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ORIGINAL ARTICLE

Energy demands during a judo match and recovery

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Objective: To assess energy demand during a judo match and the kinetics of recovery by measuring the metabolites of the oxypurine cascade, lipolytic activity, and glycolytic pathway.**Methods:** Venous blood samples were taken from 16 national judoists (mean (SEM) age 18.4 (1.6) years), before (T_1) and three minutes (T_2), one hour (T_3), and 24 hours (T_4) after a match. A seven day diet record was used to evaluate nutrient intake.**Results:** Nutrient analysis indicated that these athletes followed a low carbohydrate diet. Plasma lactate concentration had increased to 12.3 (1.8) mmol/l at the end of the match. An increase in the levels of extracellular markers of muscle adenine nucleotide catabolism, urea, and creatinine was observed at T_2 , while uric acid levels remained unchanged. High concentrations of urea persisted for 24 hours during the recovery period. Ammonia, hypoxanthine, xanthine, and creatinine returned to control levels within the 24 hour recovery period. Uric acid concentrations rose from T_3 and had not returned to baseline 24 hours after the match. The levels of triglycerides, glycerol, and free fatty acids had increased significantly ($p < 0.05$) after the match (T_2) but returned to baseline values within 24 hours. Concentrations of high density lipoprotein cholesterol and total cholesterol were significantly increased after the match.**Conclusions:** These results show that a judo match induces both protein and lipid metabolism. Carbohydrate availability, training adaptation, and metabolic stress may explain the requirement for these types of metabolism.

Judo is a dynamic, physically demanding sport that requires complex skills and tactical excellence for success.¹ It is characterised by short duration, high intensity, intermittent exercise lasting a total of 7.18 (0.2) minutes per match for men.² Sikorski *et al*² concluded that the primary source of energy during a judo match is anaerobic glycolysis. However, this is debatable.^{3,4} To obtain an understanding of the physiological capacities underlying judo performance, some studies have analysed muscle fibre composition and fibre area or have characterised elite judoists by relating their physical and physiological responses to standard human performance measures.^{1,5} The energetic requirements of a competitive match need to be analysed to provide benchmarks for developing athletes and to improve the monitoring of training.

The purpose of this investigation was to evaluate the substrates used in response to a judo match and during recovery, using biological variables that reflect the glycolytic pathway, protein catabolism, and lipolytic activity. We therefore measured plasma and/or serum concentrations of lipids and lipoprotein, extracellular markers of muscle adenine nucleotide catabolism (ammonia, hypoxanthine, xanthine, and uric acid), urea, and lactate. We also examined food intake, as nutrition has a role in substrate mobilisation.

METHODS

Subjects

Sixteen male judo competitors at interregional level (mean (SEM) age 18.4 (1.6) years, body mass 74.9 (4.7) kg, height 177.4 (5.4) cm, percentage of body fat 16.0 (1.8), fat free mass 62.9 (3.8) kg) were enrolled in the study. They had practised judo for a mean (SEM) of 10 (3.2) years. All trained for six to eight hours a week. Technical levels ranged between 2nd and 3rd Dan black belt. None were taking drugs, medication, or supplements. None had any endocrine or other medical problems that would confound the results. All were informed about the risks of the investigation before giving their written consent, and all procedures were approved by the local ethics committee.

Data were collected at the end of October during a period of weight maintenance. They came from a physical examination, assessment of heart rate during a judo match, and determination of biological variables in plasma or serum. To avoid the effects of circadian rhythm on adenosine metabolism, the experiment was performed in the morning.⁶

There was an obligatory 30 hour minimum recovery period and no physical training before the study.

Procedures

Blood samples were obtained from seated subjects at rest at 1100 (baseline values), after a judo match which began at 1130, and during the recovery period (three minutes, 60 minutes, and 24 h after the match). The sportsmen all came to the gym in the morning at 0730, where they all ate the same breakfast, which consisted of two pieces of bread with jam and a glass of orange juice.

After a 20 minute warm up period followed by 10 minutes of rest, all subjects were instructed to participate in a judo match lasting five minutes, carried out under competition conditions. Even if thrown, the athletes continued until the end of the five minute period. To create a demanding competitive environment, opponents with similar skills were matched.

Determination of characteristics

Anthropometric measurements were taken three days before the judo match. The weight and height of each subject was measured, and the percentage body fat mass was estimated from four measurements of skinfold thickness as described by Durnin and Rahaman.⁷ 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

Abbreviations: HDL-C, high density lipoprotein fraction of cholesterol; VLDL-C, very low density lipoprotein fraction of cholesterol; LDL-C, low density lipoprotein fraction of cholesterol

Table 1 $\dot{V}O_{2\text{MAX}}$, heart rate at $\dot{V}O_{2\text{MAX}}$ (HR_{MAX}), heart rate (HR) (beats/min) at rest, and the mean of the heart rate during the judo match (HRM) of male athletes (n=16)

	$\dot{V}O_{2\text{MAX}}$ (ml/min/kg)	Baseline HR (beats/min)	HRM (beats/min)	HR_{MAX} (beats/min)
	55.0 (0.5)	54.7 (0.2)	182.4 (0.4)	198.2 (0.7)
SV	49–57.5		175–185	

Values are mean (SEM). For comparison, the standard values (SV) of competitive male judo athletes are given.¹²

to the nearest 0.1 cm with an anthropometric plane. A Harpenden caliper was used to measure skinfold thicknesses—that is, biceps, triceps, subscapular, and suprailliac—on the right side of the body with the subject standing. All skinfold measurements were collected by one of the authors, an experienced anthropometrist. Each skinfold was estimated to 0.1 mm.

Dietary intake

Nutrient intakes were determined from a seven day food record kept during the week of the protocol. All participants received a detailed verbal explanation and written instructions. Subjects were asked to maintain their usual dietary habits during the recording period and 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 the methods of preparation. A list of common household measures, such as cups and tablespoons, and specific information on 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 during individual interviews. A colour photo exhibition⁸ of commonly consumed foods and their portion sizes was used during the interview to assist in estimating amounts consumed. Each subject's diet was assessed using the PRODIET 5.1 software package, a computerised database (Proform) that calculates the food composition from the French standard reference.⁹

For the assessment of maximal oxygen uptake ($\dot{V}O_{2\text{MAX}}$), subjects performed a continuous incremental test to exhaustion on an electrically braked ergometer cycle (model EO252E; Siemens, Solna, Sweden). Subjects performed a five minute warm up at 120 W followed by an initial work rate of 180 W, with increments of 30 W every two minutes until fatigue. During the second minute of each increment, an expired gas sample was collected. The Douglas bag collection system was used for this purpose. A paramagnetic oxygen analyser (Servomex 1420B, Crowborough, Sussex, UK) and an infrared carbon dioxide analyser (Servomex 1415B) were used along with a dry gas meter (Harvard Apparatus, Edenbridge, Kent, UK) to determine minute ventilation, $\dot{V}O_2$, and carbon dioxide output. The basic criteria for attainment of $\dot{V}O_{2\text{MAX}}$ were

adopted.¹⁰ Heart rate was measured continuously during both the $\dot{V}O_{2\text{MAX}}$ test and the match using a short range telemetry device (PE4000 Polar Electro, Oy, Finland).

Blood collection and biochemical analysis

Blood samples were drawn from the antecubital vein into plain vacutainer tubes in the morning at rest at 1100 (before the warm up period; T_1), and at three minutes (T_2), 60 minutes (T_3), and 24 hours (T_4) after the match which began at 1130. To minimise discomfort, all subjects were provided with an anaesthetic cream (EMLA; Astra Pharmaceuticals, Westborough, Massachusetts, USA) which was applied over the cubital region two hours before the match.

The proportion of cholesterol and triglycerides was analysed by enzymatic techniques in an Hitachi 911 (Roche Diagnostics, Mannheim, Germany) according to the manufacturer's protocol. The high density lipoprotein fraction of cholesterol (HDL-C) was measured after precipitation of the very low density lipoprotein (VLDL-C) and low density lipoprotein (LDL-C) fractions with phosphotungstic acid. LDL-C was precipitated with Biomerieux reagent.

Free fatty acids were determined by a manual technique using Wako reagents.

Blood glycerol and ammonia concentrations were measured using a test kit (Boehringer Mannheim). The plasma for these measurements was immediately separated after venepuncture and conserved at -20°C .

Uric acid, urea, and creatinine were determined by a protocol edited for Roche in Hitachi 911.

Xanthine and hypoxanthine were determined by high performance liquid chromatography, using a C18 column, and detected at 220 nm by the method of Terzuoli *et al.*¹¹ Intra-assay variability was <5%.

Fingertip capillary blood samples were collected in a capillary tube and analysed for lactate concentration using a Dr Lange LP420 Photometer (Konisburg, Germany). Blood lactate concentration was determined by enzymatic oxidation analysis.

Statistical analysis

All data were analysed using the SPSS/PC statistical package (version 10.0). They are expressed as mean (SEM). Non-parametric analyses (Friedman followed by a Wilcoxon's signed rank test to determine the location of the significant differences) were used in the statistical computations for all variables. The level of significance was set at $p=0.05$ for all statistical tests.

RESULTS

Table 1 shows $\dot{V}O_{2\text{MAX}}$ as well as heart rate monitored at rest, during the match, and at $\dot{V}O_{2\text{MAX}}$. The average heart rate monitored during the match corresponded to 92% of maximal.

Tables 2 and 3 show biological variables before and after the match, and within the recovery period. The standard laboratory values are also given. All the biological variables in our study corresponded to the standards.

Table 2 Effect of the judo match on serum lipid and lipoprotein levels

	TC (mmol/l)	TG (mmol/l)	FFA (mmol/l)	Glycerol ($\mu\text{mol/l}$)	LDL-C (mmol/l)	HDL-C (mmol/l)
Resting values (T_1)	4.50 (0.2)	0.86 (0.08)	0.40 (0.03)	74.0 (4.44)	2.9 (0.10)	1.23 (0.06)
3 min after match (T_2)	4.76 (0.1)*	1.15 (0.1)*	0.57 (0.05)*	148.4 (15.8)*	2.9 (0.1)	1.33 (0.08)*
60 min after match (T_3)	4.5 (0.2)	0.75 (0.07)	0.54 (0.03)*	117.0 (13.2)*	2.88 (0.2)	1.26 (0.06)
24 h after match (T_4)	4.22 (0.2)**	0.88 (0.1)	0.50 (0.04)	85.3 (7.0)	2.73 (0.2)	1.12 (0.06)**
Norm	3–6	0.5–1.6	0.1–0.5	50–100	2.9–4	0.75–1.6

Data are mean (SEM), n=16. For comparison, reference ranges for adults are listed (Norm).

Significantly different from resting values: * $p<0.05$; ** $p<0.01$.

TC, Total cholesterol; TG, triglycerides; FFA, free fatty acids; LDL-C, low density lipoprotein cholesterol; HDL-C, high density lipoprotein cholesterol.

Table 3 Effect of the judo match on plasma concentrations of lactate, ammonia, hypoxanthine, xanthine, uric acid, urea, and creatinine at rest and after the match

	Ammonia ($\mu\text{mol/l}$)	Hypoxanthine ($\mu\text{mol/l}$)	Xanthine ($\mu\text{mol/l}$)	Uric acid (mmol/l)	Urea (mmol/l)	Creatinine (mmol/l)	Lactate (mmol/l)
Resting values (T_1)	62.3 (4.3)	14.4 (1.0)	4.6 (1.0)	324.5 (10.8)	5.2 (0.2)	101.6 (1.5)	1.2 (0.1)
3 min after match (T_2)	141.5 (12.6)**	15.8 (1.1)*	16.0 (2.9)*	327.0 (11.0)	5.6 (0.1)**	111.6 (1.7)**	12.3 (0.8)***
60 min after match (T_3)	99.0 (11.5)**	15.6 (0.8)*	12.8 (4)*	385.8 (11.7)**	6.0 (0.2)**	107.5 (1.4)*	1.8 (0.2)*
24 h after match (T_4)	57.2 (7.8)	15.1 (1.0)	5.4 (1.1)	356.1 (16.3)*	6.0 (0.3)*	103.6 (2.4)	1.0 (0.1)
Norm	10–46	6.1–19.3		200–360	3–7.5	50–120	

Data are mean (SEM), n=16. For comparison, reference ranges for adults are listed (Norm). Significantly different from resting values: * $p<0.05$; ** $p<0.01$; *** $p<0.001$.

Effect of the judo match

The level of triglycerides, free fatty acids, and glycerol had risen significantly ($p<0.05$) three minutes after the match (T_2) (table 2). We observed a significant increase in HDL-C concentration three minutes after the exercise followed by a gradual decrease, reaching a level below the baseline at T_4 . The same pattern of response was observed for total cholesterol. No modifications in LDL-C were noted during the study.

Plasma lactate concentration was 10 times higher at T_2 than the resting level (table 3).

Levels of ammonia, hypoxanthine, xanthine, and urea had increased significantly three minutes after the match. Uric acid levels remained unchanged from T_1 to T_2 .

Creatinine concentrations increased by 10% from T_1 to T_2 .

Recovery period

Triglycerides, free fatty acids, and glycerol concentrations returned to resting values within 24 hours of the match (table 2). Plasma lactate concentration had fallen from 12.3 (0.8) mmol/l to 1.8 (0.2) mmol/l 60 minutes after the match. We noted a significant increase in uric acid levels 60 minutes after the end of the match (T_3) compared with resting values, and these concentrations remained elevated the following morning (T_4). Peak urea levels were significantly enhanced at T_3 and remained elevated 24 hours after the match (table 3). Ammonia, hypoxanthine, and xanthine concentrations remained elevated one hour after the match (T_3), but were significantly lower than those obtained at T_2 . They returned to prematch levels within 24 hours. Creatinine concentrations had returned to prematch levels within 24 hours.

Table 4 shows the main data on dietary intake and the corresponding evaluation based on French recommendations for sportsmen.⁹ Assuming that 50.1 kJ/h/kg energy was spent during judo training, and that the athletes actively trained for two hours, a mean value of 7.3 (0.5) MJ was estimated for daily energy expenditure during training.¹² Mean daily energy

expenditure (13.9 (0.6) MJ) was approximately equivalent to mean energy intake (13.4 (0.8) MJ). The contributions of protein, fat, and carbohydrate to total energy intake were 15%, 33%, and 51% respectively. The average intake of polyunsaturated fat was low.

DISCUSSION

This study examined the substrates used in response to a judo match and during recovery, using biological variables that reflect the glycolytic pathway, protein catabolism, and lipolytic activity. The average $\text{V}_{\text{O}_2\text{MAX}}$ was close to those recorded in other studies,¹² therefore our population can be considered as being representative of the national judo population.

Two major findings emerged.

(1) At the end of the match, we noted an increase in ammonia, urea, hypoxanthine, and xanthine concentrations with no change in uric acid. An increase in triglycerides, free fatty acids, glycerol, and HDL-C was also noted.

(2) All these variables returned to baseline within 24 hours except for uric acid and urea, which had increased substantially one hour after the end of the match and remained elevated the following morning (T_4).

Effect of the judo match

Our values for the lipolytic variables were in agreement with the reference norms (table 2), suggesting that judo training has no effect on this profile. This result agrees with previous studies indicating that sportsmen participating in speed or power events, such as weightlifting, have lipid values similar to those of sedentary controls.¹³

Our results also show that a judo match induced a significant increase in concentrations of triglycerides, free fatty acids, and glycerol. A rise in HDL-C levels was also noted. Limited information is available on the effect of anaerobic exercise on blood lipid concentrations.^{14 15} Results from several

Table 4 Nutritional assessment of the daily dietary intake of the judo athletes

		FRDA
Total energy intake (MJ/day)	13.4 (0.8)	12.5–14.6
Daily energy expenditure (MJ/day) with no training	7.6 (0.3)	
Daily sport activity (MJ/day)	7.3 (0.5)	
Daily energy expenditure (MJ/day)	13.9 (0.6)	
Protein (%)	15.3 (2.0)	15
Fat (%)	33.0 (4.3)	<30
Carbohydrate (%)	50.9 (4.5)	60
Total protein (g/day)	103.4 (26.0)	81
Total fat (g/day)	100.5 (31.4)	100
Carbohydrate (g/day)	333.2 (91.8)	450
Water (g)	4045.0 (350)	3500
Saturated fat (g)	38.8 (2.8)	1/3 of total fat
Monounsaturated fat (g)	31.6 (3.9)	1/3 of total fat
Polyunsaturated fat (g)	9.3 (1.9)	1/3 of total fat

Values are mean (SEM). For comparison, the French Recommended Dietary Allowances (FRDA) for sportsmen⁹ are shown.

studies show that moderate exercise intensity (40–65% $\text{VO}_{2\text{MAX}}$) leads to a rise in plasma free fatty acids.¹⁶ In contrast, increased plasma lactate levels are associated with low levels of free fatty acids during exercise at high workloads.¹⁷ The plasma lactate levels at T_2 (table 3) are in accordance with levels evaluated after a judo competition,⁷ and the heart rate recorded during the match corresponded to 92% of maximal. This intensity corresponds to that observed in national competitions.¹² These two variables are presumably an expression of a partly anaerobic power exercise, although an increase in plasma free fatty acids, glycerol, and triglycerides was observed. One can put forward several hypotheses to explain this. Firstly, it may be linked to the total duration of exercise, which includes the 20 minute warm up period and the length of the match.

It has also been shown that carbohydrate availability and nutritional and training status are factors that determine the use of lipid fuel sources by exercising muscles.¹⁶ Taking into account the daily sporting activity of the athletes, we found that their daily energy intake was equivalent to their energy requirement (13.4 (0.8) v 13.9 (0.6) respectively; table 4). However, the average carbohydrate intake was below the French recommended dietary allowance.⁹ Although the amount of carbohydrate needed by judo athletes on a daily basis is not known, a low carbohydrate intake may impair recovery, and an intake of <500 g/day may be too small to ensure rapid glycogen resynthesis after training sessions.¹⁸ It may thus be hypothesised that the low carbohydrate intake noted in these judo athletes increases the rate of adipose tissue lipolysis, resulting in a rise in plasma free fatty acids and glycerol concentrations. Moreover, athletes are used to dieting often during the year, inducing an increase in lipolysis of triglycerides in adipose tissue and circulating triglycerides.¹⁹ The increase in free fatty acids, glycerol, and triglycerides observed in our study may also be the consequence of metabolic adaptations induced by both the low glycogen stores and the hormonal adaptations elicited by training—for example, sensitivity to catecholamines, which improves lipid utilisation.²⁰ The activity of lipoprotein lipase must also be taken into account. As the activity of the skeletal muscle enzyme increases after vigorous exercise, the enhanced lipolysis in adipose tissue may increase plasma levels of free fatty acids, which in turn promote the synthesis of VLDL triglycerides in the liver. This process increases plasma triglyceride concentration, ensuring a high rate of hydrolysis by lipoprotein lipase in the working muscle.²¹

We also observed a significant increase in total cholesterol concentrations three minutes after the exercise followed by a gradual decrease to below the baseline at T_4 (table 2). The same has been observed in other studies and may be attributed to the possible haemoconcentration induced by the match and the warm up period.^{22–25} Serum creatinine levels in this study were elevated after the match, suggesting a reduction in renal plasma flow and glomerular filtration and an increase in plasma volume shifts.²⁴

Endurance training is associated with increased HDL-C concentrations.¹³ Furthermore, acute endurance activities lead to a rise in HDL-C levels. However, little is known about whether HDL-C changes as a result of short term anaerobic exercise. In this study, the HDL-C concentration was significantly above the basal level after a five minute judo match (T_2 v T_1 , $p < 0.05$). An increase in HDL-C concentration was also noted in trained swimmers after anaerobic swimming, HDL-C playing a physiological role in the free fatty acid transport system.²⁵ It has been suggested that the increase in HDL-C is induced by an increase in lipoprotein lipase, as this activity is increased after vigorous exercise.²⁶ Therefore the increase in HDL-C after the judo match in our study may also be due to an increase in lipoprotein lipase activity. Modifications in plasma volume shifts may also have induced the transient changes in HDL-C levels observed in our study.²⁷

Our results also show an increase in ammonia concentration three minutes after the match (table 3). It is well accepted that the rise in ammonia after short term intensive exercise derives from the first branch of the purine nucleotide cycle catalysed by adenylate deaminase.²⁸ These reactions take place in all muscle fibres, in relation to the intensity of the metabolic stress and glycogen availability.²⁹ Sahlin *et al*³⁰ suggested that reduced availability of muscle glycogen impairs ATP resynthesis, leads to AMP accumulation, and induces ammonia production. It has also been shown that a low carbohydrate diet induces changes in hormones, such as glucagon, catecholamines, and glucocorticoids, leading to increased protein breakdown and reduced pre-exercise muscle buffering capacity, which induces earlier AMP deaminase activation.³¹ The increase in ammonia concentrations observed in our study may be the consequence of several factors including activation of type II fibres, low carbohydrate diet, peripheral fatigue, and increased activity of the purine nucleotide cycle.³² Sutton *et al*³³ suggested that increased activity of the purine nucleotide cycle is reflected in increased plasma oxypurine levels. In this study, the whole oxypurine cascade from hypoxanthine to uric acid was activated (table 3). However, our data indicate a delay between the mean peak plasma uric acid concentration (T_1) and the mean hypoxanthine and xanthine peaks (T_2). Westing *et al*³⁴ suggested that such delays may be due to the time required for hypoxanthine and xanthine to diffuse into the tissue before uric acid can be produced, or may be the time required for activation of xanthine dehydrogenase/oxidase. In fact, it has been shown that xanthine dehydrogenase may, under various conditions, including metabolic stress, be converted into an oxidase form that generates reactive oxygen radicals. Therefore, a high activity of this enzyme may be harmful to a tissue such as muscle, which is subjected to large alterations in metabolic demand.²⁸

It has also been hypothesised that uric acid plays a role in the antioxidative defence of the muscle during exercise.³⁵ Thus, the rise in uric acid levels noted at T_3 may represent the loss of useful nucleotide precursors and may be of importance in protecting tissues against reactive oxygen species.

It has also been shown that uric acid is an indicator of carbohydrate availability and muscle glycogen stores.² In fact, low carbohydrate diets (as noted in table 4) may compromise carbohydrate utilisation so that protein catabolism may be required. Therefore the low carbohydrate diets observed in our study may lead to an increase in uric acid from purine nucleotide degradation. The rise in urea concentration observed at T_2 also suggests involvement of proteins in the oxidation of substrates during the judo match.

Recovery period

After a one hour recovery period, the concentrations of triglycerides, free fatty acids, and glycerol had decreased (table 2). These results are in accordance with previous studies.³⁶ In fact, Lithell *et al*³⁷ showed that, after exercise, skeletal muscle and adipose tissue lipoprotein lipase activity remain elevated, hepatic production of triglycerides decreases because of a decrease in free fatty acid release, and plasma triglyceride concentration decreases. It is, however, worth noting that the plasma levels of triglycerides, free fatty acids, and glycerol remained elevated compared with the resting values. This indicates that lipid mobilisation remained enhanced for at least one hour after the match. This suggests that lipolysis participates in the resynthesis of muscular glycogen stores by providing free fatty acids as fuel substrates and glycerol as a substrate for hepatic neoglucogenesis.

The extracellular markers of muscle adenine nucleotide catabolism and protein catabolism induced by the match appear to have returned to baseline levels within 24 hours of the match (table 3). These results agree with those of other studies.³⁴ The high levels of uric acid and urea noted 24 hours

after the combat suggest that the process of recovery is not complete. This has also been noted previously.^{34, 38} These authors suggested that the elevated uric acid concentrations may be important in protecting tissues against reactive oxygen species.

Conclusion

Although studies on the physiological demands of judo have previously been performed, our study presents a new perspective because the energy requirements were evaluated during a judo match, and not in a laboratory.³⁹ We show that a judo match induces both protein and lipid metabolism even if the anaerobic system is brought into action, with mean levels of plasma lactate of 12.3 mmol/l. Therefore glycogen in the muscle is not the only substrate used during a judo match. Several factors such as carbohydrate availability, training adaptation, and metabolic stress may account for the use of these substrates. These results need to be confirmed but could be taken into account when training programmes are devised.

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