Competition and Food Restriction Effects on Oxidative Stress in Judo

J. Finaud[1]
F. Degoutte[1]
V. Scislowski[2]
M. Rouveix[1]
D. Durand[2]
E. Filaire[1]

Abstract

We examined the effects of weight loss induced by restricting energy and fluid intake on antioxidant status and oxidative stress of judo athletes. Twenty male judoka were randomly assigned to one of two groups (Group A: called diet, n = 10; height 174.8 ± 1.9 cm, body weight 75.9 ± 3.1 kg; they were asked to lose ~ 5 % of their body weight through self-determined means during the week before the competition; Group B: called control, n = 10; height 176.4 ± 1.1 cm, body weight 73.3 ± 6.3 kg maintained their body weight during the week before the competition). A battery of tests was performed during a baseline period (T_1) on the morning of a simulated competition (T_2) and 10 minutes after the end of the competition (T_3). These tests included assessment for body composition, determination of lag phase (Lp) before free radical induced oxidation, maximum rate of oxidation (R_{max}) during the propagating chain reaction and maximum amount of conjugated dienes (CD_{max}) accumulated after the propagation phase, and lipidic profile. Uric acid concentrations were also evaluated in plasma. Dietary data were collected using a 7-day diet record. We noted that the athletes followed a low carbohydrate diet whatever the period of the investigation. Concerning antioxidant nutrients, we can notice that mean nutritional intakes are in the normal range values for vitamin A, C and E at T_1 and T_2. Rapid weight loss induced a significant increase in Lp values ($p < 0.05$) and uric acid concentrations without alterations in oxidative stress. Our data also showed that the competition induced the same changes of oxidative-antioxidant status whatever the dietary intake during the seven days before the competition. Moreover, the effect of the competition on the antioxidant and oxidant parameters was more pronounced than the diet. Theses results could be linked to the food containing a large proportion of PUFA and a relative low proportion of carbohydrates.

Key words
Antioxidants · food restriction · judo · oxidative stress · exercise

Introduction

The increase in reactive oxygen species production during physical exercise may disturb intracellular pro-oxidant-antioxidant homeostasis, inducing oxidative stress that initiates oxidative damage on lipid, protein, and nucleic acids [25]. Antioxidant enzymes (catalase, glutathione peroxidase, superoxide dismutase) and non-enzymatic antioxidants (vitamins E, A and C, glutathione, uric acid) can prevent exercise-induced oxidative stress [5,12]. Physical training (aerobic and anaerobic training) is known to increase the efficiency of the antioxidant defence system and thus to reduce exercise-induced oxidative stress [21,34]. Paradoxically, as physical training requires repeated bouts of physical aerobic endurance training, an increase in energy requirement increases O_2 utilization, which in turn increases free radical oxygen-derivative generation by the mitochondria of ac-

Affiliation
[1] Laboratoire de Biologie Interuniversitaire des Activités Physiques et Sportives, Bat Biologie B, Campus des Cézeaux, Aubière Cedex, France
[2] Institut National de Recherche Agronomique, Unité de Recherches sur les Herbivores, Equipe Nutriments et Métabolismes, INRA de Clermont-Ferrand, Saint-Genes-Champanelle, France

Correspondence
Edith Filaire · UFRSTAPS Campus des Cézeaux · BP 104 · 63177 Aubière Cedex · France ·
Phone: + 33 473 40 76 79 · Fax: + 33 47 34 05 62 · E-mail: efilaire@nat.fr

Accepted after revision: September 20, 2005

Bibliography
Int J Sports Med 2006; 27: 834 – 841 © Georg Thieme Verlag KG · Stuttgart · New York ·
DOI 10.1055/s-2005-872966 · Published online February 1, 2006 ·
ISSN 0172-4622

tive muscles [12]. It has also been noted that diet manipulations can reinforce antioxidant status response to exercise and training [18]. Among them, dietary restriction has been shown to modulate the production of various reactive species in animals [19]. It reduces the production of superoxide and hydroxyl radicals, and inhibits lipid peroxidation and DNA damage [36]. Dietary restriction is also shown to affect antioxidant enzymes in diverse ways, including through its ability to increase SOD, CAT, and GPX activity and expression in the liver, and attenuate the CTA and GPX activity in skeletal muscle [17]. A synergistic effect of dietary restriction and exercise has been observed to protect mitochondrial membrane fluidity against oxidative damage and suppress microsomal reactive oxidant species [16]. All these results are reported in animals, but to our knowledge, information concerning the combined influence of exercise and dietary restriction is not available in humans. Evaluating the influence of dietary restriction and exercise on stress oxidative would be very useful particularly in judo. In fact, judo athletes typically lose weight rapidly by a reduction in food intake for 4 – 5 days prior to competition, sweating through intensive exercise in plastic suits to promote water loss, fluid restrictions and even the use of diuretics [6]. In a previous study, Degoutte et al. [6] reported that a competition including five 5 min bouts induced the same modifications of physiological and psychological variables and performance whatever the dietary intake (dietary restriction or not) during the seven days before the competition. Therefore, the purpose of the present investigation was to mimic a typical major judo championship framework to examine the effects of restricted energy and fluid intake on the antioxidant status and oxidative stress before and after the competition. One can put forward the hypothesis that a dietary restriction induced lower antioxidant intakes and thus restricted-antioxidant defences, which will subsequently increase oxidative stress during rest and to a greater extent during exercise.

Materials and Methods

Subjects

Two groups of male judo competitors (n = 20) at the national level served as subjects in this investigation. Subjects were only allowed to participate in the study if they had previously practiced rapid weight reduction of their own volition more than three times in a season. Their mean period of practicing this sport was 15 years. All participated in nine hours of training per week. For all of them, their technical level ranged between 1st and 4th Dan black belt. All sportsmen fought in a category less than 81 kilograms. Controls anti-doping required by the French Federation of judo were regularly carried out. None of these sportsmen were taking any drugs or medication or taking any supplements. Medical screening indicated that none of the subjects had any endocrine or other medical problems that would confound their participation in the study. All subjects were informed about the possible risks of the investigation before giving their written informed consent to participate, and all procedures were approved by the local ethical committee.

Experimental design

The aim of our study was to examine the effects of a one-day simulated judo competition after a 1 wk weight loss period on antioxidant status and oxidative stress.

- Subjects were randomly allocated into one of two groups (A, B: n = 10/group). All members of Group A (called diet) were asked to lose ~ 5% of their body weight through self-determined means during the week before the competition. The self-determined means were at the discretion of each individual; however, post hoc analysis revealed energy and fluid restriction (– 33% and – 22% respectively).
- All members of Group B (called control) maintained their body weight.

Assessments (weight, performance, psychological test, blood samples) were made during a period of weight maintenance (T_1), after a 7 d food restriction (this food restriction was only carried out by Group A) on the morning of the competition (T_2), and at 10 min after the end of the simulated competition (T_3). The period of weight maintenance (T_1) was considered a baseline phase and judo athletes performed their regular regimens of judo and interval and resistance training. Judo training sessions normally lasted 2 h and consisted of judo specific skills and drills and randori (fighting practice) with varying intensity above and up to 90 – 95% of $\dot{V}O_{2max}$. Judoka were trained one specific training per day except Saturday and Sunday and practiced one conditioning session per week (4 hours of intensive workout, 4 hours of technical work out and 1 hour of conditioning). The experimental timeline is presented in Fig. **1**.

At T_2, the sportsmen all came to the gymnastic hall in the morning at 7 : 30, where they all had a similar breakfast after the weigh-in and the blood sampling. For breakfast, they all had two pieces of bread and one glass of orange juice. We did not evaluate the change in weight gain from weigh-in to the match. After a 20 min warm-up period followed by 10 min of rest, the subjects then participated in a simulated judo tournament with each subject wrestling for a total of five 5 min bouts, carried out under competition conditions, interspersed with 30 min seated recovery periods. Even if thrown the athletes continued until the end of the 5 min period. The first bout took place at 9 : 30 a. m. To create a demanding competitive environment, opponents with similar skills were matched. Each match was formally refereed and scored. The subjects were allowed to consume fluids between bouts as is the usual practice of judo athletes. The liquids/solids that were consumed included on average 300 ml water and energy nutriments (Punch Power®, Fareins, France). Energy nutriments included 1400 KJ. The proportion of total calories from carbohydrates, protein and lipid was 55.9%, 4.9% and 6.7%, respectively. Pulse rate during all the matches was measured using a heart rate monitor (PE4000 Polar Electro, Oy, Finland).

Anthropometric measurements

The weight and height of each subject was measured and the percentage of body fat mass was estimated from four measurements of skinfold thickness according to Durnin and Rahaman [8]. At T_1, T_2 and T_3, body mass was recorded to the nearest 0.1 kg using a portable digital scale with each athlete wearing light clothing and no footwear. Height was measured to the nearest 0.1 cm

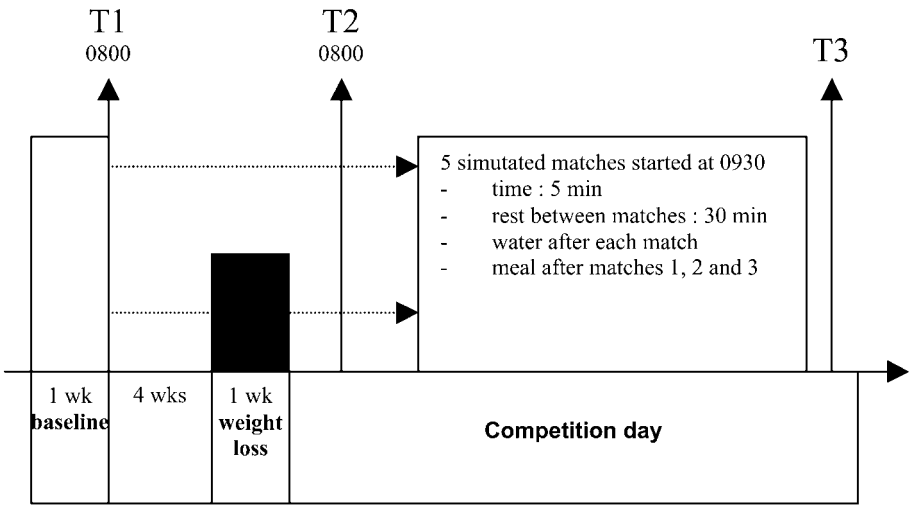

Fig. **1** Experimental timeline. The test battery was performed during a period of weight maintenance (T_1), after a 7 d food restriction (T_2), the morning of the tournament, and at the end of a simulated competition (T_3).

with an anthropometric plane only at T_1. A Harpenden caliper was used to measure the thicknesses, i.e, biceps, triceps, subscapular and suprailiac on the right side of the body with the subject in a standing position. All skinfold measurements were collected by one of the authors, an experienced anthropometrist at T_1, T_2 and T_3. Each skinfold was estimated to 0.1 mm.

Dietary intake

Values for nutrient intakes were obtained from a 7 d food record kept during a period of weight maintenance for both groups and after a 7 d food restriction for group A. The diet for food restriction was self-selected. All participants received a detailed verbal explanation and written instructions. Subjects were asked to be as accurate as possible in recording the amount and type of food and fluid consumed. They were asked to record brand names of all commercial and ready-to-eat foods consumed and method of preparation. A list of common household measures, such as cups and tablespoons, and specific information about the quantity in each measurement (grams, etc.) was given to each participant. Any questions, ambiguities, or omissions regarding the type and amount of food and beverages consumed were resolved individually with each judo athlete and controlled via direct interviews. A color photo exhibit [29] of commonly consumed foods and their portion sizes was used during the interview to assist in estimating amounts consumed. Daily energy and nutrient intakes were calculated by a computer program developed by SCDA Nutrisoft (Bilnut.4 software package, Cerelles, France). This diet analysis program accesses a French nutrient data base for standard reference [24].

Blood collection and biochemical analysis

Blood samples were drawn from the antecubital vein into plain vacutainer tubes in the morning at rest under fasting conditions at 7 : 30 a. m. at T_1 and T_2 and 10 min after the end of the tournament (T_3). To minimize discomfort, all subjects were provided with an anaesthetic cream (EMLA, Astra Pharmaceuticals, Westborough, USA) which was applied over the cubital region 1 hour before each sample. Triglycerides (TG) were analysed by enzymatic techniques in HITACHI 911 (Roche Diagnostics, Meylan, France) according to the manufacturer's protocol. The free fatty acids (FFA) were determined by a manual technique using Wako reagents. Measurements of blood glycerol were conducted using a test kit (Boehringer, Mannheim, Germany). The plasma for these measurements was immediately separated after puncture and conserved at – 20 °C. Susceptibility of plasma poly-unsaturated fatty acid (PUFA) to peroxidation was determined by monitoring the kinetics of accumulation of conjugated dienes (CD) after induction of peroxidation process by copper according to the method described by Schnitzer et al. [26]. After a 50-fold dilution of plasma sample in degazed 0.01 M phosphate buffer saline pH 7.4 (PBS), oxidation reaction was induced at 37 °C by 200 µM of freshly prepared aqueous copper chlorure solution. Absorbance of CD was continuously recorded at 245 nm using a Kontron (Uvikon 923, Saint Quentin en Yvelines, France) double-beam spectrophotometer. The kinetics of CD accumulation can be divided into three phases from which 3 parameters are calculated as described by Esterbauer et al. [9]: 1) the length of the lag phase (Lp) corresponding to the time of resistance of PUFA against oxidation, 2) the maximum rate of oxidation (R_{max}) during the propagating chain reaction, and 3) the maximum amount of CD (CD_{max}) accumulated after the propagation phase. Uric acid, urea and creatinine were determined by a protocol edited for ROCHE in HITACHI 911.

Statistical analyses

Anthropometric data are expressed as a mean and standard deviation. Blood parameters are presented as mean ± SE. Data was analysed using a multivariate analysis of variance (MANOVA) with repeated measures. Where significant main effects were observed, Tukey post hoc procedures were performed to determine pairwise differences. Statistical power was determined to be from 0.80 to 0.85 for the samples sizes used at the 0.05 alpha level. Significance was defined at $p < 0.05$. The SPSS/PC statistical package was used and the criterion for significance was set at $p < 0.05$.

Results

Values for the anthropometric parameters are shown in Table **1**.

Effect of dietary restriction

The anthropometric parameters at T_1 were not significantly different between the two groups. Group A lost a significant

Table **1** Anthropometric data for diet (Group A) and control (Group B) groups at T_1 (weight maintenance, the morning of the competition), [after a 7 d food restriction for Group A] T_2) and at T_3 (at the end of the simulated competition). (Mean ± SD)

	Group A (n = 10)			*Group B (n = 10)*		
Height (cm)	174.8 ± 1.9			176.4 ± 1.1		
Body weight (kg)	**T_1**	**T_2**	**T_3**	**T_1**	**T_2**	**T_3**
	75.9 ± 3.1	72.1 ± 1.4**	74.5 ± 3.4#	73.3 ± 6.3	74.7 ± 6.7	75.1 ± 1.6
BMI (kg/m^2)	24.9 ± 0.8	24.1 ± 0.7	24.2 ± 0.9	23.0 ± 1.3	23.6 ± 1.5	23.9 ± 1.1
FFM (kg)	64.5 ± 1.1	62.2 ± 0.6**		61.7 ± 5.3	62.5 ± 5.3	
% body fat	15.8 ± 1.1	15.0 ± .01*		15.5 ± 3.3	14.9 ± 3.0	

*: $p < 0.05$; **: $p < 0.01$ (T_2 vs. T_1); #: $p < 0.05$ (T_3 vs. T_2)

Table **2** Mean intake of micronutrients for judo athletes (Group A) at T_1 and T_2 (Mean ± ES). For comparison, French norms are listed [24]

	Group A (n = 10)		*Standard*
	T_1	***T_2***	
Energy intake			
($MJ \cdot j^{-1}$)	11.04 ± 1.9	7.02 ± 1.2**	12.54 – 14.63
($kJ \cdot j^{-1}$ kg)	145.35 ± 29.56	97.95 ± 14.94**	
Carbohydrates			
($g \cdot j^{-1} \cdot kg$)	3.9 ± 1.2	3.1 ± 1.3*	6 – 10
(%)	62.9 ± 3.7	57.2 ± 9.8**	60 – 65
Proteins			
($g \cdot j^{-1} \cdot kg$)	1.2 ± 0.1	0.8 ± 0.12**	1.5 – 3
(%)	22.2 ± 12.0	20.3 ± 5.7	15 – 20
Lipids			
($g \cdot j^{-1} \cdot kg$)	1.2 ± 0.18	1.0 ± 0.26*	1.5
(%)	26.3 ± 15.9	22.1 ± 10.9	20 – 30
Water ($g \cdot j^{-1}$)	3873.2 ± 692.5	3102.8 ± 448.3*	3500
Vitamin A (µg)	1500.0 ± 250.2	1449.0 ± 362.8	600 – 800
Vitamin E (mg)	13.5 ± 2.7	12.6 ± 1.9	12
C (mg)	135.2 ± 10.7	124.4 ± 12.8	110

T_2 vs. T_1: *: $p < 0.05$; **: $p < 0.01$

($p < 0.01$) amount of weight from baseline to postweight loss (T_2) that was equivalent to 5% of their total body weight. Percentage of body fat and fat free mass also declined significantly ($p < 0.05$ and $p < 0.01$ respectively). There were no differences in body composition parameters for Group B (Control) between T_1 and T_2. Table **2** shows the dietary intake data at T_1 and T_2 for Group A with the corresponding age-matched evaluation based on French recommendation for athletes [24]. Data for Group B are not presented in this table but there was no difference between dietary intake for Group A and Group B at T_1. The proportion of total calories from lipids and proteins corresponded to the French recommendations at T_1 and T_2. In return, carbohydrate intakes were lower than the recommendations for both groups. Concerning antioxidant nutrients, we can notice that mean nutritional intakes are in the normal range values for vitamin A, C and E at T_1 and T_2. The mean energy intake for Group A during the weight loss period (T_2) was 7.0 ± 1.2 $MJ \cdot d^{-1}$ (– 33% compared to T_1; T_1 = 11.04 ± 1.9 $MJ \cdot d^{-1}$). Intakes of protein, fat and carbohydrates decreased significantly ($p < 0.01$) during this weight loss period. The volume of fluid intake also decreased (from 3873.2 ± 692.5 ml at T_1 to 3102 ± 448.3 ml at T_2. Micronutrients are not affected by the weight loss period. The results of the blood lipid parameters are shown in Table **3**. The standard laboratory values for all parameters are given. Triglyceride concentrations were within the lower limits of the standard of the laboratory for all subjects, whatever the time of the blood sample. Dietary restriction induced a significant increase in blood glycerol and free fatty acid (FFA) concentrations ($p < 0.05$). In the same time, blood concentrations of triglycerides decreased significantly ($p < 0.05$). Table **4** presents data on oxidative stress and antioxidant parameters. Dietary restriction induced no modications in the maximum rate of conjugated dienes oxidation (R_{max}) and in the maximum amount of conjugated dienes (CD_{max}) (Group A: T_2 vs. T_1). However, weight reduction induced a significant increase in Lp values (T_2 vs. T_1: $p < 0.05$; 23%) and a significant increase in blood uric acid concentrations (10%; $p < 0.05$).

Effect of the judo tournament

Each subject participated in a simulated judo tournament wrestling for a total of five 5 min bouts, carried out under competition conditions, interspersed with 30 min seated recovery periods. Participants were allowed to consume fluids between bouts as is the usual practice of judo athletes. No fluid other than water was available. At the end of the competition, Group A had a significant increase in body mass ($p < 0.05$: T_3 vs. T_2), whereas no significant difference in body mass was seen in Group B (Table **1**). No significant differences in body mass were noted between the two groups after the competition (T_3), even if Group A weighed less heavy than Group B. A significant decrease in FFA ($p < 0.05$ in both groups) and triglycerides concentrations ($p < 0.05$ in both groups) were observed betweenn T_2 and T_3. Glycerol concentrations were significantly higher after the competition in Group B ($p < 0.01$, + 45%). A trend to an increase of glycerol concentrations was observed in Group A. For each group, (R_{max}) was significantly lower in T_3 than in T_2 (– 10%; $p < 0.01$ Group A; – 15%; $p < 0.01$ Group B). The maximum amount of conjugated dienes (CD_{max}) remained unchanged throughout the study for each group. Concerning antioxidant parameters, the

Table **3** Mean values (SE) for lipid parameters at T_1 (weight maintenance, after a 7 d food restriction for Group A), T_2 (the morning of the competition), and T_3 (at the end of the simulated competition)

	T_1	T_2	T_3	Standards
Triglycerides (mmol·l^{-1})				
– *Group A*	0.51 ± 0.06	0.45 ± 0.05*	0.26 ± 0.11#	0.5 – 1.6
– *Group B*	0.52 ± 0.07	0.53 ± 0.11	0.40 ± 0.05#	
Free fat acid (mmol·l^{-1})				
– *Group A*	0.33 ± 0.08	0.74 ± 0.80*	0.54 ± 0.11#	0.1 – 0.5
– *Group B*	0.33 ± 0.05	0.40 ± 0.05	0.26 ± 0.05#	
Glycerol (µmol·l^{-1})				
– *Group A*	83.00 ± 14.88	144.22 ± 16.34*	194.87 ± 32.00	50 – 200
– *Group B*	83.22 ± 7.04	126.2 ± 14.29	183.50 ± 18.28##	

T_2 vs. T_1; *: $p < 0.05$; T_3 vs. T_2; #: $p < 0.05$; ##: $p < 0.01$

Table **4** Evolution of maximum rate of oxidation (R_{max}), maximum amount of conjugated dienes (CD_{max}), length of the lag phase (Lp) and uric acid concentrations throughout the study (mean ± ES) for diet (Group A) and control groups (Group B). (T_1: weight maintenance, T_2: after a 7 d food restriction for the Group A: the morning of the competition, T_3: at the end of the simulated competition

	Group A (n = 10)			Group B (n = 10)		
CD_{max} (UA)	T_1	T_2	T_3	T_1	T_2	T_3
	258.1 ± 10.6	254.0 ± 12.3	251.2 ± 16.0	281.5 ± 12.4	270.3 ± 13.2	265.4 ± 11.2
R_{max} (UA)	4.2 ± 0.3	4.1 ± 0.2	3.7 ± 0.1##	4.3 ± 0.3	4.1 ± 0.3	3.5 ± 0.2##
Lp (min)	43.1 ± 4.4	52.9 ± 2.7*	73.5 ± 5.4##	48.1 ± 3.3	59.3 ± 8.9	84.1 ± 6.1#
Uric acid (µmol·l^{-1})	346.6 ± 36.7	382.2 ± 33.6*	580.1 ± 46.5#	365.3 ± 17.2	369.6 ± 20.3	613.0 ± 49.9##

*: $p < 0.05$; (T_2 vs. T_1); #: $p < 0.05$; ##: $p < 0.01$(T_3 vs. T_2)

competition induced a significant increase in Lp (+ 40% [$p < 0.01$] and + 24% [$p < 0.05$] in Group A and B, respectively) and also induced a significant increase in uric acid concentrations (52% Group A: $p < 0.05$; 66% Group B: $p < 0.01$).

Discussion

The objective of this investigation was to determine the effects of a rapid weight loss on oxidative stress before and after a simulated judo competition. Two main results emerged from this study. First, rapid weight loss induced a significant increase in Lp values ($p < 0.05$) and uric concentrations without alterations in oxidative stress. Secondly, our data showed that a competition including five 5 min bouts induced the same changes of oxidant-antioxidant status whatever the dietary intake during the seven days before the competition. In fact, we observed a decreased oxidative stress (R_{max}) after the competition in both groups ($p < 0.01$). At the same time, higher antioxidant protection, as marked by uric acid and lag phase values, are noted in both groups. Moreover, the effect of the simulated competition on the antioxidant and oxidant parameters was more pronounced than the diet.

Effect of the dietary restriction

Food records, as used in this study, are considered the standard for dietary assessment and provide a quantitative account of an individual's diet during a specific period. Although the judo athletes in this study were highly motivated, underreporting errors may have occurred. Therefore, it is important to view the report data as the mean intake for these athletes. Baseline energy intake (11.04 ± 1.9 MJ·d^{-1} at T_1) was within the average range for athletes practicing high intensity weight loss sports but low when compared with 12.5 – 20 MJ·d^{-1} reported for male endurance athletes. The average caloric intake during the food restriction period for the Group A was 7.0 ± 1.2 MJ·d^{-1} inducing a decrease in body weight (4.9 ± 0.06%), which was in accordance with other athletes in weight loss sports [30]. Dietary analysis indicated that the proportion of total calories from lipids and proteins corresponded to the French recommendations at T_1 and T_2. In return, carbohydrate intake were lower than the recommendations for both groups. Intakes of ascorbic acid, Vitamin A and E were in the normal range and were not affected by the dietary restriction. The absorption and utilization of vitamin E depends substantially upon the presence of dietary lipids, particularly polyunsaturated fatty acids (PUFA). There was no difference between groups for total dietary fat intake of PUFA and the proportion of PUFA was high. It has been shown that weight reduction may lead to alterations in lipid profile with a reduction of TG [13], as is the case in our study (Table **3**). This triglycerides decrease and the increase in FFA and glycerol concentrations may be the consequence of the increase in the increased lipolysis in adipose tissue [13] and of hormonal adaptations induced by training, i.e.,

low testosterone, increase in the sensitivity to cortisol, increase in cortisol secretion, which improves lipid utilization [13].

The reduction in diet and in intake of antioxidant rich foods was not enough to alter maximum rate of conjugated dienes oxidation (R_{max}) and in the maximum amount of conjugated dienes (CD_{max}) (as marker of oxidative stress) at rest when compared with a normal diet (Group B) (Table 3), indicating that antioxidant defences after restriction were capable of defending against resting production of reactive oxygen species (ROS). Several markers of oxidative stress have been proposed as being useful to evaluate damage of lipids. Lipid peroxidation leads to the breakdown of lipid and to the formation of a wide array of primary oxidation products such as conjugated dienes (CD) or lipids hydroperoxides (LH), and secondary oxidation products including malondialdehyde (MDA), F2-isoprostane or expired pentane, ethane or hexane [28]. Conjugated dienes measurement is also interesting because it detects molecular reorganization of polyunsaturated fatty acids (PUFA) during the initial phase of lipid peroxidation. In particular, lag phase, an index of the resistance of LDL to *in vitro* oxidation, is now widely proposed as being a reliable index of the antioxidant status in humans [22].Research on energy restriction has produced diverging results. While some workers have found beneficial effects determined by several indicators of oxidative damage [7], others report no detectable difference with a normal diet. For example, in a recent study, Suzuki et al. [29] reported that weight reduction, consisting of both intense exercise and energy restriction, might possibly cause both an increase in oxidative burst activity and decrease in neutrophil phagocytic activity in female judoists. The weight reduction-induced no oxidative stress in the present study can be related to a significant increase in the efficiency of the antioxidant system. Indeed, for the Group A, the length of the lag phase (Lp) corresponding to the time of resistance of PUFA against oxidation is higher in T_2 than in T_1. Such increases of lag phase after dietary restriction-weight loss program are reported in numerous studies [4,22]. However the population studies in these works were obese children or adults. Other studies have shown that dietary restriction in rats enhances the overall antioxidant defence system [3,19]. We also noted in our study a significant increase in uric acid concentrations ($p < 0.05$). Uric acid is an end-product of purine metabolism in humans [14]. Intense physical exercise is known to increase plasmatic concentrations of uric acid [10,14,31]. This parameter has been suggested to act as a major antioxidant *in vivo* [10]. This role is particularly important in plasma where it was shown that uric acid can contribute from 35 to 65% to the total antioxidant capacity in plasma [35]. So, we put forward the hypothesis that energy restriction in these judo athletes increases their antioxidant capacity. However, further investigation is necessary, because even if a significant increase in Lag phase and uric acid concentrations for the Group A after the dietary restriction is noted, no significant differences for these parameters between Group A and B appear at T_2. It is evident from earlier work that dietary lipids have differential effects on the activities of antioxidant capacities. In fact, Venkatraman et al. [33] indicated that consumption of PUFA, particularly of dietary n-3 lipids, not only decreased plasma TG but also significantly influences the activities of antioxidant enzymes and their mRNA expression in rats. In our study, we noted that the TG concentrations were within the lower limits of the standard of the laboratory for all the subjects (Table **3**). In the same time, the proportion of PUFA intake was high for the two groups. It can be speculated that this level of PUFA intake could also have an effect on the antioxidant capacities observed at T_2.

Effect of the judo competition

We observed that Group A again found its initial weight (T_1) at the end of the competition (T_3) with no significant differences between Group A and B being noted at T_3. The subjects were allowed to consume fluids (300 ml) and energy intake between bouts, as is the usual practice of judo athletes. Thus, between the weighing and the end of the competition, each subject could drink 1500 ml over one duration of approximately 2 h 45 min. Then, in agreement with Tarnopolsky et al. [32], the increase of the body mass observed at T_3 for Group A is probably due to the re-hydration. For Group B, the same observation is noted. Indeed, athletes of this group had a higher body weight at T_3 than that reported at T_1, even if this difference was not significant (Table **1**). Our data also showed that a competition including five 5 min bouts induced the same changes of oxidant-antioxidant status, whatever the dietary intake during the seven days before the competition. In fact, we observed a decreased oxidative stress (R_{max}) after the competition in both groups ($p < 0.01$). During the same time, higher antioxidant protection, as marked by uric acid and lag phase values, are noted in both groups. It also appears that the effect of the simulated competition on the oxidant (R_{max}) and antioxidant parameters was more pronounced than the effect of the diet. Oxidative stress and subsequent damage to cellular proteins, lipids, and nucleic acids, as well as changes to the glutathione system, are well documented in response to aerobic exercise [1]. However, far less information is available on anaerobic exercise-induced oxidative modifications. Recent evidence indicates that high intensity anaerobic work does result in oxidative modification to the above-mentioned macromolecules in both skeletal muscle and blood [11]. The increase of free radical production, specific to anaerobic exercise, may be mediated through various pathways in addition to electron leakage, such as during aerobic exercise. Xanthine oxidase production, ischemia-reperfusion, and phagocytic respiratory burst activity seems to be implicated in free radical production during anaerobic exercise. Moreover, the important increases of lactic acid, acidosis, catecholamine and post-exercise inflammation, characteristic in supramaximal exercises, are other factors which can increase the production of FR [11]. Also, it appears that chronic anaerobic exercise training can induce adaptations that act to attenuate the exercise-induced oxidative stress [23,27]. These may be specific to increased antioxidant defences and/or may act to reduce the generation of pro-oxidants during and after exercise [2]. Our results also show that the effect of the simulated competition on the antioxidant and oxidant parameters was more pronounced than the diet. In fact, maximum rate of conjugated dienes oxidation (R_{max}) was significantly lower after the judo competition and the maximum amount of conjugated dienes (CD_{max}) was unaltered in both groups. At the same time and according to Vincent et al. [34], higher antioxidant protection, as marked by uric acid and lag phase values, are noted in both groups. This increase of the antioxidant protection was higher after the competition than after the dietary restriction (+40% for the lag phase values at T_3, +23% at T_2 for Group A). De Oliveira et al. [7] reported that a carbohydrate-energy restriction (30%) increased

resistance to exhaustive exercise considerably in rats without exacerbating oxidative damage. Dietary analysis in our study indicated that the proportion of total calories from carbohydrates were lower than the recommendations for both groups. It may be hypothesis that the low carbohydrate intakes and the adaptation induced by anaerobic exercise training could be implied in the reduction of oxidative stress and in the rise of antioxidant defences observed after the competition in both groups [2,23]. Lipid peroxidation is frequently used as an indication of tissue oxidative stress as a result of free radical attack on the cell membrane. However, several factors may influence lipid peroxidation in the microsomal membranes after consumption of highly unsaturated fatty acids, including the cellular antioxidant enzyme activities, and the cellular concentrations of vitamin E, a chain-breaking antioxidant. It is possible that the level of vitamin E in this study may be adequate to protect the tissue from free radical damage. In fact, Venkatraman et al. [33] put forward the hypothesis that vitamin E from tissues may be getting mobilized to maintain serum vitamin E levels to reduce oxidation during exercise. This assumption could also explain the more significant effect of the competition on the oxidation parameters than those observed during the dietary restriction. However, further investigation are necessary to confirm this hypothesis. In conclusion, our results suggest that the combination of energy restriction and intense exercise training, which causes weight reduction before a competition, seems to enhance the antioxidant capacity of judo athletes before the start of the competition. Our data are the first to note that a competition including five 5 min bouts, induced the same changes of oxidative-antioxidant status whatever the dietary intake during the seven days before the competition. Moreover, the effect of the simulated competition on the antioxidant and oxidant parameters was more pronounced than the diet. These results could be linked to the food containing a large proportion of PUFA and a relative low proportion of carbohydrates, to an adequate nutritional intake of Vitamin E and to the adaptation induced by anaerobic exercise training. Thus, the consideration of a correct diet schedule before and during a judo competition should be taken into account.

Acknowledgements

This study was supported by a grant from the Ministère de la Jeunesse et des Sports (France).

References

[1] Alessio HM. Exercise-induced oxidative stress. Med Sci Sports Exerc 1993; 25: 218 – 224

[2] Bloomer RJ, Goldfarb AH. Anaerobic exercise and oxidative stress: a review. Can J Appl Physiol 2004; 29: 245 – 263

[3] Choi JH, Yu BP. Free radical generation and membrane fluidity of aging brain synaptosomes: modulation by dietary restriction. Free Rad Biol Med 1995; 18: 133 – 139

[4] Dandona P, Mohanty P, Ghanim H. The suppressive effect of dietary restriction and weight loss in the obese on the generation of reactive oxygen species by leucocytes, lipid peroxidation, and protein carbonylation. J Clin Endocrinol Metab 2001; 86: 355 – 362

[5] Dekkers JC, van Doornen LJ, Kemper HC. The role of antioxidant vitamins and enzymes in the prevention of exercise-induced muscle damage. Sports Med 1996; 21: 213 – 238

[6] Degoutte F, Jouanel P, Bègue RJ, Colombier M, Lac G, Pequignot JM, Filaire E. Food restriction, performance, biochemical, psychological, and endocrine changes in judo athletes. Int J Sports Med 2005. Published online April 11, 2005; DOI: 10.1055/s-2005-837505

[7] De Oliveira SL, Diniz DB, Amaya-Farfan J. Carbohydrate-energy restriction may protect the rat brain against oxidative damage and improve physical performance. Br J Nutr 2003; 89: 89 – 96

[8] Durnin JVDA, Rahaman MM. The assessment of the amount of fat in the human bofy from measurements of skinfold thickness. Br J Nutr 1967; 21: 681 – 689

[9] Esterbauer H, Striegl G, Puhl H, Rotheneder M. Continuous monitoring of *in vitro* oxidation of human low density lipoprotein. Free Radic Res Commun 1989; 6: 67 – 75

[10] Filaire E, Lac G, Pequignot JM. Biological, hormonal, and psychological parameters in professional soccer players throughout a competitive season. Percept Motor Skills 2003; 97: 1061 – 1072

[11] Groussard C, Rannou-Bekono F, Machefer G, Chevanne M, Vincent S, Sergent O, Cillard J, Gratas-Delamarche A. Changes in blood lipid peroxidation markers and antioxidants after a single sprint anaerobic exercise. Eur J Appl Physiol 2003; 89: 14 – 20

[12] Ji LL. Antioxidants and oxidative stress in exercise. Proc Soc Exp Biolo Med 1999; 222: 283 – 292

[13] Jeukendrup AE, Saris WHM, Wagenmakers AJM. Fat metabolism during exercise: a review. Part III: Effects of nutritional interventions. Int J Sports Med 1998; 19: 371 – 379

[14] Kaur H, Halliwell B. Action of biologically-relevant oxidizing species upon uric acid. Identification of uric acid oxidation products. Chem-Biol Interact 1990; 73: 235 – 247

[15] Kayatekin BM, Gönenç S, Açikgöz O, Uysal N, Dayi A. Effects of sprint exercise on oxidative stress in skeletal muscle and liver. Eur J Appl Physiol 2002; 87: 141 – 144

[16] Kim JD, Mc Carter RJM, Yu BP. Influence of age, exercise, and dietary restriction on oxidative stress in rats. Aging Clin Exp Res 1996; 8: 123 – 129

[17] Koizumi A, Weindruch R, Walfrod RL. Influences of dietary restriction and age on liver enzyme activities and lipid peroxidation in mice. J Nutr 1987; 117: 361 – 367

[18] Margaritis I, Palazzetti S, Rousseau AS, Richard MJ, Favier A. Antioxidant supplementation and tapering exercise improve exercise-induced antioxidant response. J Am Coll Nutr 2003; 22: 147 – 156

[19] Matsuo M, Gomi F, Kuramoto K, Sagai M. Caloric restriction suppresses age-dependent increases in the respired rates of hydrocarbon from rat. J Geront Biol Sci 1993; 48: B133 – B138

[20] McBride JM, Kraemer WJ, Triplett-McBride T. Effect of resistance exercise on free radical production. Med Sci Sports Exerc 1998; 30: 67 – 72

[21] Miyazaki H, Oh-ishi S, Ookawara T, Kizaki T, Toshinai K, Ha S, Haga S, Ji LL, Ohno H. Strenuous endurance training in humans reduces oxidative stress following exhaustive exercise. Eur J Appl Physiol 2001; 84: 1 – 6

[22] Mohn A, Catino M, Capanna R, Giannini C, Marcovecchio M, Chiarelli F. Increased oxidative stress in prepubertal severely obese children: effect of a dietary restriction-weight loss program. J Endocrinol Metabol 2005; 10: 2653 – 2658

[23] Ortenblad N, Madsen K, Djurhuus MS. Antioxidant status and lipid peroxidation after short-term maximal exercise in trained and untrained humans. Am J Physiol 1997; 272: R1258 – R1263

[24] Peres G. Physiologie de l'exercice musculaire et nutrition du sportif. In : Brunet-Guedj E, Comtet B, Genety J. Abrégé de Médecine du Sport. 6th edn. Paris: Masson, 2000

[25] Powers SK, De Ruisseau K, Quindry J, Hamilton K. Dietary antioxidants and exercise. J Sports Sci 2004; 22: 81 – 94

[26] Schnitzer E, Pinchuk I, Fainaru M, Schafer Z, Lichtenberg D. Copper-induced lipid oxidation in unfractionated plasma: the lag preceding oxidation as a measure of oxidation-resistance. Biochem Biophys Res Commun 1995; 216: 854 – 861

[27] Selamoglu S, Turgay F, Kayatekin BM et al. Aerobic and anaerobic training effects on the antioxidant enzymes in the blood. Acta Physiol Hung 2000; 87: 267 – 273

[28] Sjödin B, Hellsten Westing Y, Apple GS. Biochemical mechanism for oxygen free radical formation during exercise. Sports Med 1990; 10: 236 – 254

[29] Su-vi-max. Portions alimentaires, Manuel photos pour l'estimation des quantités. "Food portions, Manual photos for the evaluation of quantities". Candia e.d. Paris: Polytechnica, 1994

[30] Suzuki M, Nakaji S, Umeda T, Shimoyama T, Mochida N, Kojima A, Mashiko T, Sugawara K. Effects of weight reduction on neutrophil phagocytic activity and oxidative burst activity in female judoists. Luminescence 2003; 18: 214 – 217
[31] Svensson M, Ekblom B, Cotgreave I, Norman B, Sjöberg B, Ekblom Ö, Sjödin B, Sjödin A. Adaptative stress response of glutathione and acid uric metabolism in man following controlled exercise and diet. Acta Physiol Scand 2002; 176: 43 – 56
[32] Tarnopolsky MA, Cipriano C, Woodcroft C, Pulkkinen WJ, Robinson D, Henderson J, MacDougall JD. The effects of rapid weight loss and wrestling on muscle glycogen concentration. Clin J Sport Med 1996; 6: 78 – 84
[33] Venkatraman JT, Angkeow P, Satsangi N, Fernandes G. Effects of dietary n-6 and n-3 lipids on antioxidant defense system in livers of exercised rats. J Am Coll Nutr 1998; 6: 586 – 594
[34] Vincent KR, Vincent HK, Braith RW, et al. Resistance exercise training attenuates exercise-induced lipid peroxidation in the elderly. Eur J Appl Physiol 2002; 87: 416 – 423
[35] Wayner DDM, Burton GW, Ingold KU, Barclay LRC, Locke SJ. The relative contributions of vitamin E, urate, ascorbate and proteins to the total peroxyl radical-trapping antioxidant activity of human blood plasma. Biochim Biophys Acta 1987; 924: 408 – 419
[36] Yu BP, Chung HY. Stress resistance by caloric restriction for longevity. Ann NY Acad Sci 2001; 928: 39 – 41