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Fitness in the fit: does physical conditioning affect cardiovascular risk factors in middle-aged marathon runners?

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Objective The study was designed to assess cardiovascular risk factors in marathon runners with different degrees of fitness.

Design A total of 30 male middle-aged marathon runners were divided according to their marathon running time into fit (265 ± 8 min), fitter (222 ± 5 min) and fittest (178 ± 12 min). The three groups of 10 runners each were comparable in age, weight, and body surface area. Cardiovascular risk factors were assessed by measuring arterial pressure before and during exercise (150 watts) and determination of plasma lipoproteins, uric acid, glucose and white blood cell count before and after a marathon run.

Results All measured laboratory values such as high-density lipoprotein cholesterol ($P<0{\cdot}05$), low-density lipoprotein cholesterol ($P<0{\cdot}05$), total cholesterol (non-significant), triglycerides (non-significant), blood sugar (non-significant), uric acid ($P<0{\cdot}01$) and white blood cell count ($P<0{\cdot}05$) indicated a lower cardiovascular risk in the fastest when compared with the slowest runners. Resting blood pressure was similar in the three groups but consistently lower at all levels of exercise in the fittest when compared with the less fit runners. The fittest runners also showed greater increases in high-density lipoprotein cholesterol after the marathon run (14% vs 8% in the slowest runners, $P<0{\cdot}005$).

Conclusions We conclude that even at the extreme end of a continuum such as represented by well-conditioned, middle-aged marathon runners, cardiovascular risk factors are related to the degree of fitness, as measured by the marathon running time.

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Key Words: Exercise and cardiovascular risk factors, blood pressure and exercise, lipids and exercise.

Introduction

Regular conditioning has been well documented to exert a favourable influence on cardiovascular risk factors in physically inactive subjects and has been demonstrated to effectively reduce the incidence of non-fatal and fatal coronary heart disease[1–8]. Even in moderately active subjects, an increase in physical activity has been reported to lower arterial pressure and total cholesterol and to increase high-density lipoprotein cholesterol levels[9–15]. Marathon runners are considered to be physically very fit, yet no attempt has been made to link the degree of fitness with cardiovascular risk factors in these athletes. The present cross-sectional study was therefore designed to evaluate whether the degree of physical fitness would be related to cardiovascular risk factors even in extremely well-trained subjects such as marathon runners.

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Subjects and methods

Out of a group of a total of 100 marathon runners who participated in the Berlin Marathon (Germany), 30 male runners who had completed several marathons within the past months were recruited for the present study. According to their marathon running time, the runners were subdivided into three matched groups with no significant differences in age, weight, height, and body surface area between the three groups (Table 1): fit — slowest marathon running time (265 ± 8 min, n=10, aged 42 ± 10) years); fitter — intermediate marathon running time (222 ± 5 min, n=10, aged 39 ± 12) years); and fittest — fastest marathon running time (178 ± 12 min, n=10, aged 42 ± 9 years).

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***Table 1** Age, height, weight, body surface area, and pre-marathon training in three groups of marathon runners (mean ± 1 standard deviation)*

Group	Age (years)	Height (cm)	Weight (kg)	Body surface area (m²)	Pre-marathon training		
					km . week⁻¹	Frequency/ week	Years of training
Fittest	41·8 ± 9·2	174·3 ± 5·8	69·8 ± 6·4	1·84 ± 0·11	77 ± 18	4·8 ± 1 2	8·2 ± 6·3
Fitter	39·0 ± 12·4	176 0 ± 6·6	72·6 ± 6·3	1·88 ± 0·1	50 ± 11	3·7 ± 1·4	5 7 ± 3·6
Fit	42·4 ± 10·4	173·4 ± 4·1	71·5 ± 5·8	1·85 ± 0·09	38 ± 9	2·8 ± 0·5	7 3 ± 5·7

***Table 2** Cholesterol, high-density lipoprotein, low-density lipoprotein, triglycerides, glucose, uric acid, haematocrit and white blood cell count in three groups of marathon runners (mean+1 standard deviation)*

Group	Chol. (mg . dl⁻¹)	HDL (mg . dl⁻¹)	LDL (mg . dl⁻¹)	Triglyc. (mg . dl⁻¹)	Glucose (mg . dl⁻¹)	Uric acid (mg . dl⁻¹)	Haematocrit (%)	WBC (mrdl . l⁻¹)
Fittest	213 ± 33	64 ± 10	134 ± 25	84 ± 18	94 ± 4	4·6 ± 0·7	0·440 ± 0·025	5 2 ± 0·7
Fitter	221 ± 34	61 ± 11	144 ± 29	81 ± 29	96 ± 8	5·7 ± 0·5*	0·440 ± 0·028	6·1 ± 1·1
Fit	229 ± 29	54 ± 9*	154 ± 29*	106 ± 43	99 ± 8	5·6 ± 0·6†	0·445 ± 0·023	6 6 ± 1·4*

Chol =cholesterol; HDL=high-density lipoprotein; LDL=low-density lipoprotein; Triglyc.=triglycerides; WBC=white blood cell count. *$P<0·05$ (compared with fittest); †$P<0·01$ vs fittest.

Before the marathon run, all subjects exercised in a sitting position on a bicycle ergometer under controlled and identical conditions, using techniques given in the Proposal for International Standardization of Ergometry[16]. The exercise load started at 50 watts, with an increase of 25 watts every 2 min to a maximum of 150 watts. During this bicycle ergometry, arterial pressure readings were taken every 2 min by sphygmomanometer. Venous blood was drawn from the antecubital fossa before and immediately after a marathon run to assess high-density lipoprotein cholesterol, triglycerides, glucose, uric acid, haematocrit, and white blood cell count. Low-density lipoprotein cholesterol was calculated using the formula of Friedewald[17].

Statistical analysis within the three groups was done by analysis of variance. The significance of difference between the three groups was compared by Student's non-paired, two-tailed t-test. All data are presented as the mean ± 1 standard deviation.

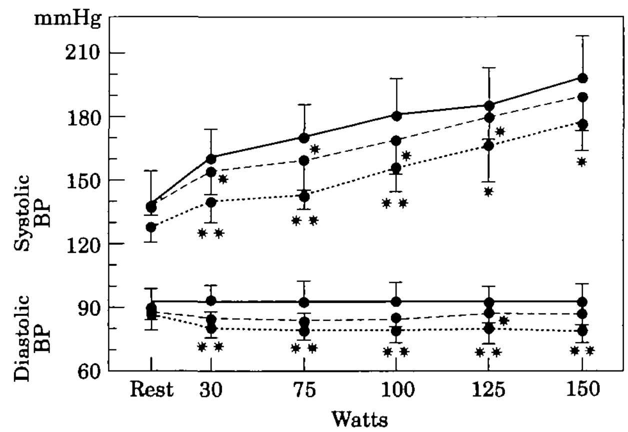

Figure 1 **Systolic and diastolic pressure at rest and during exercise in three groups of marathon runners. ● · · · · ● =fittest; ● – – – ● =fitter; ● ——— ● =fit. *$P<0·05$, **$P<0·01$ vs fit.**

Results

The pre-marathon training pattern was somewhat different for the three groups, and the fittest runners averaged more kilometres per week than the less fit contenders (Table 1). There was a statistically significant ($P<0·01$) correlation between the marathon running time and heart rate during standardized exercise (100 watts) in all groups. The fittest subjects had significantly higher high-density lipoprotein cholesterol and lower low-density lipoprotein cholesterol levels, as well as lower uric acid levels and white blood cell counts, than subjects with the slowest marathon running time (fit) (Table 2).

A similar trend that did not quite reach statistical significance was observed for total cholesterol, triglycerides, and fasting blood sugar. The three groups had similar systolic and diastolic blood pressure at rest. However, during graded bicycle exercising arterial pressure was consistently lower at all exercise levels in the fittest marathon runners when compared with their less fit counterparts (Fig. 1).

A marathon run significantly increased blood sugar, uric acid, and levels of triglycerides as well as high-density lipoprotein cholesterol (Table 3). The increase in high-density lipoprotein cholesterol was significantly ($P<0·005$) more marked in the fastest when

Table 3 ***Cholesterol, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, triglycerides, glucose and uric acid in three groups of marathon runners before and after a marathon run (mean ± 1SD) (all values in mg . dl⁻¹)***

Group	Cholesterol	HDL	Triglycerides	LDL	Glucose	Uric acid
Before the marathon run						
Fittest	213 ± 33	64 ± 10	81 ± 18	134 ± 25	94 ± 4	4·6 ± 0·7
Fitter	221 ± 34	61 ± 11	81 ± 29	144 ± 29	96 ± 8	5·7 ± 0·5
Fit	229 ± 29	54 ± 9	116 ± 43	154 ± 29	99 ± 8	5·6 ± 0·6
After the marathon run						
Fittest	222 ± 25	73 ± 8†	151 ± 38‡	120 ± 24*	103 ± 18	5·3 ± 0·8†
Fitter	223 ± 28	68 ± 15†	151 ± 37‡	125 ± 25‡	125 ± 35*	6·3 ± 0·7*
Fit	223 ± 28	59 ± 10	183 ± 41‡	128 ± 26‡	107 ± 17	6·1 ± 0·9

HDL = high-density lipoprotein; LDL = low-density lipoprotein.
*$P<0·05$; †$P<0·01$, ‡$P<0·001$.

compared with the slowest runners. In contrast, low-density lipoprotein cholesterol decreased in all groups, and no significant changes in total cholesterol were observed (Table 3). The three groups did not differ in haematocrit before and after the marathon race.

Discussion

Regular aerobic exercise training has been documented to lower arterial pressure, circulating levels of lipoproteins, blood sugar, and uric acid, and white blood cell count. Indeed, all three groups of marathon runners had high-density lipoprotein values that were 23% to 45% higher and triglyceride values that were 30% to 44% lower than those reported in age-matched groups of sedentary untrained subjects[18].

Most importantly, however, there was a 16% difference in high-density lipoprotein cholesterol and a 13% difference in low-density lipoprotein cholesterol between the fastest and the slowest marathon runners. Blood pressure, although not significantly different at rest, was lowest at rest and at all exercise levels in the fittest marathon runners. A similar pattern was observed for the white blood cell count, which has also been identified as a powerful risk factor for cardiovascular morbidity and mortality in the same studies[19–25]. The clinical significance of these provocative findings remains to be documented. However, data from the Framingham cohort and pooled data from four large epidemiological studies (which may or may not be valid for marathon runners) allow us to calculate that the difference in high-density lipoprotein cholesterol between the slowest and fastest marathon runners would translate into a decrease of coronary heart disease mortality by an impressive 45%[26,27], and the observed difference in total cholesterol between the fittest and the fit would reduce cardiovascular risk by 14%[28]. Clearly, however, caution must be exerted when data gathered in a predominately sedentary population (such as the Framingham cohort) is extrapolated to middle-aged marathon runners.

What is the exact pathophysiological mechanism accounting for the relationship between the physical fitness and cardiovascular risk factors? It has been well documented that an increase in fitness level lowers insulin resistance, thereby favourably affecting cardiovascular risk factors, such as blood pressure, glucose, and lipoptroteins[29–32]. However, in a recent study of runners, HDL cholesterol was shown to be directly related to their weekly mileage[33]. The reverse possibility, i.e. that inherent levels of insulin resistance and/or arterial pressure may dictate the maximal fitness level that a runner can achieve, must also be considered, particularly in marathon runners. In our study, the levels of arterial pressure during exercise were consistently higher in the slowest when compared with the fastest runners. Such a sustained increase in arterial pressure by 10% to 15% will translate into an increase of cardiac workload and myocardial oxygen consumption of the same magnitude. The slowest runners, therefore, wasted part of their cardiac energy maintaining a higher arterial pressure ('luxury hypertension') and therefore exercised less efficiently than the fastest runners. Thus, some degree of insulin resistance could account for both the relatively 'impaired' physical performance and the higher blood pressure, blood sugar, triglycerides, and cholesterol levels in the slowest runners when compared with the fitter ones.

Several other mechanisms have been put forward to explain the blood-pressure-lowering effect of regular aerobic exercise; among these are functional changes in the arterioles and increases in vascular capillary bed[34,35], diminished sympathetic activity[36–39], and a decrease in the beta-receptor activity[40].

Interestingly, the dose/response relationship between fitness and cardiovascular risk factors seems to be less clear at the other end of the spectrum. Duncan *et al.*[41], by studying sedentary pre-menopausal women, recently reported no difference in the cardiovascular risk profile between strollers, brisk walkers, and aerobic walkers although all three groups were significantly different from sedentary controls.

Of note, the fittest runner having the highest high-density lipoprotein levels at rest also showed the greatest increase in high-density lipoprotein cholesterol after the marathon run when compared with the less fit runners, which suggests that the fastest runners have more active plasma lipoprotein lipase than their less fit counterparts[42–44]. Although the exact clinical significance of our provocative results remains to be documented, it is intriguing that even at the extreme end of a continuum, such as that represented by middle-aged marathon runners, cardiovascular risk factors like arterial pressure, high-density lipoprotein cholesterol can be linked to the degree of fitness.

Implications

Even among middle-aged marathon runners who must be considered as very fit, cardiovascular risk factors are related to the degree of fitness.

Summary

Regular conditioning has been well documented to exert a beneficial effect of cardiovascular risk factors in sedentary people. In the present study, we studied the effects of exercise in marathon runners who are considered to be physically very fit. We were able to show that even in this well-trained group, cardiovascular risk factors, such as high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and blood pressure, were influenced by the degree of fitness as measured by the marathon running time.

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