# ADAPTATION TO DAMAGING DANCE AND REPEATED SPRINT ACTIVITY IN FEMALES

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**Brief running head** Repeated bout effect in females

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## ABSTRACT

The repeated bout effect (RBE) refers to the prophylactic effect from damaging exercise following a single prior bout of exercise**.** There is a paucity of data examining the RBE in females, and investigations employing exercise paradigms beyond isolated eccentric contractions are scarce. In light of the limited literature, this investigation aimed to determine whether two different sport-specific exercise bouts would elicit a RBE in females. Twenty-one female dancers (19 ± 1 years) completed either a dance-specific protocol (n=10) or sport-specific repeated sprint protocol (n=11). Muscle soreness (DOMS), limb girths, creatine kinase (CK), countermovement jump height, reactive strength index, maximal voluntary contraction and 30 m sprint time were recorded pre, 0, 24, 48, and 72 h post-exercise. An identical exercise bout was conducted approximately four weeks following the initial bout, during which time the subjects maintained habitual training and dietary behaviours. DOMS and 30 m sprint time decreased following a second bout of both activities ($P = 0.003$; $\eta_p^2 = 0.38$ and $P = 0.008$; $\eta_p^2 = 0.31$ respectively). Circulating CK was also lower at 24, 48 and 72 h following the second bout, independent of group ($P = 0.010$; $\eta_p^2 = 0.23$). Compared to the repeated sprint protocol, the magnitude of change in DOMS was greater following a subsequent bout of the dance protocol ($P = 0.010$; $\eta_p^2 = 0.19$). These data are the first to demonstrate that dance and repeated sprint activity resulting in muscle damage in females confers a protective effect against muscle damage following a subsequent bout.


**KEY WORDS** recovery; exercise-induced muscle damage; repeated bout effect

## INTRODUCTION

Exercise-induced muscle damage (EIMD) is associated with many sport and exercise activities. This manifests as muscle soreness, inflammation, and detriments in muscle functionality which can reduce subsequent performance potential (12). Attenuating the symptoms and/or enhancing recovery from EIMD are therefore highly desirable in physically active populations. Despite these potential issues, skeletal muscle has the ability to rapidly adapt following exercise, and indices of muscle damage are attenuated following a second bout of exercise, which is commonly termed the repeated bout effect (RBE). This phenomenon has been demonstrated to occur within 1-2 days (21), and can extend for several months after the initial bout (22). Although the mechanisms are not fully understood, the RBE has been attributed to neural, connective or cellular adaptations (including adaptation in excitation-contraction coupling and the inflammatory response); or, more likely, a combination of these (12, 17). Thus, while the symptoms associated with EIMD are often thought to be detrimental, they play a crucial role in the adaptive process; in fact associated neuromuscular adaptation is suggested to be the most effective means of attenuating future negative effects of EIMD (12, 17).

The RBE has been extensively researched in males; however data concerning females is limited. This is particularly important because oestrogen is widely considered to be responsible, in part, for attenuating symptoms of muscle damage in females and could influence a number of factors including effects on muscle membrane integrity (32). Additionally the secondary inflammatory response may be sex dependent, with reports of sex differences in leukocyte and cytokine infiltration into skeletal muscle post exercise (25, 32). As a result, the initial physiological stress and ensuing recovery and adaptation associated with EIMD in females is likely to differ compared to male populations.

Many studies investigating the RBE have employed eccentric protocols that exercise single muscle groups with isolated contractions using isokinetic dynamometry. Though these effectively induce muscle damage, they lack application to athletic paradigms. While some have explored the RBE using sport-specific protocols (for instance intermittent sprint exercise (15), drop jump protocols (8) and downhill running (4)), other muscle damaging activities that can be directly applied to sport and exercise stimuli warrant further investigation. For instance, dance and multiple sprint sports such as soccer, rugby and basketball - popular activities that are participated in both recreationally and professionally - involve eccentric-biased activities (9, 13, 24, 33). While these exercise modes differ in a number of respects (for instance dance is characterised by complex movement sequences often pre-choreographed, while the nature of repeated sprint sports means that movements are much more unpredictable), they both involve changes in velocity and direction; previously shown to elicit muscle damage. Although these activities have been shown to elicit muscle damage (1, 9, 13, 28), little is known about the adaptation associated with their participation. Given the demands of training and performance, individuals involved in these activities would benefit from an increased understanding of how repeated exposure of activity-specific stimuli can influence the symptoms of muscle damage, recovery and adaptation.

The aim of this study was to elucidate whether dance-specific and repeated sprint exercise designed to induce muscle damage can confer a protective effect against a subsequent bout of identical activity in physically active females. It was hypothesised that, despite their differences, the EIMD observed following a second bout of each activity would be lower than the first bout and that the responses of each activity would be similar.

## METHODS

### Experimental Approach to the Problem

Participants were randomly assigned to one of two muscle damaging exercise groups; either a dance-specific exercise bout or a sport-specific repeated sprint protocol. A battery of common markers of muscle damage was measured pre, immediately post (0 h) and 24, 48, and 72 h post muscle damage. In a repeated bout effect design, a subsequent bout of identical exercise and dependent measures was conducted approximately four weeks (27 ± 4 days) following the first bout to ensure that subjects had fully recovered from the initial bout and were in the same (early/mid luteal) phase of the menstrual cycle. This allowed for examination of the damage response and more specifically the repeated bout effect provided by the initial damaging bout. Participants were tested at the same time on subsequent days (± 1 h) to account for diurnal variation. Participants were asked to avoid strenuous exercise, alcohol, caffeine, nutritional supplements and any anti-inflammatory drugs or alternative treatments (for example massage or cold water immersion) 48 h prior to and for the 72 h recovery period following each exercise bout.

**Subjects**

Following institutional ethical approval, twenty-nine healthy physically active females from a university dance team volunteered for a larger scale study examining the muscle damage response to sprint and dance-specific exercise; these data are presented elsewhere (1). A sub-set of twenty-one participants (mean ± SD age 19 ± 1 y; stature 164 ± 4 cm; mass 58.6 ± 6.2 kg; and BMI 21.8 ± 2.2 $kg \cdot m^2$, respectively) completed a repeated bout of the dance-specific exercise bout (n=10) or sport-specific repeated sprint protocol (n=11) and the data of both bouts of these twenty-one participants are reported in the present study. Participants gave written, informed consent following a general health-screening questionnaire to confirm the absence of any physiological conditions which would exclude them from the study. Participants were regularly active (8.9 ± 4.1 $h \cdot week^{-1}$) including typically attending dance

rehearsals twice per week (5.9 ± 3.2 h). A 3-day food diary and activity log completed prior to testing determined that there were no differences in physical activity levels or energy and macronutrient intakes between participants. Volunteers were asked to replicate their reported diets and activity levels as closely as possible throughout the testing period and this was confirmed with a second 3-day food diary and activity log completed mid-way through the four week recovery period. Moreover participant characteristics were re-measured to ensure no differences were evident between exercise bouts (all characteristics displayed in Table 1). A questionnaire determined menstrual cycle phase; all testing took place during the early/mid luteal phase or where applicable in the 14 days prior to a withdrawal bleed. This also identified the contraceptive use of participants; 13 were using an oral combination pill (all monophasic), 5 were using a progesterone only pill/implant/injection, and 3 were normally menstruating.

[Table 1 about here]

**Procedures**

Exercise protocols

Participants completed a standardised warm-up, followed by 5 min to perform any personal stretches and prepare themselves for the assigned protocol. The subject-specific warm up on the initial day was noted so this could be replicated throughout testing. The originally described dance performance fitness test (DPFT) (26) was extended as described previously (1), to involve the repetition of a dance phrase representative of contemporary dance at a tempo of 106 bpm; with each 10 x 1 min phrase separated by a 2 min rest period. The sport specific repeated sprint test (SSRS) described in full previously (9) briefly comprises 15 x 30 m sprints departing every 65 s with a rapid 10 m deceleration phase. Both the adapted DPFT protocol and the SSRS protocol was shown to induce muscle damage previously in this

population (1). Standardised instructions and strong verbal encouragement from the investigator to encourage maximal effort was provided throughout each muscle damaging protocol. In a repeated bout effect design, each participant completed a subsequent bout of identical muscle damaging exercise approximately 4 weeks following the first bout. Protocols and measurement of dependent variables were completed in an indoor sporting facility and environmental conditions were controlled ($17.7 \pm 0.2\ ^{0}$C, $1012.1 \pm 8.4$ hpa).

Muscle soreness

Subjective delayed onset muscle soreness (DOMS) was measured using a 200 mm visual analogue scale (VAS) (9) from 'no soreness' to 'unbearably sore' anchored at each end of the scale. Participants were required to indicate on the line the level of perceived active lower limb soreness felt during a $90^{0}$ squat.

Limb girth

An anthropometric tape measure (Bodycare Products, Warwickshire, UK) was used to determine muscle swelling in the lower limbs. Girths at the calf (measured at its largest girth at baseline) and mid-thigh (located as mid-way between the inguinal crease and the superior border of the patella) of the right leg were recorded. These locations on the skin were marked with permanent marker on the initial day of each bout to ensure consistency on subsequent days. The mean of two measures at each site was used for analysis. Calf and mid-thigh girth intra-examiner %CV were <1%.

Muscle function

Subjects completed 3 countermovement jumps (CMJ) and 3 drop jumps (for measurement of reactive strength index (RSI)) using a light timing system (Optojump, Microgate, Italy). For CMJ, participants were asked to squat down and jump vertically and maximally, keeping

their hands on their hips throughout. For RSI (jump height (cm) ÷ ground contact time (s)), subjects were instructed to drop from a height of 30 cm and upon landing to perform a two footed jump maximally with minimum contact time. Each effort was separated by 60 s rest and the peak CMJ and RSI were used for analysis.

Knee extensor peak force of the participants' right leg was measured using a strain gauge (MIE Digital Myometer, MIE Medical Research Ltd, Leeds, UK) to determine maximal voluntary isometric contraction (MVC). While in a seated position, the strain gauge was wrapped immediately above the malleoli and attached to a plinth on a purpose built chair. The knee joint angle was standardised at $90^0$ using a goniometer and confirmed before each contraction. Subjects were asked to complete 3 MVCs lasting 3 s, interspersed with 30 s rest. The peak force of 3 MVCs was used for analysis.

Participants completed a single maximal effort 30 m sprint and sprint time was recorded. The sprint was initiated from a line 30 cm behind the start line in order to prevent false triggering of the timing gates (Brower telemetric timers, Brower timing systems, USA).

The 15 x 30 m sprint times of those completing the SSRS were also recorded to determine total sprint time, mean sprint time, and rate of fatigue using the following formula (7):

Fatigue Index (%) = [100 x (total sprint time / ideal sprint time)] - 100, in which total sprint time = sum of sprint times from all sprints and ideal sprint time = the number of sprints x fastest sprint time.

Creatine Kinase

Twenty participants consented to blood collection; n=10 in both groups. Where data for single time points were missing due to sampling error (9 of 210 samples [<5%]), the linear interpolation was used to complete the data set. Blood samples were collected via

venepuncture from the antecubital fossa area into a 10 mL EDTA vacutainer. The samples were centrifuged at 3000 RCF for 15 min at $4^0$ C (Alegra X-22 Centrifuge, Beckman Coulter, Bucks, UK). Plasma was extracted, and stored immediately at $-80^0$ C for later analysis. Plasma CK concentrations were determined spectrophotometrically (Roche Modular, Roche Diagnostics, UK); the analytical range for this method was 7 – 2000 IU·L and the inter- and intra-assay %CV were <2%. The reference range for adult females is 10-160 IU·L.

**Statistical Analyses**

All results are presented as mean ± standard error of the mean (SEM). To account for inter-individual variability, limb girths, CMJ, RSI, MVC and sprint performance were expressed as a percentage change relative to baseline. Statistical software (IBM SPSS v21, IBM, USA) was used for inferential analysis and significance was accepted at the $p < 0.05$ a priori. Mauchley's test assessed the sphericity of the data and where appropriate, violations were corrected using the Greenhouse-Geisser. Raw baseline values were compared between bouts for each group using paired samples t tests to assess any training effect. Total sprint time, mean sprint time, and fatigue scores were also compared between bouts in the SSRS group using paired samples t tests. A mixed factor repeated measures ANOVA (group, 2 x bout, 2 x time, 5) was performed on all variables (except total sprint time, mean sprint time, and fatigue) using group as the between-subject factor, and bout and time as within-subject factors in order to analyse differences between groups and bouts. Where significant effects occurred, LSD post-hoc analysis was performed. Partial eta squared ($\eta_p^2$) effect sizes are also presented to provide magnitude of effects; 0.010, 0.059, and >0.138 were considered to represent small, medium, and large effects respectively (27).

[Table 2 about here]

## RESULTS

There were no differences in baseline values between bout 1 and bout 2. For illustrative purposes data not presented in figures (limb girth, CMJ, RSI and MVC) are presented in Table 2. Participants completed the DPFT at a tempo of 106 bpm during both bouts and paired samples t tests determined no differences between bout 1 and 2 of the SSRS protocol for total sprint time (81.60 ± 1.00 versus 82.43 ± 0.96 s), mean sprint time (5.43 ± 0.07 versus 5.50 ± 0.06 s), and fatigue (3.82 ± 0.42 versus 3.77 ± 0.43%); demonstrating that exercise intensity was maintained. Time effects were observed for all dependent variables (all $p < 0.01$).

### Delayed onset muscle soreness

Muscle soreness increased immediately post exercise and peaked at 24 h post EIMD in both groups following bout 1 and 2 (Figure 1). However DOMS was higher during bout 1 compared to bout 2 ($F_{1, 19} = 11.5$, $p = 0.003$, $\eta_p^2 = 0.38$); where DOMS was attenuated at 0, 24, and 48 h post EIMD ($F_{2.5, 47.7} = 4.7$, $p = 0.009$, $\eta_p^2 = 0.20$). The magnitude of change in DOMS following bout 2 at 48, and 72 h was greater for DPFT compared to SSRS ($F_{2.5, 47.7} = 4.5$, $p = 0.010$, $\eta_p^2 = 0.19$).

[Figure 1 about here]

### Limb girth

There were no differences between groups or bouts for calf girth, however thigh girth was larger immediately following the DPFT compared to post SSRS independent of bout ($F_{4, 76} = 4.0$, $p = 0.005$, $\eta_p^2 = 0.18$).

### Muscle function

Reductions in CMJ, RSI and MVC peaked immediately post EIMD in both bouts with the exception of MVC in the SSRS group which reached lowest levels at 24 h post bout 1. These measures of muscle functionality progressively recovered to near baseline values with no differences between groups or bouts. Interestingly, sprint time improved ($F_{1, 19}$ = 8.6, p = 0.008, $\eta_p^2$ = 0.31) following bout 2 independent of group (Figure 2) where recovery was greater at 24, 48 and 72 h compared to bout 1 ($F_{4, 76}$ = 6.2, p < 0.001, $\eta_p^2$ = 0.25). Both groups improved sprint time beyond baseline measures at 72 h post EIMD in bout 2 (-1.5 ± 0.7 % and -1.2 ± 0.6 % of pre bout 2 sprint time for DPFT and SSRS respectively).

[Figure 2 about here]

Creatine Kinase

Circulating CK was increased and reached peak concentrations at 24 h following both bouts in both exercise groups (Figure 3). Although the SSRS group experienced higher average CK levels this was not different from the DPFT group, and CK was lower 24, 48, and 72 h following the repeated bout compared to bout 1 independent of group ($F_{2.0, 36.4}$ = 5.2, p = 0.010, $\eta_p^2$ = 0.23).

[Figure 3 about here]

## DISCUSSION

This investigation was the first to examine the RBE associated with dance and repeated sprint exercise in healthy active females. Although EIMD was evident in a repeated bout of both dance and sprint activity, this was less than the initial bout. As anticipated, there were lower levels of muscle soreness, reduced concentrations of circulating CK, and although only

evident with one variable, an attenuation of the detriments in muscle function with a second bout of both exercise protocols.

A reduction in soreness following the second bout of exercise has been observed in other investigations in females (5, 20, 23). Some authors propose that DOMS is likely related to the secondary inflammatory response (14). The increase in tissue osmotic pressure associated with inflammation results in the sensitising of afferent nociceptive fibres thought to magnify feelings of soreness and pain (14). Therefore the dampened DOMS reported following a subsequent bout of both exercise protocols (in addition to subjects becoming accustomed to the sensation of soreness (3)) is perhaps attributable to cellular adaptation relating to the secondary inflammatory response. Indeed, neutrophil and monocyte activation has been shown to be diminished with a subsequent bout of eccentric exercise (2). Interestingly, those completing the DPFT experienced greater reductions in DOMS in the later recovery period following the second bout. Intuitively, the habituation to dance-type activity may be responsible for an improved RBE in the DPFT group. A recent study in young females demonstrated that in comparison to a non-exercising group, muscle soreness was reduced following a repeated bout of eccentric exercise when regular training (similar to the damaging exercise) was undertaken between bouts (6). The authors suggested that the concomitant reduction in exercise-induced inflammation they observed may largely explain this difference. These findings suggest that perhaps the continued dance training that participants completed between bouts resulted in a sport-specific adaptation in the group performing the dance-based acute bout; as indicated by the higher reduction in DOMS. While such adaptation is plausible, since no systemic inflammatory markers were measured, this remains speculative and to be fully elucidated. In addition, a reduced inflammatory response may be merely a consequence of a reduction in the initial mechanical disruption in the repeated bout.

Thigh girth increased immediately following both bouts of the DPFT, but not post SSRS. Intuitively, this might suggest a greater blood flow and/or secondary inflammatory response associated with the dance-type activity. However, as calf girth was unaffected, this is unlikely and the differences in the nature and demands of the exercise protocols are more likely responsible for the variance in muscle swelling.

The recovery of CMJ, RSI and MVC was similar after both exercise protocols, and decrements were not mitigated by a previous bout. The decrements of these measures of muscle function were greatest immediately post exercise, with the exception of MVC during the initial bout of SSRS which reached lowest levels 24 h post exercise. The MVC response was surprising given that the combination of both muscle damage and metabolic fatigue immediately post exercise could result in the greatest decline of neuromuscular function; as demonstrated by the other functional measures. Perhaps the different exercise protocols employed in the literature are responsible for this discrepancy. While the muscles may have been put under maximum stress in the present investigation, the time under tension was far less than during traditional, isolated muscle contraction models. As a result, the fatigue component might be less evident in these sport-specific protocols. Nonetheless, the results concur with the findings of a recent study demonstrating that well trained subjects received no protection against decrements in CMJ and MVC following a second bout of intermittent-sprint exercise, despite reduced DOMS and their familiarity with the mode of exercise (15). Arguably these activities are frequently encountered during training and performance in the present study population. Though a homogenous population was recruited in order to better control for training status and free-living physical activity levels and dietary behaviours, the participants were recruited from a university dance team. Jumps and landing tasks are incorporated in most dance activities (24) and many dance movements are characterised by explosive actions (33). As physically active females recreationally participating in dance, it is

conceivable that the training status of subjects in this study may explain the apparent lack of a RBE in these measures. Indeed, the only measure of muscle function to recover more rapidly following the second bout was sprint time, and given that the study population were less accustomed with this activity compared to the other performance measures, it is perhaps unsurprising that this was observed in both exercise groups. Moreover it is possible that the exercise stimuli were not of an adequate intensity to elicit neural adaptations. Indeed neural adaptations - which manifest as an increase in motor unit activation and/or increased recruitment of slow-twitch fibres to limit myofibrillar disruption - are associated with maximal-intensity contractions (10). Having said this, while arguably small, the observed performance improvement is nevertheless an important consequence of the RBE. The present study is in agreement with a number of investigations reporting that physically active or trained participants experience a RBE (8, 10, 11, 18).

In the present investigation, a four week recovery period was chosen not only to maintain exercise intensity between bouts, but also to control for menstrual cycle phase. Many investigations in females conduct testing during the early follicular phase where oestrogen is at its lowest levels, though still higher than males. While this may conveniently control for hormonal effects, this period represents only a quarter of the menstrual cycle (31). The application of the results reported in such studies is therefore limited, particularly as recent evidence suggests that EIMD and recovery may differ between menstrual cycle phases (16, 30). If oestrogen does indeed play a role in the attenuation of muscle damage and adaptation in females, testing in the luteal phase (where oestrogen is markedly elevated) may better observe these effects. Moreover while menstrual phase in the current study was determined with participants' contraceptive use in mind, it should be noted that some forms of contraception exposes the body to synthetic forms of oestrogen, and therefore naturally occurring oestrogen may be found in lower quantities (19). Indeed, non-contraceptive users

(with potentially higher concentrations of biologically active oestrogen) have been reported to receive more protection against EIMD compared to oral contraceptive users (29). While details of contraceptive use have been provided, the lack of control of contraceptive use is a limitation of the present investigation, and the reader should interpret the results with this in mind.

Some studies examining the RBE using female subjects do not report how changes in the menstrual cycle were (if at all) accounted for and many investigations using both males and females have combined sexes in treatment groups; failing to acknowledge the potential influence of sex. It is clear that while much more research is required regarding the potential influence of sex in muscle damage, recovery and adaptation, the accurate reporting of menstrual cycle phase and contraceptive use of female participants' is essential in order to develop our understanding. Nonetheless, the current investigation is in agreement with those who have demonstrated that muscle-damaging exercise can elicit a protective affect against a subsequent damaging exercise bout in females (5, 20, 23).

## PRACTICAL APPLICATIONS

This study aimed to investigate whether physically active females receive a protective effect against indices of EIMD after an identical repeated exposure of dance-type and repeated sprint exercise. Increases in CK and decrements in muscle soreness and one measure of muscle function were attenuated after a second bout; indicative of a RBE. Perhaps the most interesting finding was that, while there were group divergences in thigh girth and muscle soreness, adaptation from exercise stimuli of this nature was similar. Though limitations warrant consideration (for instance all participants were trained in dance and their contraceptive use varied) these results add to a growing body of evidence demonstrating that

different exercise paradigms can elicit a protective effect against damage for subsequent bouts. Specifically, the findings enhance our understanding of the implications on recovery and adaptation in exercising females. These data have important practical application, suggesting that preconditioning with activity-specific exercise may reduce symptoms of damage and improve recovery with subsequent exposure of the same stimuli. Future research should examine the influence of interventions to further attenuate the damage response experienced following exercise in the female population.

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**Figure Legends**

**Figure 1.** Muscle soreness in response to muscle damaging exercise post DPFT (n = 10) and SSRS (n = 11). Values presented as mean ± SEM. $^{\phi}$denotes significant bout effect. *denotes significantly different from bout 1 independent of group. $^{\#}$denotes significantly different between groups compared to bout 1. Significance at the $p < 0.05$ level.

**Figure 2.** Sprint time in response to muscle damaging exercise post DPFT (n = 10) and SSRS (n = 11). Values presented as mean ± SEM. $^{\phi}$denotes significant bout effect. *denotes significantly different from bout 1 independent of group. Significance at the $p < 0.05$ level.

**Figure 3.** Creatine kinase in response to muscle damaging exercise post DPFT (n = 10) and SSRS (n = 10) groups. Values presented as mean ± SEM. *denotes significantly different from bout 1 independent of group. Significance at the $p < 0.05$ level.

**Table 1.** Participant characteristics, mean ± SD

| | DPFT (n = 10) | | SSRS (n = 11) | | DPFT vs SSRS P value[1] | | Bout 1 vs Bout 2 P value[2] | |
|---|---|---|---|---|---|---|---|---|
| | **Bout 1** | **Bout 2** | **Bout 1** | **Bout 2** | **Bout 1** | **Bout 2** | **DPFT** | **SSRS** |
| **Characteristics** | | | | | | | | |
| Age (y) | 20 ± 1 | 20 ± 1 | 19 ± 1 | 19 ± 1 | .147 | .147 | - | - |
| Body mass (kg) | 56.9 ± 7.0 | 56.2 ± 6.3 | 60.1 ± 5.2 | 59.9 ± 4.3 | .253 | .129 | .141 | .695 |
| Stature (cm) | 161.5 ± 3.6 | 162.4 ± 3.6 | 166.1 ± 3.0 | 166.2 ± 3.4 | .006 | .008 | .546 | .724 |
| BMI ($kg \cdot m^2$) | 21.8 ± 2.4 | 21.3 ± 2.2 | 21.8 ± 2.0 | 21.7 ± 1.9 | .976 | .786 | .119 | .679 |
| **Average daily intakes[3]** | | | | | | | | |
| Energy intake (MJ) | 6.3 ± 1.6 | 6.2 ± 1.6 | 6.9 ± 2.6 | 6.2 ± 1.8 | .502 | .980 | .908 | .320 |
| Fat (g) | 52.0 ± 20.4 | 51.9 ± 19.2 | 61.4 ± 23.2 | 56.2 ± 21.9 | .340 | .643 | .993 | .470 |
| Protein (g) | 59.4 ± 17.5 | 64.2 ± 15.4 | 69.6 ± 32.8 | 60.6 ± 18.1 | .393 | .633 | .525 | .372 |
| Carbohydrate (g) | 207.8 ± 52.3 | 194.7 ± 57.7 | 206.2 ± 89.3 | 185.1 ± 63.3 | .960 | .722 | .411 | .317 |

DPFT, dance performance fitness test; SSRS, sport-specific repeated sprint test; BMI, body mass index; [1]DPFT vs SSRS, compared by independent student's t test; [2]Bout 1 vs Bout 2, compared by paired samples t test; [3]As determined from a 3-day food diary pre testing (Bout 1) and during four week recovery (Bout 2); assessed using dietary analysis software (Nutritics LTD, Ireland)

**Table 2.** Absolute values for limb girth, CMJ, RSI and MVC following bout 1 and 2 of DPFT (n = 10) and SSRS (n = 11), mean ± SEM

| | | Thigh girth (cm) | | Calf girth (cm) | | CMJ height (cm) | | RSI (cm·s$^{-1}$) | | MVC (N) | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| **Time** | **Bout** | **DPFT** | **SSRS** | **DPFT** | **SSRS** | **DPFT** | **SSRS** | **DPFT** | **SSRS** | **DPFT** | **SSRS** |
| **Pre** | **1** | 48.5 ± 1.1 | 48.5 ± 0.8 | 35.9 ± 0.7 | 35.8 ± 0.6 | 26.9 ± 0.8 | 26.0 ± 1.0 | 66.5 ± 5.0 | 71.0 ± 8.4 | 384 ± 21 | 371 ± 18 |
| | **2** | 48.6 ± 1.2 | 48.7 ± 0.9 | 36.1 ± 0.7 | 35.7 ± 0.6 | 27.2 ± 1.1 | 24.7 ± 1.2 | 64.3 ± 7.1 | 69.7 ± 11.0 | 382 ± 21 | 382 ± 19 |
| **0 h** | **1** | 49.2 ± 1.2[a] | 48.6 ± 0.8[b] | 35.8 ± 0.7 | 35.8 ± 0.6 | 24.7 ± 1.0 | 21.6 ± 0.9 | 57.2 ± 4.7 | 58.1 ± 7.2 | 336 ± 20 | 338 ± 19 |
| | **2** | 48.9 ± 1.1[a] | 48.9 ± 0.8[b] | 36.1 ± 0.7 | 35.7 ± 0.6 | 23.5 ± 1.0 | 20.8 ± 1.0 | 56.5 ± 6.8 | 57.4 ± 8.7 | 352 ± 18 | 331 ± 14 |
| **24 h** | **1** | 48.8 ± 1.2 | 48.6 ± 0.9 | 36.1 ± 0.7 | 35.9 ± 0.5 | 25.8 ± 1.2 | 23.4 ± 1.1 | 60.8 ± 6.2 | 64.4 ± 9.4 | 339 ± 19 | 331 ± 18 |
| | **2** | 48.9 ± 1.1 | 49.0 ± 0.8 | 36.2 ± 0.7 | 35.8 ± 0.6 | 25.9 ± 1.0 | 23.1 ± 0.9 | 61.2 ± 6.9 | 65.1 ± 9.4 | 371 ± 22 | 338 ± 14 |
| **48 h** | **1** | 48.7 ± 1.2 | 48.7 ± 0.8 | 36.1 ± 0.7 | 35.8 ± 0.6 | 25.7 ± 1.0 | 23.6 ± 1.1 | 62.2 ± 7.4 | 63.1 ± 10.0 | 352 ± 22 | 337 ± 19 |
| | **2** | 48.8 ± 1.1 | 49.0 ± 0.8 | 36.1 ± 0.7 | 35.8 ± 0.6 | 26.2 ± 1.2 | 23.5 ± 0.9 | 62.6 ± 7.3 | 69.9 ± 9.6 | 378 ± 20 | 351 ± 19 |
| **72 h** | **1** | 48.7 ± 1.1 | 48.5 ± 0.8 | 36.0 ± 0.8 | 35.8 ± 0.6 | 27.1 ± 1.5 | 23.6 ± 0.9 | 65.1 ± 7.2 | 66.9 ± 9.1 | 361 ± 22 | 345 ± 22 |
| | **2** | 48.7 ± 1.1 | 48.8 ± 0.8 | 36.0 ± 0.7 | 35.7 ± 0.6 | 27.6 ± 1.3 | 24.4 ± 1.0 | 65.7 ± 6.5 | 72.6 ± 10.1 | 397 ± 19 | 358 ± 17 |

DPFT, dance performance fitness test; SSRS, sport-specific repeated sprint test; DOMS, delayed onset muscle soreness; CMJ, countermovement jump; RSI, reactive strength index; MVC, maximal voluntary isometric contraction; [a,b] Values with unlike letters were significantly different. Significance at the $P < .05$ level.

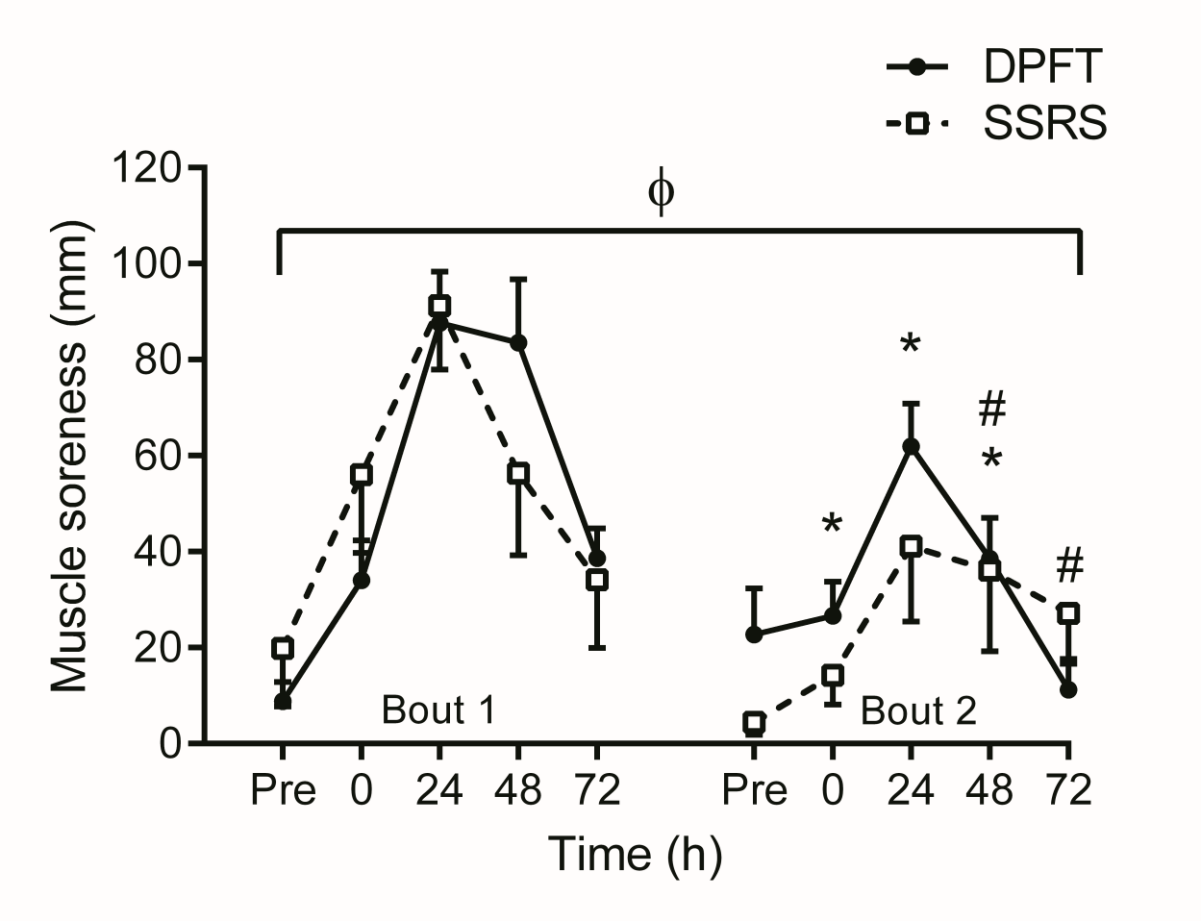


**Figure 1.** Muscle soreness in response to muscle damaging exercise post DPFT (n = 10) and SSRS (n = 11). Values presented as mean ± SEM. $^{\phi}$denotes significant bout effect. *denotes significantly different from bout 1 independent of group. $^{\#}$denotes significantly different between groups compared to bout 1. Significance at the $p < 0.05$ level.

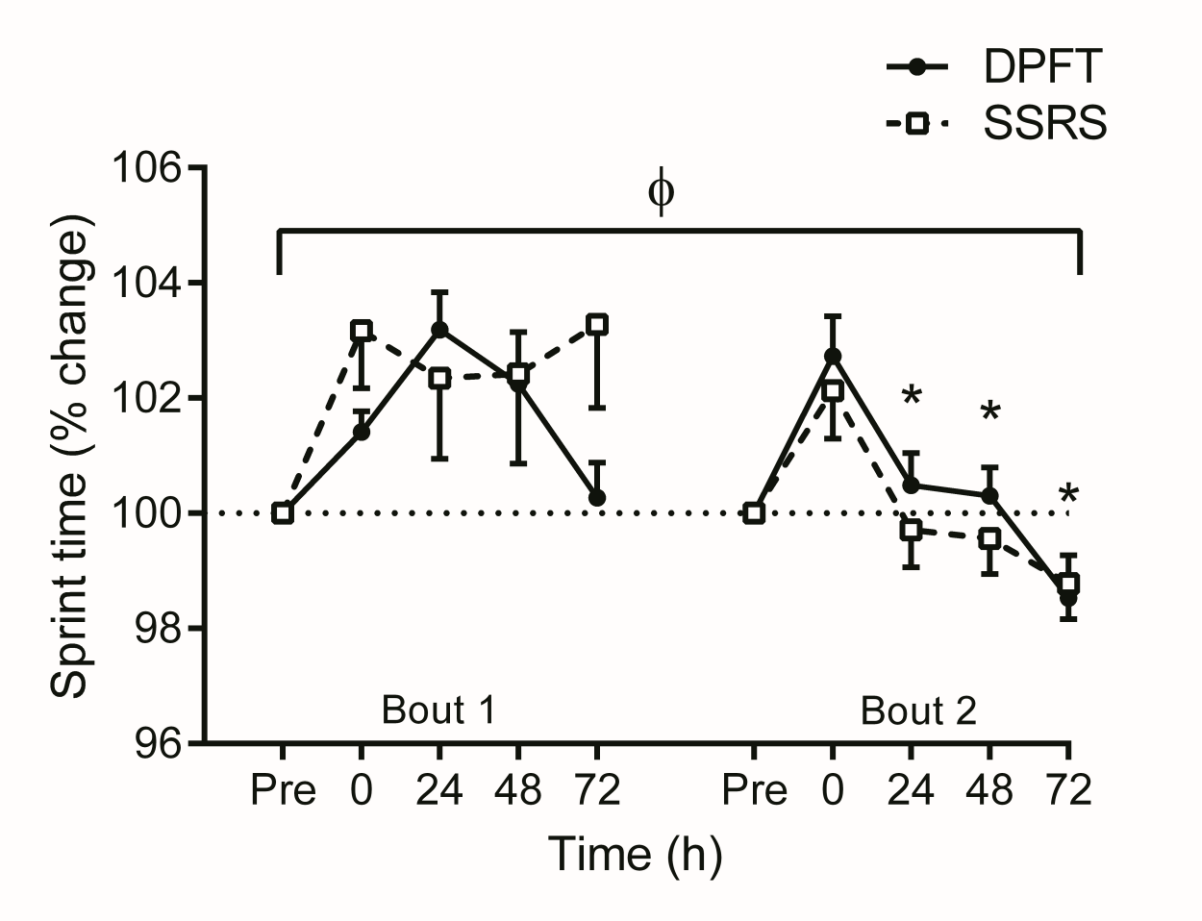


**Figure 2.** Sprint time in response to muscle damaging exercise post DPFT (n = 10) and SSRS (n = 11). Values presented as mean ± SEM. $^{\phi}$denotes significant bout effect. *denotes significantly different from bout 1 independent of group. Significance at the $p < 0.05$ level.

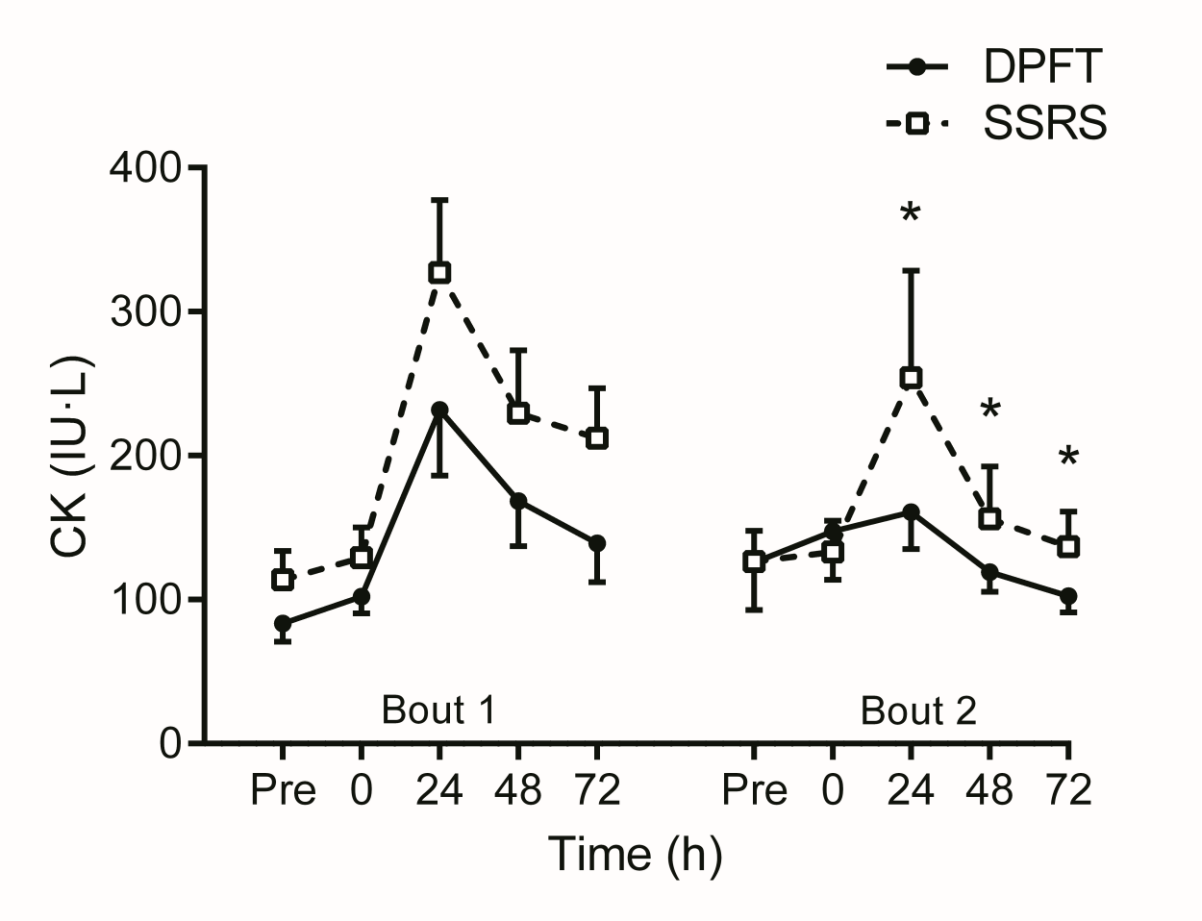


**Figure 3.** Creatine kinase in response to muscle damaging exercise post DPFT (n = 10) and SSRS (n = 10) groups. Values presented as mean ± SEM. *denotes significantly different from bout 1 independent of group. Significance at the $p < 0.05$ level.