Exercise and Leukocyte Redistribution

運動與白血球再分配

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運動與白血球再分配

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摘要

本篇綜評旨在陳述運動如何經由激活交感神經系統 (sympathetic nervous system) 和丘腦下部-腦垂體-腎上腺軸 (hypothalamic-pituitary-adrenal-axis) 而影響白血球再分配 (leukocyte redistribution)。摘要言之，長時間運動中所出現之立即性白血球增多 (leukocytosis) 主要肇因於較高之血漿兒茶酚胺 (catecholamines) 濃度，而延遲性嗜中性球增多 (delayed neutrophilia) 則受繼之昇高之血漿皮質固醇 (cortisol) 所影響。此外，生長激素 (growth hormone) 亦會導致循環嗜中性球增多現象。運動中補充碳水化合物有助於維持血漿葡萄糖濃度的穩定，進而促進耐力性運動表現，減緩血漿壓力激素濃度和白血球再分配之波動。

關鍵詞：運動、壓力激素、白血球再分配

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Introduction

The human immune system consists of a complex network of cellular and humoral factors and is regulated by stress. When homeostasis is disturbed or threatened by internal or external challenges, both the sympathetic nervous system (SNS) and the hypothalamic-pituitary-adrenal (HPA) axis become activated, resulting in increased peripheral levels of catecholamines and glucocorticoids acting in concert to maintain the steady state of internal milieu (Elenkov, Wilder, Chrousos, & Vizi, 2000). Exercise has been recognised as a reliable tool to induce reproducible and quantifiable stress responses via manipulation of type, intensity, frequency and duration (Smith & Pyne, 1997). Therefore, in recent years exercise has been applied to investigate the relationships among the endocrine, nervous, and immune systems (Ostrowski, Rohde, Zacho, & Pedersen, 1998; Suzuki *et al.*, 1999). To date, the effects of exercise on infection and immunity are not fully understood, however, it can be stated that exercise transiently alters various immune parameters and prolonged strenuous exercise can elicit reversible immunodepression (Fricker, McDonald, Gleeson, & Clancy, 1999). This may offer an "open window" to microorganisms and place athletes at a higher risk of infection after heavy exertion (Pedersen, 1999).

Many studies have indicated that regular moderate exercise is beneficial in the prevention of infectious diseases (Mackinnon, 2000; Matthews *et al.*, 2002; Suzuki & Machida, 1995). However, epidemiological studies demonstrate that endurance athletes are at increased risk of upper respiratory tract infection (URTI) after heavy training and/or competition and the vulnerable period can last up to 2 weeks (Nieman, 1997; Nieman, Johanssen, Lee, & Arabatzis, 1990; Peters, Goetzsche, Grobbelaar, & Noakes, 1993). Furthermore, exercise also results in a biphasic mobilization of total leukocytes and leukocyte subsets in the circulation (Hoffman-Goets, 1994; McCarthy & Dale, 1988), which includes initial lymphocytosis, monocytosis and neutrophilia, followed by a delayed response of neutrophilia and lymphopenia.

Therefore, the purposes of this review were to look at how the stress induced by exercise affects leukocyte redistribution and the possibility of ingesting carbohydrate beverage on preventing the impact of exercise on leukocyte mobilization.

Stress Hormone Responses and Leukocyte Mobilization to Exercise

The nervous and endocrine systems can interact with the immune system to alter the function and distribution of immune cells (Dhabhar, Miller, Stein, McEwen, & Spencer, 1994; Glaser, Rabin, Chesney, & Natelson, 1999) (Figure 1).

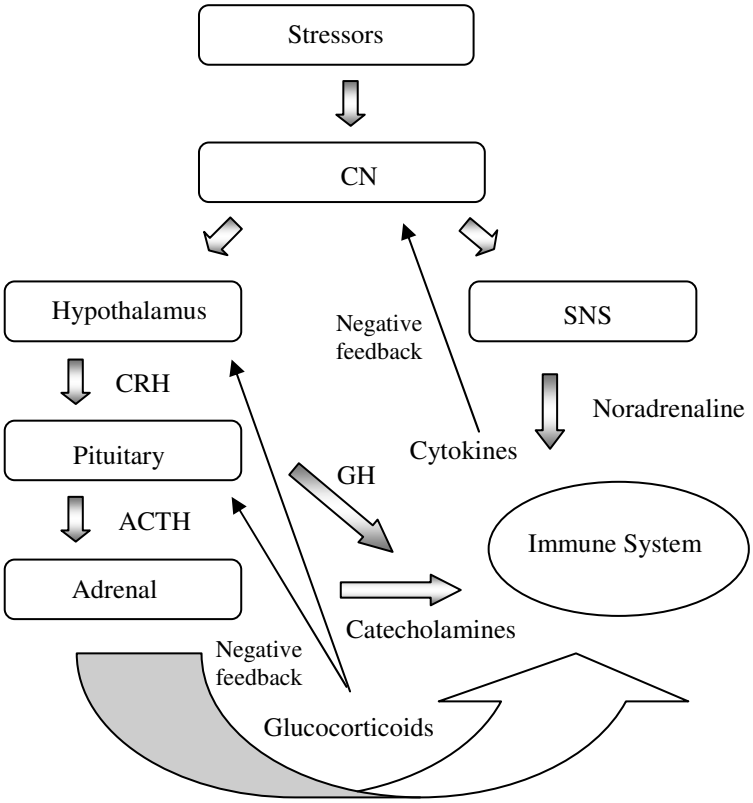

Figure 1 Communication between neuroendocrine and immune system.

(ACTH: adrenocorticotrophic hormone; CNS: central nervous system; CRH: corticotrophin-releasing hormone; GH: growth hormone; SNS: sympathetic nervous system)

Leukocyte trafficking is crucial to pathogen surveillance. It has been shown that acute exercise results in a temporary, significant, and reversible redistribution of leukocyte subsets between circulation, marginal pools and the bone marrow (Gleeson & Bishop, 1999); and that this exercise-induced mobilization is related to elevated plasma concentrations of stress hormones (Benschop, Rodriguez-Feuerhahn, & Schedlowski, 1996) (Figure 2).

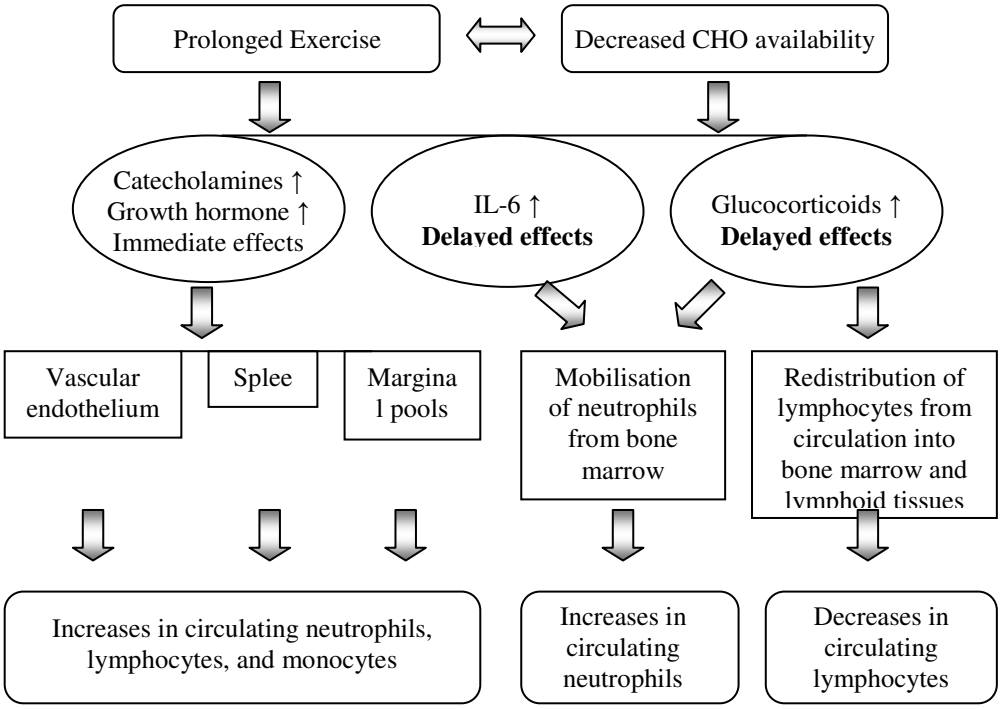

Figure 2 Mechanisms of the immediate and delayed leukocytosis induced by prolonged exercise

Catecholamines and leukocyte mobilization

During exercise, adrenaline is released from the adrenal medulla and noradrenaline is released from sympathetic nerve terminals. Arterial plasma concentrations of adrenaline and noradrenaline increase almost linearly with duration of dynamic exercise and exponentially with intensity (Kjaer, 1989). The effects of catecholamines on target cells are mediated via adrenoreceptors, which can be generally classified to four categories (α_1, α_2, β_1, and β_2) based on their different sensitivities to certain agonists. Adrenaline is a strong stimulator of β-adrenoreceptors, whereas noradrenaline predominately activates α- and β_1-adrenoreceptors (Motulsky & Insel, 1982). Since β-adrenoreceptors have been identified on T cells, B cells, NK cells, macrophages and neutrophils (Landmann, 1992), the degree to which these cells can be influenced by catecholamine signalling depends on the numbers of adrenergic receptors on the individual leukocyte subpopulations (Pedersen, 1999). The numerical orders of adrenergic receptors on lymphocyte subpopulations from highest to lowest are NK cells, $CD8^+$ lymphocytes, B cells, and $CD4^+$ lymphocytes (Rabin, Moyna, Kusnecov, Zhou, & Shurin, 1996). Therefore, after injection/infusion of adrenaline the most pronounced changes are observed for NK cells (CD16, CD56, CD57) and with subsequent smaller changes in $CD8^+$ cells, B cells, and $CD4^+$ (Benschop *et al.*, 1996).

As early as 1904, Loeper and Crouzon described a pronounced leukocytosis after a subcutaneous injection of adrenaline in man (Loeper & Crouzon, 1904). This observation was extended by subsequent studies and suggested the notion that the adrenaline response to leukocyte mobilization was biphasic, consisting of an initial lymphocytosis within 10 min, peaking at 30 min, and followed by a neutrophilia with relative lymphopenia, peaking at 2 to 4 h (Samuels, 1951). However, adrenaline infusion caused a significantly smaller elevation in neutrophil concentration than that observed following exercise while the plasma concentrations of adrenaline following administration and exercise were similar (Tvede *et al.*, 1994). Although adrenaline is an important hormone in recruiting lymphocytes and neutrophils into the circulation during intensive exercise within 90 min (Ernstrom & Sandberg, 1974; Nieman, 1997), after 90 min during exercise, its effect is lessened by the rising cortisol concentration, which attenuates lymphocytosis with a subsequent decline until exercise is finished (Nieman, 1997). It is likely that the adrenaline exerts a direct effect on neutrophil surface adhesive molecules (e.g., β_2-integrin CD11b/CD18) (Benschop *et al.*, 1996), and this together with exercise-induced haemodynamic shear forces (Foster, Martyn, Rangno, Hogg, & Pardy, 1986), work in a synergistic fashion to mobilise neutrophils from the marginal pools into the circulation, inducing the initial neutrophilia during prolonged exercise (Gannon, Rhind, Shek, & Shephard, 2001). It has been also demonstrated that the neutrophilia after adrenaline infusion was mainly recruited from spleen (Benschop *et al.*). Although lung has been suggested to be an important organ of neutrophil storage (Hogg, 1987), it did not seem to contribute significantly to the peripheral neutrophilia induced by exercise (Peters *et al.*, 1992).

Glucocorticoids and leukocyte mobilization

Increases of plasma cortisol concentrations are associated with exercise intensity and duration of above 1 h. Cortisol is the principal glucocorticoid in humans playing a major role in metabolism and immune function as a potent agent of gluconeogenesis and immunosuppression (Pedersen *et al.*, 1997). Furthermore, glucocorticoids exert a prominent role in the regulation of leukocyte redistribution (Cupps & Fauci, 1982; Dhabhar *et al.*, 1994). Glucocorticoid administration has been reported to cause neutrophilia together with lymphopenia, monocytopenia, eosinopenia, and a suppression of both NK and T cell function (Cupps & Fauci, 1982; Fauci, 1976). The significant but transient neutrophilia induced by cortisol administration is mainly caused by the influx of neutrophils from spleen and bone marrow (Toft, Helbo-Hansen, Lillevang, Rasmussen, & Christensen, 1994). Further, Nakagawa *et al.* (1998) showed after dexamethasone infusion, the circulating neutrophilia was from bone marrow (10%), marginated pools (61%) and prolongation of neutrophil intravascular half-life (29%) in rabbits. Cortisol promotes lymphocyte redistribution from the circulation to lymphoid, bone marrow, skin and injured tissue (Toft, Tonnesen, Svendsen, & Pasmussen, 1992; Wira, Sandoe, & Steele, 1990), reaching a maximum at 4 to 6 h with a return to baseline within 24 h (Calvano *et al.*, 1992; Fauci & Dale, 1975).

Growth hormone and leukocyte mobilization

Growth hormone (GH) is a classical anterior pituitary hormone promoting cell growth and metabolism (Kappel *et al.*, 1993). In terms of immunological development and function, GH promotes lymphocyte maturation and competence, NK cell activity, cytokine production and phagocyte oxidative burst activity (Berczi, 1997; Hattori *et al.*, 2001). Exercise is a powerful stimulant for GH secretion, depending on workload, duration, intensity, prior meal ingestion, and fitness level of subjects (Kanaley, Weltman, Pieper, Weltman, & Hartman, 2001). Repeated bouts of exercise on the same day appear to augment GH release (Kanaley *et al.*), whereas glucose ingestion attenuates GH secretion (Smith *et al.*, 1996). Furthermore, GH is probably at least partly responsible for exercise-induced neutrophilia because Kappel *et al*. have demonstrated that an intravenous GH injection in a physiological dose caused a neutrophilia.

Carbohydrate ingestion and exercise-induced leukocyte mobilization

Intensive prolonged exercise without nutritional supplementation may cause hypoglycaemia and subsequently evoke the increased secretion of stress hormones. Schwartz, Clutter, Shah, and Cryer (1987) reported the thresholds of plasma glucose concentrations for inducing adrenaline, GH, cortisol and hypoglycaemic symptoms were 3.8 ± 0.1 mM, 3.7 ± 0.1 mM, 3.2 ± 0.2 mM and 2.9 ± 0.1 mM, respectively. The results of previous studies have consistently demonstrated that ingestion of a CHO drink compared with placebo (PLA) during exercise is beneficial for maintaining plasma glucose concentrations, improving endurance exercise performance and attenuating the elevation of plasma stress hormones and perturbation of circulating counts of total leukocytes and leukocyte subsets (Table 1).

Table 1 CHO ingestion and exercise-induced leukocyte mobilisation

Reference	Experimental Design	Subject	Main Findings
Bishop, Gleeson, Nicholas, & Ali. (2002)	90 min high intensity intermittent running. Subjects ingested 5 mL·kg^{-1} of a 6.4% CHO or PLA before exercise and every 15-min interval during exercise.	6 male footballers	CHO ingestion attenuated the neutrophilia in the circulation compared with PLA.
Bishop, Blannin, Walsh, & Gleeson. (2001)	Subjects cycled at 75% $\dot{V}O_2$max until fatigue. Subjects ingested 5 mL·kg^{-1} of a 5% CHO or PLA before exercise and 2 mL·kg^{-1} every 15-min interval during exercise.	9 males	CHO ingestion significantly increased performance but did not affect the circulating leukocytosis, neutrophilia and lymphocytosis compared with PLA.
Henson *et al.* (2000)	2 h rowing at 82.3 % HRmax. Subjects ingested 15 mL·kg^{-1} of a 6% CHO or PLA 15-min before exercise and 4 mL·kg^{-1} every 15-min during exercise.	15 female rowers	CHO ingestion attenuated the increase in blood counts of total leukocytes, neutrophils and monocytes compared with PLA.
Nieman *et al.* (1997)	2.5 h run at 77%$\dot{V}O_2$max. Subjects ingested 750 mL of a 6% CHO or PLA before exercise and 250 mL every 15-min during exercise.	30 marathoners (24 males & 6 females)	CHO ingestion attenuated the increase in blood counts of neutrophils, lymphocytes and monocytes compared with PLA.

Summary

In this review an attempt was made to present an overview of the current knowledge of the leukocyte redistribution to exercise-induced stress. The available evidence suggests that exercise affects the immune system via stimulation of SNS and HPA-axis and the secretion of cytokines during exercise. To summarize, the immediate leukocytosis during prolonged exercise is mainly due to elevated plasma catecholamines levels, whereas the delayed neutrophilia is from the influence of elevated plasma cortisol levels. Since plasma cortisol concentration peaks at about 30 min after exercise cessation (Hansen, Wilsgard, & Osterud, 1991), it is not surprising to observe the development of a significant neutrophilia with lymphopenia within the first hour of recovery (McCarthy & Dale, 1988). Besides, GH is also partly responsible for exercise-induced neutrophilia since an intravenous GH injection in a physiological dose caused a neutrophilia (Kappel *et al*. 1993). However, CHO supplementation during exercise is beneficial for maintenance of plasma glucose concentrations and then both improving endurance exercise performance and attenuating the elevation of plasma stress hormones and perturbation of leukocyte redistribution.

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Exercise and Leukocyte Redistribution

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ABSTRACT

This review was to present an overview of the current knowledge of how exercise affects the leukocyte redistribution via stimulation of sympathetic nervous system and hypothalamic-pituitary-adrenal-axis. To summarize, the immediate leukocytosis during prolonged exercise is mainly due to elevated plasma catecholamines levels, whereas the delayed neutrophilia is from the influence of elevated plasma cortisol levels. Besides, growth hormone is also partly responsible for exercise-induced neutrophilia. However, carbohydrate supplementation during exercise is beneficial for maintenance of plasma glucose concentrations and then both improving endurance exercise performance and attenuating the elevation of plasma stress hormones and perturbation of leukocyte redistribution.

Key Words: exercise, stress hormones, leukocyte redistribution