The Subacute Effects of Exercise: Concept, Characteristics, and Clinical Implications

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NOBREGA, A.C.L. The subacute effects of exercise: Concept, characteristics, and clinical implications. Exerc. Sport Sci. Rev., Vol. 33, No. 2, pp. 84–87, 2005. Physical exercise represents a physiological stress to the organism producing acute integrative physiological adjustments, whereas repetitive and regular sessions of physical effort provoke chronic physiological adaptations that develop throughout the weeks of training and increase exercise capacity. The present work presents and discusses the concept of subacute effects of exercise, those physiological responses occurring between single bouts of exercise. Key Words: adaptation, exercise test, exertion, exercise physiology, training

INTRODUCTION

The capacity to perform physical exercise has been of paramount importance in the continuous process of animals’ adaptation to the environment throughout evolution. Endurance exercise represents a physiological stress to an organism for two main reasons. First, the increased metabolic demand associated with muscle contractions provokes a plethora of physiological adjustments that enhance oxygen and micronutrient delivery to the working muscles. These physiological responses may reach great magnitudes, because high-intensity dynamic exercise of large muscle groups can increase energy expenditure up to 20 times that of resting levels. Second, the augmented energy transfer from biochemical stores to mechanical work also increases heat release, eliciting integrative responses responsible for preserving homeothermia.

As a physiological burden, exercise is dealt by the organism with the engagement of complex homeostatic responses, which are known as acute effects or physiological responses to exercise. The magnitude of these acute responses is generally proportional to the relative stress. In other words, the same absolute exercise intensity causes greater physiological responses when performed by a less conditioned animal, because it represents a higher intensity relative to the maximum attainable by that particular organism. Therefore, the magnitude of the acute physiological responses to a standard exercise is taken as a measure of the capacity of an individual to respond to physical stress. Examples of acute effects of exercise encompass increased heart rate, pulmonary ventilation, sweating, and catecholamine secretion and decreased vagal activity, gastrointestinal motility, and splanchnic perfusion. Exposing organisms regularly to any stimuli causes physiological adaptations, and this is not different with exercise. Thus, repetitive and regular bouts of exercise trigger morphological and functional modifications, which are known as chronic effects of exercise. The main consequences of many of these physiological adaptations are an increased capacity to exercise, less severe acute responses to the same absolute workload, and higher peak performance. This concept is the main principle of exercise training, in which regular, repetitive exposure to exercise allows one to increase exercise capacity and master performance. But what happens physiologically between acute and chronic exercise? Previous publications have addressed some aspects of this issue (9,15). The purpose of the present work is to present and discuss the concept of subacute effects of exercise.

DEFINITION AND PROFILES OF SUBACUTE EFFECTS OF EXERCISE

Acute effects of exercise refer to the physiological responses occurring while exercise is performed, whereas chronic effects of exercise are those appearing after weeks or months of exercise training. Subacute effects of exercise refer to the physiological phenomenon occurring between single
bouts of physical effort, and involve the mechanisms that transfer the signals of acute stress to the adaptations that develop throughout the training period. It is important to note that some subacute effects may simply represent the residual effect of the physiological manifestations that occur during exercise and decrease progressively after exercise is interrupted. However, some subacute effects occur specifically after the effort, even if the stimulus was the exercise itself, and therefore should not be considered as residual effects of the acute physiological responses.

Three aspects of the subacute effects of exercise can be defined: 1) the direction of the induced change; 2) the relative magnitude compared with the acute effect; and 3) the interaction between the consecutive subacute effects.

**Direction of Subacute Effects of Exercise**

The physiological subacute effects of one bout of exercise may have an excitatory or an inhibitory profile, that is, a given parameter may be increased or decreased (Fig. 1). Typical subacute excitatory responses to physical effort are the elevated oxygen uptake and its physiological equivalents such as cardiac output, heart rate, and pulmonary ventilation, which remain higher than rest levels for minutes or hours after exercise is interrupted. An example of subacute inhibitory response is the postexercise decrease in blood pressure (8,12), an effect that may last several hours and that is more pronounced in those individuals with hypertension (7). Another subacute inhibitory response is the decrease in plasma volume, which is of critical importance in the understanding of orthostatic tolerance (4).

**Relative Magnitude Between Acute and Subacute Effects of Exercise**

Three different profiles can be envisioned concerning how acute and chronic responses to exercise interact. The Type 1

![Figure 1](image1.png)

**Figure 1.** Direction of the exercise-induced change. The subacute effects of one bout of exercise may increase or decrease the value of a given factor, thus disclosing either a provocative or an inhibitory action.

![Figure 2](image2.png)

**Figure 2.** Relative magnitudes of the acute and subacute effects. The subacute effects of exercise on the value of a physiological or clinical variable may be lower (Type 1, continuous line), higher (Type 2, dashed line), or much higher (Type 3, dotted line) than the acute effects observed during the exercise bout.

(Fig. 2, continuous line) profile shows that when a physiological phenomenon decreases continuously after exercise is interrupted, the subacute perturbation is lower than during exercise. Most physiological responses return to baseline quickly after exercise, and are a typical example of this type of effects, such as cardiac output, heart rate, and catecholamine release. The Type 2 subacute effect (Fig. 2, dashed line) characterizes those responses that are elicited by exercise and increases after it is stopped. Thus, the subacute effect is higher than the acute response seen during exercise. A practical example of this type of effect is the risk for mortality in individuals with ventricular ectopic beats during or after exercise (see later). The Type 3 profile includes those subacute responses that develop only after exercise (Fig. 2, dotted line). The postexercise hypotension is an example of this Type 3 response, as blood pressure increases rather than decreases during physical effort. The risk for hypoglycemia in diabetic patients fits this profile, especially those taking exogenous insulin, because hypoglycemia is prevented during physical exertion by the physiologic adrenergic activation and glucagon release, whereas after exercise, insulin sensitivity is still increased in face of basal adrenergic activity, thus leading to a decline in blood glucose levels. Another group of mechanisms involved with the increased insulin sensitivity after exercise encompasses the modification of the transcription of specific genes and the consequent alteration of the expression of different classes of protein related to glucose metabolism. In this context, special focus has been directed toward the subacute effects of exercise on the membrane transporter GLUT4 and some of its regulatory proteins (10), as well as key enzymes such as glycogen synthase and hexokinase II (5).

**Interaction Between Consecutive Subacute Effects of Exercise**

The chronic effects of physical training develop over the weeks as an adaptation to the acute stress of exercise bouts. The
Subacute effects on different variables may be superimposed simply as a temporal summation (Fig. 3, upper tracing) or as a combination with true morphological and functional adaptation (Fig. 3, lower tracing). This concept of temporal summation emphasizes the need to exercise regularly to maintain the chronic benefits of training, because interruption of the exercise sessions allows for the physiological variables to return to pre-training levels. The time necessary for the chronic effects to fade is quite different for the different variables, such as blood pressure (12) and plasma lipoproteins (11), suggesting that the proportion of temporal summation and actual adaptation may be quite different, depending on the physiological system of interest. This aspect of the subacute effects of exercise should be considered during the preparation of a training regimen, because the rest interval between exercise sessions modifies the magnitude of the metabolic stress of each bout. For example, when male athletes performed submaximal cycling for 75 min, 3 h after another bout of exercise, mean oxygen uptake was higher than the situation when no previous exercise had been performed. Because exercise intensity was the same, this observation suggests that metabolic stress was increased and exercise economy was impaired by previous effort (14).

CLINICAL IMPLICATIONS

The concept of subacute effects of exercise carries direct clinical implications. It is well known that a single exercise session causes marked changes in plasma lipid profile (11) and decreases in blood pressure (7,8). The physiological evidence supports practical recommendations, such as the need of hypertensive subjects to exercise every day to sustain the subacute effects of physical effort.

Myocardial Ischemia, Ventricular Arrhythmia, and Autonomic Function

One example of direct clinical implications of subacute effects of exercise is the occurrence of exercise-induced myocardial ischemia in susceptible patients. During physical exertion, myocardial oxygen demand is augmented because of the combination of increased heart rate, ventricular afterload, and contractility. Depending on exercise intensity, myocardial oxygen demand may exceed delivery, because coronary blood flow is restricted by atherosclerotic obstruction, thus causing ischemia. Therefore, the risk of myocardial ischemia and ventricular fibrillation is increased during exercise. However, several subacute effects of exercise reduce baseline risk of ventricular ischemia and arrhythmia, blunting its occurrence during subsequent bouts of physical effort (Fig. 4).

The work by Babai et al. (1) provided insights on the mechanisms involved with the cardioprotective subacute effects of exercise. In this study, dogs ran on a treadmill using a protocol in which the speed and slope were progressively increased to cause marked elevations in heart rate and blood pressure. Then, 24 or 48 h later, the dogs were anesthetized and subjected to a 25-min occlusion of the left anterior descending coronary artery. One single bout of exercise markedly reduced the consequences of coronary occlusion 24 h (but not 48 h) later. The exercised dogs presented only sparse ventricular premature beats and no ventricular fibrillation during ischemia/reperfusion insult, contrasting with a 36% occurrence of ventricular fibrillation in the control group (nonexercised dogs). The consequence was a much higher survival in the exercised dogs (70 vs 9%). This subacute effect of exercise was accompanied by increased arterial baroreflex sensitivity (indicating vagal activation) and by other, less marked ischemic changes, all of which had disappeared 48 h after exercise. Because the cardioprotective action of exercise were abolished by aminoguanidine, and inducible nitric oxide synthase activity was elevated threefold in left ventricular samples 24 h (not 48 h) after exercise, the subacute effects seemed to be mediated by nitric oxide. Because this subacute cardioprotection triggered by exercise...
develops hours after exercise, it can be classified an inhibitory effect (Fig. 1) of Type 3 (Fig. 2).

The mechanism involved with the increased risk in patients with ventricular arrhythmia after exercise is another example of subacute effect, and seems to be related to the impaired ability to reactivate parasympathetic drive, because patients with slower heart rate recovery are at increased risk for cardiac events (6). In this regard, parasympathetic dysfunction is an established marker of increase mortality in patients with cardiovascular disease (13), and pharmacological strategies to mimic vagal activation have shown promising results in patients with heart failure (2) and coronary artery disease (3).

CONCLUSION

The subacute effects of exercise refer to the physiological phenomenon occurring between exercise sessions, and should be considered an important physiological phenomenon with clinical implications for risk stratification and cardioprotection. Therefore, whenever a physiological change is observed along the weeks of training, it should be asked whether it represents simple temporal summation of subacute effects or is consequent to actual adaptations. Whereas in the first case, the interruption of the exercise sessions allows the physiological change to fade in hours or few days, it may take several weeks before a concrete adaptation disappears, even after the training regimen is stopped.

Acknowledgments

The development of these concepts was possible because of continuous and fruitful discussions among the members of the Laboratory of Exercise Sciences at Universidade Federal Fluminense, Niterói, RJ, Brazil. I appreciate, in particular, the assistance of Kelb Bousquet Santos and Pedro Paulo da Silva Soares for their recent scientific collaboration, and Michael Joyner for continuous support and critical appraisal of this manuscript. This work was partially supported by research grants provided from Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPq 350553/1995–5) and Fundação Carlos Chagas Filho de Amparo à Pesquisa do Estado do Rio de Janeiro (FAPERJ E-26/170:505/2001).

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