)
↑ Maximal respiratory exchange ratio	52	
↑ Submaximal respiratory exchange ratio	40,48,62	48
↓ Insulin-mediated glucose uptake	58,64-69	15
↓ Muscle GLUT-4 protein content	67,68	15
↓ Muscle lipoprotein lipase activity	70	
↑ Postprandial lipemia	66	
↓ High-density lipoprotein cholesterol	60	
↑ Low-density lipoprotein cholesterol	60	
↑ Submaximal blood lactate	8,18,42,56	
↓ Lactate threshold	42,71	
↓ Bicarbonate level	18	
↓ Muscle glycogen level	18,62,64	

↓ indicates decreased; ↑ indicates increased.

3.4 Enzymatic Activities

Skeletal muscle cytrate synthase activity decreases between 25 and 45% with short term training cessation in athletes.[42,48,52,53,55,67] A decreased muscle oxidative capacity is also reflected by the significant 12 to 27% reductions observed in β-hydroxyacil-CoA dehydrogenase,[42,55,62] malate dehydrogenase[42] and succinate dehydrogenase.[42,49] Moreover, the skeletal muscle lipoprotein lipase activity of detrained runners is reduced by 45 to 75%, whereas this enzyme's activity increases by 86% at the adipose tissue level, favouring the storage of adipose tissue.[70] These changes appear to be primarily regulated by altered protein synthesis rates.[72]

Short term training cessation has also been reported to induce small nonsystematic changes in glycolytic enzyme activities, including phosphorylase, phosphofructokinase[18,42] and hexokinase.[42] Small increments[42] and decreases[49] have been observed for lactate dehydrogenase activity. On the other hand, glycogen synthase activity decreases by 42% after only 5 days without training.[64]

Mitochondrial enzyme activities have been shown to decline to pretraining levels after short term training-detraining protocols.[38,48,59] As in highly trained athletes, these protocols induce rather insignificant changes in glycolytic enzymes.[38]

3.5 Mitochondrial ATP Production

Mitochondrial ATP production rate has not been studied in athletes during periods of training cessation. In sedentary individuals, it has been shown to decrease by 12 to 28% during 3 weeks of inactivity consecutive to 6 weeks of endurance training, as a result of a similar decline in individual mitochondrial enzyme activities. However, mitochondrial ATP production rate remained 37 to 70% above pretraining levels.[59]

3.6 Muscle Fibre Characteristics

Training cessation for 2 weeks did not change muscle fibre distribution in distance runners[49] nor in strength-trained athletes.[73] On the other hand, mean fibre cross-sectional area has been shown to decrease in soccer players[55] and weight-lifters,[53,73] mostly because of reduced fast twitch (FT) fibre area, but not to change[52] or even slightly increase[49] in distance runners.

Percentage distribution of fibre types were unaltered in recently trained individuals during 4 weeks of inactivity, but training-induced increases in the cross-sectional area of the different fibre types were reversed.[38]

3.7 Strength Performance

Strength-trained athletes showed slight but nonsignificant reductions in bench press, squat, isometric and isokinetic concentric knee extension force, and vertical jump after 2 weeks without training, but significant 8 to 13% declines in electromyogram (EMG) activity of the vastus lateralis muscle and isokinetic eccentric knee extension force.[73] Trained swimmers maintained muscular strength during 4 weeks of inactivity, but their ability to apply force in the water was markedly reduced, as shown by a 13.6% decline in swim power.[8]

Recently acquired isometric arm strength gains are lost at a quite slow rate in both ipsilateral and contralateral arms.[36] On the other hand, isokinetic strength has been reported to decline at a much higher rate, but to remain above pretraining values after 4 weeks without training, in both adults[34] and children.[74]

The main characteristics of muscular detraining in highly trained and recently trained individuals are reported in table III.

4. Short Term Hormonal Detraining

In addition to the above-mentioned decline in insulin sensitivity,[53,64-69] short term hormonal detraining is characterised by an unaltered catecholamine level at rest, during sequential hyperinsulinaemic, euglycaemic and hyperglycaemic clamps,[64,65] and after submaximal exercise.[42] Glucagon, cortisol and growth hormone (GH) levels did not change with 5 days of detraining in endurance athletes.[64,65] However, strength-trained athletes showed positive hormonal changes for anabolic processes after 14 days of inactivity, with increased GH, testosterone and

Table III. Studies of muscular characteristics of short term detraining

Detraining characteristic	Highly trained individuals (references)	Recently trained individuals (references)
↓ Capillary density	49-55	38
↓ Oxidative enzyme activities	42,48,49,52,53,55,62,67	38,48,59
↓ Glycogen synthase activity	64	
↓ Mitochondrial ATP production		59
↓ Mean fibre cross-sectional area	53,55,73	38
↓ EMG activity	73	
↓ Strength/power performance	8,73	34,74

EMG = electromyogram; ↓ indicates decreased.

testosterone/cortisol ratio, and decreased cortisol levels.[73]

Fluid-electrolyte regulating hormone levels were normalised to pretraining levels after a 6 days training-6 days detraining protocol in untrained individuals.[61]

5. Other Short Term Detraining Characteristics

It has been shown that 4 weeks of training cessation significantly lowered the flexibility of hip, trunk, shoulder and spine by 7.4 to 30.1% in male and female physical education students.[75]

Moderate regular exercise training and physical conditioning may reduce the risk of major vascular thrombotic events by reducing platelet adhesiveness and aggregability. However, the positive effects of 8 weeks of endurance training on resting and post-exercise platelet function are reversed within 4 weeks of detraining in previously sedentary women.[63]

6. Conclusion

Detraining, which should be redefined as the partial or complete loss of training-induced adaptations in response to an insufficient training stimulus, may occur during short periods (<4 weeks) of stoppage or marked reduction in habitual physical activity level.

Short term cardiorespiratory detraining is often characterised by a rapid $\dot{V}O_{2max}$ decline in highly trained athletes, but a smaller reduction in recently trained individuals. This loss in $\dot{V}O_{2max}$ is the result of an immediate reduction in total blood and plasma volumes, the latter being caused by a reduced plasma protein content. As a consequence, maximal and submaximal heart rates increase, but not enough to counterbalance the reduced stroke volume. This results in a lowered maximal cardiac output. Cardiac dimensions and blood pressure may or may not decrease during short term inactivity, and ventilatory efficiency is usually impaired. Because of this loss in cardiorespiratory fitness, the endurance performance of trained athletes declines rapidly, whereas it is readily maintained for at least 2 weeks in recently trained individuals.

From a metabolic viewpoint, short term detraining implies a higher dependence on carbohydrate as a fuel for exercising muscles, as shown by increased RERs and decreased muscle lipoprotein lipase activity. Whole-body glucose uptake is reduced, due to a decline in insulin sensitivity and a reduced muscle GLUT-4 transporter protein content, both in athletes and recently trained volunteers. Blood lactate levels at standardised submaximal intensities increase, the lactate threshold appears at a lower percentage of $\dot{V}O_{2max}$, and there is a base deficit inducing a higher postexercise acidosis. In addition, muscle glycogen levels rapidly decline.

At the muscle level, short term detraining is characterised by a lowered capillary density, unchanged arterial-venous oxygen difference and myoglobin level, significant reductions in oxidative enzyme activities resulting in a reduced mitochondrial ATP production, and nonsystematic changes in glycolytic enzyme activities. Muscle fibre distribution remains unchanged. On the other hand, fibre cross-sectional area declines in strength and sprint-oriented athletes and recently endurance-trained individuals, whereas it may increase slightly in endurance athletes. Although

strength performance in general is readily retained for up to 4 weeks of inactivity, the eccentric force and sport-specific power of athletes may suffer significant declines. The same is true for recently acquired isokinetic strength.

From a hormonal perspective, short term detraining characteristics in endurance athletes are reduced insulin sensitivity, and unaltered catecholamine, glucagon, cortisol and GH levels. Anabolic hormones may increase in strength athletes, and fluid-electrolyte regulating hormones decline to baseline after short term training-detraining paradigms.

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Correspondence and offprints: Dr *Iñigo Mujika*, Mediplan Sport, Obdulio López de Uralde 4, Bajo 01008 Vitoria-Gasteiz, Basque Country, Spain.
E-mail: imujika@grn.es