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Enzymatic and hormonal responses following a 24 h endurance run and a 10 h triathlon race

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Summary. Muscle cell leakage and hormonal changes were compared immediately after and during the 3 days following a 24 h endurance run (R_{24h}) in 8 subjects, and a 10 h triathlon non-competitive race (T_{10h}) in 6 subjects. The study showed three main differences: 1) plasma enzyme increases were considerably more significant in R_{24h} than in T_{10h}: compared with resting levels, creatine kinase increased × 120 after R_{24h} but only × 2 after T_{10h}; lactic dehydrogenase × 4, as opposed to × 1.5; and transaminases only showed an increase after R_{24h}. The plasma myoglobin increase after R_{24h} was double that found after T_{10h}; 2) for the same magnitude of plasma aldosterone and cortisol after R_{24h} and T_{10h} (3 times the resting levels), a highly significant decrease in urinary Na^+ ($p < 0.001$) and an increase in urinary K^+ ($p < 0.01$) were found only after R_{24h}; and 3) the plasma free noradrenaline level increased significantly after R_{24h} (× 2.6) whereas it was unchanged after T_{10h}. In contrast, the plasma level of conjugated dopamine increased only after T_{10h} (× 3.7, $p < 0.05$). These results suggest that long-distance running causes more muscular lesions than the triathlon, and that important factors other than aldosterone are probably involved in the regulation of urinary electrolyte excretions during T_{10h}.

Key words: Urinary electrolyte excretions — Aldosterone — Catecholamines

Introduction

The influence of strenuous athletic activity on biochemical and hormonal changes is of great interest and has been studied extensively, especially in marathon runners. However, the responses to ultra-long distance running and triathlon competitions, which are now enjoying a tremendous increase in popularity, have been less well researched. All previous studies have reported that there is always considerable muscle cell leakage after long-lasting exercise, and that it persists for 24–72 h after the exercise. Serum creatine kinase, lactic dehydrogenase and transaminase activities and serum myoglobin concentration increase dramatically after ultra-marathons (Guezennec et al. 1986; Kielblock et al. 1979; Noakes et al. 1983), after a 120 km march (Galun et al. 1984) and after an 89 km cross-country ski race (Roxin et al. 1986). The same serum evolution has been observed after triathlon competitions (Guezennec et al. 1986; Holly et al. 1986; Thomas and Mothley 1984), and after strenous training programmes (Demos et al. 1974; Kosano et al. 1986). Little work has been done on the hormonal responses to long-term exercise. Keul et al. (1981) observed after a 100 km run that plasma insulin was unaffected whereas growth hormone, aldosterone and cortisol concentrations were increased. The rises in plasma renin activity, angiotensin II and aldosterone were associated with a decrease in the urinary sodium excretion rate during a 20 day road race (Wade et al. 1985) and during prolonged physical training (Lijnen et al. 1985; Opstad et al. 1985). The return to base levels took 2–3 days after the exercise.

The purpose of this report was to compare the main changes observed after a 24 h endurance run and after a 10 h triathlon non-competitive race. Particular attention was paid to 1) the magnitude of plasma serum enzyme changes; 2) the effects of aldosterone response on renal electrolyte handling; and 3) the different patterns of plasma catecholamine responses.

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Table 1. Anthropometric and bioenergetic characteristics of the 7 men and 1 woman participating in the 24 h endurance run. Body weight loss during the race and individual performance

Subjects		Age, years	Height, cm	$\dot{V}_{O_{2max}}$, ml STPD. $min^{-1} \cdot kg^{-1}$	Body weight, kg	Body weight loss, kg	Running distance, km
	R-A	43	180	44.12	69.6	1.2	179.75
M	JC-B	42	172	57.00	69.0	3.6	164.40
E	B-C	35	179	47.96	71.0	4.4	183.64
N	E-G	54	165	39.00	63.4	1.2	132.13
	A-H	40	180	48.33	73.2	2.6	156.30
	A-R	36	164	55.83	60.8	2.8	187.75
	D-T	30	178	47.06	70.8	0	163.60
W O M A N	M-G	39	161	37.29	52.6	2.8	147.95

Materials and methods

24 h endurance run (R_{24h})

Seven men and one woman volunteered for the study after being informed of the purpose and methods of the procedure. They were healthy and had no known abnormalities. They had trained regularly, with an estimated weekly average of 80 km or 10 h. Their anthropometric and bioenergetic characteristics are presented in Table 1.

The race, which started at 1000 h and lasted for 24 h, was run on a synthetic 400 m track. The volunteers could not sleep but were allowed to walk or stop for a few minutes when they felt tired. Food and fluid intakes were selected by the runners themselves without control or restriction. The environmental temperature and relative humidity were $4.1 \pm 1.4°C$ and $69 \pm 21\%$ respectively (means $\pm$SD). The run was completed by all 8 subjects, who were weighed before and after the race. 40–50 ml of blood was sampled in the supine position from a cubital vein one week before the run (control values), immediately after the run (D0) and on the following morning (D1). A urine sample was collected just before (control values) and after the race (D0), and on the following morning (D1).

10 h triathlon race (T_{10h})

Six healthy male volunteers participated in a non-competitive triathlon race. All were in regular training (15–20 h per week). One subject (JC-B) took part in both studies. Their general anthropometric and bioenergetic characteristics are shown in Table 2. Their level of physical fitness as evaluated by maximal oxygen uptake ($\dot{V}_{O_{2max}}$) (64 ± 3 ml $\cdot$ $min^{-1} \cdot kg^{-1}$) was higher than that of the R_{24h} runners (47 ± 2 ml $\cdot$ $min^{-1} \cdot kg^{-1}$).

The non-stop race was in three stages: 1.25 h swimming in a swimming pool, 5.5 h cycling in the flat surroundings of Clermont-Ferrand, and 2.5 h running around a track. The athletes were allowed to eat and drink during the race. Weather conditions were mild and relatively constant: mean air ambient temperature 18° C, relative humidity 70% and water temperature 20° C, but the wind was strong (40 km $\cdot$ h^{-1}). Very light exercise was performed by the subjects on the days following the race. 40–50 ml of blood was drawn in the supine position from a cubital vein one week before (control values), immediately after the race (D0) and the following 3 mornings (D1, D2, and D3). All blood samples were taken between 800 and 1000 hours except for D0, which was taken at 1900 h at the end of the race. Body weight was measured before and at the end of the race.

Twenty-four hour urine collections were also made one week before (control values) and during the 3 days following the end of the race (D1, D2 and D3).

Bioenergetic measures

Maximal oxygen uptake ($\dot{V}_{O_{2max}}$) was measured by the direct triangular method on a cycle ergometer. Heart rate was obtained by continuous electrocardiogram recording.

Table 2. Anthropometric and bioenergetic data of the 6 subjects participating in the non-competitive triathlon race. Individual swimming and running distances covered during the race and changes in body weight

Subjects	Age, years	Height, cm	$\dot{V}_{O_{2max}}$, ml $\cdot$ STPD $\cdot$ $min^{-1} \cdot kg^{-1}$	Body weight, kg	Body weight loss, kg	Swimming distance, km	Running distance, km
JM-A	21	186	60.28	83.8	2.4	3.1	30.0
JC-B	42	172	57.00	68.8	2.8	3.0	32.0
C-F	35	173	62.86	63.2	2.4	3.4	31.0
P-P	25	156	64.52	50.8	1.4	4.2	29.0
M-P	23	184	78.46	74.2	2.4	4.3	32.5
P-R	24	183	61.87	72.3	1.9	4.7	30.8

Table 3. Haematological variables, serum proteins and plasma myoglobin before (control), immediately (D_0) after the race, and during the following 3 days (D_1, D_2, D_3) for the endurance run (R_{24h}) and the triathlon race (T_{10h})

		R B C, $10^{12} \cdot l^{-1}$	Haematocrit, %	M C V, μ^3	Hemoglobin, $g \cdot 100\,ml^{-1}$	Proteins, $g \cdot l^{-1}$	Myoglobin, $ng \cdot ml^{-1}$
	Control	4.77 ± 0.1	41.9 ± 1.4	88 ± 1	14.4 ± 0.4	74.5 ± 1.6	75 ± 12
		4.16 – 5.15	33.2 – 45.7	80 – 91	11.7 – 15.6	68 – 81	37 – 116
R_{24h}	D_0	4.75 ± 0.19	44.4 ± 1.7*	93 ± 1**	14.7 ± 0.5	76.5 ± 2.3	701 ± 57***
		3.83 – 5.37	36.8 – 50.4	88 – 96	12.3 – 16.3	68 – 89	456 – 906
$n = 8$	D_1	4.49 ± 0.13	40.5 ± 1.6	90 ± 2	13.6 ± 0.5*	73.0 ± 1.7	369 ± 53***
		3.90 – 5.00	36.0 – 45.9	86 – 98	11.6 – 15.1	64 – 79	66 – 544
	Control	5.07 ± 0.16	45.7 ± 0.8	91 ± 1	15.3 ± 0.3	73.2 ± 3.4	54 ± 12
		4.59 – 5.51	43.4 – 48.3	86 – 95	14.4 – 16.3	65 – 74	32 – 107
	D_0	5.25 ± 0.12	47.7 ± 0.5	91 ± 2	15.7 ± 0.2	75.8 ± 2.0	328 ± 51^{+++}
T_{10h}		4.87 – 5.71	45.5 – 49.3	85 – 96	15.2 – 16.1	69 – 83	205 – 551
	D_1	5.15 ± 0.11	44.3 ± 0.3	86 ± 2^{++}	15.0 ± 0.1	70.7 ± 1.8	166 ± 36^{++}
		4.86 – 5.64	43.4 – 45.0	80 – 92	14.8 – 15.5	66 – 78	81 – 326
	D_2	5.11 ± 0.13	44.0 ± 0.7	86 ± 1^{++}	15.0 ± 0.1	68.5 ± 1.5	52 ± 1.3
$n = 6$		4.65 – 5.57	41.7 – 45.6	82 – 90	14.5 – 15.3	62 – 73	22 – 113
	D_3	5.00 ± 0.14	43.7 ± 0.5^{+}	88 ± 2^{++}	14.3 ± 0.2^{+}	70.2 ± 1.2	44 ± 9
		4.55 – 5.36	42.1 – 45.4	82 – 93	13.9 – 15.1	67 – 74	12 – 80

n, N° of subjects. RBC, red blood cells; MVC, mean cell volume. Values are means ± SE with ranges. Comparisons with their respective control values by paired *t* test are, for R_{24h}: *, $p < 0.05$; **, $p < 0.01$; ***, $p < 0.001$, and for T_{10h}: $^{+}$, $p < 0.05$; $^{++}$, $p < 0.01$; $^{+++}$, $p < 0.001$

Chemical analysis

For both studies, blood samples were either stored at +4° C for subsequent analysis, or plasma and serum were separated by centrifugation and stored frozen at −20° C until analyzed. Plasma aldosterone, cortisol and myoglobin (MB) concentrations were determined by radio-immunoassay using "ORIS INDUSTRIE" kits. Plasma-free adrenaline (A), noradrenaline (NA) and plasma conjugated dopamine (D) were assayed by the radio-enzymatic method described by Claustre et al. (1983) for the endurance run and by a high pressure chromatography technique for the triathlon race (Cottet-Emard J. M. to be published): correlations between the two methods were highly significant for all catecholamines (Allenmark et al. 1980; Goldstein et al. 1981).

Serum and urinary sodium and potassium were measured by flame photometer, proteins by the Biuret reaction, and creatinine by the colorimetric method of Jaffé (Lepage and Galimany 1985).

All enzymatic activities were measured at 30° C: creatine kinase (CK) (EC 2.7.3.2.) with commercial reagent from Boehringer (Mannheim WG, FRG) (Szasz et al. 1976); aspartate (EC 2.6.1.1.) and alanine (EC 2.6.1.2.) aminotransferase with commercial reagents supplied by I.F.C.C. to Biomérieux (France); and lactic dehydrogenase (LDH) (EC 1.1.1.2.27.) according to the method of Wroblewski and La Due (1955).

Statistics

The Student paired *t* test was used to compare individual values within a study, and the unpaired *t* test to compare mean values between the two studies. Statistical significance was accepted at $p < 0.05$.

Results

Values are given as means ± SE

Performance. Twenty subjects took part in R_{24h}. The winner ran 219.95 km and the last subject ran 115.73 km. The individual performances of our subjects are given in Table 1. Their mean body weight loss during the run was 2.3 ± 0.5 kg (i.e. 3.6 ± 0.8% of body weight).

— In T_{10h} the distances covered by the subjects in the swimming and running trials varied according to individual fitness (Table 2). The six participants all cycled 140 km. The mean body weight loss during the race was 2.2 ± 0.2 kg (i.e. 3.2 ± 0.2% body weight) (Table 2).

Haematological and plasma variables. The significant increase in haematocrit level observed immediately after R_{24h} was probably due in part to the significant increase in mean red blood cell volume. After T_{10h} the packed cell volume remained unchanged. While the increase in serum protein concentrations was not significant in either of the studies, it was not absolutely necessary to correct the plasma hormone changes found after both races (Table 3).

— A large increase in plasma myoglobin was seen immediately after R_{24h}, from 75 ± 12 to 701 ± 57 $ng \cdot ml^{-1}$, and remained elevated on D1. The same changes were observed after T_{10h} but the response was about half the size (on D1: 328 ± 51 $ng \cdot ml^{-1}$) (Table 3).

A significant increase in plasma creatinine (above the upper limit of normal established by the laboratory technique: 120 $\mu mol \cdot l^{-1}$) was observed only after T_{10h} on D0 and D1 (Table 4).

Table 4. Creatinine in plasma and urine before and after the endurance run (R_{24h}) and the triathlon race (T_{10h}) (for details, see Table 3)

		Creatinine		
		Plasma $\mu mol \cdot l^{-1}$	Urine	
			$mmol \cdot l^{-1}$	mmol/24 h
R_{24h}	Control	103.4± 7.0	7.4±1.6	—
	D_0	111.8± 5.5	12.0±1.3***	—
	D_1	112.5± 4.5*	7.0±1.9***	—
T_{10h}	Control	106.5± 5.5	8.5±2.7	16.6±1.1
	D_0	146.7±10.8+	—	—
	D_1	125.5± 4.3+	10.7±1.1	17.4±0.7
	D_2	111.7± 4.9	7.8±1.8	15.4±1.1
	D_3	116.2± 6.2	8.0±1.7	16.0±0.9

Values are means ±SE. Comparisons with their respective control values by paired t test are, for R_{24h}: *, $p<0.05$; ***, $p<0.001$, and for T_{10h}: +, $p<0.05$

The urinary excretion rate of creatinine was normal and remained statistically unchanged after T_{10h}. The excretion rates for R_{24h} could not be calculated because 24 h urine collections were not obtained (Table 4).

Serum and urinary Na^+ and K^+ levels. In serum (Fig. 1a). There was a highly significant decrease in Na^+ from 140.9±0.6 to 136.5±0.9 $mmol \cdot l^{-1}$ ($p<0.005$) immediately after R_{24h}. The K^+ level also decreased, but the change was not statistically significant.

In contrast no significant change was observed in the Na^+ and K^+ concentrations in the T_{10h} group.

In urine (Fig. 1b). A dramatic decrease in Na^+ and an increase in K^+ were found in all the R_{24h} subjects. Maximal responses were observed on D1 for Na^+ (from 107±21 to 7±2 $mmol \cdot l^{-1}$, $p<0.001$), and immediately after the race for K^+ (from 39±10 to 92±17 $mmol \cdot l^{-1}$, $p<0.01$). These responses were notably similar in the 8 subjects.

In contrast, the mean Na^+ and K^+ concentrations and the excretion rates were not statistically altered after T_{10h}. However, as shown in Table 5,

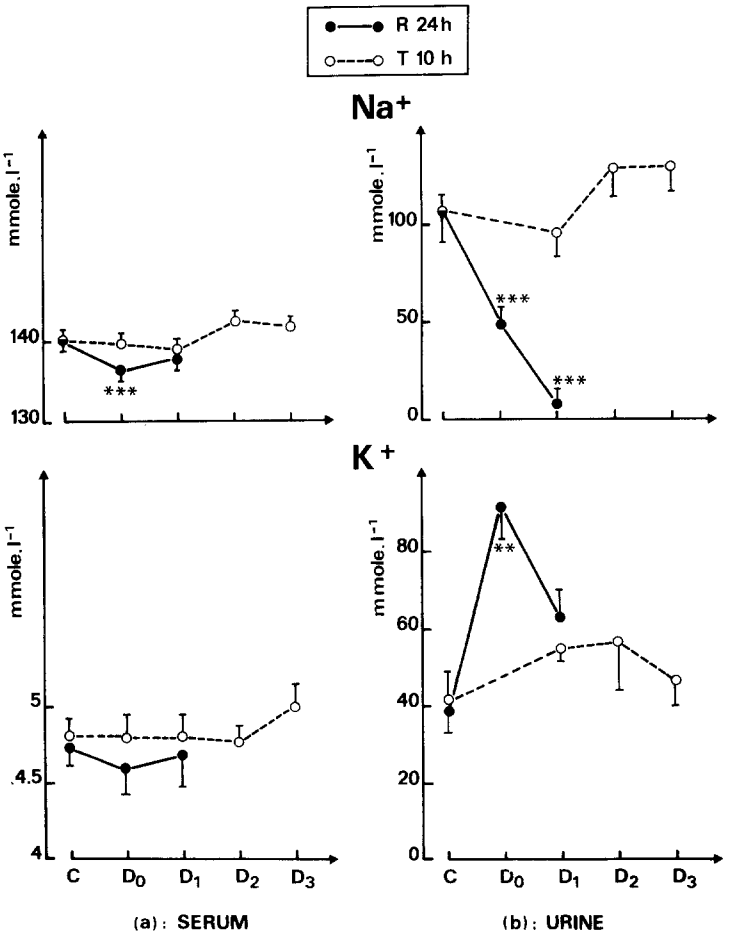

Fig. 1. Serum **a** and urinary **b** sodium and potassium concentrations before (C), immediately after (D_0) and the following 3 days (D_1, D_2, D_3) for the 24 h endurance run (●—●, R_{24h}) and the 10 h triathlon (O----O, T_{10h}). Values are means ±SE. For R_{24h} comparisons with control values by paired t test are ***, $p<0.001$. For T_{10h} no changes are significant

Table 5. Individual responses in urine flow rates and Na^+ and K^+ excretion rates before and after the triathlon race (for details, see Table 3)

Subjects	V, l/24 h					Na^+, mmol/24 h					K^+, mmol/24 h				
	Control	D_0	D_1	D_2	D_3	Control	D_0	D_1	D_2	D_3	Control	D_0	D_1	D_2	D_3
JM-A	1.50	—	2.10	3.00	3.50	197	—	248	486	487	72	—	118	132	119
JC-B	3.75	—	1.35	4.40	2.00	383	—	188	625	350	113	-	66	136	76
C-F	3.95	—	1.50	1.60	1.90	375	—	107	170	186	146	—	104	122	106
P-P	3.20	—	2.50	2.20	2.60	163	—	150	169	237	51	-	105	55	88
M-P	2.85	—	1.40	2.20	3.10	413	—	136	293	394	117	—	78	119	136
P-R	0.93	—	1.45	1.10	1.00	118	—	131	174	150	69	—	84	120	75
$\bar{m}$	2.70	—	1.72	2.42	2.35	275	—	160	319	301	95	—	92	114	100
±SE	0.50	—	0.19	0.47	0.37	53	—	21	79	53	15	—	8	12	10

V, urine flow rate. Comparisons with their respective control values by paired *t* test are no significant

individual responses varied. Three subjects exhibited a decrease in Na^+ excretion rates and one subject a small increase. Likewise three subjects showed a small decrease in K^+ level on D1 while the others showed a slight K^+ increase. On D1, urine flow rates decreased in four men and increased in two. However, the paired *t* test showed that the mean decrease was not significant (Table 5).

Plasma serum enzymatic activities (Table 6). The CK level on completion of R_{24h} was considerably elevated. Of the 8 runners, 4 exhibited CK levels, measured in international units (UI), higher than 10000 UI · l^{-1}. The runners A–R and B–C, who ran over the longest distances, had the highest CK levels. On D1, CK levels remained very high.

Of the 6 athletes participating in T_{10h}, 4 had resting levels of CK higher than the normal limit established by the laboratory technique used (upper limit of normal: 109 UI · l^{-1}). Total CK was significantly elevated in all subjects, but this increase (×2) was much smaller than that for R_{24h} (×120). The CK level still remained elevated on D1 and was at normal level on D3. All CK-MB values remained unchanged after R_{24h} and T_{10h}.

On completion of R_{24h} and T_{10h}, total LDH values exceeded the upper limit of normal (240 UI · l^{-1}) (Table 6). However, the mean level was 707 UI · l^{-1}, nearly ×4 the resting level for R_{24h}

Table 6. Plasma total creatine-kinase (CK), glutamic oxalo-acetic (GOT) and glutamic pyruvic (GPT) transaminases, and lactic-dehydrogenase (LDH) before and after the endurance run (R_{24h}) and the triathlon race (T_{10h}) (for details, see Table 3)

		CK, UI · l^{-1}	LDH, UI · l^{-1}	GOT, UI · l^{-1}	GPT, UI · l^{-1}
	Control	85± 31	162± 5	19± 3	18± 3
		50– 149	137– 178	11– 39	13– 41
R_{24h}	D_0	10347± 3012***	707± 132**	282± 69***	70± 17*
		2187–27600	367–1439	72–641	23–160
$n=8$	D_1	3208± 947***	448± 77**	159± 36**	62± 15*
		719– 8780	235– 871	48–349	21–143
	Control	148± 48	181± 7	21± 4	21± 3
		57– 377	150– 197	12– 39	13– 37
T_{10h}	D_0	291± 55^{+++}	264± 13^{+++}	25± 3	21± 2
		179– 531	231– 316	82–201	15– 28
	D_1	257± 62^{++}	186± 8	25± 4	21± 2
		151– 545	164– 215	21–111	13– 28
$n=6$	D_2	163± 37	159± 9^{+}	23± 2	20± 1
		87– 309	139– 195	18– 35	15– 25
	D_3	104± 23	172± 5	21± 2	17± 2
		59– 195	163– 195	15– 29	10– 24

n, N° of subjects. Values are means ±SE with ranges. Comparisons with their respective control values by paired *t* test are, for R_{24h}: *. $p<0.05$; **, $p<0.01$; ***, $p<0.001$, and for T_{10h}: $^{+}$, $p<0.05$; $^{++}$, $p<0.01$; $^{+++}$, $p<0.001$

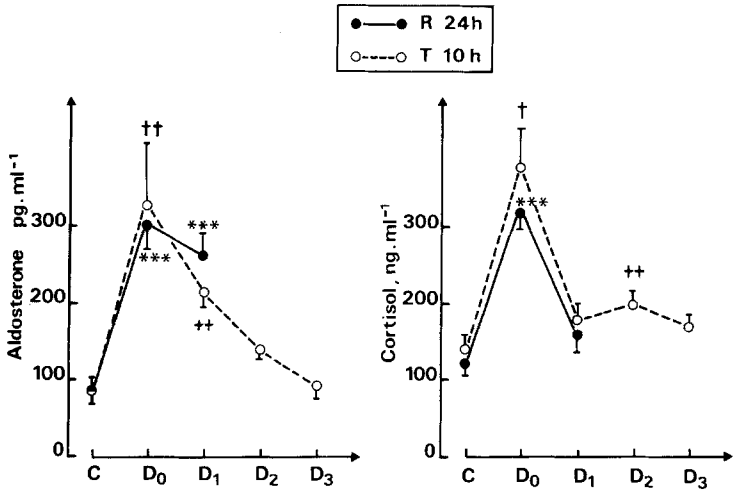

Fig. 2. Plasma aldosterone and cortisol before (C), immediately after (D_0) and during the following 3 days (D_1, D_2, D_3) for the 24 h endurance run (●—●, R_{24h}) and the 10 h triathlon (○—○, T_{10h}). Values are means ±SE. Comparisons with their respective control values by paired *t* test are, for R_{24h}: ***, $p<0.001$, and for T_{10h}: $^{+}$, $p<0.05$; $^{++}$, $p<0.01$. Comparisons of all R_{24h} and T_{10h} values by unpaired *t* test are not significant

since the mean elevation was only ×1.5 the resting level for T_{10h}. On D1 the mean LDH level was still elevated in the R_{24h} runners, while all values had returned to normal in the T_{10h} subjects. These changes in LDH activity were significantly due, in both studies, to a decrease in the H4 fraction (expressed in relative terms) from 29±1 to 20±1% and an increase in the M4 fraction from 6±1 to 17±1% on D0. There was a ×4 increase in glutamic oxalo-acetic transaminase (GOT) and ×15 in glutamic-pyruvic transaminase (GPT) activities in the R_{24h} group after the race (Table 6). In contrast, for T_{10h}, GOT and GPT levels were unchanged. On D1, GOT and GPT levels were unchanged. On D1, GOT and GPT levels in R_{24h} runners remained significantly elevated.

Hormonal responses. Immediately after both races, the mean aldosterone level increased significantly and the magnitude of the response (×3) was the same in R_{24h} as in T_{10h} (Fig. 2). The next morning, the level decreased but still remained higher than those of controls in both groups. The basal level was reached only on the third day after T_{10h}.

The increase in cortisol was the same in R_{24h} as in T_{10h} (Fig. 2). However in contrast to aldosterone responses, the levels were normal within 24 h of recovery. In T_{10h}, a slight peak was observed on D3.

The pattern of plasma NA and D responses differed between R_{24h} and T_{10h} (Table 7). The mean free NA level increased ×2.6 immediately after R_{24h}, while there was no change after T_{10h}. In contrast D increased after T_{10h} (×3.7) but remained unchanged after R_{24h}. Free A levels were not modified by either race (Table 7).

Circadian variations in hormonal levels could not explain the magnitude of responses observed. Peaks in cortisol and aldosterone occur early in

Table 7. Plasma conjugated dopamine, and free noradrenaline and adrenaline before and after the endurance run (R_{24h}) and the triathlon race (T_{10h}) (for details, see Table 3)

		Dopamine, nmol·l^{-1}	Noradrenaline, nmol·l^{-1}	Adrenaline, nmol·l^{-1}
R_{24h}	Control	34.95± 1.24	2.50±0.30	1.35±0.34
	D_0	30.82± 2.28	6.62±0.76***	1.22±0.35
	D_1	32.82± 1.77	6.25±0.45***	1.46±0.37
T_{10h}	Control	20.73± 2.12	2.43±0.21	0.71±0.08
	D_0	76.00±16.68^{+}	3.42±0.44	0.83±0.11
	D_1	35.62±15.37	2.44±0.38	0.45±0.05
	D_2	43.82±15.32	2.23±0.41	0.57±0.03
	D_3	24.36± 5.48	1.86±0.28	0.46±0.08

Values are means ±SE. Comparisons with their respective control values by paired *t* test are, for R_{24h}: ***, $p<0.001$, and for T_{10h}: $^{+}$, $p<0.05$

the morning (at 0500–0600 h) and peaks in noradrenaline, adrenaline and dopamine are nocturnal (at 2300 h) (Kuchel and Buu 1985).

Discussion

Three main differences emerged from comparisons of the physiological responses after the two different types of long-lasting physical exercise: 1) plasma enzymatic increases were considerably more significant in distance runners than in triathlon athletes; 2) for the same plasma aldosterone response, the classical effects of aldosterone on urinary Na^+ and K^+ excretion were blunted in triathlon athletes and 3) the sympathetic activities were different.

The release of muscular proteins (CK, LDH, transaminases, MG) into the circulation after endurance competitions is well known (Galun and Epstein 1984; Kielblock et al. 1979; Kosano et al. 1986; Noakes et al. 1983). These changes are related to skeletal muscular trauma. Most of the findings argue for a correlation between the severity of the trauma and the magnitude of these increases. Mechanical trauma has been suggested because exercise induced changes depend on the type of exercise performed (Hikida et al. 1983). Marching or running, during which the foot strikes the ground, always induced considerable muscle disorders, whereas swimming, even at high levels of intensity or for long periods, did not impose sufficient trauma to produce release of intracellular contents (Symansky et al. 1983).

Our comparison between a run and a triathlon race agrees with this hypothesis, especially when we compared the same subjects JC-B in the two situations. This athlete exhibited a CK level $\times 10$ higher after R_{24h} than after T_{10h} (2187 vs 233 $UI \cdot l^{-1}$). GOT and GPT remained unchanged after T_{10h} while serum levels were $\times 15$ and $\times 4$ higher after R_{24h}. A similar finding has recently been reported. Guezennec et al. (1986) found that, at the end of a 100 km run and triathlon races of the same duration (8–9 h), plasma levels in CK, LDH, and myoglobin were higher after the run. All the results suggest that running caused more muscular lesions than a triathlon in relation to mechanical trauma: the same groups of muscles were stimulated with high muscular tension at each foot impact on the ground throughout the race.

However, evidences against mechanical trauma as a cause have also been reported. Nørregaard Hansen et al. (1982) did not find significant differences in myoglobin and CK levels between runners and rowers. Moreover we found in one windsurfer after an 81 hour endurance race that CK and LDH levels were also elevated (Bedu et al. 1987).

Consequently skeletal muscle injuries depend also on other factors, such as exercise intensity and duration, and the level of physical fitness of the competitors (Haibach and Hosler 1985; Roxin et al. 1986; Thomas and Motley 1984). In accordance with numerous previous reports, we found that the fastest runners (A–R and B–C) in the R_{24h} race exhibited greater increases in enzyme levels than the others (for CK levels: 15480 and 27600 $UI \cdot l^{-1}$ respectively).

Likewise, the greater intensity of the "Iron Triathlon World Race" on Hawaii (which consisted of an 8.84 km ocean swim, 179.2 km on a bicycle and 41.92 km marathon run performed in only 9 h) induced higher GOT, GPT and LDH levels than our triathlon race (Holly et al. 1986).

On the other hand, changes in plasma enzymes are altered by training. Older subjects had higher serum myoglobin than younger ones (Roxin et al. 1986). Nørregaard Hansen et al. (1982) found that non-runners have a higher rise in serum myoglobin than runners and that three months of training reduced the muscle cell leakage. According to these authors, training may reduce muscle cell sarcolemma permeability during running and consequently prevent muscle cell release. Thus, the smaller exercise-induced changes in plasma enzyme after T_{10h} as compared with R_{24h} and previous reports after runs over the same distance were probably due in part to the high level of physical fitness of the T_{10h} competitors (mean $\dot{V}_{O_2max} = 64 \pm 3$ as against 47 ± 2 $ml \cdot min^{-1} \cdot kg^{-1}$) and to their younger age (28, compared with 40 years). Furthermore these differences in age and degree of physical fitness between the R_{24h} and T_{10h} athletes could also explain the higher level of noradrenaline in the R_{24h} runners. Lehman and Keul (1986) found that noradrenaline is increased in older subjects after exercise.

The other striking feature of this investigation was that the highly significant increase in plasma aldosterone ($\times 3$ resting level) observed immediately after T_{10h} and the days following the race was not associated with a significant decrease in Na^+ and increase in K^+ concentration and excretion rates in urine. Although individual responses varied, the magnitude of decrease in Na^+ and of increase in K^+, if they were observed, remained small. In contrast, for the same magnitude of re-

sponses in aldosterone after R_{24h} (Fig. 2), the well-known effects of aldosterone on the tubular cells of the kidneys were found in all 8 participants, and the magnitude of the changes was dramatic. Comparison of the responses in the same subject, JC-B, in the two studies was particularly significant: on D1 after R_{24h}, his aldosterone level was 239 pg · ml^{-1} with a decrease in urinary Na^+ from 191 to 19 mmol · l^{-1}. In contrast, D1 after T_{10h}, his aldosterone level was 291 pg · ml^{-1} whereas urinary Na^+ increased paradoxically from 109 to 139 mmol · l^{-1}. Most reports also describe an increase in plasma cortisol (Dessypris et al. 1980), aldosterone and renin activity during and after physical activity (Lijnen et al. 1985; Wade et al. 1985). Costill et al. (1976) reported that urinary sodium, chloride and water excretions were reduced for up to 46 hours after a single bout of exercise (60 min at 60% of $\dot{V}_{O_2max}$). After a marathon race, Viru and Körse (1971) reported that urinary Na^+ decreased while K^+ increased. Opstad et al. (1985) showed that after a 5 day military training course with lack of food and salt there was a causal connection between the decrease in urinary sodium excretion and the increase in plasma renin activity and serum aldosterone. The same relationship was found by Wade et al. (1985) in runners during a 20 day road race.

The increase in plasma aldosterone concentrations is secondary to an increase in adrenal secretion and/or to a decrease in aldosterone catabolism. The activation of the renin-angiotensin-aldosterone system was due mainly to physical exercise, dehydration (all the athletes lost an average of 2 kg, i.e. 3.5% of body mass), and probably by a small hypovolemia as shown by an increase in haematocrit and plasma protein concentration. On the other hand, the metabolism of aldosterone in the liver is directly correlated with hepatic blood flow, which is reduced by muscular exercise (Schneider et al. 1970). This hepatic catabolism was probably more reduced in R_{24h} than in T_{10h} because of the significant elevation of noradrenaline in the R_{24h} runners.

Sleep deprivation might also have altered the aldosterone response in R_{24h}. However, Opstad et al. (1985) showed that sleep deprivation had no great influence on the activation of plasma renin activity and aldosterone in young military cadets after 5 days strenuous physical exercise.

However the reasons why the 24 h urinary Na^+ and K^+ excretions were unchanged in T_{10h} remain unknown. It may have been due to a difference in cortisol response, since cortisol has, to a lesser degree, the same effect as aldosterone on NA^+ and K^+ excretion. But this explanation is unlikely, since cortisol levels were the same in both cases as shown in Fig. 2.

Other regulatory mechanisms are involved in natriuresis. Firstly the increase in dopamine (which has a natriuretic effect) observed only immediately after T_{10h} and during the two following days could explain the blunted urinary effect of aldosterone elevation. Secondly, atrial natriuretic polypeptide (ANP), which is another mechanism inducing Na^+ urinary excretion, could also explain the discrepancies in Na^+ urinary contents. Two observations lend support to this hypothesis. Firstly, exercise bouts can induce an increase in ANP (Anderson et al. 1986). Secondly, Racz et al. (1986) found that a high dose of ANP acting on human pheocromocytoma cells in-vitro resulted in a significant increase in dopamine. Although it is difficult to extrapolate the in-vivo actions of a high concentration of ANP from in-vitro conditions, we observed that dopamine was effectively elevated after T_{10h} but not after R_{24h}. Further investigations would be necessary to support this hypothesis.

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