Metabolic, haematological and antibody response during 24 hours of continuous sit-ups: Case report

Authors' Contribution:
A Study Design
B Data Collection
C Statistical Analysis
D Manuscript Preparation
E Funds Collection

Alexander Kormanovski[ABCDE], **Rafael Campos-Rodriguez**[ACDE]

Higher Medical School, National Polytechnic Institute, Mexico City, Mexico

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Abstract

Background & Study Aim: There is little information about the metabolic response during cyclical exercise requiring great strength and of extreme duration, that which represents the limit of human capacity.

Material & Methods: A weight lifter trained for and carried out a 24-hour session of continuous sit-ups with abtoner in the form of an arc. The diet during the event was carefully designed and given to the athlete every hour. The metabolic, haematological and antibody response was measured in the capillary blood samples taken every four hours during the exercise.

Results: A minimum level of glucose was observed at the midpoint of exercise, followed by a return to the basal level. During the second half of the exercise session, there was a steady and elevated level of urea. Plasmatic markers of muscular damage (CK and LDH) increased steadily during the first half of the exercise session, and remained at their maximum level (2800 U/l and 700 U/l, respectively) during the second half. Granulocytes reached their maximum level at the midpoint of exercise and diminished afterwards, whereas agranulocytes increased gradually until the end of the exercise session. IgA and IgG showed their lowest level at hour 4, and again at the end of the exercise. There was a high positive correlation between markers of muscular damage (urea, uric acid, inorganic phosphorous, and agranulocytes) during exercise.

Conclusions: 1. low carbohydrate consumption did not significantly limit performance of athlete; 2. the permeability of the membrane is the dominant factor for the CK response during.

Key words: extreme duration exercise • abdominals • blood parameters • immunity • performance • humans

Author's address: Alexander Kormanovski, Calle Hopelchen, Mn.316, Lt.2, Col. Heroes de Padierna, Del. Tlalpan, México DF, CP 14200, México; e-mail: kormanovski@yahoo.com.mx

Background

The capacity of the human organism to adapt to exercise of extreme duration is truly amazing. There are always people who want to do what seems impossible, and their experiences broaden our knowledge about the adaptive capacity of our body and mind. The fact that these events are unrepeatable does not reduce the scientific value of the knowledge obtained. We have worked with some Mexican swimmers that did various feats, including swimming for 24 hours in the open sea or a swimming pool, swimming the backstroke in the open sea for 70 km, and climbing Mount Everest. These athletes allowed us to take blood and saliva samples during their preparation and their final conquest, representing physical and mental exertion that stretches the limit of the human organism [1–3]. In the present study, we report data on the metabolic, haematological and immunological response of an athlete that did sit-up continuously for 24 hours, using "**abtoner**" in the form of an arc and beating the Guinness world record (120,000 repetitions).

Abtoner – the apparatus in the form of an arc for abdominal exercises

Material and Methods

Sit-ups – abdominals exercise

Capillary blood – from finger

Athlete position – feet fixed and hands in the abtoner

The athlete, 32 years old, is a weight lifter and gym instructor. Five years before the current study, he showed great endurance for doing **sit-ups** (abdominals) in the classic way: feet fixed and hands behind the head. He broke various records of duration and intensity of this exercise. When he decided to go for the record of 24 hours of continuous sit-ups using abtoner in the form of an arc, we assisted his training with biochemical monitoring and recommended a scientifically planned dietary regimen for the final event. The research was approved by the Institutional Ethics Committee (Superior Medical School, National Polytechnic Institute, Mexico) and National Sport Commission (CONADE).

The correct dietary regimen for exercise of extreme duration is still under discussion, due to the lack of knowledge about metabolic regulation under these conditions. We based the composition of nutrition during this event on the supposition that after 4–5 hours of exercise: (i) fats and proteins have an essential role in the supply of energy, and (ii) the release of glucose by the liver must be highly important in the maintenance of glucose homeostasis in the blood. Therefore, the amount of carbohydrates in the diet should be the minimum necessary to facilitate the use of other sources of energy, without leading to a reduced release of glucose by the liver. On the other hand, the daily diet of the athlete during training included elevated levels of protein and relatively moderate levels of carbohydrates.

The athlete carried out three trials, with biochemical monitoring, that lasted 4, 6 and 12 hours. We took advantage of each of these trials to fine tune the diet to be used for the final event. After we established the diet, the athlete got used to it during the six months of training during long session of exercise. We reduced eating during exercise to one time per hour in order to minimize the time spent on this procedure. At the odd hours, the athlete consumed 400 ml of the commercial beverage with 20 g of carbohydrates and 30 g of protein, and at the even hours 400 ml of this beverage with electrolytes and carbohydrates (10–15 g) diluted 1:1 in water. The average hourly consumption of carbohydrates was 15–20 g, and that of proteins was 15 g. With this diet during the 12–hour trial, the minimum level of glucose in the blood (57 mg/dl) was recorded at hour 10 of exercise, reaching 65 mg/dl by the end of the exercise period.

The final event of 24 hours of continuous sit-ups was carried out in Mexico City, which lies 2200 m above sea level. The exercise began at 9 am and ended at the same time the following day. During the first half of the exercise period, the athlete maintained a frequency of 85–88 rep/min, which in the second half gradually decreased to 75 rep/min. With a pulse that varied between 104 and 128 p/min, we calculated that there was between 30 and 40% VO_{2max}. A total of 120,512 sit-ups were done.

We received permission from the athlete and the organizers of event to take **capillary blood** samples every 4 hours during the 24-hour of exercise, at the time of drinking the beverage. The quantity of blood taken was limited to a maximum of 0.5 ml, which we are able to take in 30 seconds. Samples were centrifuged within 20 minutes, and the serum was stored on ice and processed 2 hours after the event. Visible haemolysis was not found in any serum sample. Various metabolic parameters were measured: glucose (Glu), lactate (LA), triglycerides (TAG), urea (UR), calcium (Ca^{2+}), inorganic phosphorous (Pi), the activity of creatine kinase (CK, 25°C) and lactate dehydrogenase (LDH, 25°C), total cholesterol (TC), HDL cholesterol (HDL-C), LDL cholesterol (LDL-C), VLDL cholesterol (VLDL-C), total proteins (TP), total albumin (TA) with the RANDOX procedure, and total globulin (TG) calculated as TP minus TA. Haematological parameters were determined on the site of the event with QBC equipment (Beckton Dickinson), including haemoglobin (Hb), haematocrit (HCT), MCHC proportion (Hb/HCT%), platelets (PLT), white blood cells (WBC), granulocytes (GR) and agranulocytes (AGR, the sum of lymphocytes and monocytes).

IgA and IgG were measured in serum and saliva by the ELISA method with two measurements/sample and three dilutions/measurement, using a commercially prepared specific isotype antiserum [2].

Statistical analysis

The Pearson correlation coefficient was calculated by comparing the behaviour of different parameters during 24 hours of exercise.

Results

There was a gradual increase in the number of hours of doing sit-ups as well as in the total sit-ups (with abtoner) per week during the last 13 weeks before the final event (Figure 1).

The basal level of blood parameters was measured before the 24-hour exercise period (Table 1). The only blood parameter out of the normal range is the CK activity. HCT is elevated, which is normal under hypoxic conditions (altitude 2300 m) and aerobic character of exercise. During 6 month of training before the event the HCT of athlete was maintained in 50±2%.

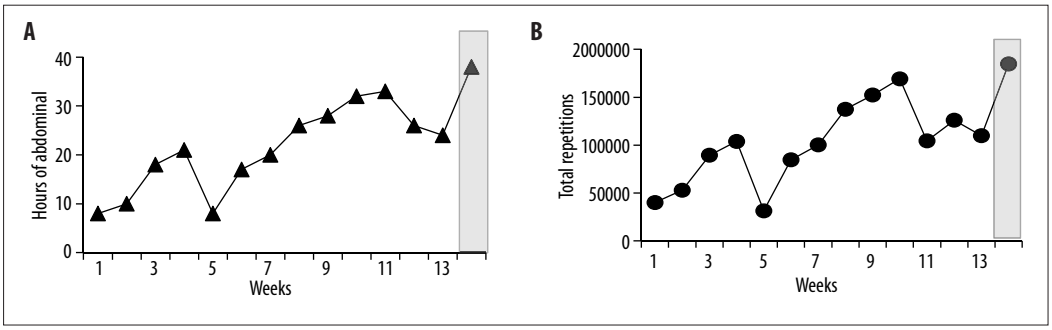

Figure 1. Total hours dedicated to sit-ups per week (**A**), and the quantity of sit-ups (using abtoner) per week (**B**), during the 13 weeks of training before the final event in week 14.

Table 1. Basal levels of blood parameters of athlete.

Parameters	Units	Level	Parameters	Units	Level
Glucose	mg/dl	97	Haematocrit	%	52.2
Triglycerides	mg/dl	78	Haemoglobin	g/dl	17.3
Urea	mg/dl	28	MCHC	%	33.1
Uric acid	mg/dl	2.5	Platelets	1000/µl	257
Lactate	mmol/l	1.5	Total WBC	1000/µl	6.2
Creatine kinase	U/l	289	Granulocytes	1000/µl	4.2
Lactate dehydrogenase	U/l	332	Agranulocytes	1000/µl	2.0
Total cholesterol	mg/dl	211	Total proteins	g/dl	6.9
LDL-cholesterol	mg/dl	144	Total albumins	g/dl	4.8
HDL-cholesterol	mg/dl	60	Total globulins	g/dl	2.1
VLDL-cholesterol	mg/dl	7	IgA	mg/ml	4.2
Inorganic phosphorous	mg/dl	3.6	IgG	mg/ml	14.1
Calcium	mg/dl	8.9	TG-IgA-IgG	mg/ml	2.7

Agranulocytes = lymphocytes + monocytes; TG – total globulins.

The parameters measured during 24 hours of exercise are presented in percentage of change from the basal level. The changes in Hb (3%) and HCT (1.5%) during exercise show that the variation in plasmatic volume is minimal (5%). The latter change was calculated according to Dill and Costill [4] and corrected to capillary blood by the procedure of Knowlton et al [5]. Therefore, plasmatic volume variation did not significantly affect the value of the measured parameters.

The level of Glu bordered on the limit of hypoglycaemia (50 mg/dl) in the eleventh hour of exercise (without clinical manifestations), with a return to the basal level by the end of the event (Figure 2A). The level of TAG was above the basal level during the whole exercise period, with the maximum increase (54%) at the midpoint (Figure 2A), which confirmed that the mobilization of TAG was greater than its use by muscles throughout the exercise. The maximum velocity of increase of TAG (8.8 mg/h) was also observed at the midpoint of exercise, followed by the maximum velocity of decrease (7.8 mg/h). The level of LA diminished as of the second hour and continued this trend until hour sixteen of exercise (Figure 2A).

Compared to the basal value, the concentration of urea increased 50% during the first two hours of exercise, and then plateaued at that level until hour seven (Figure 2B). At hour 11 this parameter again increased, this time representing 110 to 120% of the basal value, and plateaued at that level until the end of the event. These increases demonstrate the elevated level of proteic catabolism, and therefore its important role in the supply of energy during the exercise period. The uric acid response (Figure 2B) was similar to that of urea, with a high positive correlation ($r=0.94$, $p=0.001$) between these two parameters.

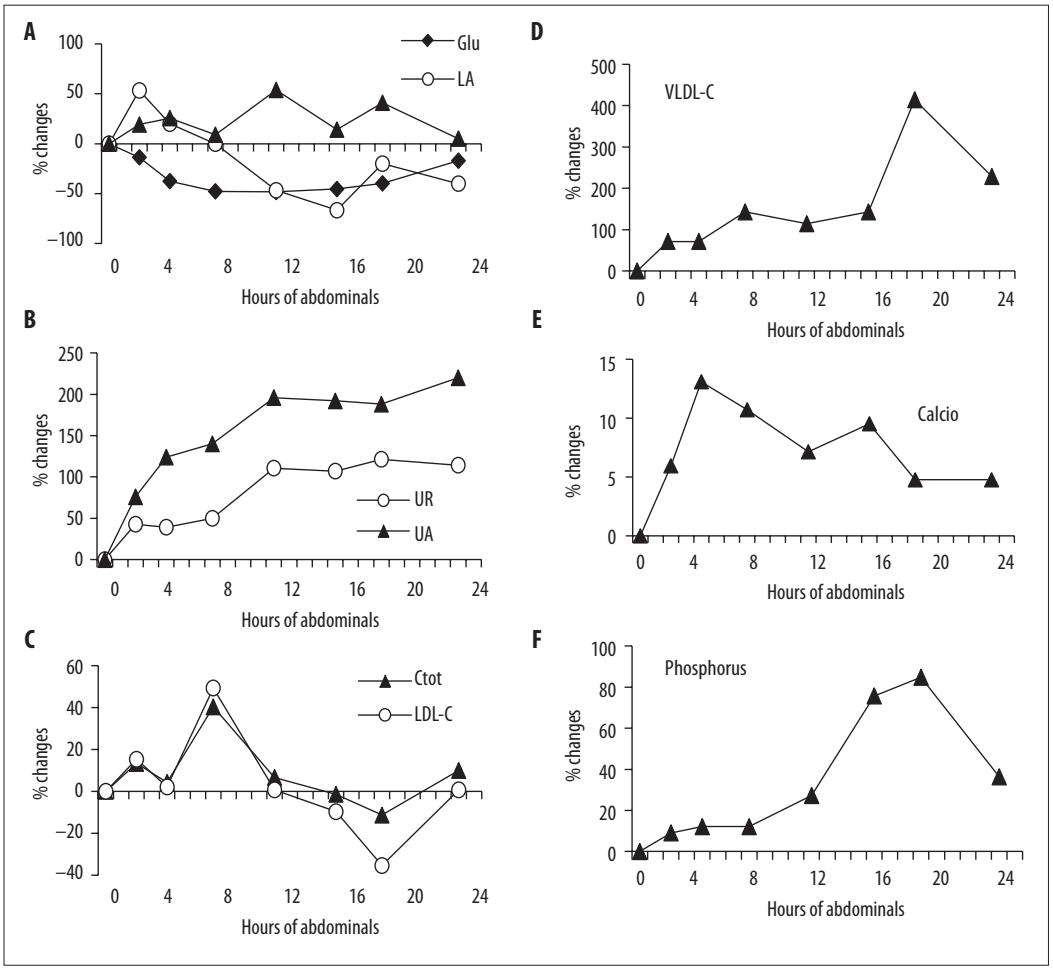

Figure 2. Percentage changes of glucose, triglycerides and lactate (**A**), urea and uric acid (**B**), total and LDL-cholesterol (**C**), VLDL-cholesterol (**D**), Ca^{2+} (**E**) and inorganic phosphorus (**F**) during 24 hours of sit-ups.

The maximum level of total and LDL cholesterol (Figure 2C) was observed at hour seven, and the minimum level was found at hour 18. From hour 7 to 18, LDL cholesterol decreased from 144 to 93 mg/dl. There were no significant changes in HDL cholesterol levels. Consequently, there was an almost100% increase in VLDL-C during the first four hours, and an increase slightly greater than 100% from hour 7 to 15. In hour 18 there was a drastic increase in the level of this parameter (Figure 2D). In hour 4 Ca^{2+} reached its maximum level (13% above the basal value), followed by a gradual decrease until the end of the exercise period (Figure 2E). A sharp increase in inorganic phosphorous (Pi) was observed between hour 15 and 18 (Figure 2F).

Two enzymes that represent markers of muscular damage are CK and LDH (Figure 3A and 3B). CK rose between the beginning of exercise and hour 14, at which time it was close to its maximum level of 2800 U/l. Similarly, LDH rose until hour 12, at which time it was close to its maximum level of 700 U/l. For the second half of the exercise period, these two parameters remained relatively constant, at or near their maximum level. There was a very high positive correlation between the behaviour of these two enzymes ($r=0.958$, $p<0.001$). Unlike CK, the elevated level of LDH in plasma can result from sources other than muscular damage, namely a high activity in erythrocytes. However, in the present study it seems that the behaviour of total LDH was determined principally by the muscular source.

WBC reached its maximum level in the middle of the exercise period and diminished afterwards (Figure 4A). Granulocytes responded in the same way, but agranulocytes increased until the end of the exercise, reaching double the basal level. Antibodies (IgG and IgA) reached their minimum level after 4 hours of exercise (40% of the basal level), followed by a return to their basal level (Figure 4B). Finally, there was a sharp decrease (more than 60%) in these parameters at the end of the exercise period.

Regarding total proteins, a 13% increase was observed at hour 4 and a 19% increase by the end of exercise (data

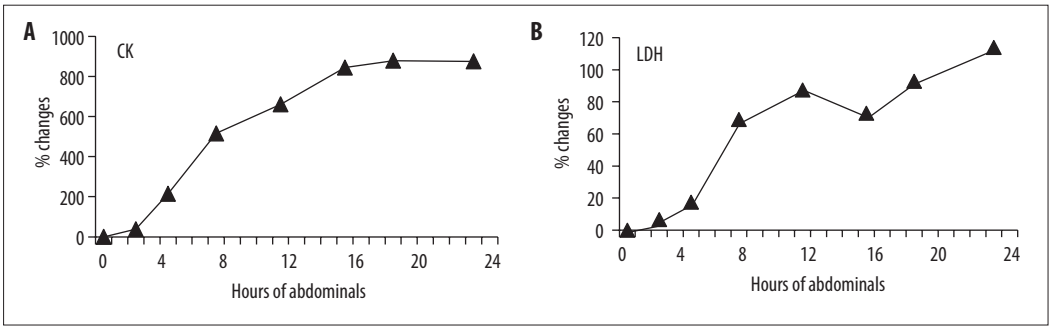

Figure 3. Percentage changes in CK activity (**A**) and LDH (**B**) during 24 hours of sit-ups.

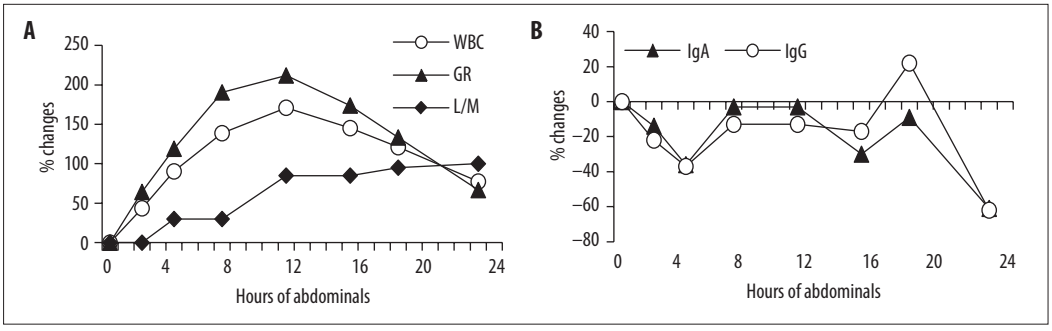

Figure 4. Percentage changes of granulocytes, agranulocytes (**A**) and immunoglobulin's (**B**) during 24 hours of sit-ups.

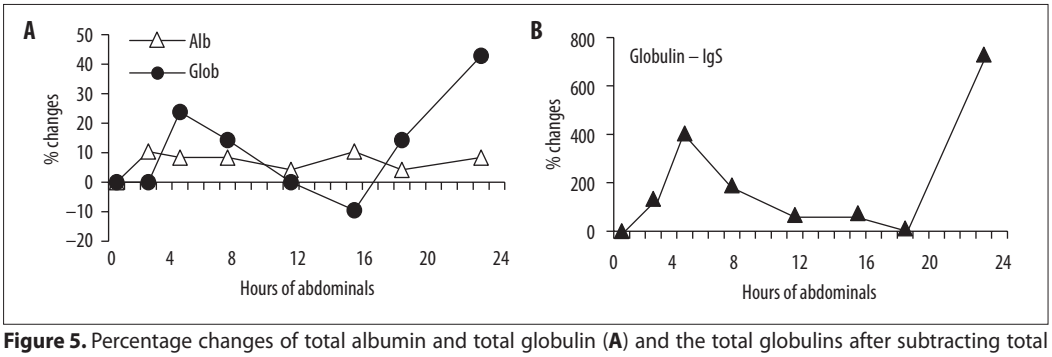

Figure 5. Percentage changes of total albumin and total globulin (**A**) and the total globulins after subtracting total immunoglobulin's (**B**) during 24 hours of sit-ups.

not shown). The response of total albumin was different from that of total globulin. Total albumin increased 10% from hour 0 to 2, and remained at that level until the end of the exercise session (Figure 5A). Total globulin, on the other hand, increased 25% between hour 3 and 4, diminished lineally until hour 15, followed by a sharp lineal increase (43%) from hour 15 to 24. The stable and elevated level of albumin during exercise is logical, due to its function as a transporter of liposoluble substances and its antioxidant effect. If the steady decrease of globulin from hour 4 to 15 can be understood as a result of catabolism, the increase from hour 15 to 24 should reflect mobilization from other tissues as an adaptation to severe physical exhaustion.

After subtracting the IgA and IgG concentrations from the total globulin concentrations, it can be seen that the behaviour of the total globulins is mainly determined by globulins other than immunoglobulin (Figure 5B). The level of globulins minus immunoglobulin increased 400% between hour 0 to 4, and 700% at the end of the exercise period.

It is interesting that a high level of positive correlation was found among various metabolic parameters, markers of muscular damage and agranulocytes (Table 2). In the case of Pi and VLDL-C, the correlation of UA with LDH is at the limit of significance (p is between 0.07 and 0.08). At least four of the seven parameters are directly related with the inflammatory process in muscular tissue.

MCHC (Hb/HCT%) was also found to be positively correlated with CK and LDH activity ($r=0.72$, $p<0.05$; $r=0.82$, $p<0.05$, respectively).The level of Glu correlated negatively with the concentration of Ca^{2+} ($r=-0.72$, $p=0.044$) and granulocytes ($r=-0.981$, $p<0.01$).

Table 2. Pearson correlation matrix between the different blood parameters.

	N	CK	LDH	Urea	UA	VLDL	AGR	Phosphorous
CK	8	1						
LDH	8	0.958**	1					
Urea	8	0.936**	0.903**	1				
Uric acid	8	0.926**	0.916**	0.939**	1			
VLDL-C	8	0.772*	0.735*	0.757*	0.668	1		
AGR	8	0.961**	0.926**	0.960**	0.924**	0.729*	1	
Pi	8	0.817*	0.643	0.809*	0.676	0.797*	0.790*	1

* $p<0.05$; ** $p<0.01$.

Discussion

The factors of diet and intensity of aerobic exercise are the main determinants for the metabolic response during exhaustive exercise [6]. Consumption of carbohydrates during prolonged exercise increases performance, which is a result of a good maintenance of homeostasis of glucose in the blood, related to oxidation of exogenous carbohydrates [7]. In the majority of studies, it has been shown that the consumption of external carbohydrates does not significantly affect the velocity of oxidation of muscular glycogen, but it does indeed reduce the release of glucose in the liver [8,9], and at high doses can even block glucose release [10,11]. This means that prolonged exercise, as of a certain hour, is carried out with a low level of muscular glycogen. The hour that this condition begins depends mainly on the intensity of exercise.

The supply of energy from carbohydrates consumed during exercise does not go beyond 20% of the energetic demand. According to various studies [12–14], the maximum velocity of oxidation of external carbohydrates, without reaching the point of producing gastrointestinal discomfort, fluctuates close to 60 g/h. The practical effect of different doses of carbohydrates is still under debate, as the relation between different sources of energy remains unclear. The majority of reports providing data on this subject are in relation to highly intense exercise lasting less than 4 hours.

We have worked with more than 30 swimmers in open waters with an average velocity between 3 and 3.6 km/h for more than 6 hours of swimming [1–3]. Only 3 of these swimmers could consume only carbohydrates (up to 50 g/h) without problems during swimming for as long as 24 hours. The rest of the swimmers felt much more comfortable with the mixture of carbohydrates (15–20 g/h) with proteins (5–15 g/h). We suppose that apart from other factors, there is a genetic predisposition to the high assimilation of CHO during exercise, because in various cases the duration, intensity and quantity of training were similar, as was the average speed of swimming.

The consumption of carbohydrates in the present study can be considered relatively low (<20 g/h). The decrease in Glu of the athlete first reached the limit of hypoglycaemia, and then returned to the basal level by the end of exercise. Taking into account that he did not present clinical symptoms of hypoglycaemia and was able to finish this extraordinary effort, could mean that the quantity of external carbohydrates did not significantly limit performance. The response of triglycerides in plasma shows an elevated mobilization as well as an elevated use of the same. There were three points (hour 4, 11 and 18) at which TAG reached its maximum levels, representing a mobilization that was able to overcome the loss in the blood flow. The greatest level of this parameter was in the middle of the exercise period (Figure 2A).

Data on the role of proteic metabolism during prolonged exercise are contradictory [15]. Prolonged and exhaustive exercise is carried out with a diminished level of muscular glycogen. Under these conditions in a recent study using stable isotopes [16], it was shown that stimulated protein catabolism and diminished its synthesis. These data coincide with the well-known increase of urea in the blood during prolonged exercise [17]. Therefore, the steady increase in ureaup to hour 11 of the present study, until reaching 100% of the basal level, can be interpreted as the contribution of proteic metabolism to the energy supply. Indeed, this is what we wanted to accomplish with the diet elaborated for this exercise. Additionally, we propose that it is likely that the presence of proteins in the intestines resulted in a more gradual assimilation of carbohydrates, due to the size of the protein molecule (more than 1000 times greater than that of Glu), which should certainly avoid rapid changes in the level of Glu and insulin during exercise.

The decrease in lactate as of the second hour probably reflects a diminishment in the flow of the components due to the aerobic glycolysis, determined by the depletion of muscular glycogen, and consequently resulting in an increase in the contribution of fats and proteins to the energy supply. The significant increase in uric acid is positively correlated to the increase in urea, adding support to the possible relation with proteic catabolism. There are data that show an antioxidant effect of uric acid [18], which would represent another positive aspect of its increase in the blood.

The behaviour of the markers of muscular damage (CK and LDH) during and after the exercise, were discussed in two reviews [19,20]. Two possible mechanisms were proposed: (i) an increase in the permeability of the muscular membrane related to the activation of the potassium channel with Ca^{2+} within the cell [21], and (ii) the destruction of the sarcomeres in zone Z [22]. The majority of studies on the exercise-induced increase in the level of CK in the blood are carried out with exercise that damages both muscular tissue and capillaries. In one study a high variation was observed (between 2360 and 25244 U/l) to the same eccentric exercise [23], which puts in doubt the assumption that CK is only and indicator of muscular damage [24,25].

Most authors consider that the destruction of muscular cells (rhabdomyolysis) is the principal mechanism of the increase of this muscle enzyme in the blood [26], and that the high variation in the CK response is attributable to the circulation of this enzyme in the lymphatic system, together with its high molecular weight [27,28]. A decrease in CK activity (but not for myoglobin) was demonstrated in athletes with one arm immobilized after a series of eccentric exercise [29]. Another study also showed a diminishment in the CK response with athletes lying in bed during 17 hours, after having run 8 km cross country [30]. These authors posed two possible explanations for the increased CK activity: (i) reduced flow of the lymphatic system, or (ii) a decreased velocity by this enzyme in leaving muscular fibres. This study can be interpreted in either of this contradictory ways.

There is little information, however, about CK dynamic during ultra-long duration exercise [31,32]. A twentyfold increase of CK activity was observed in runners during an ultra-long-distance race (1000 km in 20 days) up to day 3, which was followed by a significant decrease at the end of the race. However, the exercise was not continuous, as the runners slept between exercise sessions, and this makes interpretation of data difficult [32].

Regarding the increase in CK during aerobic exercise of extreme duration, we pose that the permeability of the membrane is the dominant factor, as opposed to the direct destruction of the same. The CK response in the present study does not contradict this supposition. Indeed, membrane permeability could be the last resort of adaptation during extreme-duration exercise, enabling the exchange of low-weight molecules between cells and the blood. On the other hand, the maximum level of CK activity in the present study (2800 U/l) is much less than muscle tissue can release with eccentric exercise [23].

Our own extensive experience measuring the CK response to extreme exercise in elite athletes of endurance sports leads us to conclude that an important part of the increased activity of this enzyme is due to an adaptation to exercise. We have observed the following: (i) the maximum level of CK in endurance sports in the absence of severe muscular injury varies between 1500 and 3000 U/l; (ii) in 13 of the 15 examples in our experience of this degree of increase in CK activity, the performance of the athlete increased significantly one to two months afterwards; and (iii) in athletes who normally have their level of CK within the normal range (<100 U/l) during training, the performance increases significantly only when, for diverse reasons, there is an increase of CK activity above this range.

In the current contribution, the response of both markers of muscular damage (CK, LDH) positively correlates with the response of metabolic parameters (urea, uric acid) and haematological parameters (agranulocytes), indirectly confirming the idea of the adaptive response to exercise rather than muscular damage as the dominant explanation for the data.

It is known that in the mioplasm of fatigued muscular fibres, the concentration of inorganic phosphorous (Pi) increases up to 10 times [33], which decreases muscular strength [34]. In one study, this type of increase in inorganic phosphorous was interpreted as a cellular mechanism of the development of muscular fatigue [35]. We suppose that the increase in the level of Pi in plasma observed in the second half of exercise in this study reflects its transfer to the blood from fatigued muscles.

In the present study, the level of Ca^{2+} in plasma reached its maximum level (13%) in hour 4 of exercise, followed by a decrease. Ca^{2+} is an important regulator of muscular contraction. It has been shown that the gradient between Ca^{2+} within the sarcoplasmic reticulum of muscular fibre and that outside of the same diminishes during exhaustive exercise [36], and we suppose that this could be related to the accumulation of Pi, that can precipitate it as calcium phosphate, and thus diminish its concentration

and consequently the capacity of its release from the sarcoplasmic reticulum, contributing to muscular fatigue. Additionally, this process should be accompanied by stability or a decrease in Ca^{2+} in the bloodstream. Ca^{2+} has a crucial role in the stimulation of glycogenolysis in muscle [37]. It is likely that the decrease in the level of Ca^{2+}in serum observed from hour 4 of sit-ups reflects its decrease within muscle tissue, which could represent depressed glycogenolysis and consequently a stimulation of the use of fat as an energy source. The behaviour of Pi and Ca^{2+} in the present study does not contradict these data (the accumulation of Pi in the second half of the event coincided with a decrease of Ca^{2+} from hour 4 to hour 24). Indeed, the change in the plasmatic concentration of Ca^{2+} was moderate (within 15%).

Conclusions

1. Relatively low carbohydrate consumption (<20 g/h) in the present study did not significantly limit performance of athlete.
2. It is possible that the permeability of the membrane is the dominant factor that determined the CK response during vary long exercise.

Acknowledgements

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