Dietary intake and the markers of muscle damage in elite basketball players after a basketball match

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Abstract:

Elite basketball games are characterized by intermittent, high intensity physical activity. Post-game muscle damage and inflammation may determine duration of recovery. The aim of this study was to examine the association between dietary intake of elite basketball players and postgame changes of creatine kinase, lipid peroxidation and blood cell counts. Fasting blood was collected the morning of a play-off match and two consecutive mornings after the match from 18 elite basketball players of four Greek A1 division teams. Dietary intake was assessed with 24-hours recalls. Despite the relatively low participation time (16.4±6.0 min) a sustained post-game increase of white blood cell, neutrophil and platelet counts was observed. Concerning the creatine kinase changes two types of volunteers, responders and non responders, were discriminated. The increments of white blood cells positively correlated with the active playing time. The nutritional habits of the participants correlated with the post-game blood cell and creatine kinase changes. An increased intake of omega-3 and monounsaturated fatty acids, folate, antioxidants vitamins, choline and calcium favorably associates with blood cell count increments indicating that dietary manipulation of post-game inflammation may have a role in the improvement of the recovery rate of elite basketball players after high intensity games.

Key words: exercise-induced muscle damage; blood cell counts, platelets, creatine kinase, polyunsaturated fatty acids, antioxidants.

Introduction

Top level basketball games are characterized by intermittent physical activity of high intensity (1). The physical demands are substantially increased by the recent changes in the rules which make the game faster, longer and increased overall intensity (2). Most top level players participate in both national and international league championships and their schedule, throughout the season, is extremely heavy since they often play 2 games per week. Moreover, several of these players are required to participate with the national team in tournaments obligations (40-45 days per year in approximation), shortly before the beginning of the pre-season period. This leaves a limited amount of time for recovery. The extent of muscle damage during the game along with the following muscle soreness, tenderness and stiffness are key factors that substantially determine the duration of the recovery time (3).

The limited recovery time results in lower training quality in the subsequent session and/or lower performance in the next game. Therefore, strategies that can diminish muscle damage during the game or its complications after the game may improve the performance of basketball players throughout the competition season. Antioxidant and nutraceutical supplementation, anti-inflammatory drug administration, stretching, massage, cryotherapy are some of the experimental approaches that have been previously tested for their ability to diminish muscle damage. However, for most of them the scientific data are either controversial or not sound enough (4,5).

The strengthening of the antioxidant and immune potential of the athletes through their nutrition seems to be a more physiological and permanent way by which they can face muscle soreness and recover faster after a game (5). However, the impact of the dietary habits on markers of muscle damage and inflammation after a team sport game is not well studied and no studies with elite basketball players have been previously conducted. Under this perspective the aim of this study was to determine the dietary habits of Greek elite basketball players and investigate whether there is a correlation with the postgame changes of exercise-induced muscle damage markers. Moreover, the impact of the dietary intake during the match day on the same markers was also assessed.

Methods

Study protocol

Eighteen Greek elite basketball players of four A1 division teams were recruited. All participants have been playing in top level competition (first and second division of the Greek league) at least 5 years. One week before the game under investigation two non-consecutive 24 h recalls (one during the weekend and one week day) were collected by trained dietitians. Subjects were asked to recall everything consumed the day before the interview and they were also asked whether the day of the 24 h recall was a typical day for their diet. Data on the type and quantity of food consumed were collected with the use of household measures, food models and food photographs. Energy and nutrient intake was assessed with the Nutritionist Pro™ software (Axxya Systems LLC, Stafford, TX) and it was based on the United States Department of Agriculture (USDA) National Nutrient Database for Standard Reference (6), Greek food composition tables (7) or the manufacturer's labels (for commercially available packaged foods). The day of the examined match another 24 h hour recall was collected from each basketball player. The assessment of the dietary intakes was calculated by the average of the three 24 h recalls while the dietary intakes of the match day were separately calculated.

The matches investigated were for the play off stage of the Greek A1 league and were taken place between 17:00-19:00 h. The morning of the match day and two consecutive mornings (10 am) after the match (15 h and 39 h postgame, respectively) fasting blood was collected from the antecubital vein of the volunteers and plasma and serum aliquots were isolated and stored at -80 °C while EDTA plasma was utilized for the complete blood count determination. The protocol was approved by the Bioethics Committee of the Harokopio University and was in accordance with the Declaration of Helsinki (1989) of the World Medical Association. All participants gave their informed written consent in order to participate in the study.

Biochemical and hematology analysis

Complete blood count was determined in EDTA plasma with a Mindray BC-3000 hematology analyzer (Mindray, Shenzhen, P. R. China). The concentrations of serum glucose, triacylglycerols, total cholesterol, HDL cholesterol, uric acid along with the activity of creatine kinase (CK) and lactate dehydrogenase (LDH) were determined with commercially available kits (Biosis, Athens, Greece) modified for 96-well plates according to manufacturer's instructions. LDL cholesterol was calculated by the formula of Friedewald (8).

Serum lipid peroxides were estimated using the thiobarbituric acid reactive substances (TBARS) assay as described previously (9).

Statistical analysis

Normality was tested with the Kolmogorov-Smirnoff criterion. Normally distributed continuous variables are presented as mean values $\pm$ standard deviation. Incremental areas under the curve (AUC) were calculated using the trapezoidal rule. Comparison of antropometric, biochemical and dietary characteristics among players playing in different positions (guards, forwards and centers) were performed by one-way analysis of co-variance (ANOVA) after testing for equality of variances (homoscedasticity). However, because of multiple comparisons, Bonferroni's correction was used to account for the increase in type I error. Correlations were evaluated using the Pearson partial correlation coefficient. For non- normally distributed variables the Spearman partial correlation was used. A repeated measures ANOVA with a post-hoc analysis for comparing mean values of the studied characteristics between the intervention groups, across time points as well as between different time points, was applied using the Bonferroni correction rule to adjust for the inflation of type I error. Linear regression models were applied to test the association between the dietary intake variables and the percent changes of the biochemical and hematological indices after controlling for several potential confounders. All reported p values are based on two-sided tests and compared to a significance level of 5%. The SPSS 14 (SPSS, Chicago, IL, USA) software was used for all the statistical calculations.

Results

Characteristics of volunteers

The majority of our volunteers (N=18) were guards (Guards: 10, Forwards: 3, Centers: 5) and their participation per game, during the previous season, exceeded 10 min for all of them. However, only three of them were key players for their teams playing more than 26 min per game during the full season. Concerning the examined game, all players played over 10 min and the majority of them had a participation time between 10-20 min (16.4 ± 6.0 min). The mean age, height and BMI of the volunteers was 24 ± 4 years, 1.99 ± 0.09 m, 24.5 ± 1.8 Kg/m², respectively.

Alterations of hematological and biochemical markers after the game

Table 1 shows the alterations of basic hematological and biochemical markers observed 15 and 39 hours after the game. A sustained 30% increase of white blood cell counts (WBC) for 2 days after the game was observed. This increase is attributed to the increase of circulating neutrophils which is accompanied by a

decrease of the percentage of lymphocytes. Moreover, a post-game sustained 13% raise of circulating platelets was also observed in our volunteers.

Table 1. Alterations of hematological and biochemical markers after the game

Measured parameter	Pre-game	Post game (15 hours)	Post game (39 hours)	p
WBC ($10^3/\mu\text{L}$)	5.1 ± 1.0 (100 ^b)	6.4 ± 0.8^a (131 $\pm$ 29)	6.4 ± 1.3^b (129 $\pm$ 24)	0.001
NEUT (%)	51.6 ± 8.2 (100)	56.3 ± 10.8 (110 $\pm$ 20)	56.7 ± 8.8^b (111 $\pm$ 9)	0.066
LYMPH (%)	42.5 ± 8.3 (100)	36.9 ± 8.1^a (88 $\pm$ 18)	38.4 ± 9.0 (91 $\pm$ 17)	0.015
RBC ($10^6/\mu\text{L}$)	4.98 ± 0.25 (100)	4.98 ± 0.26 (100 $\pm$ 3)	5.05 ± 0.27 (102 $\pm$ 3)	NS
Hg ($\mu\text{g/L}$)	16.4 ± 0.65 (100)	16.4 ± 0.75 (100 $\pm$ 3)	16.8 ± 0.71 (102 $\pm$ 3)	NS
HCT (%)	44.3 ± 1.71 (100)	44.0 ± 2.09 (99 $\pm$ 3)	44.7 ± 2.03 (101 $\pm$ 3)	NS
PLT ($10^3/\mu\text{L}$)	196 ± 35 (100)	218 ± 40^a (113 $\pm$ 17)	221 ± 38^b (114 $\pm$ 15)	0.005
Glucose (mg/dL)	74.2 ± 4.2 (100)	76.5 ± 9.8 (105 $\pm$ 15)	73.9 ± 11.0 (101 $\pm$ 19)	NS
Triacylglycerols (mg/dL)	93 ± 61 (100)	85 ± 58 (131 $\pm$ 29)	93 ± 56 (131 $\pm$ 29)	NS
Total cholesterol (mg/dL)	158 ± 28 (100)	160 ± 29 (96 $\pm$ 35)	158 ± 32 (107 $\pm$ 23)	NS
LDL cholesterol (mg/dL)	99 ± 27 (100)	102 ± 27 (103 $\pm$ 6)	98 ± 31 (99 $\pm$ 11)	NS
HDL cholesterol (mg/dL)	41 ± 12 (100)	42 ± 11 (103 $\pm$ 8)	41 ± 11 (102 $\pm$ 9)	NS
Uric acid (mg/dL)	4.8 ± 0.8 (100)	5.2 ± 1.1 (108 $\pm$ 15)	4.8 ± 0.9^c (98 $\pm$ 13)	0.016
CK (U/L) (responders)	118 ± 20 (100)	266 ± 54^a (220 $\pm$ 80)	129 ± 43^c (110 $\pm$ 12)	0.029
CK (U/L) (non-responders)	180 ± 80 (100)	184 ± 78 (105 $\pm$ 28)	156 ± 76 (87 $\pm$ 28)	NS
LDH (U/L)	183 ± 31 (100)	187 ± 34 (102 $\pm$ 8)	184 ± 38 (101 $\pm$ 14)	NS
TBARS (μM)	1.35 ± 0.34 (100)	1.37 ± 0.34 (102 $\pm$ 14)	1.41 ± 0.29 (110 $\pm$ 39)	NS

Two types of volunteers were discriminated according to the alterations of CK. The first group, called non-responders (N=8), is consisted of basketball players whose CK values did not increase after the game. The other group, called responders (N=10), showed a transient increase of CK values 15 h after the game. The percent increase of CK ranged from 15-300%.

No significant changes were observed for the other biochemical indices, apart from a small, clinically unimportant increase of uric acid at 15h post-game.

Effect of the players' position, participation in the game and somatometric characteristics on the post game alterations of cell counts and CK

In order to identify the parameters that mostly affect post game changes of cell counts and CK we initially calculated the incremental area under the curve of WBC (AUC_WBC), neutrophils (AUC_NEUT), lymphocytes (AUC_LYMPH), platelet (AUC_PLTS) and CK (AUC_CK) changes over time. The AUCs along with the percent changes of WBC, neutrophils, lymphocytes, platelets and CK 15 hours after the game (WBCPERC_15h, NEUTPERC_15h, LYMPHPERC_15h, PLTSPERC_15h and CKPERC_15h, respectively) were correlated with the somatometric indices of the players (age, height, weight, BMI) and with their participation (in minutes) in the examined game.

The magnitude of the WBC and neutrophil increases after the game, expressed either as AUC or percentage increase at 15 h compared to baseline values, was positively correlated with the participation time in the game [AUC_WBC-Participation: $p=0.518$, $p=0.028$; WBCPERC_15h – Participation: $p=0.448$, $p=0.062$; AUC_NEUT – Participation: $p=0.445$, $p=0.064$]. On the other hand CK increases in the whole sample were not affected by the participation time. Finally, one way ANOVA analysis showed that the position of the players did not affect the changes of cell counts and CK observed after the game (data not shown).

Effect of dietary habits on the postgame changes of cell counts and CK

In order to assess the contribution/preventive role of the dietary habits of the basketball players to the postgame leukocyte and platelet count increments we determined their average dietary intake by collecting three 24 h recalls from each player. The average dietary intakes per Kg of body weight according to all three 24 hour recalls along with the dietary intakes corresponding to the day of the match are shown in Table 2. The mean dietary intakes calculated from the three 24h recalls did not differ from the dietary intakes of the match day.

Table 2. The dietary intakes per Kg of body weight of the basketball players

	Average dietary intakes per Kg of body weight according to the 3 24h recalls	Dietary intakes per Kg of body weight during the match day
Energy (Kcal/Kg)	24.7 ± 7.2	24.0 ± 8.7
Protein (g/Kg)	1.5 ± 0.9	1.4 ± 1.0
Protein (%)	23.0 ± 7.4	22.7 ± 9.7
Fat (g/Kg)	0.95 ± 0.36	0.88 ± 0.33
Fat (%)	33.9 ± 5.9	33.3 ± 6.2
Carbohydrates (g/kg)	2.6 ± 0.8	2.8 ± 0.8
Carbohydrates (%)	45.3 ± 10.5	45.7 ± 11.9
Saturated fatty acids (g/kg)	0.31 ± 0.09	0.28 ± 0.13
Saturated fatty acids (% of total fat)	37.8 ± 7.6	37.2 ± 10.0
Monounsaturated fatty acids (g/Kg)	0.32 ± 0.17	0.32 ± 0.16
Monounsaturated fatty acids (% of total fat)	37.7 ± 6.9	37.9 ± 7.0
Polyunsaturated fatty acids (g/Kg)	0.20 ± 0.09	0.19 ± 0.11
Polyunsaturated fatty acids (% of total fat)	24.8 ± 6.9	25.0 ± 9.5
Cholesterol (mg/Kg)	3.9 ± 2.5	3.7 ± 3.1
Fibers (g/Kg)	0.19 ± 0.07	0.18 ± 0.09
Calcium (mg/Kg)	11.4 ± 6.3	11.9 ± 9.3
Iron (mg/Kg)	0.35 ± 0.16	0.31 ± 0.24
Magnesium (mg/Kg)	3.31 ± 1.95	3.21 ± 2.48
Phosphorus (mg/Kg)	19.0 ± 11.7	17.5 ± 11.5
Potassium (mg/Kg)	36.7 ± 20.4	34.5 ± 21.1
Sodium (mg/Kg)	35.9 ± 10.1	35.5 ± 12.4
Zinc (mg/Kg)	0.2 ± 0.09	0.2 ± 0.2
Vitamic C, total ascorbic acid (mg/Kg)	2.0 ± 2.5	2.1 ± 3.0
Thiamine (mg/Kg)	0.02 ± 0.01	0.02 ± 0.01
Riboflavin (mg/Kg)	0.06 ± 0.04	0.06 ± 0.06
Niacin (mg/Kg)	0.45 ± 0.32	0.41 ± 0.23
Folate (µg/Kg)	4.24 ± 2.05	4.10 ± 2.77
Vitamin A (IU/Kg)	42.8 ± 22.8	41.5 ± 36.3
Vitamin E, α-tocopherol (mg/Kg)	0.08 ± 0.06	0.06 ± 0.07
Vitamin D (IU/Kg)	1.93 ± 0.97	1.51 ± 1.07

The dietary intakes of our players varied considerably according to the three 24-h recalls and although the dietary needs of each players depends on its body mass and daily energy expenditure some generalization can be made. The macronutrient intake for the majority of our subjects coincide with the acceptable macronutrient distribution range which is 45-65% for carbohydrates, 10-35% for proteins and 20-35% for fat (10, 11).

Concerning the quality of the fat, the energy intake from fat should be equally distributed to saturated, monounsaturated and polyunsaturated fatty acids. Our subjects consume slightly lower amounts of PUFA in favor of SFA and MUFA. Moreover, our cohort consumed lower amounts of dietary fibers, vitamin A, vitamin D, vitamin E and magnesium and much higher amounts of phosphorus, sodium, riboflavin, niacin than the DRIs (10,11).

Subsequently, the average dietary intakes calculated from the three 24 h recalls were correlated with the percentage increases of cell counts and CK along with the respective AUCs controlling for height and participation in the game. The significant associations are shown in Table 3. In general the amount of monounsaturated fatty acids (MUFA), polyunsaturated fatty acids (PUFA), folate and calcium are the main dietary components that inversely correlate with the post game increases in blood cell counts. On the other hand dietary fibers seem to be the only dietary component which correlates with reduced post game CK increments.

Table 3. Significant correlations between the dietary intakes of the basketball players and the changes of cell counts and CK

	Dependent variable (b ± SE, r)	
	WBCPERC_24h	AUC-WBC
PUFA (%)	NS	-2.4 ± 0.8, p=0.019
Folate (µg/Kg)	NS	-13.4 ± 4.2, p=0.007
	NEUTPERC_24h	AUC-NEUT
	Energy (Kcal/Kg)	-11.0 ± 6.1, p=0.050
	Fat (g/Kg)	-248 ± 113, p=0.048
	MUFA (g/Kg)	-521 ± 241, p=0.050
	ALA (g/Kg)	NS
	Calcium (mg/Kg)	-14.1 ± 0.58, p=0.051
	CKPERC_24h	AUC-CK
	Fibers (g)	NS

Finally, the dietary intakes of the match day were partially correlated with the percentages increases of cell counts and CK along with the respective AUCs controlling for height and participation in the game. The significant associations are shown in Table 4. MUFA, PUFA, especially omega-3 PUFA such as alpha-linolenic acid (ALA) and docosahexaenoic acid (DHA), calcium, folate but also antioxidant vitamins (vitamin C, vitamin E) inversely correlated with WBC increases. Concerning CK, the saturated fat content of the diet augments CK increases while only dietary choline inversely correlates with the CK peaks.

Table 4. Significant correlations between the dietary intakes of the basketball players during the match day and the subsequent changes of cell counts and CK

	Dependent variable (b ± SE, r)	
	WBCPERC_24h	AUC-WBC
Folate (µg/Kg)	-7.3 ± 3.4, p=0.050	-10.0 ± 2.1, p=0.004
	NEUTPERC_24h	NEUT-NEUT
	Fat (g/Kg)	-255 ± 121, p=0.050
	Calcium (mg/Kg)	-11.3 ± 3.9, p=0.013
	Vitamic C, total ascorbic acid (mg/Kg)	-36.4 ± 12.8, p=0.014
	Vitamin E (mg/Kg)	NS
	Vitamin D (IU/Kg)	NS
	MUFA (g/Kg)	-585 ± 244, p=0.032
	MUFA (%)	-13.7 ± 6.4, p=0.050
	ALA (g/Kg)	NS
	DHA (mg/Kg)	NS
	PLTPERC_24h	AUC-PLT
	Choline (mg)	NS
DHA (mg)	-54141 ± 19175, p=0.014	NS
	CKPERC_24h	AUC-CK
	SFA (g)	NS
Choline (mg)	-29.4 ± 14.2, p=0.047	NS

Discussion

Modern professional basketball exerts significant physical and psychological stress to the athletes especially those playing in top level categories (1,2,12). Elite basketball players usually participate in both national and international tournaments at the same time and they often play 2-3 games per week. This leaves a limited amount of time for their recovery and nutritional strategies for the improvement of the recovery rate and the attenuation of the performance decrements are under investigation. Although intense research has already been made on the repletion of the empty glycogen stores and rehydration (5, 10) the attenuation of the muscle damage's clinical phenotype (pain, loss of muscle function, swelling, stiffness), which may equally impair performance, is much less studied in top levels athletes under real conditions of competition. Under this perspective the nutritional habits of Greek elite basketball players were correlated with increments of inflammatory cell counts and CK activity after a play-off game for the Greek A1 league.

Our cohort consisted of players of all positions but mainly guards while their average participation time in the previous games of the Greek championship were 5-15 min for the majority of them. It is therefore obvious that our volunteers were mainly bench players. Their previous participation time corresponded to the participation time of the examined games which ranged from 12-29 min.

Despite the fact that most players played approximately half a game, a significant sustained increase of WBC was observed due to the increase of neutrophil counts. It is well known that intense exercise induces a transient leukocytosis. The leukocyte increments are mainly due to an increase in the number of neutrophils in

the circulation (13). Exercise not only increases the number of neutrophils in the circulation but also functionally activates them to generate ROS (14). By this way neutrophils exert their physiological role which is the degradation of the inflamed muscle and surrounding connective tissue (15). The post exercise leukocytosis shows a biphasic pattern with an acute increase of neutrophils and lymphocytes favored by the rise of catecholamines and the release of attached neutrophils from the endothelium due to the high pressure (13). The increments observed in our study are due to the delayed phase of leukocytosis which is also characterized by lymphopenia. Delayed leukocytosis seems to reflect exercise-induced muscle damage and determine subclinical inflammation (13). Similar white blood cell counts increments were observed after an experimental basketball match in elite athletes (16).

Platelet counts also increase rapidly after high-intensity exercise (17). The sources of the newly circulating platelets seem to be the lungs, the spleen and bone marrow (18,19). Although no clear clinical significance was attributed to the transient increase of platelets after exercise we should mention that apart from their thrombotic role, platelets may contribute to the post exercise inflammatory activation through their interaction with leukocytes and endothelial cells (20).

In attempt to identify the factors that affect the magnitude of post exercise leukocytosis we clearly demonstrated that the participation in the game is positively correlated with increments of WBC and neutrophils along with the decrements of lymphocytes. It seems that the higher the participation in the game, the most potent the inflammatory response due to higher physiological stress, eccentric loads and catecholamine releases.

The fact that the CK changes after the game did not show associations with the participation time or the WBC changes implies that the inflammatory response should be dissociated from the extent of muscle damage. In fact almost half of our players were non responders showing no elevations of their CK values despite the fact that for most of them WBC increased substantially (5-85 %). The reasons for the discrimination of athletes into high and low responders is not clear but muscle anatomy and genetic reasons may play a role (21). Concerning the responders, CK increases in the range of 20-300% were observed despite the relatively low participation time of our athletes. Previous studies have shown that basketball simulated activities of 10 min impose an intense biochemical response in amateur players accompanied by much higher CK elevations (22). Such striking increments of CK are not expected in our study since our elite athletes have been adapted to this kind of exercise.

Nutrition seems to play a crucial role for the recovery of an athlete after a demanding game such a top level basketball game. However, the impact of the dietary habits on muscle damage induced inflammation and oxidative stress is much less studied, especially, in elite athletes. Moreover, the acute effect of the dietary intakes during the match day on the post exercise inflammatory response has never been studied before. Tables 3,4 demonstrate significant associations between the quality/quantity of the diet and blood cell counts or CK. The associations of the quality and quantity of the fat consumed by the athletes (either long term or the day of the match) seem to be the most prominent ones. Specifically, both the percentages of PUFA, MUFA and the absolute quantities (in g/Kg body weight/day) inversely correlate with the increments of white blood cells, neutrophils and platelets implying that an increased consumption of unsaturated fatty acids in favor of saturated fatty acids may act beneficially on the post-game inflammation. Concerning PUFA, an increased the ratio of omega-3/omega-6 fatty acids can exert anti-inflammatory actions by several mechanisms such as modulation of eicosanoid synthesis, inhibition of cytokine release and reduction of cyclooxygenase-2 expression (23). However, supplementation studies with omega-3 fatty acids or fish oils before exercise showed conflicting results concerning their ability to attenuate post exercise inflammation (24). Probably, the long term dietary consumption of omega-3 from sources (e.g seed oils) that are also rich in antioxidant vitamins (such as vitamin E) may confer better anti-inflammatory protection than a short term supplementation. The inverse correlation of ALA and DHA with neutrophil increments (Table 3,4) implies a favorable role of omega-3 in post-exercise inflammation.

A strong negative correlation of MUFA (both absolute amount and % of fat content) with WBC and platelet counts was also observed in our study. Since the major dietary MUFA source in Greece is the olive oil-derived oleic acid both a direct anti-inflammatory effect of oleic acid (Basu25) or indirect anti-inflammatory effects of olive oil micronutrients (26) can be attributed to the observed protective effects of MUFA.

Another group of micronutrients that seem to have a protective role against the post-game WBC increments is the antioxidant vitamins, especially vitamin C and vitamin E. It is well known that strenuous exercise is accompanied by an increased production of reactive oxygen and nitrogen species that may propagate inflammatory responses (27). Under this perspective, several research groups attempted to attenuate postexercise inflammation by supplementing various combinations of antioxidants of different doses and different supplementation schemes before and during exercise. Until now the results are conflicting and inconclusive (28) while some studies have shown that antioxidant supplementation not only does not protect against exercise induced oxidative stress but it may delay the recovery of the damaged muscle (29). On the other hand a more natural and mild modulation of the exogenous antioxidants intake through diet may be more advantageous, since it avoids the pro-oxidant effects of supranatural intakes of antioxidants and allows the intake of a wide variety of micronutrients which seem to have distinct antioxidant and anti-inflammatory roles thus acting in a synergistic fashion.

A rather novel finding of our study is the inverse association of folate intake with WBC increments. It is well known that folate reduces homocysteine levels since it is an essential cofactor of its metabolism (30). On the other hand, athletes have higher levels of homocysteine than controls (31) and exercise induces a transient hyperhomocysteinemia (32). Since elevated levels of homocysteine may lead to oxidative stress, cytoplasmic calcium influx, cellular apoptosis and endothelial dysfunction (33) adequate amounts of dietary folate may prevent the post exercise increases of homocysteine and by this way the inflammatory processes which activates.

The inverse correlation of choline with CK increments confirms found in our study coincide with the findings of a study which showed that choline deficiency in humans can lead to elevated circulating CK levels. The authors of this study showed that choline deficiency lowers the percentage of phosphatidylcholine in myocytes. This in turn resulted in more fragile myocytes and increased leakage of CK from them (34). Moreover, choline is necessary for the synthesis of neurotransmitter synthesis such as acetylcholine while it acts as a precursor of phosphatidylcholine one of the major phospholipids of cell membranes (35).

Among all minerals calcium seems to have the strongest impact on post exercise neutrophil increments. Calcium deficiency increases intracellular calcium levels, minimizes calcium gradients, induces oxidative stress and by this way impairs muscle contractions (36). Animals studies have shown that exercise may cause an augmented oxidative stress in calcium deficient rats through activation of neutrophils (37).

Conclusions

Despite the relatively low participation time, a significant increase of white blood cell and platelet count was observed in Greek elite basketball players after a play-off game, while CK increments were observed in only half of the players. The nutritional habits of the participants, either long term or during the day of the game seem to associate with the post-game inflammatory response and muscle damage. An increased intake of unsaturated fatty acids, especially omega-3 fatty acids, folate, antioxidants vitamins, choline and calcium act favorably on the observed WBC and CK increments indicating that daily nutritional practices, may play a significant role in the attenuation of exercise induced inflammation and improve the recovery rate after high intensity games.

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