

Comparison of muscle damage parameters between the training session and after a fight amongst judokas

GANDOUZI IMED¹, TURKI MOUNA², AYEDI FATMA³, SOUISSI NIZAR⁴

^{1,2}Research Unit Molecular Bases of Human Pathology UR12ES17, Faculty of Medicine, Sfax University, TUNISIA

^{2,3}Laboratory of Biochemistry; CHU Habib Bourguiba; Sfax, TUNISIA

⁴National Observatory of Sport, TUNISIA

Published online: January 31, 2020

(Accepted for publication: December 28, 2019)

DOI:10.7752/jpes.2020.01009

Abstract:

Judo is an intermittent sport based on resistance efforts, high isometric intensity and strong concentric and eccentric muscle contractions. During a training or a fight, the hand-to-hand contact and falls cause traumatic shocks, fatigue and localized damage to muscle tissue. The purpose of this study was to compare the variations of the parameters of damage and muscular inflammation between a typical training session and a fight among the judokas. To achieve this purpose, we focused on judo athletes of regular sporting activity. Three blood samples were taken from each athlete: at rest, after a 90 min training and after a 5 min fight for the determination of muscle damage parameters (creatinine kinase, lactate dehydrogenase, transaminases), muscle inflammation (C-reactive protein) and lactic acid. Mean values of creatinine kinase and dehydrogenase differed significantly between fight and rest ($p=0.015$, $p<0.001$ respectively). The transaminase levels after the fight were also higher compared to rest, but no statistically significant difference was found. However, we noticed significant increases in white blood cells, neutrophils, lymphocytes monocytes and eosinophils ($p<0.001$, $p=0.032$, $p<0.001$, $p<0.001$ and $p=0.47$ respectively). Mean C-reactive protein values were much higher after the fight but with no statistically significant difference for the three samples ($p=0.710$). A high significant difference for lactic acid was found among all specimens ($p<0.001$). Unlike a training session, the judo fight requires a preponderant solicitation of anaerobic glycolysis and it causes a more acute muscle damage. Indeed, the effectiveness of the training sessions is questioned in terms of Intensity, Volume, Type of exercise, Sector sought.... Therefore, the right way to prepare for the fight is to train in fight.

Key Words: Randori, inflammation, creatine kinase, lactate dehydrogenase, leukocytosis, C-reactive protein.

Introduction

The primary purpose of high athletic performance training is to guarantee a competitive physical readiness on the day of the competition. In general, in most sport disciplines, the duration of the competition is lower than that of the training, and so is the frequency. In individual sports, as well as in fight sports, the athlete trains every week for maybe two or three competitions annually. However, the athlete is much more tired during the competition than training. Similarly, sports accidents are more frequent during the competition. Clinically, this fatigue is defined by biochemical responses inducing muscle damage, which can be transformed into muscular inflammations according to the intensity and the type of training and the duration of the exercise. Indeed, the concentric, eccentric and static physical exercises seem to be able to damage the muscles (Koch et al., 2014). Resistance exercise induces rapid leukocytosis in healthy men (Hulmi et al., 2010). The local inflammatory response is accompanied by a systematic response known as the "acute phase response". This response includes the production of a large number of C-reactive proteins (CRP), α 2-macroglobulin and transferrin (Pedersen & Hoffman-Goetz, 2000).

Several researchers have studied muscle damage and inflammation following physical exercises or competitions (Callegari et al., 2017; Gioldasis, 2016; Teixeira et al., 2015). During intense exercise, muscle damage is mainly caused by a strong muscle contraction (Takarada, 2003), especially after a resistance exercise or other exercise requiring mainly eccentric actions (Brown et al., 1997; Koch et al., 2014). The eccentric, contraction-intensive intermittent sports based on direct impact during exercise, such as football, rugby, field hockey ... are more traumatic than other types of sports (Bauduer et al., 2011; Ehlers et al., 2002; Junge, 2004; Takarada, 2003). In fact, all muscular actions (concentric, eccentric, static) seem to be able to damage the muscle (Clarkson et al., 1986).

Generally, the duration of the competition is less than the duration of the training, notably in combat sport. However, there is no clear explanation for the fatigue and accidents, especially muscular, which are higher

during the competitions. Indeed, judo is an intermittent sport based on resistance efforts, high isometric intensity and strong concentric and eccentric muscle contraction. The duration of a judo fight is much shorter than the training time. In spite of this, it is very remarkable that the judoka during the competition is more tired than during the training and the frequency of muscular accidents is much higher during the competition. To our knowledge, no study has compared the acute biochemical effects of competition on skeletal muscle to those of a weekly workout. Yet, such knowledge could help athletes to judge their level of training and to know whether a given level of training is sufficient to prepare their physical condition for the requirements of the competition. As a result, our goal is to compare variations in damage parameters and muscle inflammation between a typical workout and a fight in judokas in order to determine the best method to prepare for a competition

Materials & methods

Participants

For this prospective study, 17 participants were recruited from a club in the southern region of Tunisia. They had a regular activity. Their coaches informed us that they had been training for at least 6 months without stopping and that most of the judokas belonged to school and university sport. One judoka did not finish all the experimental protocol because he had a fracture in the arm during the training. All judokas presented their verbal and written agreements to participate in this research. This study had the agreement of the research ethics committee of Habib Bourguiba University Hospital of Sfax.

Test protocol, Measurements and Instruments

All anthropometric data and physical examinations of judokas were collected at the beginning of the protocol. Clinical characteristics were determined at the cardiology department of CHU Hedi Chaker Tunisia. The weight was determined directly using a scale. The size was measured using a height chart.

The heart rate was deduced from a resting electrocardiogram (ECG). Three blood samples were taken from all participants. The first sample was taken at rest, the second just after a weekly training session of 90 min and the third sample was taken just after a round of 5 min randori where all the regulations of the competition were respected except the stop of the fight with ippon. All fights were played until exactly 5 minutes and the winner is the one who scores more points. Enzyme parameters (Creatine Kinase (CK), Lactate Dehydrogenase (LDH) and transaminases (ALAT and ASAT), ionic (Sodium Na, Potassium K and Cl chloride), renal (Urea and Creatinine), inflammatory (CRP), blood count (BC) and concentration lactic acid (AL) and blood counts were measured at each sampling.

The three samples were made simultaneously in similar conditions. They were all done in the club training room and after 7 o'clock in the evening. Three-day rest was required between the second and third samplings. All samples were taken at almost the same time to reduce the effect of diurnal variation. The biochemical parameters were realized on Cobas C501 and BC on Sysmex.

Statistical analysis

Statistical analyses were performed using SPSS (V23). The results were presented as mean \pm one time standard deviation. Normality was tested for all measured biochemical values; then averages of measured parameter values were compared using bidirectional ANOVA. If normality was not satisfied even after a log transformation, the nonparametric Friedman test was used to compare the means of the values. A value of $p < 0.05$ was considered significant. Post hoc tests were used to compare parameters two by two.

Results:

Seventeen young judokas with a mean age of 18.35 ± 2.0 participated to this study (see Table 1). Their clinical characteristics are determined in table 1.

Table 1: Clinical characteristic of judokas

Characteristics	Mean \pm SD
Age (years)	18.31 \pm 2.02
Size (cm)	174.06 \pm 7.03
Weight (kg)	68.62 \pm 16.88
Body mass index (kg/m ²)	22.37 \pm 3.67
Body area (m ²)	1.82 \pm .25
Fat Index (%)	14.63 \pm 4.77
HR rest (bat.min-1)	65.19 \pm 11.61

SD = Standard deviation; HR=heart rate

Table 2 shows the changes in the blood ionogram resulting from a weekly workout and a judo fight. Significant differences between the three blood samples for electrolyte measurement (Sodium, Potassium, and Chloride) were found ($p < 0.001$ for all variables measured).

Table 2: Variation of the blood ionogram resulting from a weekly training session and a judo fight. The p value serves to test intergroup differences

Electrolytes	ANOVA (p value)	Rest (Mean ± SD)	Training (Mean ± SD)	Combat (Mean ± SD)
Sodium (Na)(mmol/l)	< 0.001*	142±1.87	137.88±1.69	141.68±2.15
Potassium (K) (mmol/l)	<0.001*	4.12±0.47	3.69±0.31	3.53±0.25
Chloride (Cl) (mmol/l)	<0.001*	100.29±1.10	96±1.69	95.87±1.36

* Significant at $p < 0.05$; Mean ± SD: mean ± standard deviation

Post hoc analysis showed a significant difference between Na after training and in the two other samples at rest and after combat. However, for K, the difference was significant between rest and the two other samples. Likewise, resting Cl was significantly different from training and combat (Table 3).

Table 3: Post Hoc Test: Scheffe inter-specimen difference in blood electrolytes between the three samples (rest, training and combat)

Electrolytes	Rest # Training (p)	Rest #Combat (p)	Combat # Training (p)
Sodium (Na) (mmol/l)	<0.001*	0.896	<0.001*
Potassium (K) (mmol/l)	0.004*	<0.001*	0.444
Chloride (Cl) (mmol/l)	<0.001*	<0.001*	0.968

* Significant difference at $p < 0.05$, #: against

Table 4 shows the changes in muscle damage marker enzymes resulting from a weekly workout and a judo fight. The comparison of values obtained from the three samples of the LDH levels showed a significant difference ($F(2.47) = 8.52, p < 0.001$). Similarly, and following a logarithmic transformation to verify the normal distribution condition of the values, the CK and LA levels at rest, after training and after competition were significantly different ($F(2.47) = 3.48, p = 0.039, F(2.47) = 89.66, p < 0.001$ respectively). However, no statistically significant difference was found by comparing the AST and ALT levels for the three blood samples, although the values found after the fight were higher than the others.

Table 4. Variations of the muscle damage marker enzymes between the three samples (rest, training and combat). The value of p to test intergroup differences by ANOVA

	ANOVA (p value)	Rest (Mean ± SD)	Training (Mean ± SD)	Combat (Mean ± SD)
LDH(U/L)	<0.001*	205.7±42.25	234.94±46.77	287.18±77.71
CK [†] (U/L)	0.039*	2.43±0.26	2.54±0.26	2.69±0.33
ASAT [†] (U/L)	0.42	1.44±0.17	1.46±0.15	1.52±0.17
ALAT [†] (U/L)	0.39	1.2±0.26	1.28±0.26	1.33±0.29
L A [†] (mmol/l)	< 0.001*	0.17±0.15	0.61±0.3	1.12±0.08

[†]: value after logarithmic transformation; * significant difference at $p < 0.05$; Mean ± SD: mean ± standard deviation

The post hoc analysis reported a significant difference between the value of LDH obtained after combat and the two other samples: at rest and after training. CK values were significantly different between fighting and rest alone. For lactic acid, post hoc analysis showed significant differences between all samples. (Table 5)

Table 5. Post Hoc Test: Scheffe inter-sampling difference for the muscle damage marker enzymes between the three samples (rest, training and combat)

	Rest # Training (p)	Rest #Combat (p)	Combat # Training (p)
LDH (U/L)	0.33	<0.001*	0.041*
CK [†] (U/L)	0.52	0.039*	0.32
ASAT [†] (U/L)	0.94	0.44	0.63
ALAT [†] (U/L)	0.68	0.40	0.88
LA [†] (mmol/l)	<0.001*	<0.001*	<0.001*

[†]: value after logarithmic transformation; * significant difference at $p < 0.05$, #: against

Table 6 shows the variation of the CBC blood count between training and judokas competition. Significant differences were found between WBC, NE, LY, MO, and EO recorded at rest, after training and after combat ($F(2.47) = 15.24, p < 0.001, F(2.47) = 3.72, p = 0.032, F(2.47) = 42.30, p < 0.001, (F(2.47) = 28.80, p < 0.001$ and ($F(2.47) = 3.42, p = 0.041$) respectively). For BA values, even the logarithmic transformation did not satisfy the condition of the normal distribution. For that, we used the nonparametric Pearson test for continuous quantitative values. A significant difference was found between the three samples ($p = 0.007$). On the other hand,

no statistically significant difference was recorded for the variations of the rates of the other ions (RBC, HGB, PLT and HC).

Table 6: Hematologic variation resulting from a weekly workout and a judo fight. The value of p to test intergroup differences by ANOVA

	ANOVA (p value)	Rest (Mean ± SD)	Training (Mean ± SD)	Combat (Mean ± SD)
WBC (103µL)	<0.001*	8.18±2.06	9.45±3.30	13.50±3.10
NE (103µL)	0.032*	3.76±1.59	4.60±2.20	5.66±2.17
LY (103µL)	<0.001*	2.97±.65	3.90±1.28	6.58±1.42
MO (103µL)	<0.001*	.58±.17	.70±.19	1.09±.23
EO (103µL)	0.041*	.27±.16	.33±.33±	.47±.30
BA (103µL)	0.007* "	.029±.04	.029±.047	.088±07
RBC (106µL)	0.189	4.95±.29	5.04±.26	5.13±.27
HGB (g.dL-1)	0.668	14.01±.88	14.39±1.02	13.90±2.53
PLT (103µL)	0.254	282.00±76.07	308.47±78.39	327.93±82.43
HC (%)	0.154	42.14±2.56	43.05±3.14	44.07±2.54

* ": Significant at p<0.05 by applying the nonparametric Pearson test; *: significant at p<0.05 by applying ANOVA; Mean ± SD: Mean ± standard deviation; WBC: White blood cells; NE: neutrophils; LY: lymphocytes; MO: monocytes; EO: eosinophils; BA: basophils; RBC: Red blood cells; HGB: hemoglobin; PLT: Platelets; HC: Hematocrit

Post hoc analysis showed a significant difference between post-combat WBC and other blood samples. Similarly for lymphocytes (LY) and monocytes (MO), the blood sampling rates were significantly different after the fight for both, comparing them with values at rest and after training. For neutrophils (NE) and eosinophils (EO), the significant difference was found only between the value recorded after the fight and that at rest. (Table 7)

Table 7. Post Hoc Test: Difference between Scheffe and BC (inflammatory index) between the three samples (rest, training and combat)

	Rest # Training (p)	Rest #Combat (p)	Combat # Training (p)
WBC (103µL)	0.439	<0.001*	<0.001*
NE (103µL)	0.474	0.032*	0.561
LY (103µL)	0.078	<0.001*	<0.001*
MO (103µL)	0.246	<0.001*	<0.001*
EO (103µL)	0.746	0.047*	0.210
BA (103µL)		0.007* "	
RBC (106µL)	0.602	0.200	0.712
HGB (g.dL-1)	0.800	0.981	0.696
PLT (103µL)	0.623	0.258	0.779
HC (%)	0.634	0.147	0.574

* ": Significant at p<0.05 by applying the nonparametric Pearson test; *: significant at p<0.05 by applying ANOVA; #: against; WBC: White blood cells; NE: neutrophils; LY: lymphocytes; MO: monocytes; EO: eosinophils; BA: basophils; RBC: Red blood cells; HGB: hemoglobin; PLT: Platelets; HC: Hematocrit

The level of C-reactive protein (CRP) as markers of muscle inflammation was not significantly different between the three blood samples (F (2.47) = 0.345, p = 0.710). (Table 8)

Table 8. Variation in CRP as an index of muscle inflammation between training and combat

	ANOVA (p value)	Rest (Mean ± SD)	Training (Mean ± SD)	Combat (Mean ± SD)
CRP [†] (m g/l)	0.710	-0.233± 0.43	-0.339 ± 0.53	-0.370 ± 0.52

* Significant difference at p < 0.05; Mean ± SD: Mean ± standard deviation; †: values after logarithmic transformation.

Discussion

The fight damaged the skeletal muscle of the judoka by causing an increase in the rate of CK and LDH. This exercise strongly requires anaerobic lactic glycolysis. The judo fight did not cause muscle inflammation despite the increase in the rate of CRP which remained insignificant. In contrast, the training session generally did not show significant changes in these parameters of damage and muscle inflammation. The judo fight caused leukocytosis in response to a muscle damage stimulus involving local muscle inflammation. Our results demonstrated that this leukocytosis depended on these monocytic, lymphocyte and polymorphonuclear

subcomponents. In 1932, Edward (Edwards & Wood, 1932) described leukocytosis as increasing the total number of white blood cells in response to difficult muscular work and the increase appears to be proportional to the intensity and duration of the exercise. This can be explained by the release of epinephrine and norepinephrine (NOR) (Prestes et al., 2008) in response to the innervation of lymphoid organs by sympathetic nerve fibers. Indeed, norepinephrine (NOR) recruits lymphocytes (Pedersen & Hoffman-Goetz, 2000) and monocytes (Steppich et al., 2000).

The total numbers of LE, LY, MO and NE increased during and immediately after resistance exercise (Kraemer et al., 1996). In contrast, Hulmi et al. (Hulmi et al., 2010) did not find a significant difference in LE after resistance exercise for subjects trained in resistance for 2 weeks. Simonson and Jackson (Simonson & Jackson, 2004) described a very low number of BAs in the peripheral circulation and this type of cell appears to be unresponsive to the stress of exercise. In our study, we have found an increase in WBC, NE, LY, MO, EO and BA.

Indeed, BA has bactericidal and phagocytosis properties that are mainly involved in the destruction of parasites, but less than NE. Therefore, the fight exerts a very intense inflammatory stress on the skeletal muscles. In addition, BA is considered the cell for immediate allergic manifestations. Despite a number of BA that is the lowest of the polynuclears (0 to 1% of the total GB), its increase represents a sign of the high inflammatory effect caused by combat which is not the case during training.

However, neither the fight nor the training session resulted in a significant change in the erythrocytes of hematocytes and platelets. Our results were different from those of Teixeira et al. (Teixeira et al., 2015) who found that a resistance exercise caused a significant increase in hematocrits, erythrocytes, hemoglobins, and platelets. This can be explained by the fact that in a judo fight, the actions are performed in apnea soliciting, thus, the anaerobic sector in which the low or no use of O₂.

For electrolytes, sodium decreased only after training, conversely to a longitudinal study of Gioldasis (Gioldasis, 2016) in which there was an increase in sodium levels during preparation and during the competition period for footballers. This is probably due to a much longer work time during training than the fight (5mn vs 90 min) which it is able to clear enough sweat that can cause a decrease in sodium.

The potassium level, decreased after training and continue decreasing after the fight conversely at the Meyer and Meister (Meyer & Meister, 2011) study that recorded an increase in potassium during an entire season of Football. However other studies like that of Gioldasis (Gioldasis, 2016) did not find any change of the potassium level during the preparation and the football competition period. Indeed the role of potassium is crucial and must remain almost constant. Its decrease may be due to muscle fatigue that damages skeletal muscle and may limit athletic performance.

The lack of chloride, which contributes to blood osmosis, can induce fatigue and muscle cramps. Consequently, training like fighting in judo has caused a decrease in chlorine levels, which can be a sign of muscle damage.

In addition, physical exercise and resistance can result in localized damage to muscle tissue. This damage can be seen in the sarcolemma, the basal lamina, as well as in the contractile elements and the cytoskeleton. Usually, the damage is accompanied by the release of enzymes such as CK and LDH, myoglobin and other proteins in the blood (Koch et al., 2014). Creatine kinase (Koch et al., 2014), lactate dehydrogenase, aldolase, myoglobin, troponin, aspartate aminotransferase and carbonic anhydrase CAIII are the most useful serum markers for muscle injury (Brancaccio et al., 2010; Gioldasis, 2016).

Thus, CK and LDH, as reliable indicators of muscle damage, are significantly higher after combat. Studies have found similar results: Callegari et al. (Callegari et al., 2017) observed a significant increase in LDH and more pronounced CK levels after resistance exercise with a multiple series protocol. Teixeira et al. (Teixeira et al., 2015) showed that a resistance exercise for beginners resulted in muscle damage characterized by an increase in CK. Studies have shown an increase in CK levels following a rugby match (Gill, 2006). This increase in CK rate is proportional to the number of tackles (Bauduer et al., 2011) and it is highly correlated with the number of tackles per match ($r = 0.922$) (Takarada, 2003).

Brancaccio et al. (Brancaccio et al., 2010) have shown that "Crush injuries, direct hits and intense physical exercise are the most common causes of muscle damage". U Zuliani et al. (Zuliani et al., 1985) showed that a real boxing fight (with actual wrist cost) caused muscle damage by significant increase in CK and LDH. On the other hand, an amber fight (boxing fight without touch) gave no change in the parameters of muscular damage.

Indeed, the judoka, during a fight, performs an intermittent effort of an average of 11 sequences of actions of a duration of $\approx 2.52'$ min of which 8 standing and 3 on tatami interspersed by 7 sequences of pause of a total duration of $\approx 1.41'$ min (lluís Castanerlas & Planas, 1997). In addition, the fight is based on body contact that is standing, during the projection especially when contacting the tatami or during the ground fight.

Based on these results and by analogy with studies done on boxing, rugby ..., the increase of CK and LDH may be due to muscle damage induced by body-body and body-tatami contact after a free fall in addition to a brief intense effort, intermittent isometric resistances. In addition, the shocks are more aggressive if they result from a projection by supporting the weight of Uke (the striker in judo) who has just heaped the body of Tori

(who is attacked in judo) at the time of his contact with the tatami. However, no significant differences in CK and LDH were observed after the training. This can be explained by the fact that the effort released during a judo training session is unable to damage the muscles since the recovery times are quite long and the contact less aggressive.

For ALT and ASAT our results are confirmed by the results obtained by H. Bruunsgaard et al. (Bruunsgaard et al., 1997) who tested 9 subjects who did eccentric and concentric ergometer exercises. They found significant increases after the eccentric exercise of the CK and LDH levels. In contrast, ASAT and ALAT concentrations did not change after both types of contractions. These results are also confirmed by Zuliani et al. (Zuliani et al., 1985) who showed that contact and non-contact boxing did not result in significant changes in ASAT and ALAT levels. This may be due to release thresholds of these enzymes that have not been reached (Zuliani et al., 1985). Zuliani et al. (Zuliani et al., 1983) showed that ASAT levels increase in response to prolonged intense muscle stress for hours which is not the case for combat (5min) and even if training exceeds one hour the intensity of work is usually not high enough.

The significant differences found between resting lactate levels and those measured after training (doubled 2.6 times) and after combat (8.45 times) certify that judo is an effort that strongly demands the anaerobic sector, which is confirmed by many experimental results in all the previous studies. However, after combat these values are multiplied by ≈ 2.6 compared to the training. This can be explained by the fact that the anaerobic metabolism is solicited in a major and dominating way during the fight and by the intersection and the alternation of different metabolism during the training.

The damage to the musculoskeletal tissue led to an inflammatory response deduced in our study by a leukocytosis but the rate of CRP did not show any change after the two types of exercises (training and combat). In fact, CRP is an inflammatory marker of the acute phase produced by hepatocytes in response to IL-6 (Michigan et al., 2011). Nakajima et al. (Nakajima et al., 2010) suggested that oxidation and degranulation of circulating neutrophils would be responsible for increasing CRP immediately after a resistance exercise (SRE). In our studies, despite the significant rise in NE after combat, there was no significant change in CRP. Indeed, Kruger et al. (Krüger et al., 2011) did not find an increase in CRP immediately after an intense resistance exercise. They recorded that the increase in CRP became significant after 3 hours of exercise. On the other hand, a study by Laskowski et al. (Laskowski et al., 2011) on judokas who had a 3-day training, the most important increases of pro-inflammatory cytokines as well as IL-6 are recorded after a randori training.

These results can be explained in two points. Either by the fact that the combat exercise in our study is not sufficiently motivating to push the judoka to give the maximum efforts since it is not a real competition and we must, perhaps, do another study when the judokas are taken in a real competition or, more probably, because the CRP rate will increase after a few hours of recovery.

This proves that the judoka during a fight that exercises intermittent actions of high intensity and short durations with very strong eccentric and concentric muscular contractions capable of causing muscular inflammation. In addition, judo training should be based on randori mainly with intermittent eccentric and concentric contraction resistance exercises.

Conclusion:

Judo is an intermittent sport with a very high static component based on contact. The judo fight caused damage and inflammation of the skeletal muscles. The muscular lesions after combat is caused by; Effect of contact and shocks, Intermittent effort (resistance, static, concentric and eccentric very high muscular contraction...), anaerobic glycolysis solicited in a major and preponderant way during the fight (in training recovery is more important). It was therefore necessary to repeat the blood sampling after recovery, which is a weak point in this study.

Professional judokas are forced to undergo a very intense competition season. Such intense competition may result in overtraining, muscle damage and increased inflammatory response. Therefore, the training period must prepare professional athletes to adapt to changing conditions during a season of intense competition. Indeed, the effectiveness of the training sessions are questioned in terms of Intensity, Volume, Type of exercise, Solicited Sector...

In this study, we raised two key questions: Does the training really affect the needs of the athlete during the competition? Are physical aptitude tests sufficient to predict the athlete's condition or is it time to talk about the importance of biochemical tests to: have a real idea about the athlete's condition, schedule a specific training for competitions, anticipate and protect against especially muscular accidents?

We therefore suggest that judo training should be based essentially on randori with intermittent exercises of eccentric and concentric contraction resistance. Therefore, in our opinion the right way to prepare for the fight is to train in combat. Further experiences are scheduled in the coming months to validate our suggestions.

Conflict of interest:

The authors report no conflict of interest.

References:

- Bauduer, F., Monchaux, C., Burtin, M.-L., Dubroca, B., & Mathieu, J.-P. (2011). Déséquilibres biochimiques, déshydratation, récupération et rugby professionnel – données du suivi longitudinal de la Ligue nationale de rugby et de l'exploration par bioimpédance multifréquence. *Science & Sports*, 26(1), 19-24.
- Brancaccio, P., Lippi, G., & Maffulli, N. (2010). Biochemical markers of muscular damage. *Clinical Chemistry and Laboratory Medicine*, 48(6).
- Brown, S. J., Child, R. B., Day, S. H., & Donnelly, A. E. (1997). Exercise-induced skeletal muscle damage and adaptation following repeated bouts of eccentric muscle contractions. *Journal of Sports Sciences*, 15(2), 215-222.
- Bruunsgaard, H., Galbo, H., Halkjaer-Kristensen, J., Johansen, T. L., MacLean, D. A., & Pedersen, B. K. (1997). Exercise-induced increase in serum interleukin-6 in humans is related to muscle damage. *The Journal of Physiology*, 499 (Pt 3), 833-841.
- Callegari, G. A., Novaes, J. S., Neto, G. R., Dias, I., Garrido, N. D., & Dani, C. (2017). Creatine Kinase and Lactate Dehydrogenase Responses After Different Resistance and Aerobic Exercise Protocols. *Journal of Human Kinetics*, 58(1).
- Clarkson, P. M., Byrnes, W. C., McCormick, K. M., Turcotte, L. P., & White, J. S. (1986). Muscle soreness and serum creatine kinase activity following isometric, eccentric, and concentric exercise. *International Journal of Sports Medicine*, 7(3), 152-155.
- Edwards, H. T., & Wood, W. B. (1932). A study of leukocytosis in exercise. *Arbeitsphysiologie*, 6(1-2), 73-83.
- Ehlers, G. G., Ball, T. E., & Liston, L. (2002). *Creatine Kinase Levels are Elevated During 2-A-Day Practices in Collegiate Football Players*. 37(2), 6.
- Gill, N. D. (2006). Effectiveness of post-match recovery strategies in rugby players. *British Journal of Sports Medicine*, 40(3), 260-263.
- Gioldasis, A. (2016). Biochemical Changes from Preparation to Competitive Period in Soccer. *International Journal of Science Culture and Sport*, 4(16), 150-150.
- Hulmi, J. J., Myllymäki, T., Tenhumäki, M., Mutanen, N., Puurtinen, R., Paulsen, G., & Mero, A. A. (2010). Effects of resistance exercise and protein ingestion on blood leukocytes and platelets in young and older men. *European Journal of Applied Physiology*, 109(2), 343-353.
- Junge, A. (2004). Injuries in youth amateur soccer and rugby players—Comparison of incidence and characteristics. *British Journal of Sports Medicine*, 38(2), 168-172.
- Koch, A. J., Pereira, R., & Machado, M. (2014). *The creatine kinase response to resistance exercise*. 14(1), 10.
- Kraemer, W. J., Clemson, A., Triplett, N. T., Bush, J. A., Newton, R. U., & Lynch, J. M. (1996). The effects of plasma cortisol elevation on total and differential leukocyte counts in response to heavy-resistance exercise. *European Journal of Applied Physiology and Occupational Physiology*, 73(1-2), 93-97. 5
- Krüger, K., Agnischock, S., Lechtermann, A., Tiwari, S., Mishra, M., Pilat, C., Wagner, A., Tweddell, C., Gramlich, I., & Mooren, F. C. (2011). Intensive resistance exercise induces lymphocyte apoptosis via cortisol and glucocorticoid receptor-dependent pathways. *Journal of Applied Physiology*, 110(5), 1226-1232.
- Laskowski, R., Ziemann, E., Olek, R., & Zembron-Lacny, A. (2011). The Effect of Three Days of Judo Training Sessions on the Inflammatory Response and Oxidative Stress Markers. *Journal of Human Kinetics*, 30(1).
- Iluís Castanerlas, J., & Planas, A. (1997). ESTUDI DE L'ESTRUCTURA TEMPORAL DEL COMBAT DE JUDO. *Educació Física i Esports*, 47, 8 (32-39).
- Meyer, T., & Meister, S. (2011). Routine Blood Parameters in Elite Soccer Players. *International Journal of Sports Medicine*, 32(11), 875-881.
- Michigan, A., Johnson, T. V., & Master, V. A. (2011). Review of the Relationship between C-Reactive Protein and Exercise. *Molecular Diagnosis & Therapy*, 15(5), 265-275. <https://doi.org/10.1007/BF03256418>
- Nakajima, T., Kurano, M., Hasegawa, T., Takano, H., Iida, H., Yasuda, T., Fukuda, T., Madarame, H., Uno, K., Meguro, K., Shiga, T., Sagara, M., Nagata, T., Maemura, K., Hirata, Y., Yamasoba, T., & Nagai, R. (2010). Pentraxin3 and high-sensitive C-reactive protein are independent inflammatory markers released during high-intensity exercise. *European Journal of Applied Physiology*, 110(5), 905-913.
- Pedersen, B. K., & Hoffman-Goetz, L. (2000). Exercise and the Immune System : Regulation, Integration, and Adaptation. *Physiological Reviews*, 80(3), 1055-1081.
- Prestes, J., de Ferreira, C., Dias, R., Frollini, A., Donatto, F., Cury-Boaventura, M., Guerreschi, M., Pithon-Curi, T., Verlengia, R., Palanch, A., Curi, R., & Cavaglieri, C. (2008). Lymphocyte and Cytokines after Short Periods of Exercise. *International Journal of Sports Medicine*, 29(12), 1010-1014.
- Simonson, S. R., & Jackson, C. G. R. (2004). Leukocytosis occurs in response to resistance exercise in men. *Journal of Strength and Conditioning Research*, 18(2), 266-271. <https://doi.org/10.1519/R-12572.1>
- Stappich, B., Dayyani, F., Gruber, R., Lorenz, R., Mack, M., & Ziegler-Heitbrock, H. W. L. (2000). Selective mobilization of CD14⁺ CD16⁺ monocytes by exercise. *American Journal of Physiology-Cell Physiology*, 279(3), C578-C586.

- Takarada, Y. (2003). Evaluation of muscle damage after a rugby match with special reference to tackle plays. *British Journal of Sports Medicine*, 37(5), 416-419.
- Teixeira, A. D. O., Paulitsch, F. D. S., Umpierre, M. D. M., Moraes, M. B. de, Rosa, C. E. da, & Signori, L. U. (2015). **Inflammatory response after session of resistance exercises in untrained volunteers.** *Acta Scientiarum. Health Sciences*, 37(1), 31.
- Zuliani, U., Bonetti, A., Franchini, D., Serventi, G., Ugolotti, G., & Varacca, A. (1985). Effect of Boxing on Some Metabolic Indices of Muscular Contraction. *International Journal of Sports Medicine*, 06(04), 234-236.
- Zuliani, U., Mandras, A., Beltrami, G. F., Bonetti, A., Montani, G., & Novarini, A. (1983). Metabolic modifications caused by sport activity : Effect in leisure-time cross-country skiers. *The Journal of Sports Medicine and Physical Fitness*, 23(4), 385-392.