

CHAPTER

12

DETRAINING AND RETENTION OF TRAINING-INDUCED ADAPTATIONS

EDWARD F. COYLE

REVERSIBILITY OF ADAPTATIONS INDUCED BY TRAINING

Physical training exposes the various systems of the body to potent physiologic stimuli. These stimuli induce specific adaptations that enhance an individual's tolerance for the type of exercise encountered in training. The level of adaptation and the magnitude of improvement in exercise tolerance is proportional to the potency of the physical training stimuli.

Inherent to these observations is the concept of the reversibility of the adaptations induced by training. The "reversibility concept" holds that when physical training is stopped (i.e., detraining) or reduced, the bodily systems readjust in accordance with the diminished physiologic stimuli. The focus of this chapter is on the time course of loss of the adaptations to endurance training as well as on the possibility that certain adaptations persist, to some extent, when training is stopped. Because endurance exercise training generally improves cardiovascular function and promotes metabolic adaptations within the exercising skeletal musculature, the reversibility of these specific adaptations is considered. Another approach to the study of the effects of reduced activity is to examine the exercise responses of people before

and after prolonged bedrest. The idea that postural fluid shifts rather than inactivity account for the loss of cardiovascular function after bedrest is discussed.

CARDIOVASCULAR DETRAINING

MAXIMAL OXYGEN UPTAKE

Endurance training induces increases in maximal oxygen uptake (i.e., $\dot{V}O_{2max}$), cardiac output, and stroke volume.^{1,2} When sedentary men participate in a 7-week, low-intensity training program (20 min/day⁻¹; 3 days/week⁻¹), $\dot{V}O_{2max}$ levels increase by 6%, with a return of $\dot{V}O_{2max}$ values to pretraining levels with 8 weeks of detraining.³ Moderate endurance training increases $\dot{V}O_{2max}$ by 10 to 20%, yet again $\dot{V}O_{2max}$ may decline to pretraining levels when training is stopped.⁴⁻⁶ Values of $\dot{V}O_{2max}$ decline rapidly during the first month of inactivity, whereas a slower decline to untrained levels occurs during the second and third months of detraining.⁴⁻⁶ Therefore, the available evidence suggests that the increases in $\dot{V}O_{2max}$ produced by endurance training involving exercise of low to moderate intensities and

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durations are totally reversed after several months of detraining.

Investigators have not yet examined and then exposed untrained individuals to several years of intense endurance training and subsequent inactivity to determine if extreme training results in a persistent elevation of $\dot{V}O_{2\max}$ above untrained levels. Our present knowledge is limited to findings of studies involving already trained endurance athletes who agreed to cease training so that reversibility of their physiologic

adaptations could be studied periodically.⁷ Figure 12-1 is a display of the time course of the decline in $\dot{V}O_{2\max}$ (and its components of maximal stroke volume, cardiac output, and arteriovenous O_2 difference) when people become sedentary after training intensely for approximately 10 years.

The $\dot{V}O_{2\max}$ value was relatively high in trained subjects (i.e., $62 \text{ ml}/\text{min}^{-1}$ at 0 days without training) and it declined a total of 16% after 84 days of detraining. A rapid decline of 7% occurred in the first 12 to 21 days with a further decline of 9% during the period from 21 to 84 days.⁷ The rapid, early decline in $\dot{V}O_{2\max}$ was related to a reduction in maximal stroke volume measured during exercise in the upright position. Most of the decline in stroke volume occurred during the first 12 days of inactivity. Adaptive increases in maximal heart rate compensated somewhat for this loss of stroke volume. The decline in $\dot{V}O_{2\max}$ during the 21- to 84-day period was associated with a decline in maximal arteriovenous O_2 difference.

The 84-day period of detraining resulted in a stabilization of $\dot{V}O_{2\max}$ and maximal stroke volume. Thus, the subjects appeared to have detrained for a sufficient length of time to display a complete readjustment of cardiovascular response in accordance with their sedentary lifestyle. Note that maximal stroke volume during upright exercise in the detrained subjects was virtually the same as that observed in people who had never engaged in endurance training (Table 12-1). The idea that this finding does not necessarily imply a loss of heart function is subsequently discussed. Although maximal cardiac output and stroke volume declined to untrained levels, $\dot{V}O_{2\max}$ levels in the detrained subjects remained 17% above that of untrained individuals, primarily because of an elevation of maximal arteriovenous O_2 difference. The persistent elevation of $\dot{V}O_{2\max}$ values in the detrained subjects, the result of an augmented ability of the exercising musculature to extract oxygen, may be related to the observation that these subjects displayed no loss of the increased capillary density derived from the training and only a partial loss of the increase in muscle mitochondria (see *Detraining and Muscle Metabolism*).

STROKE VOLUME AND HEART SIZE

Prolonged and intense endurance training is thought to promote an increase in heart mass,

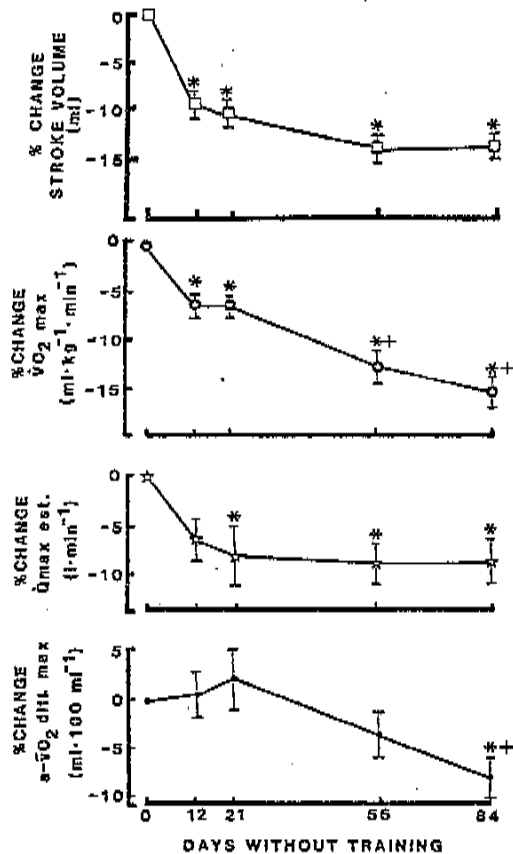


FIGURE 12-1. Effects of detraining upon percent changes in stroke volume during exercise, maximal O_2 uptake ($\dot{V}O_{2\max}$), maximal cardiac output ($\dot{Q}_{\max\text{est}}$), and maximal arteriovenous O_2 difference ($a-v\dot{O}_2\text{diff}\text{max}$). *, significantly lower than trained (day 0). +, significantly lower than 21 days. (Modified from Coyle EF, et al.: Time course of loss of adaptations after stopping prolonged intense endurance training. *J Appl Physiol*, 57:1857-1864, 1984.)

Table 12-1. Comparison of Untrained People and Detrained Subjects after 84 Days of Detraining

Population	$\dot{V}O_{2\max}$ ($\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$)	SV_{\max} (ml)	HR_{\max} (beats $\cdot \text{min}^{-1}$)	a- $\dot{V}O_2$ Difference ($\text{ml} \cdot 100 \text{ ml}^{-1}$)
Untrained people	43.3	128	192	12.6
Detrained subjects	50.8*	129	197	14.1*
Percent difference from untrained	+17*	+1	+3	+12*

* Values for detrained subjects are significantly ($p < 0.05$) higher than untrained subjects. (Modified from Coyle EF, et al.: Time course of loss of adaptations after stopping prolonged intense endurance training. *J Appl Physiol* 57:1857-1864, 1984.)

and researchers believe detraining results in a decline in heart mass.^{1,8,9} What is not clear, however, is whether the training-induced increases in ventricular volume and myocardial wall thickness regress totally with inactivity. Athletes who become sedentary have enlarged hearts and an elevated $\dot{V}O_{2\max}$ level in contrast to people who have never trained.¹⁰

One of the most striking effects of detraining in endurance-trained individuals is the rapid decline in stroke volume. To gain information regarding the cause of this large and rapid decline, Martin et al. measured stroke volume during exercise in trained subjects in both the upright and supine positions and again after 21 and 56 days of inactivity (Figure 12-2).

Simultaneous measurements of the diameter of the left ventricle were obtained echocardiographically. The large decline in stroke volume during upright cycling was associated with parallel reductions in the diameter of the left ventricle at end-diastole (i.e., LVEDD). When the subjects were evaluated during exercise in the supine position, a condition that usually augments ventricular filling because of the drainage of blood from the elevated legs, reduction in LVEDD was minimal. As a result, stroke volume during exercise in the supine position was maintained within a few percent of trained levels during the 56-day detraining period.

ROLE OF BLOOD VOLUME

Along the same lines, results of recent studies indicate that the rapid reduction with detraining of stroke volume during exercise in the upright position is related to a decline in blood volume (Fig. 12-3).¹² Intense exercise training usually results in an increase in blood volume by approximately 500 ml through the expansion of plasma volume.^{13,14} This adaptation is gained after only a few bouts of exercise, and it is quickly reversed when training ceases.^{13,14} The decline in stroke volume and the increase in heart rate during submaximal exercise, which normally accompanies several weeks of de-

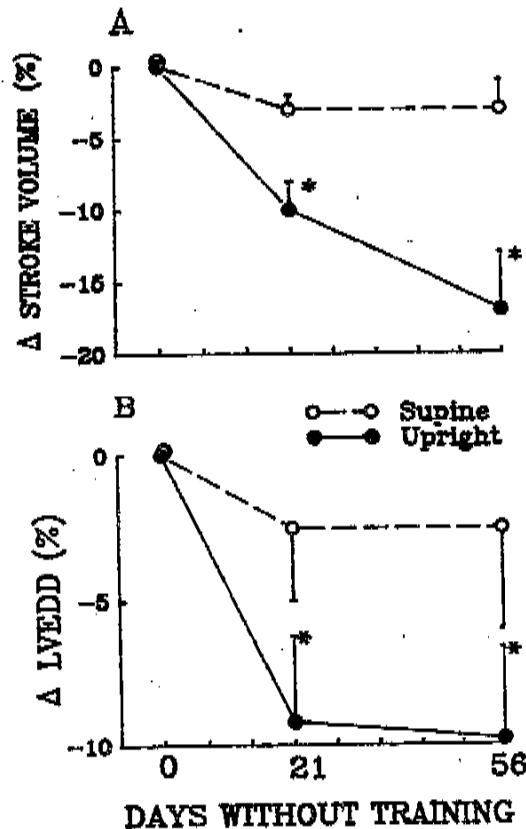


FIGURE 12-2. Percentage decline in exercise stroke volume (A) and left ventricular end diastolic diameter (LVEDD) (measured using echocardiography) (B) during exercise in upright and supine positions when trained and after 21 and 56 days of inactivity. *, responses in upright position are significantly ($p < 0.05$) lower than in supine position and lower than when trained (i.e., day 0). (From Martin WH, Coyle EF, Bloomfield SA, Ehsani AA: Effects of physical deconditioning after intense training on left ventricular dimensions and stroke volume. *J Am Coll Cardiol*, 7:982-989, 1986.)

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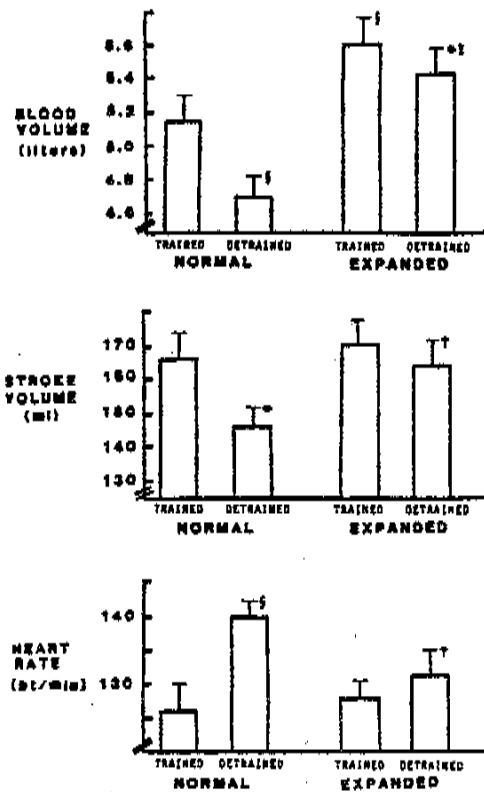


FIGURE 12-3. Responses to upright exercise with normal and expanded blood volume when trained and detrained. Significantly different from trained normal (*, $p < 0.05$; §, $p < 0.01$). Detrained with expanded blood volume significantly different from detrained with normal blood volume (†, $p < 0.05$; ‡, $p < 0.01$). (From Coyle EF, Hemmert MK, Coggan AR: Effects of detraining on cardiovascular responses to exercise: Role of blood volume. *J Appl Physiol*, 60:95-99, 1986.)

training, can be essentially reversed, and values return to near trained levels when the blood volume of detrained men expands to a level similar to that when the subjects were trained (Fig. 12-3).¹²

The observation that stroke volume during exercise is maintained at near trained levels when blood volume is high suggests that the ability of the heart to fill with blood is not significantly altered by detraining. If ventricular mass does indeed decline, a thinning of the ventricular walls and not a reduction in LVEDD is probably involved.¹¹ Thus, the reduction in intrinsic

cardiovascular function is apparently minimal after several weeks of inactivity in men who had been training intensely for several years.¹² The large reduction in stroke volume during exercise in the upright position is largely a result of reduced blood volume and not of a deterioration of heart function.¹²

DETRAINING AND MUSCLE METABOLISM

ENZYMES OF ENERGY METABOLISM

Endurance exercise training induces enzymatic adaptations in the exercising musculature that result in slower rates of glycogen utilization and lactate production and improved endurance during submaximal exercise.¹⁵ One of the more important alterations is an increase in the activity of mitochondrial enzymes, which results in an increased ability to metabolize fuels in the presence of oxygen. Moderate endurance training (2 to 4 months duration) increases mitochondrial enzyme activity by 20 to 40% from untrained levels.^{5,16} When moderate training ceases, however, and the stimuli for adaptation are removed, the increases in mitochondrial activity are quickly and totally reversed. Mitochondrial activity returns to pretraining levels within 28 to 56 days after the cessation of training.^{5,16}

The pattern of change in enzyme activity observed when individuals who trained intensely for 10 years stopped training for 84 days is provided in Figure 12-4.¹⁷ Mitochondrial enzyme activity in trained subjects (i.e., citrate synthase, succinate dehydrogenase, malate dehydrogenase, and β -hydroxyacyl-CoA dehydrogenase), which is initially twofold higher than those in untrained persons, declines progressively during the first 56 days of detraining and stabilizes at levels that are 50% higher than the values obtained from sedentary control subjects. The half-time of decline is approximately 12 days (i.e., declines one half the distance between trained and detrained in 12 days). Therefore, prolonged and intense training, in contrast to training programs that last only a few months, appears to result in only a partial loss of mitochondrial enzyme activity and thus a persistent elevation of activity above untrained levels. This elevation occurred almost entirely because of a persistent 80% elevation above untrained

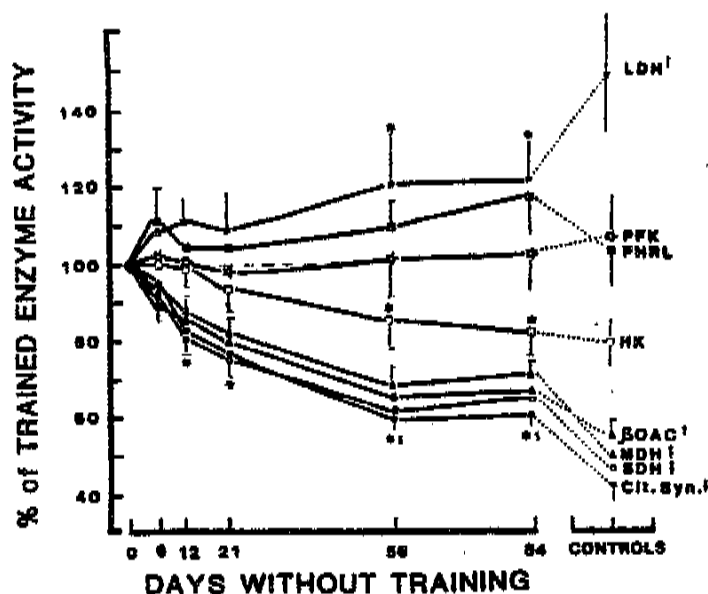


FIGURE 12-4. Enzyme activity during detraining period and comparison to sedentary control subjects. Values expressed as percentage of trained values. Cit. Syn., citrate synthase. SDH, succinate dehydrogenase. MDH, malate dehydrogenase. BOAC, B-hydroxyacyl-CoA dehydrogenase. HK, hexokinase. LDH, total lactate dehydrogenase. PHRL, phosphorylase. PFK, phosphofructokinase. *, significantly different from trained ($p < 0.05$); significantly different from 21 days ($p < 0.01$); ‡, control significantly different from 84 days ($p < 0.05$); †, control significantly different from 84 days ($p < 0.001$). (From Coyle EF, et al.: Effects of detraining on responses to submaximal exercise. *J Appl Physiol*, 59:853-859, 1985.)

levels in the mitochondrial enzyme activity in fast-twitch muscle fibers.¹⁸

MUSCLE CAPILLARIZATION

Endurance training promotes increased capillarization of the exercising musculature, which theoretically both prolongs the transit time of blood flow through the muscle and reduces diffusion distances, thus improving the availability of oxygen and nutrients to the muscle while also allowing for better removal of metabolic waste products. Moderate endurance training of several months' duration increases muscle capillarization by 20 to 30% above pretraining levels.^{5,19} Results of preliminary studies indicate that certain indices of muscle capillarization remain somewhat higher than pretraining levels 8 weeks after the cessation of moderate training.⁵

More prolonged and intense training increases muscle capillary density by 40 to 50% from untrained levels.^{7,19} No indication exists

that increases in muscle capillary density in highly trained people are reversed during 3 months of detraining.⁷

MUSCULAR ADAPTATIONS THAT PERSIST WITH DETRAINING

The detraining responses in the skeletal musculature of highly trained people who regularly engaged in intense exercise for several years apparently differ from those in individuals who have trained for only a few months. No loss of the increase in muscle capillarization occurs with the cessation of prolonged intense training, although such a loss does occur when moderate training is stopped. The cessation of moderate training results in a complete reversal of the training-induced increases in mitochondrial enzyme activity, whereas only a partial decline and therefore a persistent elevation of mitochondrial activity above untrained levels occurs with the cessation of exercise after prolonged intense endurance training.^{3,5,16,17}

EXERCISE RESPONSES OF DETRAINED SUBJECTS

Currently, scant evidence is available to imply that the cardiovascular or skeletal musculature adaptations derived from mild and moderate endurance training are maintained above pretraining levels with cessation of training for more than approximately 8 weeks. Therefore, a person should be stressed to the same degree during exercise of a given intensity whether untrained or after a prolonged detraining period. This hypothesis has yet to be fully evaluated, however, and one factor to consider is the possibility that people may perceive exercise to be more comfortable when they are in the detrained state having already experienced physical training, as compared to the untrained condition.

In agreement with the findings that individuals who exercised intensely on a regular basis for several years remain superior in the detrained state with respect to their muscle metabolism and intrinsic heart function (i.e., stroke volume when ventricular filling is high) compared with untrained people, it appears that these detrained people can exercise more intensely before becoming inordinately stressed. One indication of this ability is the observation that detrained persons not only possess a $\dot{V}O_{2\max}$ level that is well above untrained values, but also they maintain the ability to exercise at a high percentage of $\dot{V}O_{2\max}$ before lactic acid begins to accumulate in the blood.¹⁷

BEDREST DECONDITIONING

Prolonged bedrest causes severe cardiovascular deconditioning, manifested by orthostatic intolerance and large reductions in maximal O_2 uptake and stroke volume during exercise.²⁰⁻²² This loss of cardiovascular function after bedrest results more from fluid shifts induced by a reclining posture than from inactivity. This reasoning is supported by the observation that the deterioration in cardiovascular function that results from 2 to 3 weeks of bedrest can be elicited after only 20 hours of bedrest with head-down tilt.²³ The primary cause of the cardiovascular dysfunction, especially during exercise in the upright position, appears to involve an altered distribution of body fluids and changes in intra-

vascular pressures. The factors responsible for the altered distribution of fluids may include autonomic nervous system dysfunction, a moderate reduction in blood volume (i.e., 500 ml), and altered venous compliance.²⁰

Several methods can be used in an attempt to counteract the deterioration of cardiovascular function, which is most severe during exercise in the upright position after bedrest.²⁰ Blood volume expansion significantly improves cardiovascular status but it does not restore normal function. Treatments designed to induce venous pooling (i.e., lower body negative pressure and reverse gradient garments) during bedrest to stimulate conditions experienced in the upright position significantly reduce the deterioration in cardiovascular dysfunction.^{20,24} Exercise in the supine position during bedrest, involving either dynamic or isometric contractions, tends to reduce cardiovascular deterioration; it is not capable, however, of preventing dysfunction.^{20,25} Perhaps the best countermeasure for minimizing deconditioning of the cardiovascular system with bedrest is the mere exposure to the orthostatic stress encountered in the upright position.

SUMMARY

When physical training ceases (i.e., detraining), the bodily systems readjust in accordance with the diminished physiologic stimuli, and many training-induced adaptations are reversed to varying extents. The available evidence to date suggests that the increases in $\dot{V}O_{2\max}$ produced by endurance training of low to moderate intensities and durations are totally reversed after several months of detraining. When people detrain after several years of intense training, they display large reductions (i.e., 5 to 15%) in stroke volume and $\dot{V}O_{2\max}$ during the first 12 to 21 days of inactivity. These declines do not indicate a deterioration of heart function, but instead are largely a result of reduced blood volume and the ability to return venous blood to the heart. The $\dot{V}O_{2\max}$ of endurance athletes continues to decline during the 21 to 56 days of detraining because of reductions in maximal arteriovenous O_2 difference. These reductions are associated with a loss of mitochondrial enzyme activity within the trained musculature, which declines with a half-time of

approximately 12 days. Endurance athletes, however, do not regress to levels displayed by individuals who never participated in exercise training. Levels of mitochondrial enzyme activity remain 50% higher than those of sedentary subjects, skeletal muscle capillarization is maintained at high levels, and $\dot{V}O_{2\max}$ and the maximal arteriovenous O_2 difference stabilize at a point that is 12 to 17% higher than untrained levels after 84 days of detraining. The aims of future studies should be to determine if these superior physiologic abilities of people who cease prolonged and intense training are maintained for longer than 84 days and if these abilities relate to persistent effects of physical training or to inherent genetic predispositions.

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