The supplementation of omega-3 lipids and medium chain triglicerides do not affect metabolic indicators in the exhaustion test

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ABSTRACT

Objective: This study compares the effects of strength training (ST) with or without supplementation of omega-3 lipids (W-3) or medium chain triglycerides (MCT) in metabolic indicators in the exhaustion test (ET). **Methods:** The subjects 12 males with minimum 11 months of ST of experience were divided in group W-3 (GW-3: n = 7, 26.7 ± 6.0 years old; 82.6 ± 10 kg) and group MCT (GMCT: n = 5, 18.8 ± 1.3 years old; 74.6 ± 9.7 kg). There were 2 moments of ET: after 28 days only with ST and blood collection samples before (M1b) and after (M1a), and after 28 more days of ST plus supplementation with 4 g/d of W-3 or 4 g/d of MCT repeating the same procedure (M2b and M2a). **Results:** The hematocrite (Ht), osmolality (Os), sodium (Na^+) and PCO_2 showed no significant change at any moment, group or Δ M2 ($P > 0.05$). The PO_2 increased significantly after ET in two moments and groups ($P < 0.05$). The glucose and HCO_3 had significant increase in M1 to GW-3 and HCO_3 in M2 to GMCT ($P < 0.05$), without other changes. The LDH increased significantly only in M2 to GW-3 ($P < 0.05$) and pH decreased in M1 to both groups ($P < 0.05$), without other significant changes. **Conclusions:** The ET *per se* altered the major metabolic indicators but a great standard deviation occurred. The tests induced acidosis without influencing fat acids supplementation.

Key words: Strength training. Acidosis. Lipids. pH.

INTRODUCTION

The tolerance to exhaustion in intense exercises depends on the strength increase and muscular resistance induced by the resistance training with overloads above the usual, also known as overload principle[1]. The deposition of contractile protean material and metabolic alterations for the muscular energy output depends on energetic dietetic factors[2], adequate amounts of protein ingestion[3], favorable hormonal alterations[4], among other factors. Some lipids may have positive effects in these processes[5].

Fatigue and exhaustion are many times used as synonymous. However, the fatigability and exhaustion are different with regard to the type of exercise and consequent alteration on the energetic metabolism. The central fatigue is due to the neurotransmitter depletion of the central nervous system (CNS), impairing the electrochemical impulses into the muscle, while the peripheral fatigue is mostly caused by the glucose reduction and metabolic growth, reducing the pH[1]. The fatigue may be defined as *"a progressive decline of the capacity of generating muscular strength for the physical activity"*, and the exhaustion as *"a status in which the capacity of generating muscular strength declines until the target strength (TS)"*. TS is the percentile goal of the maximum voluntary strength (MVS) defined as the training or test aim and serves as point of exhaustion definition[6].

In both fatigue and exhaustion, several indexes are used in order to quantify their respective intensities. Some important metabolic indicators are: hemoconcentration (hematocrite and osmolality), sodium (Na^+), acidosis (pH and bicarbonate (HCO_3)), anaerobic metabolism (lactate dehydrogenase (LDH)), oxygen pressure (PO_2), carbon dioxide pressure (PCO_2) and energetic substrates (glucose)[7].

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During intense exercises, the glucose produces high amounts of lactic acid, releasing hydrogen ions (H^+) that may increase from 100 nEq.L^{-1} (pH = 7.0) up to over than 300 nEq.L^{-1} (pH = 6.5) in the muscle, and from 40 nEq.L^{-1} (pH = 7.4) up to 100 nEq.L^{-1} (pH = 7.0) in the plasma comparing rest to maximum exercise. The H^+ formed attaches to the NAD^+ forming NADH, which needs to be reduced once again into NAD^+ in order to pursue the glucose hydrolysis and oxidation. In the absence of oxygen, the NADH is reduced into lactate through the action of the enzyme LDH. The increase on the concentration of H^+ in exhausting activities reduces pH and turns the NAD^+ available in order to attach to the enzyme 3-phosphate glyceraldehyde dehydrogenase that oxidizes the 3-phosphate D-glyceraldehyde and the phosphate dihydroxyacetone, formed from 1,6-biphosphate D-fructose, stopping the glycolysis[1], summarizing the reaction:

3-phosphate D-glyceraldehyde + NAD^+ + Pi $\rightleftarrows$ 1,3- biphosphoglycerate + NADH + H^+

The objective of this study was to verify the metabolic alterations induced by maximum muscular exhaustion tests (ET) *per se* and whether omega-3 lipids (W-3) or medium chain triglycerides (MCT) would affect the response of those indicators after 28 days of supplementation.

METHODS

Subjects – Twelve male subjects who volunteered for this study were divided in two groups: group W-3 (GW-3: n = 7, 26.7 ± 6.0 years old; 82.6 ± 10 kg; average ± standard deviation) and group MCT (GMCT: n = 5, 18.8 ± 1.3 years old; 74.6 ± 9.7 kg; average ± standard deviation). The selection was performed in muscular exercises academies from the city of Botucatu, SP, Brazil, through personal interviews. A minimum of 11 continuous months of hypertrophic training in resistance exercises was required, not smoker, not alcoholic beverage user, not anaerobic steroid user or similar and not to carry metabolic disease historical.

All participants were properly informed and signed up approval declaration according to the regulations of the Research and Ethics Committee from the State of São Paulo University Medical School – Unesp, Botucatu. Those documents were conducted to the mentioned Committee and approved for the accomplishment of this study.

Alimentary ingestion and dietetic protocol – Alimentary investigation questionnaires were applied (24-hours reminiscent, 3-days alimentary record and alimentary habits), from a program containing the food centesimal composition. The usual diet was adjusted, containing 1.5 g of protein/kg of weight/day, with a total of 30 non-protein kcal per gram of protein. At the second month of training, the individuals were given 4 g/day W-3 (n = 7), or 4 g/day of MCT (n = 5) as the only alterations.

Physical activity protocol – The training protocol was of 5 weekly days as follows: 3 days of continuous training, 1 day of rest followed by two more days with loads between 70 and 85% from the RM, with recovering time between 30 seconds and 1 minute, based on the methodologies combination for hypertrophy[8]. The same protocol was used during the entire study.

The sessions involved the following muscular groups: 1) – chest, shoulder, triceps, wrist and abdomen; 2) – back, biceps, forearm; 3) – thighs, gluteal, lumbar and leg calf; 4) – the same groups of session 1 and 2; 5) – the same groups of session 3, previously described[9].

Moments – Two exhaustion tests (ET) were performed; one of them after the first month of training (moment 1 – M1) without W-3 or MCT supplementation and sample collecting before (M1b) and after (M1a) the ET and, the other one after the second month of training (moment 2 – M2), with supplementation of 4 g/day of W-3 or 4 g/day of MCT and sample collecting before (M2b) and after (M2a) the ET.

Exhaustion test (ET) – The exhaustion test consisted of tests used in preceding studies of the researching group from the Nutrition and Metabolism Center *(CeMeNutri)*[9], State of São Paulo University Medical School – Unesp, Botucatu, Brazil. The ET was applied after the first month of training without supplementation (M1) and after the second month of training with supplementation (M2).

The test consisted of applying decreasing loads starting at 80% of 1 RM, performing the highest number of repetitions as possible until the muscular failure (the repetition non-execution), the load was reduced of 20% (80/60/40 and 20% of RM) through external aid with no exercise interruption and the continuity of this process was repeated until it remained only 20% of the maximum load (TS) and the fatigue occurred[8].

Exercises selected: 1) – straight supine (chest, shoulder and triceps); 2) – ducking in the Hack® machine (Hack is the name given to the ducking exercise performed in machine, with support for the back and a ball bearing little car, in respect to the exercises inventor (quadriceps, adductor magnus and gluteal)); 3) – low paddling in pulley (back, shoulder and biceps). Rests in between exercises were not allowed.

Samples attainment – The venous blood collecting was performed before (M1b and M2a) and shortly after (M1b and M2a) the ET, through puncture of the cubital vein with disposable needles and syringes. After the separation of the serum or plasma, this material was conducted to the Clinical Analysis Section of the General Hospital of this institution for the analyses of hematocrite (micro-hematocrite technique), sodium (Na^+), arterial gasometry for the pH, bicarbonate (HCO_3), osmolality, enzymes lactate dehydro-

genase (LDH), glucose (oxidase glucose method), PO_2 and PCO_2.

Statistical analysis – The results of the moments (M1b, M1a, M2b, M2a) are presented on tables and figures as average value ± standard deviation. The study of the variables consecutive measuring was performed by means of the repeated measures analysis complemented with the construction of simultaneous confidence intervals (95%) for the construction of moments, through the test of Friedman for dependent samples. The comparison between groups was performed by the delta of M2b and M2a (Δ M2) using the Mann-Whitney test for independent samples. All statistical conclusions were performed at 5% of significance. The tests were performed through the Graph Pad in Stat statistical program.

TABLE 1
Averages before and after endurance in moment 1 (before supplementation – M1b and M1a) and in moment 2 (after 28 days of supplementation with W-3 or MCT – M2b and M2a). Significant increase after endurance test in relation to the basal is indicated by: * $P < 0.01$ e ** $P < 0.001$. Data presenting no sign: $P > 0.05$

	M1b	M1a	M2b	M2a
Hematocrite (%)				
GW-3				
Average	42.43	52.43	43.33	49.0
Sd	5.85	3.40	4.45	4.42
GMCT				
Average	44.20	52.60	44.80	52.20
SD	7.49	4.03	5.71	2.16
Osmolality (mOsm)				
GW-3				
Average	277.71	290.71	282.33	289.17
SD	15.40	9.65	2.42	6.79
GMCT				
Average	279.40	291.60	279.0	291.75
SD	4.03	7.16	2.23	4.99
Sodium (mmol/L)				
GW-3				
Average	140.71	147.14	144.67	147.17
SD	7.63	6.06	1.36	2.38
GMCT				
Average	142.80	148.80	142.80	148.80
SD	2.58	4.08	1.48	2.48
Oxygen pressure (mmHg)				
GW-3				
Average	26.97	59.42*	31.65	65.34**
SD	6.78	11.70	8.77	10.83
GMCT				
Average	26.94	55.04*	33.20	62.90**
SD	4.36	8.77	18.78	7.23
Carbon dioxide pressure (mmHg)				
GW-3				
Average	48.60	43.94	51.37	39.21
SD	6.55	16.59	6.77	9.21
GMCT				
Average	46.30	47.46	40.28	52.64
SD	7.37	14.11	12.30	11.34

RESULTS

The hematocrite, osmolality and Na^+ had no statistical significance for any group, in the Δ M2 between groups and with treatment ($P > 0.05$). The PO_2 increased significantly after exhaustion in both moments and groups (M1: $P < 0.01$ and M2: $P < 0.001$), without difference in the Δ M2 between groups or treatment with lipids ($P > 0.05$). No significant statistical difference for PCO_2 before and after exhaustion was observed in any moment, in the Δ M2 between groups or after treatment ($P > 0.05$; table 1).

The glucose increased significantly only after exhaustion in M1 for GW-3 ($P < 0.05$), but not in M2 and with no difference for the GMCT and in Δ M2 between groups or after treatment (figure 1). The HCO_3 decreased after exhaustion in M1 of GW-3 ($P < 0.05$) and in M2 of GMCT ($P < 0.01$), without significant difference in the Δ M2 between groups or with treatment ($P > 0.05$; figure 2). The LDH increased significantly after exhaustion in M2 for GW-3 (P

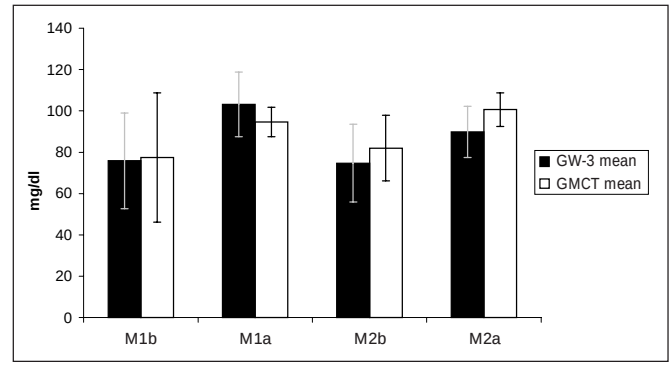

***Fig. 1** – Glucose results (average and standard deviation at 5% significance). Two moments were evaluated (M1 – after 28 days of strength training, and M2 – after 28 more days of strength training plus supplementation of 4 g/day of W-3 or 4 g/day of MCT), with two data collecting at each moment: before ET (M1b and M2b – first and third set of bars from left to right) and after ET (M1a and M2a – second and fourth set of bars from left to right). A significant increase after the endurance test in M1 for GW-3 (P < 0.05) was observed. No significance on the other variables compared was found (P > 0.05).*

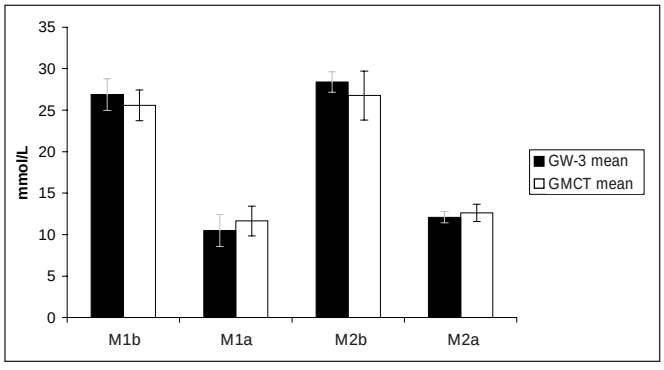

***Fig. 2** – HCO_3 results (average and standard deviation at 5% significance). Two moments were evaluated (M1 – after 28 days of strength training, and M2 – after 28 more days of strength training plus supplementation of 4 g/day of W-3 or 4 g/day of MCT), with two data collecting at each moment: before ET (M1b and M2b – first and third set of bars from left to right) and after ET (M1a and M2a – second and fourth set of bars from left to right). A significant decrease on the HCO_3 after the endurance test in M1 for GMCT (P < 0.05) and in M2 for GW-3 (P < 0.01) was observed.*

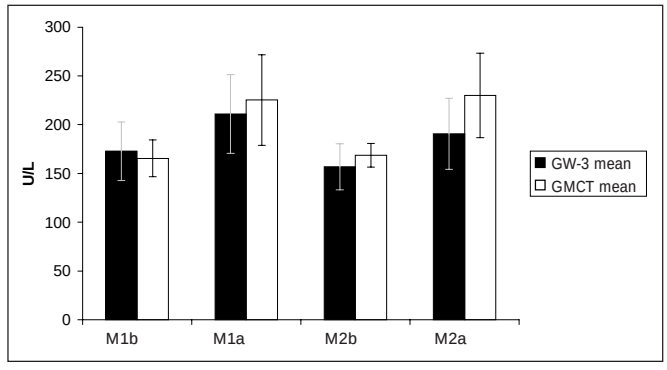

***Fig. 3** – LDH results (average and standard deviation at 5% significance). Two moments were evaluated (M1 – after 28 days of strength training, and M2 – after 28 more days of strength training plus supplementation of 4 g/day of W-3 or 4 g/day of MCT), with two data collecting at each moment: before ET (M1b and M2b – first and third set of bars from left to right) and after ET (M1a and M2a – second and fourth set of bars from left to right). A significant increase of LDH after the endurance test in M2 for GW-3 (P < 0.05) was observed. No significance on the other variables compared was found (P > 0.05).*

< 0.05), but without difference in M1 and in both moments of GMCT, in Δ M2 between groups or with treatment (figure 3). The pH decreased significantly after exhaustion of M1 for both groups ($P < 0.05$). No differences in M2 for both groups, in the Δ M2 between groups and with treatment were verified (figure 4).

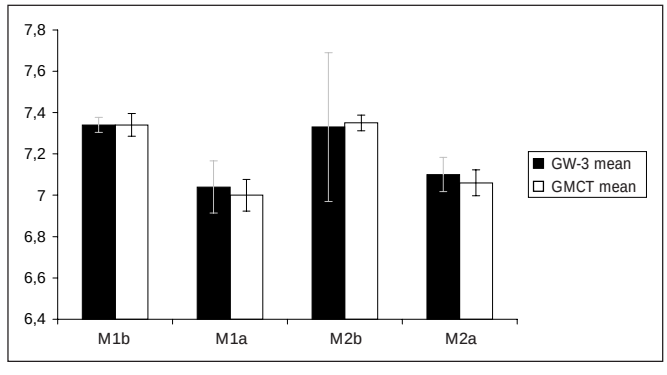

***Fig. 4** – pH results (average and standard deviation at 5% significance). Two moments were evaluated (M1 – after 28 days of strength training, and M2 – after 28 more days of strength training plus supplementation of 4 g/day of W-3 or 4 g/day of MCT), with two data collecting at each moment: before ET (M1b and M2b – first and third set of bars from left to right) and after ET (M1a and M2a – second and fourth set of bars from left to right). A significant decrease of pH after the endurance test in M1 for GW-3 (P < 0.01) and for GMCT (P < 0.05) was observed. No significance on the other variables compared was found (P > 0.05).*

DISCUSSION

The results of this study have demonstrated that the exhaustion *per se* is an important synchronizer of metabolic alterations and the supplementation with lipids W-3 or MCT showed no alterations in those indicators. The non-alteration of some expected results as well as the significance variation between groups and moments may be due to a large standard deviation.

The metabolic alterations obtained with the dynamic or isometric contraction of a given member indicate exhaustion and local biochemical alterations, but do not represent the systemic stress[6]. In the ET, the several energetic metabolisms involved with the load progressive decreases and different types of muscular fibres show additional difficulties for the assessment of alterations that occur in each fatigues phase.

The ET intended to reach a large muscular volume that represents the systemic stress, while most studies regarding to muscular fatigue have employed isometric contractions of small well-defined muscular groups[6]. The muscular groups directly requested in the exercise were the ones causing more metabolic alterations in the evaluation of the impact on the systemic stress, evaluated by our group. The exercises were selected based on previous results of alterations of 3 variables (glucose, HCO_3, NH_3). The highest alterations were caused by: back = biceps = triceps; followed by the pectoral muscular groups and quadriceps and the muscular groups causing the lowest alterations were: leg calf, posterior thigh and shoulders[9].

No significant alterations for hematocrite, osmolality and Na^+ were verified for any group between before and after ET, in Δ M2 between groups and with treatment ($P > 0.05$). It is believed that the great variation of data has been re-

sponsible for such result. The hematocrite and osmolality could have been increased due to the water influx that follows the sodium and to the potassium efflux unchaining the cell membrane action potential[10]. The proton movement in and out the cell should be followed by the flux of cations or by the co-flux of anions in order to maintain the electroneutrality. The Na^+ increase ratio in similar situations may be attributed to the Na^+-H^+ change mechanism, which has demonstrated to be an important pH regulator in the muscles of rats, in both the rest and in the exhaustion recovery[11], fact not observed in this study.

There was an increase on the glucose only after the exhaustion of M1 of GW-3 and M2 of GMCT. An increase on the glucose in all moments after the exhaustion tests was expected through the exacerbation of the sympathetic discharge, mobilizing the hepatic glucose[12]. Another reason is that the non-lactic anaerobic system, derived from the ATP (adenosine triphosphate) breakage and creatine phosphate (CP) stored in the muscle lasts around 10 seconds in maximum activity. As the activity continues, the anaerobic glycolysis provides ATP, ending by the formation of lactate[13], what leads to the plasmatic pH drop observed in the M1 of this study. Boobis *et al.*[14], observed that the anaerobic energy produced during 6 and 30 seconds of maximum activity in ergometer cycle was of 63 and 189 mmol ATP.kg^{-1} d.w., with the glycolysis contribution estimated in 53 and 64%, respectively. Once the average of the repetitions summation for the three exercises was of 151 times and of 3 seconds per repetition, on average, the total time spent for the performance of the test was of 453 seconds, in other words, with an important participation of the anaerobic glycolysis, once the intensity was maximal.

The LDH increased significantly after exhaustion in M1 only for GW-3, with no difference for the other comparisons. As for the glucose, an increase of LDH after the ET was expected, once intense activities with formation of lactic acid activate the Cori cycle, which is the transportation of the flowed out lactate of the muscle through the red cells for the circulation until the liver, which utilizes LDH for the re-conversion into pyruvate and through the gluconeogenesis, to form more glucose[15]. Maybe the blood collecting shortly after exhaustion has not been long enough for the gluconeogenesis to be significant, associated to a great variability of data.

In endurance exercises, the two hydrogen atoms extracted from the substrate during the glycolysis are transferred into the NAD^+, forming NADH that, associated to the production of H^+, exceeds the processing velocity of the respiratory chain. The continuous release of anaerobic energy in the glycolysis depends on the NAD^+ availability for the oxidation of 3-phosfoglyceraldeid, otherwise, the glycolysis fast rhythm would "exhaust". In anaerobic conditions, the NAD^+ is generated as "excessive" pair of H^+ are combined with the pyruvate in an additional stage catalyzed by the enzyme lactate dehydrogenase (LDH), forming lactic acid through a reversible reaction[12], what corresponds to an increase of LDH observed in this study, in M1 of GW-3. When this occurs, the associated hydrogen ions cause a decrease on the intracellular and plasmatic pH[16], what occurred in M1 for both groups. It is not yet conclusive if lipids may have affected the non-significance in the pH drop in M2 (figure 4), by the absence of the control group, once there was a significant increase of LDH of GW-3 and a decrease of HCO_3 of GMCT after the ET in M2. The excessive increase of ions H^+ and the plasmatic pH drop may occur regardless the lactate transportation[11], inhibiting the glycolysis and therefore the muscular contraction[17], what may have been relevant in the muscular exhaustion observed in the ET. A significant decrease on the HCO_3 always after the ET was expected, once it is used in order to buffer the lactic acid[17], what not always occurred.

The PO_2 increased significantly always after the ET in M1 and M2, regardless the lipids supplementation. The increase on the PCO_2 stimulates the respiratory centers to the hyperventilation[12] that, in turn, increases the PO_2. The concentrations of O_2, CO_2 and the respiratory frequency increased proportionally in a progressive exercise until the anaerobic threshold, when the ventilation increases exponentially, dissociating from the other curves[18].

The increase on the PCO_2 was expected by the maximum intensity of the test. The CO_2 is produced from both the aerobic metabolism at the end of the respiratory chain along with H_2O, and from the anaerobic metabolism, due to production of lactic acid. The dissolved CO_2 reacts chemically with H_2O in order to produce carbonic acid, which is spontaneously dissociated into H^+ and HCO_3^- that is dissociated into H^+ and CO_3^{2-}. The classical equations of the blood [La^-] increase are correlated to the equimolar decrease of the plasmatic [HCO_3^-] for the lactic acid buffering, given by the equations[19]:

$$LaH + HCO_3^- \rightleftarrows La + H_2CO_3$$

$$H_2CO_3 \rightleftarrows CO_2 + H_2O$$

This is in agreement with results observed in M1 of GW-3 and M2 of GMCT for the significant reduction of HCO_3. The non-statistical significance for the PCO_2 may be due either by the great variability of data or by the fast stimulus its increase causes in respiratory centers, hyperventilating and quickly eliminating the blood excess through respiration.

Another possible explanation for the non-increase on the PCO_2 is that the lactate remains being released from the

muscular cell into the blood during the exercise recovery[20], being important source to produce CO_2. The venous blood collecting shortly after test may not have been long enough for the increase on the PCO_2. However, Bangsbo *et al.*[11] observed significant increase on the PCO_2 in the femoral vein shortly after endurance unilateral exercise in ergometer cycle. The data of HCO_3 observed in the mentioned study corroborate with results of our study after the ET in M1 for GW-3 and M2 of GMCT, but not for the M1 of GMCT and M2 of GW-3.

In short, the exhaustion test *per se* was the main synchronizer of changes observed despite some discrepancies. Increases on the acidosis, drops on the pH and HCO_3 and increases on PO_2 were observed, when compared between before and after the ET, although not always in both groups and moments. Possibly, the great standard deviation may have affected the statistical power. There were no changes induced by the supplementation of W-3 or MCT in the exhaustion metabolic indicators.

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