Abstract: Resistance exercise produces transient perturbations in immunity, including alterations in circulating leukocyte numbers, cytokine concentration, and some measures of cell function. These changes are typically interpreted as being transiently detrimental to host defense. The mechanisms responsible for these immune fluctuations appear to be neuroendocrine-mediated alterations in cell trafficking and function and microtrauma-mediated alterations in cytokine release. Alterations in immunity following resistance exercise appear to be similar in pattern but smaller in magnitude than those typically seen after long, vigorous endurance exercise and are resolved within a few hours. However, resistance exercise–induced changes in immunity may become clinically relevant after repeated exercise bouts with insufficient recovery. Regular training appears to attenuate the immune response to resistance exercise. Care should be taken to ensure that resistance training is planned, with adequate variation in intensity and volume over time, to ensure recovery between sessions and to avoid chronic systemic inflammation.

Keywords: strength training; immunity; leukocytes; cytokines

The immune system . . . displays substantial perturbations in response to a single bout of exercise.  

P hysical exercise provides a challenge to homeostasis throughout the body. The immune system, like many other physiological systems, displays substantial perturbations in response to a single bout of exercise. As early as 1902, Larrabee documented the impact of exercise on the immune system, noting an increase in blood neutrophil levels in runners following the Boston marathon.1

Many studies have now documented a stereotypical immune response to endurance exercise, consisting of a biphasic alteration in circulating immune cell numbers,2,3 reduced natural killer (NK) cell activity,2 reduced mitogen-induced lymphocyte proliferation,4 a reduced salivary immunoglobulin (Ig) secretion,5 and elevated circulating cytokines.6 Several published review articles have summarized the immune response to endurance exercise, characterizing it as a transient perturbation that is increased in severity with increasing volume and intensity of exercise.7–10

Resistance exercise is a popular form of physical exercise, currently performed by roughly 20% of the US population.11 Unfortunately, relatively fewer studies have examined the immune response to resistance exercise. Based on available data, the acute immune response to resistance exercise appears to be of a similar pattern, although less marked than the response induced by heavy endurance exercise. Two major mechanisms drive
respiratory tract infection (URTI) with high volumes and intensity of endurance exercise. Based on these studies, a J-shaped curve has been postulated to explain the relationship between exercise and risk of URTI (Figure 1). According to this model, those who engage in frequent, high-intensity exercise increase their odds of contracting URTI as compared with sedentary individuals. Conversely, those who engage in moderate exercise regimens would experience fewer URTIs than sedentary individuals.

To date, there has been no epidemiological study that has specifically examined the incidence of URTI in athletes who engaged in resistance exercise versus sedentary controls. The only epidemiological data currently available that have focused on immunity in strength-power athletes is Fahlman and Engel's 2005 investigation. In this study, a significantly greater percentage of American football player subjects reported having cold symptoms than did a control group at several time points during the season. Thus, it appears that high volumes of either aerobic or anaerobic exercise can have similar consequences in terms of challenging host defense.

Circulating Leukocyte Counts
Circulating leukocyte counts consistently display a characteristic, biphasic shift following heavy endurance exercise of at least 1 hour duration at an intensity ≥60% VO\textsubscript{2max}. Several studies have established that high-intensity aerobic exercise causes a unique, biphasic perturbation in the circulating leukocyte count. Immediately postexercise, total leukocytes, represented evenly by neutrophils and lymphocytes, with a small contribution of monocytes, increase 50% to 100% above resting preexercise values. Within 30 minutes of recovery, the lymphocyte count dips 30% to 50% below preexercise levels, remaining low for 2 to 6 hours. Eosinophils also egress from circulation, while basophils remain largely unaffected. As this occurs, circulating neutrophil numbers increase markedly and are maintained for a prolonged period. Moderate-intensity exercise (<60% VO\textsubscript{2max}) has repeatedly demonstrated a much smaller degree of postexercise leukocytosis, lymphocytosis and neutrophilia and a less-pronounced lymphocytopenia during recovery when compared with higher intensity activity.

Resistance exercise studies have generally found that leukocytes change in distribution in a similar manner, both immediately postexercise and during recovery, to that observed during endurance exercise studies despite a substantially shorter duration of effort. Figure 2 illustrates a comparison of lymphocyte counts between aerobic exercise and resistance exercise. Note the similar pattern of leukocyte distribution (with the exception of a more pronounced neutrophilia after endurance exercise) despite the fact that the exercise bouts vary greatly (150 minutes vs 20 minutes) in duration.

The leukocytosis during and after exercise is accomplished by flushing leukocytes out of marginal pools. The mechanisms responsible for mediating this process are not fully understood, but available evidence implicates sympathetic nervous activity, catecholamines, and cortisol as mediators of the process. Although these changes in circulating cell numbers are thoroughly documented to occur, their clinical significance is unclear. It is not known whether the increase in circulating cells is a positive response, increasing the availability of cells to become involved in immune reactions, or if it is a negative response, meaning that cells have been diverted from areas in which they were previously involved in immune reactions. Furthermore, the number of immune cells in circulation reflects only a small fraction (0.2%) of total leukocyte mass, with the balance of leukocytes existing in lymphoid, bone marrow, and other tissues. Lastly, a simple accounting of circulating leukocyte numbers does not address the function of those cells.

Functions of Circulating Immune Cells

Natural Killer Cells
Natural killer (NK) cells are large, granular lymphocytes that can mediate cytolytic reactions against neoplastic and virally infected cells. They serve as a component of the innate immune system and a first line of immune defense, as they have the cytotoxic capability without prior sensitization. NK cell cytotoxic function is commonly measured by incubating NK cells...
with a target cell line, then measuring the degree of cytotoxic killing by the release of radiolabeled chromium from lysed target cells.

Following prolonged (>60 minute) aerobic exercise at an intensity >60% VO$_{2\text{max}}$, NK cell activity (NKCA) increases immediately after exercise but then decreases below resting values for several hours during recovery. Similarly, NKCA is substantially reduced immediately after and during the 2 hours of recovery following exhaustive exercise involving large muscle mass, multijoint (barbell squats) movements. The postexercise increase and recovery decrease in NKCA have been attributed to the redistribution of NK cells from the circulation to other tissues. However, in Nieman's study, recovery NKCA was reduced even when adjusted for lytic units per NK cell, controlling for fluctuations in cell numbers. Thus, it appears that intense, exhaustive resistance exercise that involves a large amount of muscle mass reduces recovery NKCA via some other mechanism, possibly prostaglandins released by activated monocytes and neutrophils. No decrease in NK cell activity is apparent when exercise is limited to smaller muscle mass, single-joint (knee extensions, calf raises) movements, likely due to the smaller metabolic and hormonal demand of such exercise.

**Lymphocyte Proliferation**

T lymphocytes coordinate the response of many components of cell-mediated immunity via their activity and their release of many soluble factors, such as cytokines. B lymphocytes function to produce immunoglobulins, and their function is in part dependent on an interaction with T-helper cells. The functional capacity of T and B lymphocytes is commonly assessed through the proliferative response of these cells to various mitogens, such as phytohemagglutinin (PHA) and pokeweed mitogen (PWM) in vitro. When lymphocytes are exposed to a foreign pathogen, their ability to divide is an important component of the adaptive immune system. In the laboratory, researchers incubate lymphocytes with various types of mitogens and then measure the level of cellular proliferation.

Most studies investigating the lymphocyte proliferative response to intense, long-endurance exercise have found a temporary impairment in the lymphocyte proliferative response following exercise. Studies of resistance exercise have also consistently found a decrease in mitogen-induced lymphocyte proliferation immediately after exercise. Decreases in mitogen-induced lymphocyte proliferation following exercise are commonly confounded by changes in circulating cell numbers. Immediately after exercise, because of an increase in circulating NK cells and monocytes, the proportion of immune cells that respond to mitogen decreases in both whole blood and isolated peripheral blood mononuclear cells. For example, Green found that when adjusted per T cell, PHA-stimulated T-cell proliferation was not altered after heavy (60-minute run at 90% of the ventilatory threshold) endurance exercise. However, Dolf et al found that PWM-stimulated B-cell proliferation after exercise was significantly decreased, even when adjusted per B cell. Thus, it appears as though a true decrease in lymphocyte function can be induced by heavy resistance exercise. From more recent research, it appears that exercise (at least endurance exercise) decreases lymphocyte function through transient increases in apoptosis (programmed cell death), rather than decreases in mitosis. Furthermore, the extent of postexercise lymphocyte apoptosis is intensity dependent, with a threshold intensity of approximately 40% to 60% of VO$_{2\text{max}}$ required to induce significantly greater apoptosis than at rest.
To date, the effect of resistance exercise on lymphocyte apoptosis has not been directly studied. Since the effect of exercise on apoptosis appears to be intensity dependent, one could infer that resistance exercise would likely induce substantial lymphocyte apoptosis and that apoptosis is likely the mechanism behind previously reported findings\(^{21,30,37}\) that resistance exercise decreased mitogen-induced lymphocyte proliferation.

**Neutrophil and Monocyte Activity**

Neutrophils and monocytes play an important role in innate or nonspecific immunity. Neutrophils comprise approximately 60\% of all circulating leukocytes. They migrate to sites of infection where they bind, engulf, and destroy pathogens via phagocytosis involving both oxidative and nonoxidative means. Monocytes move from circulation to injured tissue, where they are transformed into macrophages. And when activated, they become an integral component of both the local and systemic inflammatory process. Together, neutrophils and monocytes act as a first line of defense to eliminate infectious agents and are involved in the muscle tissue inflammatory response to exercise-induced injury.\(^{15}\) Neutrophil and monocyte function can be expressed as a measure of phagocytosis (ability to engulf pathogens) or the oxidative burst (ability to kill pathogens once engulfed).\(^{9}\) Acute endurance exercise activates phagocytic neutrophils and monocytes, increasing phagocytosis, but high-intensity activity downregulates oxidative burst activity.\(^{27,44,45}\)

Every study that has measured cell counts after resistance exercise has documented increases in circulating number of neutrophils and monocytes.\(^{21,23,24,46}\) To date, no one has studied neutrophil or monocyte function following resistance exercise.

**Other Components of Immunity**

**Cytokines**

Cytokines are soluble glycoproteins that are produced by several cell types, including immune cells, endothelial cells, myocytes, and adipocytes. They mediate communication within and between cells, organs, and organ systems throughout the body in immune, inflammatory, and several other responses. Heavy physical activity produces a rapid, transient increase in cytokine production and entails increases in both proinflammatory (interleukin [IL]-2, IL-5, IL-6, IL-8, tumor necrosis factor [TNF]-α) and anti-inflammatory (IL-1ra, IL-10) cytokines.\(^{7}\)

As would be expected, studies indicate that acute resistance exercise increases both proinflammatory\(^{23,46,48}\) and anti-inflammatory\(^{24}\) cytokines in circulation. Chan et al\(^{16}\) documented decreases in mitogen (PHA)-stimulated IL-2 and IL-5 isolated peripheral blood mononuclear cells following resistance exercise.

**Immunoglobulins**

Immunoglobulins (Ig) are a class of glycoproteins secreted by B cells, which appear in bodily secretions, such as serum, tears, and saliva. Igs that react with a specific antigen are referred to as antibodies. Antibodies serve to bind to the surface antigens of pathogens, thereby stimulating the activation and differentiation of other immune cells. There are 5 classes (based on basic structure) of Ig. IgG is the major class of Ig found in serum. IgA is the major Ig class found in saliva. Studies of the immunoglobulin response to exercise have focused on both serum and salivary antibodies. Salivary concentrations of IgA have been shown to correlate more closely with URTI than serum antibodies.\(^{31}\) Salivary immunoglobulins are the first barrier to colonization by microorganisms causing URTI.\(^{52}\) Immunoglobulin A (IgA) is the predominant immunoglobulin in mucosal fluids, serving to inhibit the attachment and replication of pathogens and neutralize viruses and toxins. In addition, low resting levels of salivary IgA have been correlated with an increased risk of URTI among competitive swimmers\(^{63}\) and American football players.\(^{15}\)

Several studies have found that heavy exercise can elicit a postexercise decrease in salivary IgA levels.\(^{51,55}\) Suggested mechanisms behind an exercise-induced decrease in salivary IgA include changes in the transport of IgA across the mucosal epithelium or sympathetically mediated vasoconstriction in the oral submucosa and consequent reduction in the migration of cells synthesizing and secreting IgA.\(^{55}\)

However, this finding is not consistent with others reporting either no change\(^{36}\) or an increase\(^{57}\) in postexercise IgA. A likely explanation for these discrepant findings is the debate over the best method of expressing salivary IgA changes during exercise. Raw IgA concentrations do not account for changes in saliva composition typically associated with exercise.\(^{39}\) IgA:protein has been the traditional method to correct for the drying effects of exercise on oral surfaces. However, exercise typically produces an increase in the total protein content of saliva; thus, apparent decreases in salivary IgA:protein following exercise may reflect changes in the total protein content of the saliva sample, rather than fluctuations in IgA.\(^{55,56}\) Reflective of this confusion, the 3 available studies of the effects of resistance exercise on salivary IgA have thus far found a decrease in salivary IgA expressed relative to total salivary protein\(^{44}\) or no change\(^{46}\) or an increase\(^{41}\) in raw salivary IgA. Potteiger et al\(^{17}\) found no change in serum IgGs after resistance exercise.

**Mechanisms Behind the Immune Response to Resistance Exercise**

**Cortisol and Catecholamines**

Cortisol has been related to many of the immunosuppressive and cell-trafficking changes experienced during recovery from long-endurance exercise.\(^{2,30,42}\) Glucocorticoids administered in vivo have been reported to cause neutrophilia, eosinopenia, lymphopenia, and suppression of NK and T-cell function, all of which occur during recovery from prolonged, high-intensity aerobic exercise.\(^{52}\)

Immediately following a long, intense bout of aerobic exercise, plasma catecholamines are elevated. Epinephrine and norepinephrine play an important role in recruiting lymphocytes to the circulation. Epinephrine infusion induces changes in immunity similar to those induced by heavy endurance exercise.
including NK cell activity, mitogen-induced lymphocyte proliferation, and increases in IL-6, and it is also associated with lymphocyte apoptosis. However, epinephrine and norepinephrine’s effects are lessened after intensive exercise of >90 minutes’ duration. Immediately after exercise of 2.5 to 3 hours’ duration, the lymphocyte count is virtually unchanged from resting values. This contrasts sharply with the marked increase in lymphocyte count that is measured after exercise of <90 minutes’ duration. Thus, the hormonal milieu after exercise of >90 minutes appears to favor cortisol-mediated, rather than catecholamine-mediated, effects.

In contrast, several studies of the immune response to resistance exercise have failed to establish any connection between cortisol and measured exercise-evoked perturbations in immunity. Nieman et al. found NK cell activity/cell decreased by 40% after exercise despite no significant increase in cortisol. Kramer et al. found no difference in leukocyte counts after 8 sets of 10RM leg presses between cortisol responders (those who experienced substantial elevation in plasma cortisol after exercise) and nonresponders. Ingestion of carbohydrate has been shown to significantly alter the immune response to long-endurance exercise, with significantly reduced recovery lymphopenia, attenuated increase in proinflammatory and anti-inflammatory cytokines, and attenuated reduction of PHA-induced lymphocyte proliferation observed following long-endurance exercise with carbohydrate versus placebo. The proposed mechanism behind these differences in the immune response to exercise following carbohydrate ingestion is the inverse relationship between glucose and cortisol. Studies of carbohydrate ingestion and the immune response to resistance exercise, however, have found less dramatic results. Specifically, carbohydrate ingestion yielded minimal or no difference in lymphocyte proliferation, salivary IgA, plasma cytokines, or muscle cytokine mRNA for TNF-α or IL-1β. Carbohydrate ingestion has been found to decrease postexercise leukocytosis and lymphocytosis and to attenuate decreases in mitogen-induced IL-2 and IL-5 secretion from isolated peripheral blood mononuclear cells. However, none of these findings were linked to changes in cortisol, with cortisol either not increasing or the increase not being different between carbohydrate and placebo conditions. Furthermore, it should be noted that typical resistance exercise bouts, with rest periods of 2 to 3 minutes or greater between sets, do not generally produce a substantial increase in cortisol. The experimental protocols for the previously cited studies were all designed specifically to produce an increase in cortisol by employing a high volume of multijoint exercises with short rest intervals between sets. Thus, it is unlikely that postexercise increases in cortisol play a clinically significant role in any resistance exercise-induced alterations in immune function.

The rise in the catecholamines epinephrine and norepinephrine following resistance exercise has been less extensively studied. Nieman et al. directly measured epinephrine and norepinephrine concurrently with measures of immune function following resistance exercise. They reported that although norepinephrine levels rose substantially (>400% above resting), epinephrine concentrations after sets of exhaustive resistance exercise rose only modestly (0.77 nmol/L) and were closer to those observed after treadmill walking at 50% VO2max (0.568 nmol/L) than those measured after running at 80% VO2max (1.29 nmol/L). Miles et al. measured blood lactate in response to 6 sets of 10 repetitions of squats at 75% 1RM as a proxy measure of epinephrine. They found significantly greater increases in T-helper, T-cytotoxic, NK, and B-lymphocyte numbers after exercise in subjects in the highest quartile of lactate increase after exercise versus those in the lowest quartile of lactate response. Heavy resistance exercise strongly activates the sympathetic nervous system. The sympathetic nervous system is strongly linked to immune function, with innate immune cells expressing both α- and β-adrenergic receptors and T-cytotoxic and B-lymphocytes expressing β2-adrenoreceptors. Given the lack of findings regarding a cortisol effect, the hormonal milieu after resistance exercise appears to favor sympathetic nervous activation rather than cortisol-mediated effects.

**Muscle Damage**

Resistance exercise can induce significant microtrauma to muscle fibers. When mechanical forces placed on the myofiber exceed the structural capacity of the membrane, microtears in the sarcolemma occur. Consequently, intramuscular components, such as enzymes, leak out of the damaged muscle fiber and into circulation. Serum concentrations of the muscle enzyme creatine kinase (CK) have been used as indicators of muscle damage following resistance exercise and may indicate the status of the muscle cell membranes. Tissue damage leads to activation of the immune system. The overall response is characterized by a movement of fluid, plasma proteins, and immune cells from the circulation to the injured tissue. Neutrophils and monocytes are the primary immune cell types involved in this process. Their actions are coordinated to a large degree by cytokines.

The extent of muscle damage appears to affect the acute immune response chiefly with regard to circulating cytokine levels. Muscle damage—inducing (as measured by CK concentrations) eccentric cycling exercise yielded greater increases in plasma IL-6 than a concentric-only bout, despite a matching metabolic and hormonal demand between the two. In addition, the increases in IL-6 were correlated to increases in CK. Also, increases in IL-6 two hours after resistance exercise are related to the extent of DOMS reported 24 hours after exercise. Relative to strenuous endurance exercise, eccentric-only resistance exercise (4 sets of 12 repetitions of leg curl and bench press) induced a smaller rise in cytokines, which occurred later after the termination of exercise. However, any substantial rise in cytokines may be clinically meaningful.

From a clinical standpoint, the relationship between exercise-induced skeletal muscle microtrauma and the production
of cytokines might be the most relevant immune response to resistance exercise. Smith has proposed a model implicating cytokines as the key effectors behind the physiological symptoms associated with the overtraining syndrome. According to her theory, overtraining occurs if local inflammation, induced by acute exercise, is not resolved before subsequent bouts of exercise are performed. If insufficient recovery is given before subsequent bouts of exercise are performed, the local inflammation can become chronic local inflammation, and eventually, chronic systemic inflammation. With chronic systemic inflammation, monocytes become activated to release large quantities of proinflammatory cytokines. In particular, the proinflammatory cytokines IL-1β, TNF-α, and IL-6 are implicated as central to this theory of overtraining. IL-1β and TNF-α are secreted at the onset of inflammation, and they act systemically on the liver to induce acute-phase protein synthesis, the hypothalamus to change the body temperature set point, assisting in the control of fever, and higher centers of the brain. IL-6 displays both proinflammatory (mediating the acute-phase response) and anti-inflammatory effects (synthesis of glucocorticoids). Chronically elevated levels of these cytokines can induce “sickness” behavior, characterized by a loss of appetite, weight loss, sleep disturbances, depression, and so forth, via direct activation of the central nervous system. Furthermore, cytokines would increase glycogen and acute protein synthesis in the liver and activate the sympathetic nervous system and the hypothalamic-pituitary-adrenal axis, altering blood levels of catecholamines, glucocorticoids, and gonadal hormones. Immune suppression would possibly result as well, as a consequence of the anti-inflammatory factors released in conjunction with the proinflammatory response to tissue trauma.

Influence of Training Status

The most effective way to ameliorate any potentially immunosuppressive effects of exercise on immune function appears to be engaging in a regular training program. After the first exposure to eccentric exercise, subjects typically display substantial evidence of microtrauma. Upon a second exposure to the same exercise stimulus, markers of inflammation and muscle damage are often markedly reduced. This phenomenon is termed the repeated-bout effect. The repeated-bout effect is a well-documented illustration of an organism’s adaptation to exercise stress. Documentation of the repeated-bout effect includes a lower proinflammatory cytokine profile and lower postexercise neutrophilia following a second exposure to the same exercise stimulus. Adaptations behind the repeated-bout effect are thought to include a shift toward greater recruitment of slow-twitch motor units and the generation of new sarcomeres in series, thereby reducing the extent of microtrauma, and a down-regulation of inflammation, which would limit the extent of postexercise cell damage in the days following the exercise.

In cross-sectional comparisons, trained subjects show less evidence of tissue trauma following the same exercise stimulus than untrained subjects. For example, Newton et al found smaller fluctuations in plasma CK activity and arm circumference and a quicker recovery of strength in trained than untrained subjects following 10 sets of 6 maximal voluntary eccentric elbow flexions. Markers of oxidative stress following sprinting or weightlifting exercises were found to be lower in anaerobically trained athletes than those previously reported in untrained individuals. Mooren et al observed that athletes with a high VO2max (≥60 mL·kg⁻¹·min⁻¹; assumed to be more highly trained) had no increase in apoptotic lymphocytes following a marathon, while less well-trained athletes (VO2max ≤55 mL·kg⁻¹·min⁻¹) experienced substantially elevated lymphocyte apoptosis above resting levels. Thus, the lymphocytes of well-trained aerobic athletes appear to be more resistant to exercise-induced apoptosis.

Potteiger et al found trained women displayed no reduction in PHA-stimulated lymphocyte proliferation following a whole-body resistance exercise routine, while a significant reduction in lymphocyte proliferation was noted for untrained women who exercised at the same relative intensity. In contrast, Dohi et al found lower B-cell proliferation after 6 sets of squat at RM loads in better-trained subjects than in those who were less well trained. A likely reason for these discrepant findings is that subjects in the study by Dohi et al exercised to their repetition maximum on each set, and the better-trained subjects were working at a higher absolute intensity, as evidenced by a trend toward greater cortisol release in the trained group. Similarly, Miles et al found no change in the postexercise mitogen-induced T- or B-cell proliferation following 6 months of resistance training. However, again, the trained subjects were exercising at a higher workload at the end of the study. Thus, it appears that resistance training potently ameliorates postexercise fluctuations in immunity when compared with an exercise stimulus of the same absolute, submaximal workload.

While it may attenuate the immune response to acute exercise, regular training does not appear to greatly affect resting measures of immunity. Most measures of resting immune function show little or no difference between athletes and untrained controls. Several studies show that athletes display a reduced neutrophil activity, which is thought to reflect an adaptation to limit chronic inflammatory response caused by regular exercise. Available studies of resistance training have found no effect on resting NKCA or lymphocyte proliferation.

Clinical Relevance of Immune Response to Resistance Exercise

The acute changes in immunity following heavy exercise (including resistance exercise) are typically looked on as potentially markers of a transient immunosuppression. This has led to the formulation of the open window theory of immunosuppression, which proposes that athletes who train rigorously repeatedly induce a short-term downregulation of...
immunosurveillance. As a consequence, foreign pathogens are given a foothold to infect the host.

Acceptance of the open window theory is tempting and is bolstered by the availability of epidemiological data showing that hard-training athletes do report more sicknesses than sedentary controls. However, as Nieman has pointed out, no one has yet linked the transient alterations in immunity after heavy exercise with an increased risk of sickness. Until that correlation is made, the open window theory is still subject to challenge.

Resistance exercise does produce transient perturbations in immunity. The mechanisms responsible for these alterations appear to be neuroendocrine-mediated alterations in cell trafficking and function and microtrauma-mediated alterations in cytokine release. Neuroendocrine-mediated alterations in immunity following resistance exercise appear to be smaller in magnitude than those typically seen after long, vigorous endurance exercise and are resolved within a few hours. Microtrauma-mediated rises in circulating proinflammatory cytokines after a single bout of resistance exercise are also typically smaller in magnitude than those observed after long-endurance exercise. However, they may become clinically relevant after repeated resistance exercise bouts with insufficient recovery.

Regular training attenuates the immune response to resistance exercise. Care should be taken to ensure that resistance training is planned, with adequate variation in intensity and volume over time to ensure recovery and avoid chronic systemic inflammation.

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