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The effect of maximal exercise on the activity of neutrophil granulocytes in highly trained athletes in a moderate training period

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Summary. Leucocyte cell counts and the phagocytic and chemotactic activities of neutrophil granulocytes were investigated in highly endurance-trained long-distance runners ($n = 10$) and triathletes ($n = 10$) during a moderate training period and compared with untrained subjects ($n = 10$) before and up to 24 h after a graded exercise to exhaustion on a treadmill. After exercise a leucocytosis was noted with a significant increase in lymphocyte ($P \leq 0.01$) and neutrophil ($P \leq 0.01$) counts in all groups. In neutrophils the number of ingested inert latex beads was significantly increased ($P \leq 0.01$) from 0.21 (SD 0.09) to 0.45 (SD 0.22) in controls, from 0.20 (SD 0.12) to 0.56 (SD 0.16) in long-distance runners and from 0.25 (SD 0.08) to 1.03 (SD 0.42) particles per cell in triathletes 24 h after exercise, compared with resting values. The capability of neutrophils to produce microbicidal reactive oxygen species fell ($P \leq 0.05$) immediately after exercise in all subjects and then increased by 36 (SD 8)%, 31 (SD 6)% and 19 (SD 9)% in controls, runners and triathletes respectively up to 24 h after exercise ($P \leq 0.05$) compared with pre-start values. With respect to the absolute number of neutrophils, ingestion capacity, production of superoxide anions and chemotactic activity, no significant differences were found between athletes and control subjects at rest and after exercise. These data indicate, on the one hand, no impairment of the granulocyte system during a moderate training period in long-distance runners and triathletes but, on the other, that the prolonged activation of the phagocytosis reaction after exercise might impair the granulocyte system in periods of intensive training with high training frequency.

Key words: Exercise – Neutrophils – Leucocytosis – Phagocytosis – Chemotaxis

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Introduction

Neutrophil granulocytes play an important role in the immune system. They form a "first line of defence" against invading microorganisms by destroying them or impairing their development (Sawyer et al. 1989).

Neutrophil defects, such as neutropenia, Chediak-Higashi syndrome or chronic granulomatous defect, are often associated with recurrent infections (Curnutte 1988; Weingarten and Bokoch 1990) and indicate a relationship between neutrophil granulocytes and susceptibility to infection. Frequent intense training has been shown to impair the immune response of athletes' lymphocytes and natural killer cells and for this reason might increase susceptibility to infection (Mackinnon and Tomasi 1986; Fitzgerald 1988; Liesen et al. 1989; Fitzgerald 1991). In addition to lymphocytes, the main cellular component of the immune system, it thus seems important to investigate the influence of acute exercise and training on neutrophil granulocytes.

Recent results (McCarthy and Dale 1988; Hansen et al. 1991; Heath et al. 1991) have shown a biphasic pattern of neutrophil counts with increasing levels immediately and 2–4 h after exercise.

There are few data available concerning neutrophil functions – chemotaxis and phagocytosis – in relation to exercise, despite the ability of neutrophil granulocytes to protect the body against infection (Gaudry et al. 1990). Contradictory results with unchanged (Lewicki et al. 1987) as well as increased (Rodriguez et al. 1991) phagocytic activity immediately after exercise have been reported. Smith et al. (1990) showed that phagocytic activity remains increased for 6 h after submaximal exercise. In the present study the activity and cell count of neutrophil granulocytes obtained from highly trained long-distance runners and triathletes were determined and compared with values obtained from untrained control subjects before and after a graded exercise to exhaustion on a treadmill. In order to determine whether a single exhausting exercise has a prolonged effect on the neutrophil granulocyte system, cell count and phagocytosis

Table 1. Anthropometric and performance data of subjects

		Controls	Runners	Triathletes
Age (years)		25.5 (3.2)	28.6 (5.2)	28.8 (4.5)
Height (cm)		185 (6.6)	181 (4.7)	183 (7.5)
Body mass (kg)		80.6 (9.8)	66.5 (5.9)	73.8 (5.9)
$\dot{V}O_{2max}$ ($ml \cdot min^{-1} \cdot kg^{-1}$)		53.3 (5.5)**	72.6 (6.0)	67.7 (3.5)
V_{max} ($km \cdot h^{-1}$)		15.6 (1.5)**	20.4 (1.3)	19.4 (0.9)
f_c ($beats \cdot min^{-1}$)	pre-start	65 (14)**	51 (8)	55 (6)
	post-exercise	199 (3)	195 (4)	198 (3)
MAP (mm Hg)	pre-start	104 (9)**	90 (10)	89 (6)
	post-exercise	125 (16)	121 (15)	123 (12)

Values are means (SD). Significant differences between athletes and control subjects are indicated as follows: * $P \leq 0.05$; ** $P \leq 0.01$; $\dot{V}O_{2max}$, maximum rate of oxygen uptake; V_{max}, maximum running velocity; f_c, heart rate; MAP, mean arterial blood pressure

as well as chemotactic activity were investigated up to 24 h after exercise.

Methods

Exercise protocol. Participants in this study were male long-distance runners ($n = 10$) and triathletes ($n = 10$) of national and international standard as well as untrained medical students ($n = 10$) serving as controls. At the time of the study the long-distance runners were undergoing an extensive training period with endurance running of 115.0 ± 39.8 $km \cdot week^{-1}$. The triathletes were also investigated during an extensive training period with endurance running of 66.9 ± 17.3 $km \cdot week^{-1}$, swimming of 11.1 ± 3.4 $km \cdot week^{-1}$ and cycling of 246.4 ± 116.1 $km \cdot week^{-1}$.

Medical examination showed the subjects to be in good health. They had been without medication for at least 6 weeks before the study and had refrained from eating and drinking alcohol, coffee or tea for 12 h before the test. Having been informed about the study design all subjects gave their written consent.

All subjects underwent a graded exercise test (Laufergotest LE1; Jäger, Würzburg, FRG) to exhaustion on a treadmill at a constant slope of 2.5%. The treadmill velocity started at $5 \; km \cdot h^{-1}$ for 5 min, followed by an increment of $3 \; km \cdot h^{-1}$ for 3 min and was finally increased by $2 \; km \cdot h^{-1}$ every 3 min until exhaustion. Blood was collected before the exercise (after 0.5 h rest in the supine position), immediately after, and 0.5 h and 24 h post-exercise. To assess aerobic capacity, heart rate, mean arterial blood pressure (MAP), oxygen uptake and running velocity were determined. Heart rate was measured by ECG (Servomed SM 104; Hellige, Freiburg, FRG). MAP was calculated using the following formula:

MAP = 1/3 (systolic blood pressure − diastolic blood pressure) + diastolic blood pressure.

Oxygen uptake was determined by the open-circuit method (Siregnost FD 85S; Siemens, Mannheim, FRG). Criteria for exhaustion were considered to be the subjects' behaviour, a heart rate of more than 190 $beats \cdot min^{-1}$, levelling off of the maximal oxygen consumption ($\dot{V}O_{2max}$) and a respiratory quotient of more than 1.1.

Anthropometric and exercise data of all subjects are summarized in Table 1.

Preparation of neutrophil granulocytes. Blood (40 ml) was collected from the antecubital vein into tubes containing 8 ml of 4.5% Na-ethylenediaminetetraacetate (pH 7.4). The samples were kept on ice for further preparation. Cells were prepared at 4° C in sterile tubes (Becton Dickinson, New Jersey, USA) to avoid activation of neutrophil granulocyte functions. Platelet-rich plasma was removed from the blood by centrifugation at 200 *g* for 10 min and the pellet resuspended in an equal volume of phosphate buffered saline (Gibco BRL, Eggenstein, FRG). Neutrophils were separated from lyphocytes by standard Ficoll-Hypaque (Biochrom, Berlin, FRG) density-gradient centrifugation at 450 *g* for 30 min (Boyum 1968). Remaining erythrocytes were destroyed by hypotonic lysis in the presence of 20 ml of 0.2% NaCl for 15 s, followed by the addition of equal volumes of 1.6% NaCl to restore isotonicity. After centrifugation for 10 min at 300 *g* the supernatant containing lysed erythrocytes was removed. This step was repeated until the pellet was free of erythrocytes, i.e. when the opaque mixture became translucent. Neutrophils were washed twice in 12 ml Hanks balanced salt solution (HBSS; Gibco BRL), counted and then suspended in HBSS and adjusted to a concentration of $1 \cdot 10^6$ $cells \cdot ml^{-1}$. Differential blood counts using prestained slides (Sangodiff G; Merck, Darmstadt, FRG) showed the cell suspensions to comprise more than 98% neutrophils. Viability was checked by the fluorescence assay according to Ford and Hunt (1979) and found to be always above 95%. The cell counts of leucocytes, lymphocytes and neutrophils in each blood sample were determined with the Coulter Counter T 660 (Coulter Electronics, Krefeld, FRG). Changes in plasma volume were determined by the method of Dill and Costill (1974).

Latex assay. As previously described by Schroeder and Kinden (1983), $1 \cdot 10^6$ $neutrophils \cdot ml^{-1}$ were incubated with fluorescent monodisperse latex beads (diameter 0.7 µm; Serva, Heidelberg, FRG) at 37° C for 30 min (250 $beads \cdot cell^{-1}$). Cells were centrifuged at 100 *g* for 5 min and washed in 6 ml HBSS. The number of ingested latex beads in 200 neutrophils was counted by fluorescence microscopy immediately after washing. The phagocytic index (PI) was calculated according to the following formula:

PI = number of ingested particles · 200 $neutrophils^{-1}$

Superoxide anion assay. Superoxide anions (O_2^-) were determined as previously described by Gaudry et al. (1990) using phorbol-12-myristate-13-acetate (PMA; Sigma, Deisenhofen, FRG). A 2 ml volume of the cell suspension ($2 \cdot 10^6$ neutrophils) was incubated with $1 \; mg \cdot ml^{-1}$ ferricytochrome C (horse heart type III, Sigma) for 30 min at 37° C with or without $1 \; \mu g \cdot ml^{-1}$ PMA. The O_2^- concentration was calculated from the absorption at 550 nm against a cell-free blank multiplied with the molar extinction coefficient of $21 \cdot 10^3$ $M^{-1} \cdot cm^{-1}$ (Massey 1959). Negative controls without PMA showed no effect on O_2^- production.

Chemotaxis assay. The mobility of neutrophil granulocytes was measured by the agarose-plate method, as previously described (Nelson and Herron 1988). Briefly, $5 \cdot 10^5$ cells were allowed to migrate under an agarose gel toward a chemotactic stimulus of $1.1 \cdot 10^{-7}$ M *N*-formyl-methionyl-leucyl-phenylalanine (Sigma) for 4 h in a CO_2 incubator (B 5060 EK/CO_2; Heraeus Christ, Hanau, FRG) at 37° C. The chemotactic index (CI) was calculated using the following formula:

CI = (directional migration – random migration) · random migration^{-1}

Statistical analysis. The experimental data established in duplicate are given as means (SD) unless otherwise stated. All data were analysed statistically using the SAS statistical analysis program (version 6.04; SAS Institute, Cary, USA). Comparisons between groups were performed using Student's unpaired *t*-test; for comparisons within groups, the paired *t*-test was used.

Results

The exercise test

Heart rate and MAP at rest were significantly lower ($P \leq 0.01$) in the long-distance runners and triathletes than in controls, whereas $\dot{V}O_{2max}$ and the maximal running velocity were significantly higher ($P \leq 0.01$). Table 1 shows that all athletes were highly trained.

Quantification of blood lymphocytes

Immediately after exercise the total number of leucocytes (WBC), lymphocytes and neutrophil granulocytes increased significantly compared with resting values in all subjects ($P \leq 0.01$) (Table 2). This initial increase of leucocytes was followed 30 min after exercise by a significant lymphopenia compared with pre-start levels. Additionally, cell counts were determined in the triathlete group after 24 h of recovery. At this time all cell counts had reached pre-exercise levels. Neither at rest nor after exercise were significant differences found between groups.

Table 2. Cell counts of circulating leucocytes, lymphocytes and neutrophils

[Cells · nl^{-1}]		Leucocytes	Lymphocytes	Neutrophils
Pre-start	Controls	5.56 (0.84)	2.17 (0.29)	3.23 (0.76)
	Runners	5.44 (0.82)	1.90 (0.46)	3.41 (0.83)
	Triathletes	4.80 (1.27)	1.61 (0.56)	2.98 (1.18)
Post-exercise	Controls	8.74 (1.66)**	4.01 (0.96)**	4.17 (0.86)**
	Runners	9.30 (1.57)**	4.31 (1.24)**	4.25 (0.86)**
	Triathletes	8.66 (1.86)**	3.75 (0.99)**	4.31 (1.67)**
0.5 h recovery	Controls	4.91 (0.68)**	1.50 (0.17)**	3.27 (0.58)
	Runners	4.91 (0.94)*	1.55 (0.35)*	3.31 (0.94)
	Triathletes	5.26 (1.72)	1.24 (0.40)**	3.92 (1.87)
24 h recovery	Controls	ND	ND	ND
	Runners	ND	ND	ND
	Triathletes	4.84 (1.15)	1.64 (0.56)	3.03 (0.79)

Values are means (SD). Significant differences compared with resting values are indicated as: * $P \leq 0.05$; ** $P \leq 0.01$; ND, not determined

Phagocytosis assays

The complex phagocytosis reaction was measured by latex ingestion and O_2^- production. Cell viability was 96 (SD 3) % and remained unchanged throughout the tests. The PI, as a measure of ingestive capacity, increased significantly immediately after exercise in neutrophil granulocytes from 0.21 (SD 0.09) to 0.33 (SD 0.14), from 0.20 (SD 0.12) to 0.28 (SD 0.13) and from 0.25 (SD 0.08) to 0.38 (SD 0.13) particles · cell^{-1} in controls, long-distance runners and triathletes, respectively (Fig. 1). PI was even further increased 24 h after exercise in all groups: 0.45 (SD 0.22) particles · cell^{-1} in controls, 0.56 (SD 0.16) in long-distance runners and 1.03 (SD 0.42) in triathletes, compared with resting values. At 24 h after

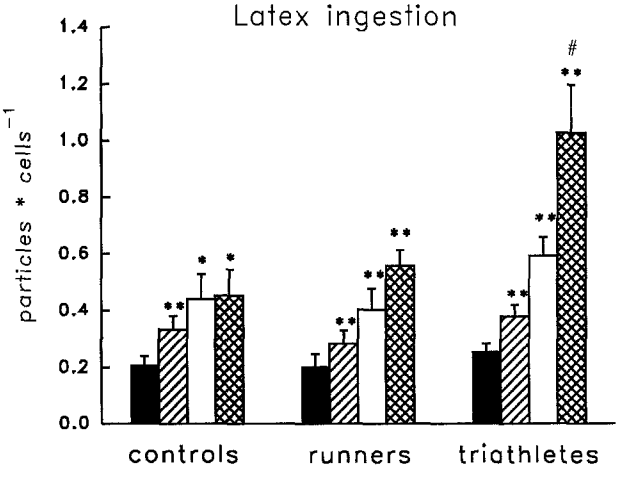

Fig. 1. Ingestion capacity of neutrophils before (■) and immediately after (▨) a graded exercise to exhaustion on a treadmill and after 0.5 h (□) or 24 h (▩) recovery; values are means, *error bars* indicate SEM; significant differences compared with resting values are indicated as follows: * $P \leq 0.05$, ** $P \leq 0.01$; significant differences between controls and triathletes are indicated as # $P \leq 0.05$

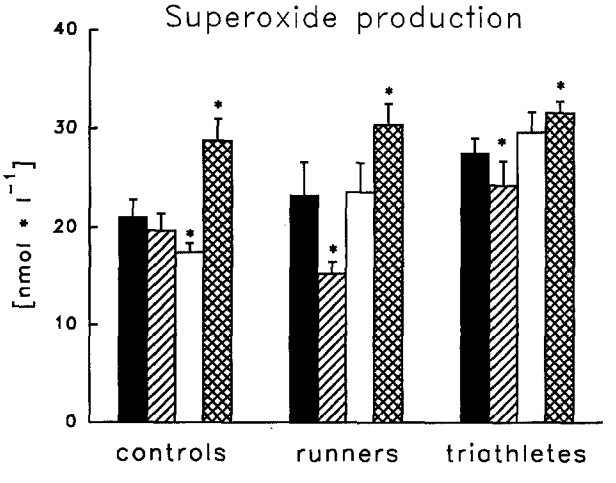

Fig. 2. Superoxide anion production of neutrophils before (■) and immediately after (▨) exercise and after 0.5 h (□) and 24 h (▩) recovery; values are means, *error bars* indicate SEM; significant differences compared to resting values are indicated as * $P \leq 0.05$

Table 3. Chemotaxis of peripheral blood neutrophils

		Chemotaxis (CI)
Pre-start	Controls	1.81 (0.94)
	Runners	2.01 (0.93)
	Triathletes	1.43 (0.41)
Post-exercise	Controls	1.85 (1.49)
	Runners	1.56 (1.04)
	Triathletes	1.74 (0.61)
0.5 h recovery	Controls	2.05 (0.84)
	Runners	2.38 (1.20)
	Triathletes	1.55 (0.69)
24 h recovery	Controls	2.26 (0.96)
	Runners	2.58 (1.10)
	Triathletes	1.39 (0.15)

Values are means (SD). CI, chemotactic index

exercise a higher ingestion capacity was observed in long-distance runners (NS) and triathletes ($P \leq 0.05$) compared with controls.

The estimation of the phagocytic process by determination of O_2^- production demonstrated a higher (but not significant) activity in triathletes and long-distance runners at rest compared with controls (Fig. 2). In contrast to the latex ingestion O_2^- production was significantly reduced after exercise in all subjects ($P \leq 0.05$). However, 24 h after exercise O_2^- production was significantly increased ($P \leq 0.05$) by 36 (SD 8) % in controls, by 31 (SD 6) % in long-distance runners and by 19 (SD 9) % in triathletes compared with resting values.

Chemotaxis

The chemotactic capacity and random migration of neutrophils were not significantly modified by exhaustive exercise or by a recovery period of 24 h. Only small differences between groups at rest and after exercise were observed. Results of the agarose assay are shown in Table 3.

Discussion

It is assumed that mild exercise improves the immune system (Nieman et al. 1990; Nehlson-Cannarella et al. 1991). Recent investigations in top athletes, however, indicate that intense training during a competitive period may suppress the immune system and increase susceptibility to infections (Simon 1984; Keast et al. 1988; Pedersen 1991). To elucidate this phenomenon most researchers have determined lymphocyte cell counts and their in vitro response to mitogens, which is reduced for a short time after an exhaustive exercise bout (Nehlson-Cannarella et al. 1991; Frey et al. 1992). Less attention has been paid to date to neutrophil granulocytes, although they contribute importantly to the immune system by their support of specific immune reactions (Thompson 1988).

Our results indicate that graded exercise to exhaustion on a treadmill is associated with an increase in circulating neutrophils, an increase in phagocytic ingestion and a marginal decrease in bacterial killing immediately after exercise. The increase in neutrophils is known to result from a selected redistribution of neutrophils from marginated pools and due to increased catecholamine levels after exercise (McCarthy and Dale 1988; Hansen et al. 1991). Catecholamines are also suggested to play a role in the impairment of "bacterial killing" immediately after exercise, since they increase (5- to 10-fold) during exercise (data not shown) and correlate roughly with the decrease in bacterial killing ($r = -0.57$; $P \leq 0.05$). Furthermore, recent work by Macha et al. (1990) demonstrated a 40% decrease in bactericidal capacity after addition of post-exercise plasma to pre-exercise neutrophils and they suggested there might be an inhibitory factor in the plasma. Moreover, Yamazaki et al. (1989) demonstrated that O_2^- production in neutrophils decreased in vitro after treatment with catecholamines. They also showed that this effect was diminished after β-blockade, which supports the hypothesis of catecholamines being an inhibitory factor in bacterial killing. However, this decrease in neutrophil function immediately after exercise is only transient, as has been shown previously for the impairment in specific immune functions (Frey et al. 1992), and might therefore reflect immunomodulation rather than real immunosuppression.

The major result of this study was that phagocytic ingestion and bacterial killing increased up to 24 h after exercise. At this time the cell counts of all subjects had already returned to pre-exercise levels.

There are several possible explanations for this prolonged activation: not all work-induced microtraumatic events or inflammatory irritations may have been eliminated completely (Smith et al. 1990) or the post-exercise increase of cortisol (Hansen et al. 1991) may lead to a selective release of higher-activity neutrophil granulocytes from the bone marrow (Lew 1990). This latter interpretation can be supported by the finding that neutrophil granulocytes are widely heterogeneous (Gallin 1984; Miyagawa et al. 1990) and that after acute bacterial infection a subpopulation of higher-activity neutrophil granulocytes has been observed (Bass et al. 1986).

Since an increase in phagocytic activation was obtained 24 h after exercise and since athletes had even higher values in both phagocytic tests compared with untrained subjects, it is assumed that this function is not impaired in the athletes during a moderate training period. The lack of significant differences between trained and untrained subjects at rest regarding neutrophil cell counts and phagocytic tests as well as chemotaxis further supports this assumption.

Lowered neutrophil cell counts and phagocytic activity in sportsmen at rest compared with controls have been demonstrated recently (Lewicki et al. 1987; Smith et al. 1990), in contrast to our data. Unfortunately these authors supply no information about the training period. This is an important parameter and might explain the conflicting results, since the prolonged activation of phagocytic reactions obtained 24 h after a single session

of exhaustive exercise might impair the granulocyte system of top athletes in periods of increased training intensity and frequency. This must be further elucidated by investigating the same cohort of athletes during a period of intense training.

In conclusion, our results indicate activation of the phagocytosis persisting 24 h after exhaustive exercise but no difference in the granulocyte system between athletes in a period of moderate training and untrained subjects.

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