Editorial

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Acquired immunity and moderate physical exercise: 5th series of scientific evidence

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With the exception of passive immunity, humans have two main types of immunity—innate and acquired immunity. The former evolved in primitive organisms, and the latter appeared in vertebrates. Innate immunity reacts quickly (between several hours to days), while the latter reacts slowly (days to years). The cellular components of innate immunity consist of macrophages, natural killer (NK) cells, and mast cells, while the cellular components of acquired immunity are made up of T and B cells. Both cells are from leukocytes, which destroy disease-causing organisms. Leukocytes are produced or stored in many locations in the thymus, spleen, and bone marrow. For this reason, they are also called lymphocytes, which allow the body to remember and recognize previous invaders and help the body destroy them. Lymphocytes start out in the bone marrow and either stay there and mature into B cells (a term that may have originated from "Bone marrow"), or they leave for the thymus gland where they mature into T cells (a term that may have originated from "Thymus gland"). The B lymphocytes are comparable to the body's military intelligence system, seeking out their targets and sending defenses to lock onto them. T cells are like the soldiers, destroying the invaders that the intelligence system has identified. The effector mechanisms of innate immunity are alternative pathways, cytokines, chemokines, and cell-mediated cytotoxicity. The transcription factor of innate immunity is either nuclear factor kappa-light-chain-enhancer of activated B (NF-κB) or positive c-Jun N-terminal kinases/activation protein 1. Meanwhile, the effector mechanisms of acquired immunity include antibodies, cytotoxic T cells, classical complement activation, antibody-dependent cell-mediated cytotoxicity, cytokines, and chemokines, while transcription factors include Janus kinase/signal transducer and activator of transcription or NF-κB.

The effects of exercise on innate immunity were covered in the previous editorials, so this issue will discuss the role of exercise on acquired immunity. Acquired immunity is also known as the second kind of protection for the human body and is also known as adaptive or active immunity. As the term implies, acquired immunity develops throughout our lives. It involves the lymphocytes and develops as people are exposed to diseases or immunized against diseases. Acquired immunity recognizes specific molecular structures and depends on the generation of large numbers of antigen receptors (i.e., T-cell receptors and immunoglobulins) by somatic rearrangement processes in blast cells. Once T cells recognize foreign antigens presented to them, they initiate acquired immune responses against precisely these antigens. These responses include cytotoxic T lymphocytes directly attacking antigen-bearing cells, stimulating B cells to produce antibodies against the antigens, and inducing inflammation in the area where the antigen is present, in addition to enhanced innate responses. All these responses cooperate to eliminate foreign particles and microorganisms. However, when there are improper responses by the immune system, they can result in autoimmune diseases or allograft rejection (Hansson et al., 2002).

Although T and B lymphocytes, the detector cells of acquired immune responses, differ entirely from those of innate immunity, the effector pathways overlap to a great extent. Thus, T-cell activation leads to the secretion of cytokine interferon-γ, which primes macrophages, lowering their threshold for toll-like receptor-dependent activation. In addition, T cells can produce tumor necro-

sis factor-α, a proinflammatory cytokine with NF-κB cells activating capacity. Moreover, the activated T cells express a cluster of differentiation (CD) 40 ligand, which ligates its receptor, CD 40, on macrophages, B cells, and many other cells including dendrite cells, epithelial cell cells, and skeletal muscle cells (Schönbeck and Libby, 2001). By involving inflammatory cells in the effector phase, T cells with the T helper-1 (Th1) tend to promote and amplify the same kind of inflammatory responses also induced when innate immune cells recognize pathogen-associated molecular patterns through their pattern-recognition receptors. The most common changes accompanying the adaptive immune system include the reduction of T- and B-cell proliferation, repertoire degeneracy, increase of memory cells, decreased numbers of naive cells, and a shift from Th1 to T helper2 (Th2) response.

Regular aerobic exercise may improve the changes in acquired immunity which can strengthen resistance against external pathogens (Malaguarnera et al., 2008). It is noteworthy that Th1 is characterized by a proinflammatory status and a resistance to infectious agents, whereas Th2 is characterized by an anti-inflammatory status. In other words, regular exercise refers to a series of processes that can improve physical fitness and overall health. It strengthens the body's organs, thereby providing protection from disease. Specifically, regular exercise plays an important role in protecting the body from external antigens by enhancing adaptive immune function. Recently, there have been many studies on the relationship between exercise and acquired immune cells such as T cells (CD4+ and CD8+), B cells, and NK cells. According to several previous studies, the acquired immune function of the human body can activate T, B, and NK cells only by activating the musculoskeletal system. In other words, when exploring the possible mechanisms by which adaptation to physical exercise can occur, the immune system appears to be of importance because physical exercise exerts both systemic and local effects on the immune system (Malm, 2002). A few new hypotheses are presented: First, the primary mechanism governing skeletal muscle adaptation to physical exercise is one of noninflammatory origin. Second, interleukin-10 may function as one of the signals transmitted by skeletal muscle cells when substrate levels are low. Third, a creatine kinase may have immuno-modulatory actions, thereby serving as a messenger molecule between skeletal muscles and the immune system (Malm, 2002).

As of now, previous comprehensive studies have shown that exercising with an intensity that is too low does not significantly improve immune function, whereas excessively high exercise intensity causes a suppression of the body's immune function. Ultimately, it can be said that moderate exercise intensity creates an intramuscular environment that can activate immune function in a beneficial way.

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CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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