Responses of m-GOT and humeral immune biomarkers to exhaustive endurance running and antioxidant supplementation in elite distance runners

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Abstract
This investigation examined the responses of mitochondrial aspartate aminotransferase (m-GOT) and immunoglobulins (IgA, IgG, IgM) to exhaustive endurance running and to antioxidants supplementation. Twelve female elite distance runners were divided into two groups (EG, CG) and participated in treadmill running test. EG was supplied with antioxidants for 12 weeks. Blood samples were collected before (Pre-test) and after (Post-test) 12 weeks at baseline (T1), exhaustion (T2), 30 minutes after exhaustion (T3) and 60 minutes after exhaustion (T4). m-GOT was not changed significantly, which suggested that the mitochondrial function of elite distance runners might not be hurt easily. There were significant differences of IgA, IgG and IgM within groups. In the Pre-test, EG and CG showed a significantly higher level in T2 than in T3. In the Post-test, EG didn't reveal such similarity but, compared to EG, CG showed a significantly higher level in T2 than in T3. These results suggest that antioxidant supplementation may have some beneficial effects on the humoral immune responses of elite distance runners. Correlation, factor- and regression-analysis showed that IgG has a significant relationship with all variables. These findings suggest that IgG may play important roles not only in maintaining of mitochondrial function but also in mediating immune responses.

Keywords: treadmill running test, mitochondrial damage, immunoglobulins

INTRODUCTION

Human beings require oxygen to generate energy in mitochondria in their cells. For this, it is often forgotten that oxygen can also be changed into toxic agents, such as reactive oxygen species (ROS) [1]. ROS not only mediate tissue injury but also regulate metabolism, signal transduction, and, ultimately, tissue function [2]. That is, ironically, various ROS-mediated actions actually protect cells against ROS-induced oxidative stress and re-establish or maintain “redox balance”, also termed “redox homeostasis” [3].

 Nonetheless, overproduction of ROS results in oxidative stress, a deleterious process that can be an important mediator of damage to cell structures, including lipids and membranes, proteins, and DNA. In contrast, the beneficial effects of ROS and RNS (reactive nitrogen species, e.g. nitric oxide) occur at low/moderate concentrations and involve cellular responses to noxia, for example, defense against infectious agents, functioning in a number of cellular signaling pathways, and the induction of a mitogenic response [3]. That is, free radicals play crucial roles not only in maintaining health and mediating disease but also in maintaining normal biological functions. Therefore, many studies have been performed for the purpose of determining the mechanism of ROS generation [4,5], the method of decreasing the exercise-induced oxidative stress [6], and a way of attenuating ROS toxicity [7].

Aerobes survive in the presence of ROS since they have evolved antioxidant defenses [8]. Given that ROS may act as toxins, mediators, and modulators of inflammatory gene activation, efforts have focused on the investigation of antioxidant molecules as potential therapeutic agents [9]. Antioxidants are defined as substances that, when present at low concentrations compared to those of an oxidizable substrate, significantly delay or prevent oxidation of that substrate [10]. Antioxidants work by preventing the formation of new free radical species, by converting existing free radicals into less harmful molecules, and by preventing chain reactions [1].

Overload training performed by well-trained endurance
athletes increases oxidative stress through the accumulative effect of repeated high-intensity exercise [6]. Oxidative stress refers to the cellular injuries and pathological changes that occur when there is an imbalance favoring oxidants over antioxidants within a living organism [11]. Athletes who maintain a very high volume of exercise, as a result of greater production of ROS, may be particularly susceptible to oxidative damage [12]. Oxidative damage of biomolecules is postulated to be a major causal factor of various physiological function disorders [13].

Therefore, exhaustive endurance exercise practiced by elite middle to long distance runners is likely to trigger oxidative stress. Consequently, many studies have focused on the relationship between exercise, antioxidants, and oxidative stress [14,15]. Further, the actual methods of reducing oxidative stress caused by exhaustive endurance exercise must involve supplementation with antioxidants. The potential of dietary antioxidants as an endogenous defense against lipid peroxides produced during exercise has received increasing attention in recent years. However, the results of human studies on the effects of supplementation with antioxidant vitamins on lipid peroxidation or enzyme muscle damage are controversial [16].

Meanwhile, moderate physical activity, when performed on a regular basis, presents a number of benefits to the whole organism, especially regarding immune system function [17], increased oxygen consumption, and energy expenditure during physical work [18]. Free radical production is increased during exercise, and oxidative damage occurs in several tissues [19]. That is, free radicals generated during exhaustive exercise cause subsequent oxidative damage to cells. This kind of tissue injury may be accompanied by immune responses. An increased systemic concentration of stress hormones as well as some cytokines may contribute to the depression of immune cell function typically observed after prolonged exercise [20]. These studies suggest that there are some interactions between exercise-induced oxidative stress and immune responses.

Immunoglobulins play important roles in humoral immunity. Humoral immunity, in particular secretion of neutralizing antibodies, is of central importance to protecting the body against acutely cytopathic viruses, whereas noncytopathic viruses have found ways to coexist with the immune system to avoid antibody-mediated elimination. Secreted protective antibodies of humoral memory provide an efficient line of defense against reinfection [21]. Immunoglobulin A (IgA) plays a central role in local immunity [22]. Immunoglobulin G (IgG) is the most abundant of the immunoglobulins in serum and is the most important antibody component [23]. IgG also attaches promptly to surface receptors on macrophages (or phagocyte). Intense endurance exercise suppresses salivary immunoglobulins. This exercise-induced decrease is specific for the secretory antibodies IgA and IgM [24]. IgA and IgG concentrations were correlated positively with serum IL-6 levels, which is a marker of inflammation [25].

Mitochondria consume about 90 percent of the oxygen used by the body and are a particularly rich source of ROS [26]. That is, mitochondria are an active and continuous source of ROS during respiration. Increased production of ROS during endurance training is the result of augmented electron transport through the respiratory chains, which makes the mitochondria a potential target for oxidative damage [27]. In addition to the well-established role of mitochondria in energy metabolism, regulation of cell death has recently emerged as a second major function of these organelles [28]. m-GOT, a general marker for cell damage [29], is located in the mitochondrial matrix and is useful for investigating the permeability of the mitochondrial inner membrane [30]. When mitochondria injury becomes serious, the rate of m-GOT that flows out into the serum is increased [31].

Therefore, the objective of this study was to investigate the relationships between exhaustive endurance exercise, antioxidant supplementation, humoral immune response, and mitochondrial damage in well-trained distance runners.

METHODS AND MATERIALS

Human subjects

Twelve healthy young women, who ran for an average of nearly 20 hours per week, and were thus well-trained distance runners (age 17.75 ± 0.62 years; body weight 51.42 ± 2.02 kg; height 165.71 ± 3.36 cm; athletic career 5.12 ± 0.71 years; HRrest 48.67 ± 3.47 beats/min; HRmax 189.67 ± 2.71 beats/min) volunteered to participate in the present study. All subjects signed a written informed consent form. Twelve subjects were randomly divided into two groups (experimental group, EG, n = 6; comparative group, CG, n = 6). EG was supplied by oral administration of antioxidants (671.14 mg a-tocopherol; 400 mg Vitamin C) every day for 12 weeks. CG was not given any supplemental antioxidants. All subjects abstained from alcohol and caffeine consumption for at least 24 hours, and did not exercise for the 24 hours prior to testing.

Experimental exercise protocol

In the present study, all subjects carried out experimental