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Stress Adaptation in Athletes: Relation of Lipoprotein Levels to Hormonal Response

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TSOPANAKIS, A., A. STALIKAS, E. SGOURAKI AND C. TSOPANAKIS. *Stress adaptation in athletes: Relation of lipoprotein levels to hormonal response.* PHARMACOL BIOCHEM BEHAV **48**(2) 377–382, 1994.—Increased physical stress is produced in acute exercise conditions before and during a physical trial. The effects of the physical stress on lipid and lipoprotein parameters as well as on testosterone and cortisol levels were examined in male elite athletes. In a sample of 22 measured athletes, 11 showed increases and 11 showed decreases in testosterone levels. Subsequently these subjects were treated as two separate groups for statistical purposes in order to characterise the source of individual differences in response to a stressor. Group 1 showed a 16.1% significant increase in testosterone levels, 13.3% in total cholesterol and low density lipoproteins, and a 105% increase in testosterone/cortisol ratio immediately after an acute bout of physical stress of 30 s. Group 2 showed a −25.8% significant decline in testosterone levels and no significant change in either total cholesterol or lipoprotein concentrations, followed by a significant correlation of all lipid and hormonal parameters to psychophysiological factors, such as skin temperature. A measurement of testosterone/cortisol and total cholesterol and high density lipoprotein (HDL-C) levels after a bout of acute physical stress may give a picture of the ability to "respond quickly" to stress, which will be useful in assessing the performance of the elite athlete.

Stress Lipoproteins Lipids Testosterone Cortisol Physical exercise

DIFFERENT kinds of stress provoke significant physiological alterations, some of which could be of pathogenic importance. Changes in the physiological state of humans influence not only the level of blood cholesterol but also the incidence of coronary heart disease (CHD) (4,20).

Changes in lipid and lipoprotein levels are closely related to atherosclerosis (9). It has been reported that sustained stress affects cholesterol and reduces high density lipoprotein (HDL-C) levels in animals (1,23,27,29), while a decreased ratio of HDLs over very low density lipoproteins plus low density lipoproteins [HDL/(VLDL + LDL)] has been associated with various types of mental stress (10,16).

Individual variations in serum cholesterol and lipoprotein levels are influenced by body weight, age, genetics, gender, and sex hormones (7,21,24,31). In addition the extent of exposure to stressors, as well as the psychological makeup of the individual, can also underlie such variability. Testosterone secretion is affected by stress (19,21). Finally, stress-stimulated cortisol secretion has been implicated (21) in the testosterone response to stress (19–21).

The readiness of the elite athlete for physical performance is of paramount importance, despite the increased stress conditions before and during the games. Various ways by which HDL-C levels could be increased by exercise have previously been investigated (6,26,28).

To examine the longitudinal physiological response to lipid metabolism factors under the above conditions and the possibility of finding a simple way to measure such a readiness and resistance or adaptation to stress were the purposes of this pilot study.

It has been found in primates that immediately after the administration of the same stressor a number of the animals exhibit "stress resistance" (expressed in higher testosterone and HDL-C levels) and "stress sensitiveness" (expressed in lower

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testosterone and HDL-C levels) (22,23). Furthermore, the diminished HDL-C levels were associated with elevated basal cortisol concentrations. We decided to investigate the possibility of a similar response in humans by examining selected elite athletes.

Under a specially designed stress model produced by acute exercise, we measured the alteration of total cholesterol (TC), HDL-C, LDL-C, TC/HDL-C ratio, triglycerides (TGs), testosterone (TES), and cortisol (COR) levels as well as the psychological stress load of elite athletes. The article describes changes in these measures during the early poststress period, as well as relevant individual differences. Finally we discuss the possible relations between them.

METHODS

Athletes and Stress Conditions

Twenty-two male elite athletes (age 20 ± 2 years), training for at least 4 years (10 h/week) (sport specialisation: handball [n = 11], volleyball [n = 11]) participated voluntarily in this study. Before they were selected they completed a questionnaire regarding their diet, medication, and smoking habits. The athletes selected did not smoke, drink, or take any medication, and all had similar diets. They all performed the Wingate test, which consists of a 30-s maximal effort trial on a cycle ergometer with a 0.077 kp/kg body weight load, after a 2–3-s initial cycling. The test took place in the morning (10:00 to 12:00 AM) for all athletes, to take into account the same hormonal rhythmic fluctuations. The athletes were told what to do immediately before they performed the test and after they had rested quietly for a while.

Venous blood was drawn (*at resting position*) before the test and immediately after the test and was processed the same day. All athletes had abstained for 12 h from lipid consumption. The study took place at a time when no major games took place.

In order to characterise the source of individual differences in response to a stressor, TES values were measured before and after the Wingate test. In a sample of 22 subjects, 11 showed increases and 11 decreases irrespective of their specialization; these subjects were treated as two separate groups (1 and 2) for statistical purposes. All other parameter values followed this pattern.

Biochemical Methods

Serum was collected, placed in an ice bath (4°C), and centrifuged at 1500 × g. TC was determined enzymatically (2) (Boehringer Mannheim, Germany, Cat. No. 237564), TGs enzymatically (30) (Human, Germany, Cat. No. H5008), and HDL-C by $MgCl_2$-Na phosphotungstate precipitation (14) (Boehringer Mannheim, Cat. No. 543004). TES and COR were assayed by radioimmunoassay (RIA) (using Immunochem kits, Cat. No. 07-289101 and 07-221102, respectively, ICN Biochemical, CA).

Psychophysiological Methods

Two different measurements were obtained using special equipment in order to measure stress before and immediately after the cycloergometer test.

First skin temperature (ST) was measured before and after the Wingate test using a hypersensitive thermal electronic end probe (DIEHL-Thermotron, Germany) attached to the fingertips. The higher the measurement value following the test, the higher the stress load or sensitivity of the individual (25).

Second, skin conductance was measured by using a Psychogalvanic Reflex (PGR) Monitor (Schuhfried, GES, MBH A2340 MODLING, Germany) attached to a register, which applied a steady potential of 0.5 V. Once again we obtained measures before and immediately after the Wingate test. The equipment has a sensitivity of four scales (2–12) with a maximal registration for a conductance change of 0.1 μS (= 0.1 μmho). The higher the values of skin conductance level (SCL), the higher the stress load or sensitivity of the individual (15,18,25).

Statistics

To analyse the data we used the SPSS$^+$ program (1990). Group parameters were compared for differences by using parametric tests, Student's t test, and paired t test. Since the number of subjects was smaller than 20 in each group, nonparametric tests Mann–Whitney and Wilcoxon were performed for a further verification with the same results. Furthermore, to correlate parameters of group 1 and 2 between them we performed correlation tests.

TES values before and after the Wingate test were used to define and classify the two groups of athletes. This should be kept in mind when differences in ratios are compared. However, the high correlation (r) values signify that there is not only this parameter to be taken into account in the differences among the groups.

RESULTS

Somatometric parameters are exposed in Table 1. No significant differences were found between groups for any of these measures.

The Wingate test produced a profound but individual effect on the poststress TES levels of the athletes (irrespective of their specialization): Half of the subjects showed increases, while the other half showed decreases in TES (Table 2). The athletes were treated as two separate groups, based on this particular random differentiation of TES values and for statistical purposes, in order to characterise the source of individual differences in response to a stressor: Group 1 included those individuals with *increased poststress TES levels* and group 2 included those with *decreased levels*. All other parameter values were classified by following this pattern. The reason for this separation was based on Sapolsky's report that "stress resistant" primates exhibit higher TES and lipid levels immedi-

TABLE 1

SOMATOMETRIC PARAMETERS OF ALL GROUPS

Parameters	Group 1 (n = 11)	Group 2 (n = 11)
Age	21.4 ± 2.8	22.2 ± 2.1
Weight (kg)	86.6 ± 4.1	88.6 ± 6.8
Height (cm)	192.9 ± 6.9	189.6 ± 6.9
RBW kg/100 (m − 1)	0.94 ± 0.07	0.99 ± 0.07
BMI (kg/m^2)	23.36 ± 1.60	24.66 ± 1.72

Values are expressed as mean ± SDs. Group 1 = athletes with poststress increased TES levels (volley ball: n = 6; hand ball: n = 5). Group 2 = those with decreased TES poststress levels (volleyball: n = 5; handball: n = 6). The numbers of athletes in the two groups were randomly equal. RBW = relative body weight—that is, BW/[100(H − 1)], where H = height; BMI = body mass index (i.e., BW/H^2).

TABLE 2

SERUM TOTAL CHOLESTEROL (TC), TRIGLYCERIDES (TGs), HIGH (HDL-C) AND LOW (LDL-C) DENSITY LIPOPROTEINS, TESTOSTERONE (TES), CORTISOL (COR), SKIN TEMPERATURE (ST), AND SKIN CONDUCTANCE LEVEL (SCL)

	Group 1 (n = 11)		Group 2 (n = 11)	
Parameters	Rest	Poststress	Rest	Poststress
TC (mg/dl)	163.31* ±22.44	184.99 ±30.23†	183.64 ±23.24	195.17 ±28.06
TG (mg/dl)	77.27 ±16.11	80.10 ±16.84	77.61 ±20.12	84.76 ±25.57
HDL-C (mg/dl)	50.16 ±10.94	52.61 ±9.17	47.68 ±11.56	50.58 ±15.90
LDL-C (mg/dl)	70.99 ±15.16	80.48 ±18.83‡	81.04 ±13.78	81.74 ±20.49
TES (μg/dl)	0.745 ±0.067	0.865 ±0.108†§	0.723 ±0.185	0.537 ±0.160†
COR (μg/dl)	21.23 ±4.13	12.81 ±5.04¶	20.73 ±3.84	11.85 ±2.16¶
ST (°C)	31.69 ±3.61	32.26 ±2.63	31.25 ±3.21	33.15 ±1.97‡
SCL (μS)	0.305 ±0.140	0.287 ±0.132	0.309 ±0.143	0.248 ±0.089

Values are means ± SDs. Group 1 and group 2 athletes at rest and following the Wingate test (poststress). *$p < 0.05$, between-group comparison for the same condition (Student's t test). †$p < 0.01$, ‡$p < 0.05$, within-group comparison with rest (paired t test). §$p < 0.001$, between-group comparison for the same condition (Student's t test). ¶$p < 0.001$, within-group comparison with rest (paired t test).

TABLE 3

RATIOS OF THE PARAMETERS AND THEIR STATISTICAL SIGNIFICANT DIFFERENCES BEFORE AND AFTER STRESS

	Group 1 (n = 11)		Group 2 (n = 11)	
Parameters	Rest	Poststress	Rest	Poststress
ST/SCL (°C/μS)	127.19 ±65.13	135.54 ±65.53	118.27 ±43.88	150.37 ±53.86
TC/HDL	3.43 ±0.44*	3.72 ±0.64†	3.99 ±0.72	4.11 ±0.99
TES/COR ($\times 10^3$)	36.20 ±9.46	74.07 ±21.19‡§	35.39 ±8.98	46.85 ±18.23†
HDL/TES ($\times 10^{-3}$)	68.61 ±20.13	61.92 ±15.34*¶	73.59 ±25.95	97.38 ±42.88§
TC/TES ($\times 10^{-3}$)	220.57 ±31.57*	214.74 ±34.43#	281.43 ±80.54	375.18 ±14.73§
TC/COR ($\times 10^{-3}$)	8.26 ±2.06	17.09 ±5.24¶	9.40 ±2.02	17.54 ±5.31¶
HDL/COR ($\times 10^{-3}$)	2.54 ±0.67	4.88 ±1.48§	2.44 ±0.74	4.48 ±1.68§

Groups were separated as in Table 1. ST = skin temperature, SCL = skin conductance level, TC = serum total cholesterol, HDL = high density lipoprotein, TES = testosterone, COR = cortisol. *$p < 0.05$, between-group comparison for the same condition (Student's t test). †$p < 0.05$, within-group comparison with rest (paired t test). ‡$p < 0.01$, between-group comparison for the same condition (Student's t test). §$p < 0.001$, ¶$p < 0.01$, within-group comparison with rest (paired t test). #$p < 0.001$, between-group comparison for the same condition (Student's t test).

ately after stress, while "stress sensitive" exhibit lower ones (22). We desired to examine if an analogous event takes place in humans and whether such a differentiation is associated with lipid levels in elite athletes.

Displayed in Table 2 are the statistical differences in the two groups (see Table 1) of the lipid and hormonal parameters before and after stress. In group 1 it was found that after stress TC and LDL-C increased significantly, while HDL-C and TGs did not show significant change. TES values increased significantly, while COR decreased. ST and SCL remained unchanged.

In group 2 we found that TES and COR levels decreased significantly; ST increased. All other main parameters remained unchanged.

Table 3 shows the statistical differences in the two groups of various ratios between parameters that displayed high statistically significant correlation coefficients (see Table 4) and that we considered would be of importance in our study. The ratio TC/HDL is the well-known coronary risk factor ratio of the Framingham study, while the ratio TES/COR is extensively used lately in exercise biochemistry for evaluation of performance. Furthermore, one of the ratios might prove to be of diagnostic value. So in light of the above, we present these ratios.

In group 1 the ST/SCL ratio remained unchanged after stress. TC/HDL increased significantly in the poststress period, as did also TES/COR, HDL/TES, TC/COR, and HDL/COR.

In group 2 ST/SCL and TC/HDL remained unchanged after stress; TES/COR, HDL/TES, TC/TES, TC/COR, and HDL/COR increased significantly.

In the same two tables there are displayed the statistically significant differences between groups 1 and 2 at rest and (Student's *t*-test differences). TC at rest and TC/HDL at rest were significantly lower in group 1. Also, TC/TES rest was significantly lower. Poststress TES and also the ratio TES/COR were significantly higher.

The poststress ratios HDL/TES and TC/TES were significantly lower.

Table 4 presents the statistically significant correlations found between different parameters in the two groups at rest and after stress.

In group 1 at rest it appears there is a negative significant correlation of ST to TGs, while a positive one exists between TC/COR to TES and HDL/COR to LDL.

In group 2 both psychophysiological parameters were significantly correlated to TGs and cholesterol (TC or LDL). Also, both TES and COR were significantly correlated to TC and HDL.

After stress, however, there appears to develop a differentiation between the two groups of athletes.

In group 1 the ratio TES/COR to HDL and TES/COR to LDL are both negatively correlated, as well as the ratio TC/COR to TES. COR to TES is positively correlated.

It should be noted that only in group 2 does there appear to be a significant correlation between the psychophysiological variable (ST) and hormonal or lipid parameters: In fact all the correlations (save COR to TC and HDL) include this factor, while in group 1 the only correlation appears to be between TES, COR, and TC or lipoproteins with an increase of 105% in the ratio of TES/COR.

Table 5 was drawn after the total statistical evaluation of the results and is based on the differential profile of percentage differences of the statistical values, found among groups after stress, between TES and lipid levels. As is seen (except with TES/TC), all lipid parameters and ratios in group 1 increase significantly in the poststress period, especially the TES/COR ratio, while all the equivalent ones in group 2 decrease and the lipid ones remain unchanged (except the minor increase of the TES/COR ratio).

DISCUSSION

Acute physical exercise can be conceived of as a gross physical stress, resulting in marked psychophysiological and biochemical changes in order to maintain the internal milieu (8,15,18,25).

TABLE 4

CORRELATION COEFFICIENTS (*r*) BETWEEN THE DIFFERENT PARAMETERS OF THE TWO GROUPS AT REST AND AFTER STRESS

Group 1 (n = 11)		Group 2 (n = 11)	
STr : TGr	−0.58*	STr : LDLr	0.60*
[TCr/CORr] : TESr	0.58*	STr : [TCr : CORr]	0.54*
		[STr/SCLr] : TGr	0.53*
[HDLr/CORr] : LDLr	0.76†	SCLr : TGr	−0.66*
		[HDLr/CORr] : TCr	0.78*
		[HDLr/TESr] : TCr	0.73*
TESst : CORst	0.60*	STst : LDLst	0.67*
[TESst/CORst] : HDLst	−0.62*	STst : [TCst/HDLst]	−0.58*
[TESst/CORst] : LDLst	−0.62*	STst : TESst	−0.60*
[TCst/CORst] : TESst	−0.56*	STst : [HDLst/TESst]	0.70†
[LDLst/CORst] : TESst	−0.61*	STst : [TCst/TESst]	0.55*
		[HDLst/CORst] : TCst	0.64*

ST = skin temperature, TG = triglycerides, LDL = low density lipoprotein, TC = serum total cholesterol, COR = cortisol, TES = testosterone, SCL = skin conductance level, HDL = high density lipoprotein, r = at rest, st = poststress (i.e., at maximal effort). *$p < 0.05$. †$p < 0.01$ within group comparison with test (paired *t*-test). Groups 1 and 2 were separated as in Table 1.

TABLE 5

COMPARISON (%) OF REST TO POSTSTRESS MEAN VALUES WITH SIGNIFICANT DIFFERENCES (Tables 2 and 3) OF LIPIDS, TESTOSTERONE (TES), AND RATIOS OF THE TWO GROUPS

	Group 1 (n = 11)	Group 2 (n = 11)
TC	↑ + 13.3%	NS
LDL-C	↑ + 13.3%	NS
TC/HDL-C	↑ + 8.5%	NS
TES	↑ + 16.1%	↓ −25.8%
TES/TC*	NS	↓ −22.8%
TES/HDL*	↑ + 9.7%	↓ −21.6%
TES/COR	↑ +104.6%	↑ +32.4%
ST	NS	↑ + 6.1%

TC = serum total cholesterol, LDL-C = low density lipoprotein, HDL-C = high density lipoprotein, COR = cortisol, ST = skin temperature. COR parameters are not included here because they do not differ significantly between the groups (group 1 = −39.7%, group 2 = −42.8%). Groups 1 and 2 were separated as in Table 1. *These ratios are the reciprocals from those in Table 2.

We and other investigators have observed lower HDL-C levels in animals exposed to various stressors (16,23,27,29). Also, epidemiological data in humans support the finding that stress can change lipid levels (3).

The present study demonstrates that highly trained, elite athletes exposed to an identical stressor display a wide range of TES responses. When divided into two groups based on the direction of these TES responses, differences were found between other parameters, such as TC and LDL-C levels and the ratios TES/HDL-C, TC/HDL-C, and TES/COR (Table 5). This differentiation cannot be the result of age, body weight, training, or diet, since all athletes had similar ages and body weights (Table 1) and had analogous diets and training conditions. They also underwent the same test, so they received the same stress conditions; their COR levels fell equally in both groups, suggesting an analogous general stress response in the total of athletes.

It also cannot be attributed to hemoconcentration, since it would be similar for all parameters and positive for all athletes. COR levels cannot be held responsible for these discrepancies, since they are the same in both groups. The rise of TES levels in group 1 also cannot be explained by decreased sensitivity to TES-inhibiting effects of COR, since if this was the only mechanism it would merely slow their decline, not lead to elevation. So the rise itself probably results from the stress-induced release by the sympathetic system of catecholamines which affect blood flow, since acute physical effort provokes the excitation of the sympatho-adrenomedullary axis (12). Other researchers have reported increased TES concentrations after acute exercise of short duration (13).

Pharmacological inhibition of catecholamine release completely abolished the transient rise in TES concentrations in stress-resistant primates (22). It has also been reported in the rat that catecholamines might transiently increase TES concentrations in stress-resistant males by acting on testicular vasculature to increase blood flow, which may lead to increasing concentrations of TC—as our present results also show—of glucose or increase the efficiency of removal of newly synthesised TES (5).

The correlation between TES and lipids in group 1 suggests that there may be a differentiation between the groups, as far as the effectiveness of response to stress is concerned. The increasing TC and lipoprotein levels cannot be explained by the lipolytic effect of COR, since in both groups these levels declined after stress. Also, it has been reported that acute manipulation of glucocorticoid concentrations failed to alter these variables (23). However, it might be explained by an increased secretion rate and a concomitant decline of liver elimination and turnover of these variables (13).

TES synthesis has as a precursor cholesterol. It would seem that a mobilisation of TES synthesis is probably followed by an analogous one of cholesterol and lipoprotein metabolism in order to satisfy the demand. Individuals having this "quick response" ability may be able to cope better under maximal effort stress conditions, since it is known that TES mobilises in its turn the rate of energy supply to muscle cells under heavy energy need.

Cholesterol and lipoprotein metabolism in association with TES secretion seems to be more readily mobilised under maximal effort stress conditions in individuals more adaptable to it and is also reflected in the TES/COR ratio increase of 105% only in group 1 (Table 5). Equally, the correlation of psychophysiological parameters (ST) to lipids present only in group 2 after stress may further support this suggestion.

However, it has been reported (11) in humans and in primates "resistant" to stress (21) that COR levels increased after stress, but that they fell immediately after due to accurate feedback from blood to brain as soon as the threshold level was reached. The decline in poststress COR by both groups of our athletes may indicate an analogous response to stress of the hypothalamus–pituitary adrenocortical axis.

What is the extent of the effectiveness of the mechanism leading to a successful stress response and how can it be interpreted, in terms of physical performance, by using a simple measurement or test? From the above results we propose that after an acute physical exercise test, measuring TES, COR, TC, and HDL-C levels and their ratios immediately after may give a picture of the ability of an organism to "respond quickly" to stress.

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