

**Mixed martial arts induces significant fatigue and muscle damage
up to 24 hours post-combat**

Running Title: 24-hr follow-up of simulated mixed martial arts combat

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Abstract

This study investigates the physiological/physical responses to a simulated mixed martial arts (MMA) competition over 24 hr. Twelve fighters performed a simulated MMA competition, consisting of three 5-min MMA matches. Physiological/physical data were assessed before (T_{rest}), directly after round 1 (T_{rd1}), round 2 (T_{rd2}) and round 3 (T_{rd3}), and then 30-min ($T_{recovery30min}$) and/or 24-hr ($T_{recovery24h}$) post-competition. Heart rate (HR), rating of perceived exertion (RPE) and blood lactate concentration ($[La^-]$) were assessed at T_{rest} , T_{rd1} , T_{rd2} and T_{rd3} . Biological data were collected at T_{rest} , T_{rd3} , $T_{recovery30min}$ and $T_{recovery24h}$. Physical tests were performed at T_{rest} , $T_{recovery30min}$ and $T_{recovery24h}$. HR, RPE and $[La^-]$ were high during competition. Leukocytes, hemoglobin, total protein and glycemia were increased at T_{rd3} compared with all other time points ($p < 0.05$). Cortisol was increased at T_{rd3} compared with T_{rest} and $T_{recovery24h}$ ($p < 0.05$). Testosterone was higher at T_{rd3} and $T_{recovery30min}$ than T_{rest} ($p < 0.001$). Higher values of uric acid were noted during recovery periods ($p < 0.001$). Lactate dehydrogenase was lower at T_{rest} compared with T_{rd3} , $T_{recovery30min}$ and $T_{recovery24h}$ ($p < 0.05$). Countermovement jump was higher at T_{rest} than $T_{recovery30min}$ ($p = 0.020$). Consequently, MMA is a high-intensity intermittent combat sport that induces significant fatigue and muscle damage, both of which are still present 24-hr post-competition.

Keywords: Combat sport; Exercise intensity; Recovery; Biochemical data; Fighter.

INTRODUCTION

Mixed martial arts (MMA) is a combat sport characterized by a combination of fighting styles borrowed from other combat sports. The techniques used during the fight can be grouped into three types, based on the component sports: strikes, clinch and grappling, and submission techniques [2,12,21]. MMA has received significant media exposure and is currently growing in worldwide popularity, especially with the Ultimate Fighting Championship® (UFC) events. Generally, the competitions have three to five rounds lasting 5 min each, with 1 min of passive recovery between each round. The winner is defined by a judge's decision, knockout, technical knockout, submission or disqualification.

As MMA is a mixture of several combat sports, the training load for these fighters is likely to be very high [2]. Many top-level competitors train several times per day, 7 days per week, with the average number of training and practice sessions being 11 sessions per week [2]. For this reason, overtraining is a serious risk for the MMA athlete, and the risk of musculoskeletal injury increases as the intensity and duration of training sessions rises [24]. This may explain why the majority of studies have focused on the injury profile [24]. However, few studies have investigated the cardiovascular and biochemical responses, even though understanding the demands during competition is paramount to reduce the risk of injury and optimize the athlete's performance [2,18,25,34].

Most of the results in the literature have stemmed from the component sports [22,39] because of the complications of conducting physiological measurements during MMA combat. Some authors have focused on the analysis of blood lactate concentration ($[La^-]$) and the rating of perceived exertion (RPE) during MMA training and competition [1]. The results have demonstrated the high metabolic demands of training for and competing in this sport. Del Vecchio et al. [12] conducted a time-motion analysis of MMA, analyzing work rates in stand-up and ground grappling situations in different rounds, and classified the effort of MMA action as low- or high-intensity. The results showed that the ratio between high-intensity to low-intensity in MMA competitions was 1:2 to 1:4, with intermittent high-intensity actions lasting 6 to 14 s followed by low-intensity efforts lasting 46 to 62 s, suggesting that the MMA athlete needs to be prepared to maintain high-intensity activity for the duration of each round.

Additionally, the literature raises interesting questions about the metabolic profile of MMA athletes because the levels of anaerobic and aerobic fitness may be similar to one of the component sports or may reflect a mixture of multiple sports. This is due to the intermittent nature of the sport and the open tasks involving striking skills (punches, kicks, knee and elbow attacks), as well as grappling skills (tackles, twists, throwing techniques using legs) and submission on the ground [20,26,41]. Recently, Kirk et al. [20] showed that MMA has greater workloads than non-contact team sports. It was also shown that the MMA fighter's energetics may more closely match those of striking-based athletes as opposed to grappling-based ones, while the MMA effort-pause ratio sits in the middle of the striking and grappling spectrum.

Despite the great workloads of MMA matches and, only one study [8] investigated the effects of MMA-simulated competition on physiological and physical parameters and reported increased muscle damage 48 hr after the match compared with pre-combat values. The same investigators [8] also compared simulated and official competition and reported that the two conditions induced comparable glycolytic activation and muscle damage biochemical markers immediately after the matches. This suggests that simulated competitions can be used to infer the physiological and biochemical responses to official competitions. Lindsay et al. [23] demonstrated values of inflammatory and physiological stress biomarkers following 90 min of contest preparation training session with sequential MMA bouts. MMA training involves sparring several times per training camp [23] without real referential of an MMA championship, what could interfere in performance and recovery processes.

No study has yet developed a complete, validated physiological and physical profile of the MMA fighter based on metabolic activity and performance and the post-competition passive recovery effect. Therefore, this study aimed to investigate the cardiovascular and biochemical responses and physical performances for 24 hr following a simulated MMA competition. The hypotheses were that MMA competition would induce high fatigue and muscle damage and that a 24-hr passive recovery period would be insufficient to return to baseline values.

METHODS

Experimental Approach to the Problem

To study the effects of MMA competition, the cardiovascular and biochemical responses and physical performances have been collected in 12 fighters before, during and after a simulated MMA competition. These data were then compared to test the impact of MMA competition on athletes.

Subjects

Twelve male MMA athletes (age: 26 ± 5 years, height: 182 ± 7 cm, body mass: 86.2 ± 10.9 kg) were recruited as participants for this study. These athletes competed in local and regional competitions once a month and were regularly training (technical and tactical) 3-4 times a week in the weeks before the experimental protocol.

Before the experiment, all participants attended a briefing meeting and signed an informed consent document to ensure that they understood the testing procedures and the risks and benefits associated with the study. All testing procedures were in line with the Declaration of Helsinki. All participants were more than 18 years old, had previous experience with professional UFC events, rules, and procedures used during the experimental championship. No interferences were made in the training, nutritional or hydration status of participants, and they maintained the weight loss recovery time pattern of 24 hours between official weigh-in and the bout following MMA rules.

Procedures

The MMA bouts were performed on ring, following fighting area requirements and equipment described in unified rules and other MMA regulations. The ring had 24 feet square within the ropes. The corner had a blue designation and the corner directly opposite had a red designation. The ring floor had 18 inches beyond the ropes and the floor was padded with closed-cell foam, with 1-inch layer of foam padding. This padding extended beyond the ring ropes and over the edge of the platform, with a top covering of canvas, tightly stretched and laced to the ring platform. The ring platform had 3 feet above the floor of the building and had suitable steps for the use of the unarmed fighters. Ring posts was made of metal, with 2 inches in diameter, extending from the floor of the building to a height of 60 inches above the ring floor, and had properly padded in a manner approved by three experts in MMA. Ring posts had 18 inches from the ring ropes. There had five ring ropes, with one inch in diameter and wrapped in soft material. There had no one obstruction or object, without limitations.

The MMA athletes performed three 5-min MMA rounds separated by 1 min of passive recovery. They were divided into pairs with a difference of body mass of no more than 10%. This protocol reproduced the real MMA combat. The experimental protocol consisted of six testing time points (Figure 1): before (T_{rest}), directly after the first (T_{rd1}), second (T_{rd2}) and third (T_{rd3}) rounds, and 30-min ($T_{recovery30min}$) and/or 24-hr ($T_{recovery24h}$) post-competition.

****Figure 1****

The time interval between rounds was set in order to respect the recovery period of a real MMA fight as described in the literature [37]. The initial testing time point (T_{rest}) consisted of heart rate (HR), RPE, arterial blood pressure, $[La^-]$ and other blood data (*i.e.*, hematocrit, hemoglobin, erythrocyte, leukocyte, platelets, total protein, total cholesterol, triglyceride, glucose, cortisol, testosterone, cortisol:testosterone ratio, uric acid, creatine kinase and lactate dehydrogenase) and physical performances (*i.e.*, squat and countermovement jumps, hand grip, sprints) at baseline (Figure 1). After each round (T_{rd1} , T_{rd2} , T_{rd3}), HR, RPE and $[La^-]$ were assessed again (Figure 1). Moreover, just after the competition protocol (T_{rd3}), arterial blood pressure was measured. At $T_{recovery30min}$, participants completed the identical testing procedures as at baseline (Figure 1). The following day ($T_{recovery24h}$), arterial blood pressure and physical performances were measured again (Figure 1).

Measures

Heart rate (HR) was measured with a HR chest monitor and wrist watch receiver (S810, Polar® Kempele, Finland). Before the experimental protocol, the participants were fitted with the HR monitor, which was worn before, during and after competition. HR measures were obtained after a seated rest period of 5 min at T_{rest} and $T_{recovery30min}$. Moreover, HR was recorded in the 1-min recovery period after each round (T_{rd1} , T_{rd2} and T_{rd3}). The HR represents the mean value of HR measured during the last 60 s of each period.

The modified 0-10 category ratio scale [14] was used to measure RPE. This scale is used to transform athletes' perceptions of effort into numerical scores between 0 and 10. Competitors were familiarized with the scale in the week before the experimental

procedure. Instructions about this scale were read before each testing session. Overall RPE was collected at T_{rest} , T_{rd1} , T_{rd2} , T_{rd3} and $T_{recovery30min}$.

Systolic (SBP) and diastolic (DBP) blood pressures were also recorded at baseline (T_{rest}), just after competition (T_{rd3}), and 30-min and 24-h post-competition ($T_{recovery30min}$ and $T_{recovery24h}$) using an automated oscillometric device (705 IT, Omron[®], Kyoto, Japan) positioned on the participant's left arm after he had been sitting quietly for 5 min.

Blood lactate concentrations ($[La^-]$) were measured with the Lactate Pro analyzer (Arkray[®], Tokyo, Japan) from a fingertip blood sample (5 μ L). The samples were collected on five occasions: T_{rest} , T_{rd1} , T_{rd2} , T_{rd3} and $T_{recovery30min}$. Prior to sampling, the fingertip was sterilized with an alcohol swab and a lancet was then used to pierce the skin. The initial drop of blood was discarded and the following drop was measured. Before each blood sampling, the Lactate Pro analyzer was calibrated and used according to manufacturer's guidelines.

Just before simulated competition (T_{rest}), immediately post-competition (T_{rd3}), and 30-min ($T_{recovery30min}$) and 24-hr ($T_{recovery24h}$) post-competition, 4 ml of blood from the antecubital vein was taken using vacutainer tubes containing coagulant gel [13]. The blood samples remained at rest and were stored on ice until centrifuged at 3000 rpm for 15 min at 4°C for serum separation. After centrifugation, aliquots of serum and plasma were stored at -80°C for later analysis. Hematocrit, hemoglobin, red and white blood cell and platelet counts were made with a multichannel automated blood cell analyzer (Gen system-2, Coulter T540, Beckman Coulter[®], Brea, CA, USA).

Moreover, samples were analyzed for total protein, glucose, total cholesterol and triglyceride concentrations using kits from Spinreact[®] (Girona, Spain). Cortisol and testosterone were determined by radio-immunoassay (Immunotech RIA, Beckman Coulter[®], Brea, CA, USA). Serum samples were also analyzed for uric acid, creatine kinase (CK) and lactate dehydrogenase (LDH) using an automated analyzer (Vitros 5.1, Ortho Clinical Diagnostics[®], Rochester, NY, USA) [13]. All blood analyses were performed by a trained medical team.

The squat jump (SJ) and countermovement jump (CMJ) were performed using an infrared jump system (Optojump, Microgate[®], Bolzano, Italy) interfaced with a microcomputer to assess the explosive power of the leg muscles [37] at T_{rest} , $T_{recovery30min}$ and $T_{recovery24h}$. Participants were asked to perform the SJ standing upright with good balance and the trunk as vertical as possible, feet parallel and shoulder-width apart, and hands on the hips throughout the test with a knee angle around 90°. The trial was not considered valid if any movement was perceived with the increased knee flexion at the start of the jump. For the CMJ, participants started from an upright standing position and made a preliminary downward movement by flexing the knees and hips, with a knee angle around 90° at the end of the countermovement. The best of three trials was recorded for SJ and CMJ.

The static maximal grip strength was measured using the Jamar Handgrip Dynamometer (Model J00105, Lafayette, IN, USA). This dynamometer features a dual pointer system to retain the maximum effort. The testing range on the dual scale is 0-100 kg. Before taking the measurements, the participants were requested to stand in a comfortable position with the dominant arm outstretched. They then squeezed

the dynamometer as hard as possible, without moving the rest of the body. The reading was taken from the dynamometer scale when the pointer no longer moved. Only the best of three trials was recorded.

Sprint ability was evaluated by a 10-m standing-start maximal run on an outdoor synthetic court, using telemetric photoelectric cells placed at 5 m and 10 m (Racetime 2, Microgate[®], Bolzano, Italy). The participants commenced the sprint when ready 0.5 m behind the first timing gate. Timing began when they crossed the first pair of photocells, and they then ran as fast as they could to complete the 10-m distance. They completed three runs interspersed by 1 min of recovery and the best time was selected. During the recovery period, the participants walked back to the starting line and then waited for the next sprint. The analysis used the affiliated split times for the 5 and 10 m.

Statistical analyses

Descriptive data are presented as mean and standard deviation (SD). One-way ANOVAs with repeated measures were used to compare data. Normal Gaussian distribution was verified by the Shapiro-Wilk test. When these data did not pass the test for normality, we transformed the data using the logarithm function. Moreover, the sphericity was checked by the Mauchly test. When the assumption of sphericity was not met, the significance of *F*-ratios was adjusted according to the Greenhouse-Geisser procedure. When significant differences were identified, the Bonferroni post hoc test was performed. Furthermore, the Eta squared (η^2) was calculated as effect size. The 95% confidence intervals were also calculated. A Pearson (*r*) correlation

test was used to examine the relationships between physiological, biochemical and neuromuscular measures. Statistical significance was set at $p \leq 0.05$, and all analyses were performed with the Statistical Package for the Social Sciences (release 20.0, Chicago, IL, USA).

RESULTS

HR at T_{rest} and $T_{\text{recovery30min}}$ was significantly lower compared with T_{rd1} , T_{rd2} and T_{rd3} ($p < 0.001$ for all comparisons, Figure 2). Significant differences were noted for RPE between each comparison ($p < 0.05$), except between T_{rd1} and $T_{\text{recovery30min}}$ (Figure 2). Concerning $[La^-]$, there were significant differences between all time points ($p \leq 0.05$), except between T_{rd2} and T_{rd3} ($p > 0.05$, Figure 2).

****Figure 2****

SBP changed across time ($p = 0.002$, $\eta^2 = 0.349$), with lower values at $T_{\text{recovery30min}}$ compared with T_{rd3} ($p = 0.004$) and $T_{\text{recovery24h}}$ ($p = 0.040$, Table 1). Conversely, DBP did not change ($p = 0.210$, $\eta^2 = 0.127$, Table 1).

****Table 1****

Hematocrit changed across time ($p < 0.001$, $\eta^2 = 0.626$), with higher values at T_{rd3} compared with T_{rest} ($p = 0.009$), $T_{\text{recovery30min}}$ ($p = 0.002$) and $T_{\text{recovery24h}}$ ($p < 0.001$, Table 1). Moreover, significantly higher values at T_{rest} were noted in comparison with $T_{\text{recovery24h}}$ ($p = 0.037$, Table 1). As presented in Table 1, hemoglobin changed similarly to hematocrit.

The erythrocyte count did not change ($p=0.360$, $\eta^2=0.091$, Table 1). However, the leukocyte count changed across time points ($p<0.001$, $\eta^2=0.620$), with higher values at T_{rd3} compared with T_{rest} ($p=0.002$), $T_{recovery30min}$ ($p=0.029$) and $T_{recovery24h}$ ($p=0.001$, Table 1). Platelets differed across time ($p<0.001$, $\eta^2=0.837$) similarly to leukocytes (Table 1).

Total protein ($p<0.001$, $\eta^2=0.682$) and glycemia ($p<0.001$, $\eta^2=0.445$) also varied, with increased values at T_{rd3} compared with all other time points ($p<0.001$, Table 1). Total cholesterol changed between time points ($p<0.001$, $\eta^2=0.561$), with higher values at T_{rest} and T_{rd3} compared with $T_{recovery30min}$ and $T_{recovery24h}$ ($p<0.01$). The only significant difference in triglycerides was noted between T_{rd3} and $T_{recovery30min}$ ($p=0.024$).

Cortisol changed across time ($p=0.002$, $\eta^2=0.362$), with increased values at T_{rd3} compared with T_{rest} and $T_{recovery24h}$ ($p<0.05$). Testosterone was also affected by time ($p<0.001$, $\eta^2=0.941$), with higher values post-competition compared with all other moments ($p<0.001$), except between T_{rest} and $T_{recovery24h}$ ($p=0.164$). No significant change in cortisol:testosterone ratio was detected ($p=0.063$, $\eta^2=0.182$).

Uric acid also varied across time ($p<0.001$, $\eta^2=0.636$), with higher values during recovery periods compared with all other time points ($p<0.001$), except between T_{rd3} and $T_{recovery24}$ (Table 1).

No change in CK was detected ($p=0.119$, $\eta^2=0.160$), but LDH changed ($p=0.010$, $\eta^2=0.288$), with lower values at T_{rest} compared with T_{rd3} , $T_{\text{recovery30min}}$ and $T_{\text{recovery24h}}$ ($p<0.05$, Table 1).

****Table 2****

For SJ, no effect of time was observed ($p=0.114$, $\eta^2=0.195$). CMJ was affected by the time of measurement ($p=0.040$, $\eta^2=0.275$), with higher values at T_{rest} than $T_{\text{recovery30min}}$ ($p=0.020$, Table 2). No significant change in handgrip or sprint (on 5- and 10-m) performance was detected ($p>0.05$, Table 2).

Concerning correlations during T_{rest} , statistical analysis showed associations between cortisol and hematocrit ($p=0.038$, $r=0.60$), hemoglobin ($p=0.043$, $r=0.59$), red ($p\leq 0.001$, $r=0.87$) and white blood cells ($p=0.058$, $r=0.58$). Glucose presented an inverse correlation with cortisol ($p=0.019$, $r=-0.66$) and with total cholesterol ($p=0.020$, $r=-0.66$) at T_{rest} .

White and red blood cells demonstrated correlations with hematocrit ($p=0.003$, $r=0.78$, and $p=0.004$, $r=0.76$, respectively) and hemoglobin ($p=0.012$, $r=0.62$, and $p=0.032$, $r=0.62$, respectively) during T_{rest} , respectively.

At T_{rest} , analysis showed a correlation between $[La^-]$ and total protein ($r=0.87$, $p\leq 0.001$), while RPE had a negative association with hematocrit ($r=-0.64$, $p=0.024$), hemoglobin ($r=-0.73$, $p=0.007$) and with white blood cells ($r=-0.65$, $p=0.023$).

During T_{rest} , SBP demonstrated correlations with sprints of 5 m ($r=0.66$, $p=0.020$) and 10 m ($r=0.69$, $p=0.012$).

SBP and DBP blood pressures were correlated during T_{rest} and T_{rd3} moments ($r=0.86$, $p\leq 0.001$ and $r=0.86$, $p=0.010$).

Regarding correlations at T_{rd3} , LDH showed association with CK ($r=0.76$, $p=0.004$), uric acid ($r=0.76$, $p=0.018$) and white blood cells ($r=0.59$, $p=0.045$). Uric acid had association with hematocrit ($r=0.70$, $p=0.012$) and hemoglobin ($r=0.63$, $p=0.027$) during T_{rd3} . In addition, white blood cells demonstrated correlation with hematocrit ($r=0.61$, $p=0.035$), LDH ($r=0.59$, $p=0.045$) and glucose ($r=0.58$, $p=0.048$), while hematocrit was correlated with hemoglobin ($r=0.80$, $p=0.002$) at T_{rd3} . At T_{rd3} , RPE was inversely correlated with SJ ($r=-0.70$, $p=0.017$), SBP ($r=-0.69$, $p=0.013$) and DBP ($r=-0.67$, $p=0.017$).

Regarding $T_{recovery30min}$, glucose had correlation with cortisol ($r=0.75$, $p=0.005$), uric acid ($r=0.68$, $p=0.014$) and white blood cells ($r=0.73$, $p=0.007$). Hemoglobin was associated with hematocrit ($r=0.69$, $p=0.018$), with white ($r=0.63$, $p=0.030$) and red ($r=0.78$, $p=0.003$) blood cells at $T_{recovery30min}$. LHD and $[La^-]$ demonstrated correlation with cortisol ($r=0.84$, $p\leq 0.001$ and $r=0.67$, $p=0.016$) and glucose ($r=0.82$, $p\leq 0.001$ and $r=0.85$, $p\leq 0.001$), respectively. $[La^-]$ was correlated with uric acid ($r=0.77$, $p=0.003$) and white blood cells ($r=0.67$, $p=0.016$), HD ($r=0.66$, $p=0.020$) and inversely related with RPE ($r=-0.66$, $p=0.020$). RPE was related with hematocrit ($r=-0.77$, $p=0.003$), white ($r=-0.73$, $p=0.007$) and red ($r=-0.65$, $p=0.022$) blood cells during $T_{recovery30min}$. White blood cells demonstrated a correlation with hematocrit

($r=0.64$, $p=0.024$) and uric acid ($r=0.68$, $p=0.016$), while total protein was inversely correlated with total cholesterol ($r=-0.64$, $p=0.024$) at $T_{\text{recovery}30\text{min}}$.

Concerning $T_{\text{recovery}30\text{min}}$ and $T_{\text{recovery}24\text{h}}$ moments, hand grip was inversely correlated with cortisol ($r=-0.70$, $p=0.011$ and $r=-0.61$, $p=0.046$). At $T_{\text{recovery}24\text{h}}$, 5 m sprint was correlated with testosterone ($r=0.67$, $p=0.022$), CK ($r=0.63$, $p=0.038$) and total protein ($r=0.62$, $p=0.044$). At $T_{\text{recovery}30\text{min}}$, white blood cells was correlated with 10 m sprint ($r=0.62$, $p=0.031$) and inversely associated with CMJ ($r=-0.59$, $p=0.042$).

Regarding $T_{\text{recovery}24\text{h}}$, testosterone was correlated with total protein ($r=0.73$, $p=0.007$), glucose was inversely associated with hematocrit ($r=-0.75$, $p=0.009$) and red blood cells ($r=-0.74$, $p=0.006$), and LDH was associated with CK ($r=0.78$, $p=0.003$). Glucose was inversely related with hematocrit ($r=-0.74$, $p=0.006$), while hematocrit was correlated with hemoglobin ($r=0.82$, $p\leq 0.001$) and red blood ($r=0.69$, $p=0.013$) during $T_{\text{recovery}24\text{h}}$.

Concerning T_{rest} , $T_{\text{recovery}30\text{min}}$ and $T_{\text{recovery}24\text{h}}$ 5 m sprint was associated with 10 m sprint ($r=0.89$, $p\leq 0.001$, $r=0.94$, $p\leq 0.001$ and $r=0.88$, $p\leq 0.001$), respectively. CMJ was correlated with hand grip at T_{rest} ($r=0.64$, $p=0.035$). During $T_{\text{recovery}24\text{h}}$, CMJ was inversely associated with 10 m sprint ($r=-0.68$, $p=0.021$).

DISCUSSION

This research investigated the physiological and physical responses to a simulated MMA competition over 24 hr. The results confirmed our hypotheses because the MMA competition induced significant fatigue and muscle damage immediately post-competition that were still present 24 hr later. To the best of our knowledge, this is the first study to analyse the fatigue effects on neuromuscular, physiological and biochemical variables previously and after an MMA tournament and during 24h of recovery time, thus increasing the external validity of the present results.

The data show that the simulated MMA competition induced high HR, RPE and $[La^-]$ (Figure 2). The unique MMA environment can explain these results. Preceding reports indicated that the predominant energy source in combat sports is aerobic [16,19]. It has been confirmed that the aerobic system plays a significant role in determining performance during high intensity exercise, with a maximal exercise effort of 75 seconds deriving approximately equal energy from the aerobic and anaerobic energy systems [16]. In addition, it was described that there is a significant contribution of the glycolytic and lactic systems in determinants MMA actions [1], suggesting that anaerobic metabolism and recovery time are relevant to MMA. Therefore, key engagements depend on powerful actions and optimal aerobic condition to support performance throughout the combat [6,17,32,38,40]. Therefore, MMA must be considered a high-intensity intermittent combat sport which energy systems contribute sequentially with a predominance of the oxidative system to supply the energy cost of combats.

Regarding the physiological responses, the post-competition HR (184 ± 14 bpm, Figure 2) was comparable to that reported in earlier studies of elite judo, karate and

taekwondo athletes, with the studies including between 1 and 7 simulated matches and reporting mean values closed to 185 bpm [32-33]. Moreover, in our study, the acute $[La^-]$ responses to the MMA rounds seem to be similar to those in other combat reports on karate [5], Brazilian jiu-jitsu [42], judo [11] and boxing [31]. Nevertheless, although our post-competition $[La^-]$ (13.6 ± 1.4 mmol/L, Figure 2) is similar to that of a recent study on karate (14.0 ± 1.8 mmol/L) [34], these values are rather low compared with those of studies specifically investigating MMA matches, in which a range of 13.8 to 20.7 mmol/L was found [1,7,17]. This indicates that, compared with the literature, the fighters of our study did not reach very high $[La^-]$, probably because of a lower competition level.

The post-competition RPEs were between "somewhat hard" and "maximal" (mean RPE= 6.5 ± 2.1 on the modified CR-10 scale of Foster et al. [14]). Previous authors attempting to develop a fitness profile of MMA competitors during simulated matches also reported RPEs (obtained from another Borg scale) between "somewhat hard" and "maximal" [1,9]. After MMA tournament, our RPEs demonstrated a strong and inversely association with squat jump height results and CMJ. Therefore, the present RPE results converge with the literature.

The results show that MMA causes tissue damage, the production of stress hormones, and alterations in the measure and function of several circulating immune cells (Table 1). The significant increase in leukocyte and hemoglobin counts immediately following competition suggests that MMA matches cause graded and well-defined amounts of muscle trauma and may therefore serve as an experimental model for inflammation and sepsis patterns, as shown in another study [30].

Coswig et al. [8] analyzed blood glucose concentration in MMA fighters during simulated matches and found a higher glucose concentration post-competition, comparable to our finding and that of James et al. [17]) (Table 1). This increased glucose may be due to enhanced glycogenolysis and gluconeogenesis in response to the energy demands during MMA combat [8].

Glucose presented a strong correlation with cortisol at acute recovery moment. First, this occurrence may be associated with anticipatory cortisol performance [4,28] occasioning increased blood glucose concentration because of the stimulus of gluconeogenesis (*e.g.*, hepatic glucose production from lactate) [8]. Previously, an association between blood glucose and cortisol has been described [9], and greater energy availability seems to be a positive anticipatory feature correlated with improved fighter effectiveness [15]. Second, when measurements were compared, we found a moderate effect size for glucose concentration, which could indicate anticipatory preparation for the next round by sympathetic system activation, consequently raising cortisol and glycogenolysis levels [9,42]. On the other hand, the present results are also in agreement with previous studies showing an increase in cortisol and testosterone (Table 1) immediately after combat sport matches [33,35]. Despite cortisol levels produced in MMA training session were lower than our data, immediate cold water immersion could attenuate concentration changes in saliva cortisol, as in urinary neopterin and total neopterin in a recent study [23]. This contribution indicates the requirement of future studies with recovery methods that may reduce fatigue and injuries after MMA tournaments.

The post-competition results showed higher values of uric acid formation (Table 1). However, the values were lower than those previously reported from heavy weight

judo athletes [29]. Uric acid formation results from the rise in purine nucleotide degradation and fast-twitch fiber utilization in conditions of high energy demand, confirming that MMA requires a high anaerobic contribution.

The increased level of enzymatic activities of LDH and CK were strongly correlated in our results and were similar to those in a previous report [9], suggesting the existence of muscle damage by cell extravasations of LDH and CK from the fragmentation of the sarcolemma due to the efforts with high intensity [7]. CK showed no difference between pre- and all other time points (Table 1); however, the peak reported in the above-cited study was likely reached 24 to 48 hr after exercise [9]. Moreover, these authors reported that the higher metabolic stress resulted in greater leakage of muscle damage markers (*e.g.*, LDH), which usually continues to increase for several hours post-competition. In the present study, the MMA time points were associated with severe muscle damage, as evident by the significant increase in LDH, even after the 24-hr recovery period (Table 1). The variations in the number of punches and kicks to the lower body are known to significantly contribute to the variation in muscle damage markers in MMA fighters [43]. Muscle damage responses after MMA competitions can partially be attributed to the number of incidents of blunt force trauma. However, in addition to this trauma resulting from physical collisions, the opponent's failed upright kicks also contribute to the variance of the observed CK response [43].

Regarding physical performance, the explosive power of the leg muscles was non-significantly lower for SJ (-3%) and significantly lower for CMJ at $T_{\text{recovery30min}}$ compared with T_{rest} (-6%, Table 2). These low differences may be due to the prevalence of the short-lasting power actions of the lower limbs, like kicks, that make up only 5% or less of the total standing combat time [20]. On the other hand,

static maximal handgrip strength is crucial for grappling actions, predominantly during the groundwork phase, and is associated with the ability to bring the opponent to submission, choke and/or lock positions [27]. Our study revealed a strong inverse relationship between maximum isometric handgrip strength and cortisol levels, without significant effect. Only a non-significant reduction of 6.9% in handgrip performance after the simulated competition was noted. Another report on grappling modalities indicated decreases for approximately 10%, especially in the dominant hand [3], explained by higher overloads held on the dominant side in combat sports (in judo). A Brazilian jiu-jitsu study, however, reported no differences in upper limb performance after three successive matches [10]. It should thus be noted that the scant literature on combat sports (especially MMA) shows discrepant results on the effects of competition and recovery on the power output of the upper limbs, even though this body segment contributes substantially in strike and takedown actions, among others, during matches [6].

The present study has some limitations. Indeed, we chose to measure physical performances (SJ, CMJ and handgrip and sprint performances) at T_{rest} , $T_{recovery30min}$ and $T_{recovery24h}$, but not T_{rd3} . The results revealed a significant effect of the simulated competition only for CMJ, with lower values at $T_{recovery30min}$ compared with T_{rest} . Although this cannot be proved, we assume that the high workload during the competition significantly reduced the physical performances just after the protocol (at T_{rd3}). Physical performances were not measured at T_{rd3} because these maximal tests might have influenced the biological data at $T_{recovery30min}$ (reflecting not just the effect of simulated competition on the biological data, but the effect of simulated competition combined with the physical tests).

Another potential limitation is that we did not analyze the technical-tactical actions, but these analyses have already been reported in the literature [9,17,20,26], whereas accurate analyses of physiological demands and recovery are still scarce.

Practical Applications

This study attempted to develop a complete and physiological and physical profile of the MMA fighter based on a 24-hr follow-up of a simulated MMA competition. From the correlations between biomarkers, RPEs and neuromotor tests, it is possible to evaluate and to create specific contextual training according, MMA tournament demands instead of long time of training session during contest preparation. The data demonstrate that MMA is a high-intensity intermittent combat sport that induces significant fatigue and muscle damage immediately post-competition, both of which are still present 24 hr later. These findings may be used in the future to develop appropriate preparation, creating high-intensity interval training, alternating short periods of intense strike or grappling actions with low intensity or recovery periods and, after the bout, recovery strategies such as active recovery, cold water immersion or compressive garment to recover faster. Given the importance of improving the recovery of MMA fighters, different recovery modality could be used after high-intensity combat. Recovery sessions should be conducted based on several criteria such as the nature of the sport, fatigue levels of athletes and time required to recover, and should be taken into consideration by official MMA organizations. More specific applied research is needed to establish and refine recovery strategies for MMA athletes after intense competition.

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Disclosure of interest

The authors report no conflicts of interest.

Table 1. Biomarkers and blood pressures from before the simulated competition (T_{rest}), after the 3rd round (T_{rd3}), as well as 30 min ($T_{recovery30min}$) and 24 h ($T_{recovery24h}$) after the simulated competition. **Legend.** ^asignificantly different from T_{rest} ($p < 0.05$); ^bsignificantly different from T_{rd3} ($p < 0.05$); ^csignificantly different from $T_{recovery30min}$ ($p < 0.05$); ^dsignificantly different from $T_{recovery24h}$ ($p < 0.05$).

Variables	T_{rest}	T_{rd3}	$T_{recovery30min}$	$T_{recovery24h}$
Hematocrit (%)	43.6 ± 1.7 ^{b,d}	45.0 ± 1.3 ^{a,c,d}	42.8 ± 1.5 ^b	42.0 ± 1.3 ^{a,b}
Hemoglobin (g/dL)	14.5 ± 0.8 ^{b,d}	14.9 ± 0.7 ^{a,c,d}	14.3 ± 0.7 ^b	14.0 ± 0.5 ^{a,b}
Erythrocyte ($10^{12}/L$)	4.975 ± 0.252	5.084 ± 0.232	4.907 ± 0.273	4.824 ± 0.264
Leukocyte ($10^9/L$)	7.717 ± 2.476 ^b	12.642 ± 4.212 ^{a,c,d}	9.883 ± 4.561 ^b	7.617 ± 2.134 ^b
Thrombocyte ($10^9/L$)	243 ± 67 ^b	302 ± 72 ^{a,c,d}	240 ± 64 ^b	235 ± 60 ^b
Total protein (g/L)	74 ± 3 ^b	78 ± 3 ^{a,c,d}	73 ± 4 ^b	72 ± 4 ^b
Total cholesterol (mmol/L)	4.5 ± 0.4 ^{c,d}	4.6 ± 0.4 ^{c,d}	4.2 ± 0.3 ^{a,b}	4.2 ± 0.4 ^{a,b}
Triglyceride (mmol/L)	1.27 ± 0.68	1.55 ± 0.74 ^c	1.21 ± 0.53 ^b	1.21 ± 0.59
Glucose (mmol/L)	5.1 ± 0.8 ^b	7.5 ± 1.8 ^{a,c,d}	6.0 ± 1.6 ^b	5.4 ± 1.0 ^b
Cortisol (ng/dL)	88.3 ± 19.6 ^b	131.2 ± 56.4 ^{a,d}	117.3 ± 46.3	79.3 ± 23.2 ^b
Testosterone (ng/dL)	3.5 ± 0.3 ^{b,c}	4.6 ± 0.3 ^{a,c,d}	3.7 ± 0.3 ^{a,b,d}	3.4 ± 0.3 ^{b,c}
Cortisol:Testosterone ratio (ng/dL)	25.5 ± 1.8	28.5 ± 3.4	32.1 ± 3.5	23.6 ± 1.6
Uric acid ($\mu\text{mol}/L$)	308 ± 53 ^{b,c,d}	342 ± 54 ^{a,c}	414 ± 58 ^{a,b,d}	344 ± 48 ^{a,c}
Creatine kinase (u/L)	432 ± 606	505 ± 668	468 ± 566	598 ± 451
Lactate dehydrogenase (u/L)	169 ± 70 ^{b,c,d}	230 ± 116 ^a	228 ± 94 ^a	226 ± 75 ^a
Systolic blood pressure (mmHg)	124 ± 10	130 ± 15 ^c	116 ± 10 ^{b,d}	125 ± 9 ^c
Diastolic blood pressure (mmHg)	73 ± 8	76 ± 8	72 ± 7	75 ± 7

Table 2. Physical performances from before the simulated competition (T_{rest}), and after 30 min ($T_{recovery30min}$) and 24 h ($T_{recovery24h}$) recovery period after the simulated competition. **Legend.** ^asignificantly different from T_{rest} ($p < 0.05$); ^bsignificantly different from T_{rd3} ($p < 0.05$).

Variables	T_{rest}	$T_{recovery30min}$	$T_{recovery24h}$
Squat jump (W/kg)	12.5 ± 1.3	12.1 ± 1.1	12.0 ± 1.1
Countermovement jump (W/kg)	13.1 ± 1.5 ^b	12.5 ± 1.3 ^a	12.7 ± 1.2
Hand grip (kgf)	52.0 ± 10.6	48.4 ± 10.1	53.7 ± 9.5
5-m sprint (s)	1.22 ± 0.08	1.26 ± 0.12	1.26 ± 0.11
10-m sprint (s)	2.03 ± 0.08	2.10 ± 0.15	2.11 ± 0.20

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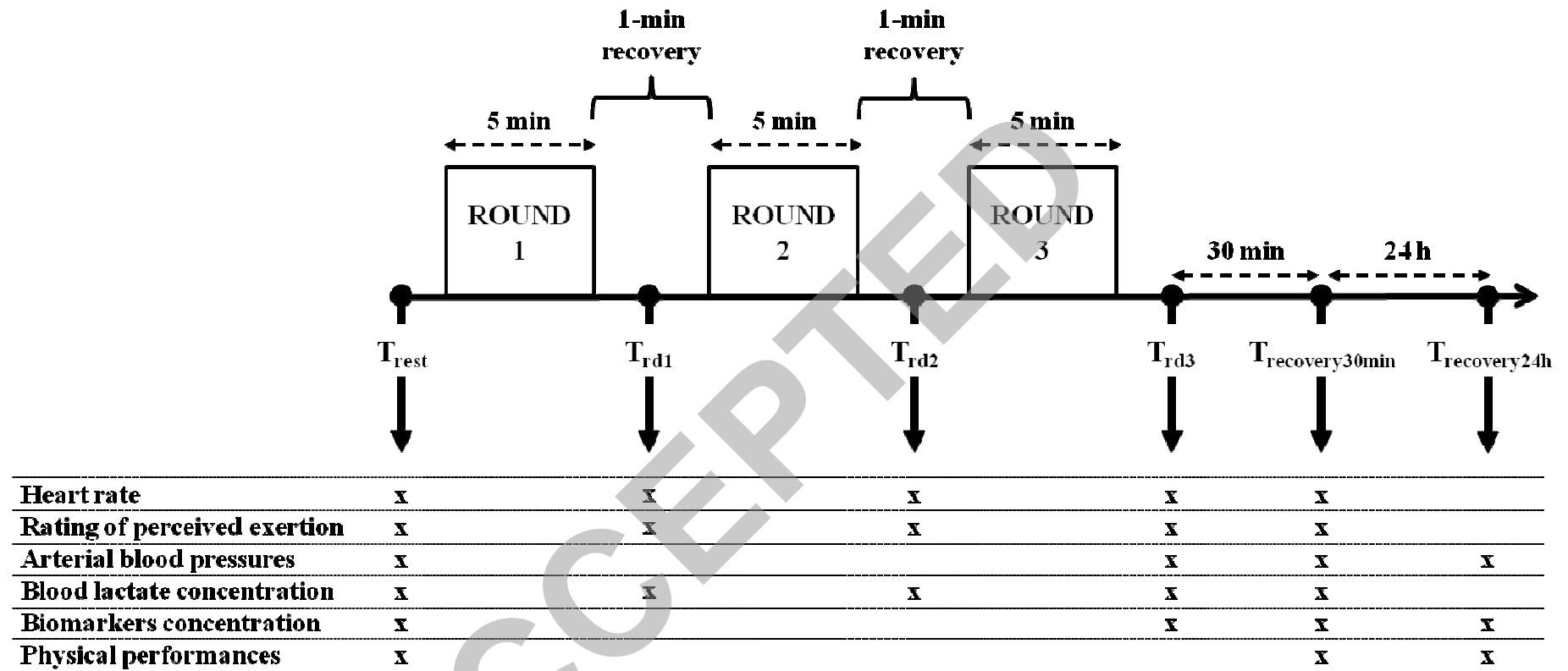


Figure 1. Schematic representing study design.

Legend. T_{rest} , testing session before the simulated competition; T_{rd1} , testing session after the 1st round; T_{rd2} , testing session after the 2nd round; T_{rd3} , testing session after the 3rd round (*i.e.*, just after the simulated competition); $T_{recovery30min}$, testing session 30 min after the simulated competition; $T_{recovery24h}$, testing session 24 h after the simulated competition.

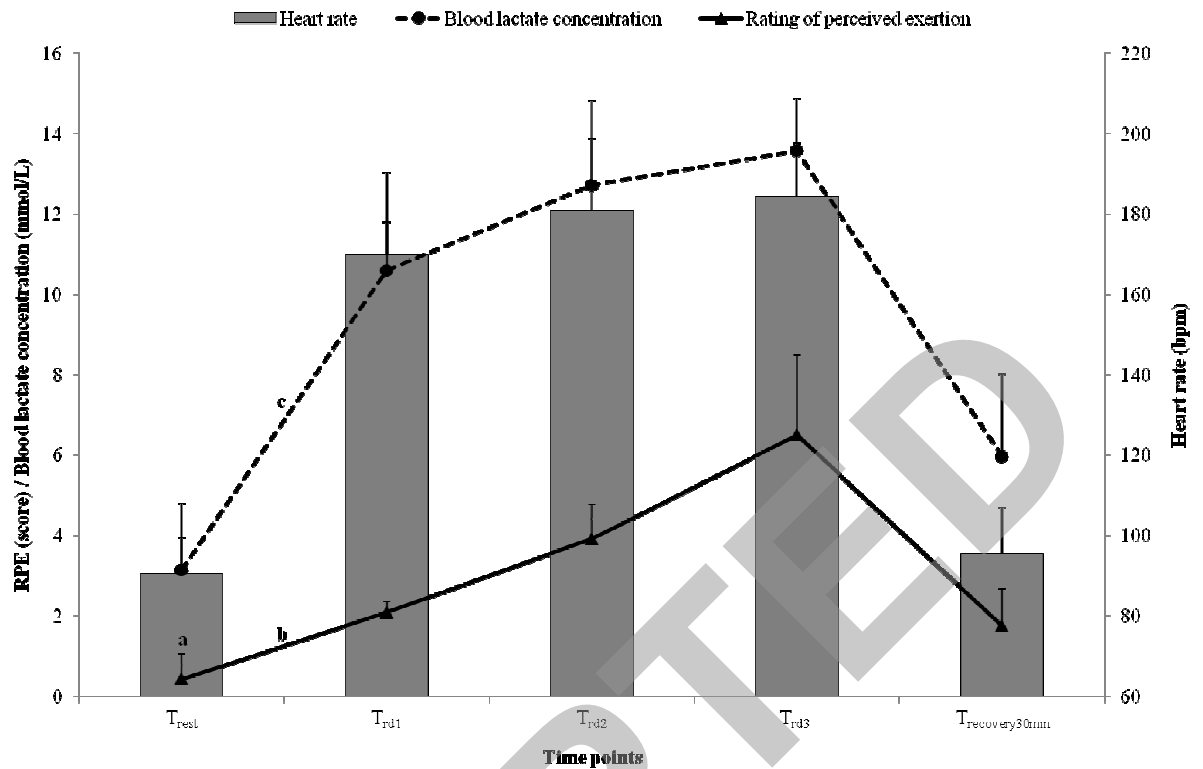


Figure 2. Heart rate, rating of perceived exertion and blood lactate concentration from before the simulated competition (T_{rest}), after the 1st (T_{rd1}), 2nd (T_{rd2}), and 3rd rounds (T_{rd3}), as well as 30 min after the simulated competition (T_{recovery30min}). **Legend.** ^aHeart rate was significantly different from all time points ($p < 0.001$), except for between T_{rest} and T_{recovery30min}, T_{rd1} and T_{rd2}, T_{rd1} and T_{rd3}, and between T_{rd2} and T_{rd3} ($p > 0.05$); ^bRating of perceived exertion was significantly different from all time points ($p < 0.05$), except for between T_{rd1} and T_{recovery30min} ($p > 0.05$); ^cBlood lactate concentration was significantly different from all time points ($p \leq 0.05$), except for between T_{rd2} and T_{rd3} ($p > 0.05$).