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Recovery kinetics following different sprint training protocols: Resisted versus unresisted sprints

by

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Master Thesis, submitted to the professorial committee for the partial completion of Master Science Degree in ''Exercise and Health: Evaluation and Prescription''. Department of Physical Education and Sport Science, University of Thessaly.

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ΣΧΟΛΗ ΕΠΙΣΤΗΜΗΣ ΦΥΣΙΚΗΣ ΑΓΩΓΗΣ ΚΑΙ ΑΘΛΗΤΙΣΜΟΥ

ΤΜΗΜΑ ΕΠΙΣΤΗΜΗΣ ΦΥΣΙΚΗΣ ΑΓΩΓΗΣ ΚΑΙ ΑΘΛΗΤΙΣΜΟΥ

Η κινητική της αποκατάστασης μετά από διαφορετικά πρωτόκολλα προπόνησης ταχύτητας: Επιταχύνσεις με ή χωρίς αντίσταση

Της

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ABSTRACT

Liakou Christina: Recovery kinetics following different sprint training protocols: Resisted versus unresisted sprints

Sprinting is one of the most important physical capacities for many sports, and sprint-training is incorporated in athletes' training-programs. However, sprint training may lead to exercise induced muscle damage (EIMD), and performance deterioration the following days. Nevertheless, data regarding the recovery kinetics of EIMD, metabolism, and performance after acute sprint training is limited. The aim of the present study was to examine the recovery kinetics after different sprint-training protocols. In a crossover design, ten healthy men and women athletes aged 20.4 (2.5) [Mean (SD)] years performed: a) control trial (CT, no training), b) unresisted sprint training (UNR), c) resisted sprint training with an additional load of 10% of their body weight (BW) (R10), and d) resisted sprints training with an additional load of 20% of their BW (R20). Lactate increased post-training similarly in all sprint-training protocols (p=0.000), but not in CT. Creatine kinase (CK) increased up to 72h in UNR (p=0.009) and R10 (p=0.007), and up to 48h in R20 (p=0.017) while no changes were observed in CT. Delayed onset of muscle soreness (DOMS) of knee extensors (KE) of both dominant (DL) and non-dominant limb (NDL) increased up to 48h in UNR (p=0.046) and R20 (p=0.011) trials, and up to 72h in R10 trial (p=0.038). DOMS of knee flexors (KF) of both DL and NDL increased up to 48h in UNR (p=0.038) and R10 (DL: p=0.038; NDL: p=0.023) trials, while up to 72h in R20 trial (DL: p=0.046; NDL: p=0.025). Isokinetic eccentric torque of the KE of NDL decreased at 24h (p=0.039) post-training in all trials. 10-m sprint time increased (p=0.009) and average

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speed at 10-m sprint decreased (p=0.016) at 48h only in R20 trial. 30-m sprint time increased at 24h (p=0.012), 48h (p=0.001) and 72h (p=0.054) only in RST20 trial, while average speed at 30-m sprint decreased at 24h (p=0.033) and 48h (p=0.011) in all sprint training trials. Countermovement jump did not change after sprint training. Acute sprint-acceleration training, both unresisted and resisted may induce EIMD. Higher loads induce greater metabolic demands, EIMD symptoms and performance decline and this needs to be considered by coaches for effectively designing the training programs of their athletes to optimize athletic performance and minimize injury risk. We suggest that short acceleration sprints and jumps may be repeated 48h after unresisted and 10%BW-resisted sprint-training, while more than 72h of recovery are needed after 20%BW-resisted sprint-training.

Key words: speed, power, exercise-induced muscle injury, performance

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ΠΕΡΙΛΗΨΗ

Λιάκου Χριστίνα: Η κινητική της αποκατάστασης μετά από διαφορετικά πρωτόκολλα προπόνησης ταχύτητας: Επιταχύνσεις με ή χωρίς αντίσταση

Η ικανότητα για σπριντ είναι πολύ σημαντική για πολλά αθλήματα, και η προπόνηση σπριντ ενσωματώνεται ως βασικό συστατικό στα προπονητικά προγράμματα των αθλητών. Ωστόσο, η προπόνηση ταχύτητας, μπορεί να οδηγήσει σε ασκησιογενή μυϊκό τραυματισμό (EIMD) και σε μείωση της απόδοσης τις επόμενες ημέρες. Όμως, τα δεδομένα σχετικά με την κινητική αποκατάστασης του EIMD, του μεταβολισμού και της απόδοσης μετά από οξεία προπόνηση ταχύτητας, είναι περιορισμένα. Σκοπός της παρούσας μελέτης, ήταν να εξετάσει την κινητική της αποκατάστασης έπειτα από διαφορετικά πρωτόκολλα προπόνησης σπριντ. Στη διασταυρούμενη αυτή μελέτη, συμμετείχαν δέκα υγιείς άνδρες και γυναίκες αθλητές και αθλήτριες, ηλικίας 20,4 (2,5) [Μέσος όρος (SD)], που πραγματοποίησαν τα παρακάτω πρωτόκολλα: α) Συνθήκη ελέγχου (ΣΕ) χωρίς προπονητική παρέμβαση, β) προπόνηση σπριντ χωρίς αντίσταση (ΠΣΧΑ), γ) προπόνηση σπριντ με πρόσθετο φορτίο στο 10% της σωματικής τους μάζας (ΣΜ) (ΠΣ10%) και δ) προπόνηση σπριντ με πρόσθετο φορτίο το 20% της ΣΜ (ΠΣ20%). Η συγκέντρωση του γαλακτικού οξέος αυξήθηκε μετά την προπόνηση με παρόμοιο τρόπο σε όλα τα πρωτόκολλα προπόνησης (p=0,000). Η δραστικότητα της κρεατινικής κινάσης (CK) αυξήθηκε έως και τις 72h στην ΠΣΧΑ (p=0,009) και ΠΣ10% (p=0,007) και έως τις 48h στην ΠΣ20% (p=0,017), ενώ δεν παρατηρήθηκαν αλλαγές στη ΣΕ. Ο καθυστερημένος μυϊκός πόνος (DOMS) των εκτεινόντων του γόνατος (ΕΓ) τόσο του κυρίαρχου (ΚΠ) όσο και του μη κυρίαρχου ποδιού (ΜΚΠ) αυξήθηκε έως και τις 48h στα πρωτόκολλα ΠΣΧΑ

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(p=0,046) και ΠΣ20% (p=0,011), και έως τις 72h στο πρωτόκολλο ΠΣ10% (p=0,038). Ο DOMS των καμπτήρων του γόνατος (ΚΓ) τόσο του ΚΠ και του ΜΚΠ αυξήθηκε έως και τις 48h στα πρωτόκολλα ΠΣΧΑ (p=0,038) και ΠΣ10% (ΚΠ: p=0,038; ΜΚΠ: p=0,023), ενώ έως και τις 72h στο πρωτόκολλο ΠΣ20% (ΚΠ: p=0,046, ΜΚΠ: p=0,025). Δεν υπήρξαν αλλαγές στον DOMS στη ΣΕ. Η ισοκινητική έκκεντρη ροπή των ΕΓ του ΜΚΠ μειώθηκε στις 24h (p=0,039) μετά την προπόνηση σε όλα τα πρωτόκολλα. Ο χρόνος στο σπριντ 10-m αυξήθηκε (p=0,009) και η μέση ταχύτητα στα 10-m σπριντ μειώθηκε (p=0,016) στις 48h μόνο στο πρωτόκολλο ΠΣ20%. Ο χρόνος στα 30-m σπριντ αυξήθηκε στις 24h (p=0,012), 48h (p=0,001) και 72h (p=0.054) μόνο στο πρωτόκολλο ΠΣ20%, ενώ η μέση ταχύτητα στα 30-m σπριντ μειώθηκε στις 24h (p=0,033) και 48h (p=0,011) σε όλα τα πρωτόκολλα προπόνησης. Η απόδοση στο κάθετο άλμα με αντιμετάθεση, δεν μεταβλήθηκε μετά την προπόνηση σε καμία χρονική στιγμή. Συμπερασματικά, η οξεία προπόνηση σπριντ, τόσο χωρίς αντίσταση όσο και με αντίσταση μπορεί να προκαλέσει ασκησιογενή μυϊκό τραυματισμό. Τα υψηλότερα φορτία φαίνεται να έχουν μεγαλύτερες μεταβολικές απαιτήσεις ενώ παράλληλα προκαλούν εντονότερα συμπτώματα EIMD και μείωση της απόδοσης κάτι που θα πρέπει να ληφθεί υπόψη από τους προπονητές, για τον αποτελεσματικότερο σχεδιασμό των προγραμμάτων προπόνησης των αθλητών, τη βελτιστοποίηση της αθλητικής απόδοσης και την ελαχιστοποίηση του κινδύνου τραυματισμού. Προτείνεται ότι, η προπόνηση ταχύτητας και αλμάτων μπορούν να επαναληφθούν 48 ώρες μετά την προπόνηση σπριντ χωρίς αντίσταση και με αντίσταση έως 10% της ΣΜ, ενώ απαιτούνται περισσότερες από 72 ώρες αποκατάστασης μετά από προπόνηση σπριντ με αντίσταση 20% της ΣΜ.

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Λέξεις κλειδιά: ταχύτητα, ισχύς, ασκησιογενής μυϊκός τραυματισμός, απόδοση

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15/10/2023 05:31:37 EEST - 167.114.118.212

CONTENTS

AKNOWLEDGEMENTS ……...4

ABSTRACT ……5

ΠΕΡΙΛΗΨΗ …….7

LIST OF TABLES ……..13

LIST OF FIGURES ……..13

1. INTRODUCTION ……..14

2. SCIENTIFIC BACKGROUND ……17

2.1. Training methods for sprint improvement …….17

2.1.1. Unresisted acceleration training ……18

2.1.2. Resisted acceleration training ……19

2.1.3. Maximal velocity training……..21

2.1.4. Sprint specific endurance training……22

2.1.5. Speed endurance training……23

2.2. Exercise induced muscle damage ……23

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

2.2.1. Exercise induced muscle damage symptoms……26

2.3. Sprint training and EIMD ……29

2.3.1. Recovery kinetics after unresisted sprint training ……..29

2.3.2. Recovery kinetics after resisted sprint training ……..31

3. METHODS ……33

3.1. Participants ……33

3.2. Study design ……34

3.3. Exercise protocols ……35

3.4. Blood sampling ……..36

3.5. Evaluation of anthropometric characteristics, body composition and aerobic capacity ……..37

3.6. Performance evaluation ……..38

3.7. Muscle damage ……..39

3.8. Metabolism evaluation ……39

4. STATISTICAL ANALYSIS ……...40

5. RESULTS ……41

5.1. Baseline characteristics of the participants ……41

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

5.2. Lactate ……42

5.3. Creatine kinase ……..44

5.4. Delayed onset of muscle soreness ……..46

5.4.1. Muscle origin of the dominant and non-dominant limb ……..46

5.4.2. Muscle belly of the dominant and non-dominant limb ……..50

5.4.3. Muscle insertion of the dominant and non-dominant limb ……54

5.5. Performance ……57

5.5.1. Sprinting performance ……57

5.5.2. Isokinetic strength performance ……..58

5.5.3. Countermovement jump performance ……64

6. DISCUSSION ……70

7. CONCLUSIONS ……..78

8. REFERENCES ……..79

Appendix 1: Ethics approval ……93

Appendix 1B: Candidate's responsibilities throughout the study ……..95

Appendix 2B: Skills acquired during the MSc program ……..95

Appendix 2C: Academic activities during the MSc program ……..96

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

LIST OF TABLES

Table 1. Baseline characteristics of the participants ……41

Table 2. Changes in DOMS at muscle origin during recovery …….49

Table 3. Changes in DOMS at muscle belly during recovery ……53

Table 4. Changes in DOMS at muscle insertion during recovery …….56

Table 5. Changes in isokinetic strength during recovery ……..61

Table 6. Changes in countermovement jump performance during recovery …….65

LIST OF FIGURES

Figure 1. Study design ……..35

Figure 2. Changes in lactate ……43

Figure 3. Changes in creatine kinase ……..45

Figure 4. Changes in 10m-sprint time and average speed ……59

Figure 5. Changes in 30m-sprint time and average speed ……60

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

1. INTRODUCTION

Sprinting, consists of a main form of speed. Defining sprinting, it is a series of coupled flight and support phases, known as strides, orchestrated to displace the athlete's body down the track, at maximum acceleration or velocity, or both (Seitz, Reyes, Tran, Saez de Villarreal, & Haff, 2014). Sprinting requires an athlete to move forward with maximum velocity and has been described as rapid, unpaced, maximal effort running of 15 seconds or less (Ross & Leveritt, 2001). The 100-m track and field sprint event is probably the most characteristic example, in which the fastest sprinter classically wins the race (Rumpf, Lockie, Cronin, & Jalilvand, 2016). However, sprinting gives a critical advantage to other individual athletic events, as well as in team sports. For example, long-jumpers with an effective acceleration in run up may have a greater potential for a longer jump (Bridgett & Linthorne, 2006). Similarly, sprinting activities are very frequent in soccer and rugby games in all positions (Haugen, Tønnessen, Hisdal, & Seiler, 2014) with faster players being more effective in better positioning themselves against the opponent and gain the ball (Haugen et al. (2014), while straight sprinting mostly precedes goal-scoring situations during a match (Faude, Koch, & Meyer, 2012). Additionally, chronic sprint-training improves acceleration, maximal speed (Alcaraz, Carlos-Vivas, Oponjuru, & Martínez-Rodríguez, 2018); (Rumpf et al., 2016), agility, and power (Alcaraz et al., 2018; Rumpf et al., 2016; Sinclair et al., 2021). Thus, sprinting improvement consists one of the most basic goals, and sprint-training consists one of the main components of the training program for many athletes (Haugen, McGhie, & Ettema, 2019; Petrakos, Morin, & Egan, 2016).

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15/10/2023 05:31:37 EEST - 167.114.118.212

Sprinting, is performed through the stretch-shortening cycle and highly includes the component of eccentric muscle contraction (Hennessy & Kilty, 2001). However, eccentric muscle activities may lead to a phenomenon that is called exercise induced muscle damage (EIMD), the following days. EIMD, is manifested through physiological and biochemical symptoms, including elevated concentration of muscle proteins into the circulation, delayed onset of muscle soreness (DOMS), and reduction of muscle performance (Baird, Graham, Baker, & Bickerstaff, 2012; Deli et al., 2017; Ispirlidis et al., 2008; Jamurtas et al., 2005).

Even though sprint training is used by a wide range of sports, research evidence regarding the recovery kinetics after sprint training are scarce. So far, only limited data exist regarding unloaded sprint training, while only one study has examined the recovery kinetics after resisted sprint training (Bachero-Mena, Sánchez-Moreno, Pareja-Blanco, & Sañudo, 2020). In that study, the load varied from 0% to 80% of body weight (BW) and training resulted in elevation of creatine kinase (CK), lactic acid, neuromuscular fatigue, decrease of jumping ability and maximal speed, up to 24 h post-training (Bachero-Mena et al., 2020). Even though the above study introduced signs of muscle damage after resisted sprint, recovery kinetics were limited up to the first 24 hours following training. However, information regarding an athlete's capacity to recover after sprint training the following days is critical for coaches and athletes, in order to effectively design the appropriate training program and incorporate the training components of a microcycle, as well as avoid injuries, and maximize performance.

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The aim of the present study was to investigate recovery kinetics of exercise-induced muscle damage and performance indices following different sprint training protocols compared to a control trial.

Study Hypotheses

1. Sprint-acceleration training will increase metabolism.
2. Sprint-acceleration training will provoke EIMD.
3. Sprint-acceleration training will result in muscle performance deterioration.
4. Resisted sprint training will provoke greater EIMD and performance deterioration compared to unresisted sprint training.

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15/10/2023 05:31:37 EEST - 167.114.118.212

2. SCIENTIFIC BACKGROUND

2.1. Training methods for sprint improvement

Linear sprinting is composed of a series of distinct phases that is the start, the acceleration, maximum speed, and deceleration (Haugen, McGhie, et al., 2019), and several training methods have been developed in order to improve these phases and subsequently the overall sprinting performance (Alcaraz et al., 2018; Haugen, Seiler, Sandbakk, & Tønnessen, 2019; Haugen et al., 2014). Sprint training is mainly classified as sprint-specific training methods and sprint-non-specific training methods, while a combination of sprint-specific and non-specific methods are used (Rumpf et al., 2016). Sprint-specific training includes free sprinting (unloaded running on a flat surface), resisted sprinting (sled pulling or pushing, running with bands or uphill running), as well as assisted sprinting (running with a towing device, downhill running). Specifically, regarding sprint-acceleration, several sprint distances are recommended, usually within the range of 10 to 50 m depending on the sport and event characteristics but also on the athletes' training experience and level, and both unresisted and resisted sprinting are implemented (Haugen, McGhie, et al., 2019; Petrakos et al., 2016; Rumpf et al., 2016). Sprint-non-specific training includes strength, power and plyometric training (Rumpf et al., 2016). However, non-specific-sprint training, has been reported to have less training efficacy than specific-sprint training for distances of 0-10m and 0-20m but also for longer sprint distances (0-30m and >30m) (Rumpf et al., 2016). Nonetheless, sprint-non-specific training may enhance some secondary elements, that lead to an overall better sprint performance such as force production (Morin et al., 2012), running technique, step length

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(Barr, Sheppard, Agar-Newman, & Newton, 2014)but also strength and power (BAKER & NANCE, 1999)

In the following sections, literature review regarding sprint-training will mainly focus on sprint-specific training methods.

2.1.1. Unresisted sprint-acceleration training

Sprint-acceleration training with unresisted sprinting, consists of a basic training component for many athletes. Unresisted sprint training incorporates weight bearing sprints performed without any additional load. Research evidence has shown that, unresisted sprints can improve running speed over 20m, as well as some technical parts of sprinting (Rumpf et al., 2016). For example, a 6-week training program with unresisted sprint increases step length and decreases ground contract time in young and well trained individuals (sport science students), with no previews experience with specific sprint training (Kristensen, van den Tillaar, & Ettema, 2006). Similarly, 6 weeks of unresisted sprint training decreases flight time and step frequency, as well as contact time in field sport men athletes (rugby or Australian football players) (Lockie, Murphy, Schultz, Knight, & Janse de Jonge, 2012).

Acceleration is the first segment of sprinting. The faster an athlete accelerates, the sooner he/she reaches the maximum speed, thus, it is important to overcome this phase and enter the maximal speed phase, having gained the possible maximum acceleration (Haugen, Seiler, et al., 2019). Typically, a training program that aims to the improvement

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of the acceleration phase, includes distances ranging from 10-30m long performed in a number of sets, and full recovery required between sets (Haugen, Seiler, et al., 2019). There are also techniques that aim to improve the starting phase of the acceleration, comprising blocks or crouch starts, or several jumps (Haugen, Seiler, et al., 2019). An example of an acceleration session could be runs over 20m, from a crouched start, with 2-min recovery while an elite sprinter can execute sprints over 40 m from starting blocks with 7-min recovery in between sets (Haugen, Seiler, et al., 2019; Petrakos et al., 2016; Rumpf et al., 2016).

2.1.2. Resisted sprint-acceleration training

Resisted sprint training is one of the most used strategies to improve acceleration (Bachero-Mena et al., 2020; Haugen, Seiler, et al., 2019; Mangine et al., 2018). Resisted sprints, are a form of speed training in which an external resistance is applied to the natural sprint performance (uphill running, sled pull or push, parachute running) (Haugen, Seiler, et al., 2019; Petrakos et al., 2016; Rumpf et al., 2016). Resisted sprinting includes uphill sprints, sled sprints or sprints with motorized devices (Cahill et al., 2019; Haugen, Seiler, et al., 2019). Sled sprints are of the most investigated methods consisting of straight-line sprint efforts whilst towing a sled device with an additional load, prescribed either as a percentage of body mass, a targeted reduction in velocity compared with unresisted sprint velocity or as an absolute load (Petrakos et al., 2016).

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Several loads have been proposed for resisted sprint training for improving performance and acceleration, however, the most effective load has yet to be determined, as different loads result in improvement of sprint performance. According to the percentage of reduction that is caused on maximum speed time under unloaded sprinting, the prescribed load can be defined as light (<10% velocity decrement), moderate (10-15%), heavy (15-30%), and very heavy (>30%) loads (Petrakos et al., 2016). To prescribe the external load for sprint training, the demands of the event or sport, or the daily training goal should be considered. Thus, for track and field sprinters, loads that do not decrease unresisted sprint velocity by more than 10-12% are mainly recommended, while for field or other sport athletes who overcome external resistance while blocking and tackling or while moving an external mass loads of 20-30% of BM are usually proposed to improve acceleration (Alcaraz et al., 2018). However, loads that decrease sprint velocity by even ~50% corresponding to 69%-96% BM, have also been suggested as optimal to improve horizontal force and power production during early sprint acceleration (Cronin & Hansen, 2006; Cross, Samozino, Brown, & Morin, 2018; Morin et al., 2012; Zafeiridis et al., 2005). Regarding the optimal distances, 10-30m are recommended, with a total volume of 50-200m, and full recovery after each repetition (Haugen, Seiler, et al., 2019). Resisted sprinting seems to be more efficient for distances 0-10 and 0-20m, which means that it can improve acceleration during the first running meters (Rumpf et al., 2016). This method is often used in the preparatory training phase of an athlete, for boosting acceleration and power, and leads to an increase in stride

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length during the unresisted running (Costello, 1985; Cronin & Hansen, 2006; Haugen, Seiler, et al., 2019).

Different loads may lead to different sprint adaptations. For example, 6 weeks of resisted sprint training with a load of 12.6% BM decreased maximal speed and improved force production and linear speed in a distance up to 10m in men athletes (Lockie et al., 2012). In another study (Bachero-Mena & González-Badillo, 2014), that used a 7-week, 14-session, sled-resisted sprint training with 3 different loads (5%, 12.5%, and 20% BM), it was shown that for the improvement of the initial phase of the acceleration up to 30m, loads of ~20% of BM should be used, while for improvement of high-speed acceleration phases, loads of ~5-12.5% of BM should be preferred. Additionally, a heavier load of 40% of BM has also been reported to improve performance in the initial acceleration phase, that is from 10-20m and 20-30m in a distance of 30m (Wibowo & Abdullah, 2017) .

Comparing unresisted with resisted sprint training, although both methods are reported to improve sprint acceleration, evidence suggests that resisted sprint training is more effective in improving sprint acceleration performance over unresisted sprint training (Sinclair et al., 2021; Zafeiridis et al., 2005), or over traditional strength and power training (Petrakos et al., 2016).

2.1.3. Maximal velocity training

The desideratum for a sprint runner or a speed athlete, is to manage the preservation of speed, for as long as possible. The aim of maximal speed training, is to

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15/10/2023 05:31:37 EEST - 167.114.118.212

maintain speed over 30 m, the point where speed is near 100% (Hanley, Walker, Paradisis, Merlino, & Bissas, 2021). Maximal velocity sprint training typically begins with a flying for about 20-40m followed by a 30 – 50 m sprint with maximum intensity (>98%), for as long as the maximum speed is not decreased, with full recovery between sets. The overall session volume, is proposed not to exceed 150m (Haugen, Seiler, et al., 2019). Except from flying starts, training during maximal velocity could start with a 3-point start or block starts, that focus both on acceleration and maximal speed. Maximum velocity training may also include distances from 40m to 80m with rest periods of 3-4min between repetitions (Rumpf et al., 2016). Sprint-non-specific methods are also used to develop maximal speed, such as strength training with loads of 60-100%, and power training consisting of explosive exercises with loads of 50-90% (Rumpf et al., 2016). Also, plyometric training, with explosive bodyweight exercises, including repeated vertical jumps, has been reported to improve speed in distances of 10m to 40m (Ochoa, Fernandez-Gonzalo, & de paz, 2014; Rimmer & Sleivert, 2000; Seitz et al., 2014).

2.1.4. Sprint-specific endurance training

The aim of sprint-specific endurance training is to improve the ability of an athlete to maintain maximum velocity for as long as possible. During the deceleration phase, there is a reduction on the rate of steps, as a result of central nervous system fatigue (Haugen, Seiler, et al., 2019). This method of training aims to preserve maximum speed, and delay the deceleration phase (Girard, Mendez-Villanueva, & Bishop, 2011; Haugen,

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15/10/2023 05:31:37 EEST - 167.114.118.212

Seiler, et al., 2019).Runs during this session, usually last about 7-15sec at an intensity of 95-100%, with full recovery between repetitions. The sets usually involve standing starts, with each repetition ranging from 80-150m. The total training volume ranges from 300m to 900m depending on age, experience and training state of each individual, with and intensity over 95% (Girard et al., 2011; Haugen, Seiler, et al., 2019).

2.1.5. Speed Endurance

The difference between sprint specific endurance training and speed endurance training is volume and intensity. In speed endurance, intensity is submaximal, and it is often used during preparation phase. For example, a typical training could include distances of 60-80 m performed at 90-95% intensity, with 2-4 min rest or more between repetitions (it depends on the total volume), and a total volume varying from 600m to 2000m (Girard et al., 2011; Haugen, Seiler, et al., 2019).

2.2. Exercise-induced muscle damage

Strenuous exercise, especially when unaccustomed and with an eccentric component included, may result in exercise-induced muscle damage (EIMD) (Twist & Eston, 2005). EIMD is accompanied by several symptoms the following days. Alongside with morphological and ultra-structural changes to muscle architecture (Fridén, 1984), muscle damage is manifested by muscle soreness, force and power output decline (Priscilla M. Clarkson & Hubal, 2002), deterioration of range of motion (ROM) (Cleak &

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15/10/2023 05:31:37 EEST - 167.114.118.212

Eston, 1992), deterioration in running economy (Chen, Nosaka, Lin, Chen, & Wu, 2009; Petrakos et al., 2016), alterations in position sense and reaction angle (Vassilis Paschalis et al., 2007; V. Paschalis et al., 2010), and connective tissue damage (Brown, Child, Day, & Donnelly, 1997; Tofas et al., 2008). It can also induce muscle swelling (Howell, Chleboun, & Conatser, 1993), increased flux of muscle proteins into the circulation (Jamurtas, 2018), migration of monocytes and neutrophils into the injured area (Peake, Neubauer, Della Gatta, & Nosaka, 2017; Smith, 1992; Tidball & Villalta, 2010), and increased oxidative stress in blood and muscle (Vassilis Paschalis et al., 2007; Theodorou et al., 2011).

In EIMD, the initial injury is thought to occur either via mechanical or metabolic stress (Kendall & Eston, 2002). The initial mechanical disruption of (Fridén & Lieber, 1992), is followed by impaired excitation-contraction coupling and calcium (Ca^{2+}) signaling, and finally, activation of calcium-sensitive degradation pathways (Peake et al., 2017). The initial myofibril disruption has been attributed to high forces (Tiidus & Ianuzzo, 1983), high tensile stresses (McCully & Faulkner, 1985), or an imbalance in a nearby sarcomere tension (Fridén & Lieber, 1992). During eccentric contractions, fewer fibers are activated to exert a given amount of force as compared to concentric or isometric contractions; therefore, larger forces per muscle fiber are developed, resulting to greater damage. Moreover, during eccentric contractions, some sarcomeres more resistant to stretching than others force weaker sarcomeres to absorb more stretch. With repeated eccentric contractions, the weaker sarcomeres first, and then the stronger sarcomeres are overstretched. If the latter fail to withstand the stretching force during the relaxation

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15/10/2023 05:31:37 EEST - 167.114.118.212

phase, damage will occur (Jamurtas, 2018). After the initial trauma, progressive myofibril degeneration is observed in some fibers suggesting a second phase of events, with release of intracellular proteins and infiltration of the tissue by neutrophils and macrophages (Tidball & Villalta, 2010). This secondary injury occurs as a result of the disruption of the membrane of the sarcoplasmic reticulum or sarcolemma and disturbance of Ca^{2+} homeostasis (Belcastro, Shewchuk, & Raj, 1998). Increases in intracellular Ca^{2+} concentration leads to additional degradation of muscle fibers due to activation of calcium-dependent proteolytic (Proske & Allen, 2005). These processes are accompanied by inflammatory responses and associate with fiber breakdown and repair (Proske & Allen, 2005). Neutrophils invade the injured tissue within several hours following the initial insult, in order to destroy the damaged muscle tissue, heading towards the muscle inflammation area (Peake et al., 2017; Trappe et al., 2011). Additionally, the activated macrophages produce pro-inflammatory cytokines such as IL-1β, IL-6, and TNF-α within 24 hours and can be present for several days (Beaton, Tarnopolsky, & Phillips, 2002). These cytokines act along in order to deal with inflammation (Moldoveanu, Shephard, & Shek, 2001; Zhang & An, 2007). Furthermore, reactive oxygen and nitrogen species (RONS) that are produced in excess by neutrophils and macrophages, consequently leading to oxidative stress, contribute to the initial damage, but they also facilitate the restoration of the injured muscle by degrading the damaged tissue, and probably by enhancing the repair processes (Tidball & Villalta, 2010). Finally, the injured muscle regenerates via the activation of satellite cells which proliferate in response to injury and

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15/10/2023 05:31:37 EEST - 167.114.118.212

give rise to regenerated muscle (Bazgir, Fathi, Rezazadeh Valojerdi, Mozdziak, & Asgari, 2017; Owens, Twist, Cobley, Howatson, & Close, 2019).

2.2.1. Exercise-induced muscle damage symptoms

One of the most remarkable symptoms of EIMD, is the force loss. Force reduction correlates with research findings of muscle injury (Chatzinikolaou et al., 2014), and seems to be one of the most reliable indices of EIMD (Abernethy, Wilson, & Logan, 1995). Due to force reduction an athlete may have a lower performance on the following days, until the symptoms recover. This phenomenon is usually spotted immediately after an intensive bout of exercise, and can be detected days after exercise (Draganidis et al., 2015). The magnitude of force loss, depends on the type, intensity and volume of exercise, with high-force eccentric exercise provoking greater loss (Newham, Jones, & Clarkson, 1987). Performance reduction, may be present for over 72 hours, especially when exercise volume is high, and without previous familiarization (Twist & Eston, 2005).

Delayed onset muscle damage (DOMS) is another well-known phenomenon accompanying EIMD. It is characterized by muscle pain, discomfort and stiffness, detected usually after 24 hours, and peaking at 48 to 72 hours after exercise (MacIntyre, Reid, & McKenzie, 1995). As a result of this discomfort, DOMS can affect athletic performance, by causing a reduction in range of motion, and attenuation and peak torque (Cheung, Hume, & Maxwell, 2003). There are many theories trying to give an explanation about the pain

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15/10/2023 05:31:37 EEST - 167.114.118.212

stimulus, associated with DOMS, such as lactic acid, muscle spasms, inflammation and other proposed models (Cheung et al., 2003).

CK is an indirect marker of EIMD. CK is an enzyme expressed in various tissues and cell types and is located inside the muscle (Baird et al., 2012). CK is released by the muscles into the lymphatic system, where it is transported to the thoracic duct and enters the blood stream (Havas, Komulainen, & Vihko, 1997). Under situations of muscle trauma, CK diffuses into the extracellular space due to destruction of the cytoplasmic membrane and its activity increases in the circulation (Noakes, 1987). CK activity usually peaks at 24h-48h post EIMD, and depending on the extend of the injury, it recovers after several days post-exercise (Priscilla M. Clarkson & Hubal, 2002; Deli et al., 2017; Jamurtas et al., 2005; Papanikolaou et al., 2021). Increased CK has been reported amongst others, after eccentric exercise (Deli et al., 2017), downhill running (Jamurtas et al., 2013), speed endurance training (Tzatzakis et al., 2019), and small-sided games (Papanikolaou et al., 2021).

It is worth noting that, CK activity presents high interindividual variability, such that some individuals are found to have high levels of serum CK compared to other individuals of similar characteristics both at rest and when exposed to the same exercise protocol even when other non-modifiable factors such as gender, age, and training status are accounted for (Baird et al., 2012). More specific, resting CK activity is higher in trained compared to untrained individuals (Hortobágyi & Denahan, 1989; Koutedakis et al., 1993; Vincent & Vincent, 1997), and in males compared to females (Borges & Essén-Gustavsson, 1989; Strømme, Rustad, Steensland, Theodorsen, & Urdal, 2004)). Following exercise,

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15/10/2023 05:31:37 EEST - 167.114.118.212

trained individuals, tend to have lower CK activity compared to untrained (Vincent & Vincent, 1997). What is more, among athletes, the ones with the best physical condition, tend to have lower CK activity the following days (Maxwell & Bloor, 1981). Regarding gender effect, teen girls demonstrated lower CK activity than boys after an all-out 100m swim (Fu, You, & Kong, 2002), probably due to differences in oestrogen activation, that stabilize the membrane and CK leakage from muscles (Tiidus & Ianuzzo, 1983). Additionally, some athletes tend to be “low responders”, while others tend to be “high responders” (Brancaccio, Maffulli, & Limongelli, 2007). A breakpoint at 300-500 IU/l of CK serum release after exercise has been reported (Totsuka, Nakaji, Suzuki, Sugawara, & Sato, 2002). The levels of CK released are associated with distinctive individual muscular characteristics. In high CK responders, the cross-sectional area and volume of the quadriceps femoris muscle, seem to be lower than low responders (Totsuka et al., 2002). After endurance exercise, variability in each subject may be due to differences in the degree of physical workload in performing exercise. Also, individual muscle strength is an important factor affecting serum CK activity (Totsuka et al., 2002). Overtraining may also explain why some athletes tend to be ‘’high responders’’, although in order to substantiate this statement, a large increase in CK must be observe with a combination of reduced exercise tolerance (Hartmann & Mester, 2000). For ‘’low CK responders’’, low % of body fat, and muscle mass may account for the absence of response (Heled, Bloom, Wu, Stephens, & Deuster, 2007). Considering the above, an issue has been raised on the validity of CK activity to reflecting EIMD (Baird et al., 2012). However, increased levels of CK may lead to a further injury and prolonged fatigue, while persistent increased CK levels

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15/10/2023 05:31:37 EEST - 167.114.118.212

may indicate subclinical disorders (Baird et al., 2012; Brancaccio et al., 2007), thus, it is imperative to monitor the changes of the enzyme due to intense exercise.

2.3. Sprint training and EIMD

Sprinting is implemented through the stretch-shortening cycle (SSC), where the pre-activated muscle is first stretched (eccentric action) and then followed by the shortening (concentric) action. Thus, sprint training includes the component of the eccentric muscle contraction, which may cause EIMD and consequently the related symptoms (Kristoffersen et al., 2018). Most of the evidence indicating that sprint training may cause EIMD, comes from studies that used unresisted sprints (Hedayatpour & Falla, 2015; Wackerhage, 2003), while limited data exists on resisted sprint training (Bachero-Mena & González-Badillo, 2014).

2.3.1. Recovery kinetics after unresisted sprint training

Unresisted sprint training may result in EIMD and fatigue (Thomas et al., 2018), although no effect of sprint training has also been reported (Grazioli et al., 2020). However, data regarding recovery kinetics following acute sprint training comes from studies that incorporated repeated sprint training (Howatson & Milak, 2009; Keane, Salicki, Goodall, Thomas, & Howatson, 2015; Thomas et al., 2018; Woolley, Jakeman, &

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15/10/2023 05:31:37 EEST - 167.114.118.212

Faulkner, 2014), while limited data exist on linear sprint training (Bachero-Mena et al., 2020; R. J. Johnston, Watsford, Austin, Pine, & Spurrs, 2015).

Maximal repeated sprints (15 x 30m) have been reported to result in deterioration of the potentiated quadriceps twitch force up to 48h, fatigue up to 72h, and decline in maximal voluntary activation up to 24h post training in male athletes (Thomas et al., 2018). In another study, a sport-specific repeated sprint training (15 x 30m with 10-m deceleration zone) resulted in CK and DOMS elevation up to 72h, whereas a decline in maximal voluntary contraction up to 48h post-training in collegiate field-based team sports male athletes (Howatson & Milak, 2009). Similarly, a higher volume of multiple sprints (40 x 15m with a 5-m deceleration zone) increased CK, aspartate aminotransferase and DOMS, while decreased sprint performance, vertical jump, and agility for up to 72h post-training in physically active males (Woolley et al., 2014). As with male athletes, female athletes also present a similar pattern of EIMD after repeated sprint training. Female collegiate athletes, demonstrated elevated CK and DOMS up to 72h, while reduced CMJ height and sprint performance up to 48h and 72h, respectively, after a repeated sprint protocol (15 x 30m) (Keane et al., 2015). On the other hand, repeated sprint training of moderate volume (15 x 20m) of sprints, either in line or with two changes of direction, did not induce any changes in jumping performance, CK levels, knee extension maximal and explosive strength and also quadriceps and hamstrings echo, at any time point up from post to 72h post-training in well-trained collegiate athletes (Grazioli et al., 2020). It seems that training volume but also training status, critically affects the occurrence and magnitude of EIMD during recovery.

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

As far as we know, only two studies addressed EIMD after linear sprint training. The first study (M. Johnston, Cook, Crewther, Drake, & Kilduff, 2015), used a maximal speed training protocol (6 x 50m with 5-min rest between efforts) to examine the neuromuscular and endocrine responses. The authors reported an elevation of lactate immediately post and of CK and DOMS at post and up to 24h post-training, while a decline of testosterone and cortisol at 2h post-training. Jumping performance parameters exhibited a bimodal pattern of recovery, with several of them declining immediately post, recovering at 2h post, and suffering a secondary decline at 24h post-training. The second study (Bachero-Mena et al., 2020), is also the only one that incorporated resisted sprints, thus the results are presented in the next section.

2.3.2. Recovery kinetics after resisted sprint training

Literature is poor in respect to recovery after resisted sprint training. As far as we know, the only study that investigated recovery kinetics of EIMD after resisted linear sprint training, was that of Bachero-Mena et al., (Bachero-Mena et al., 2020). In that study, fifteen male participants performed 8 × 20m sprints with 2-min rests between sprints under 5 different loading conditions: 0%, 20%, 40%, 60%, and 80% BM. Results showed that lactate increased significantly post-training after all loading conditions, however, as sled loadings increased, higher lactate concentrations were observed. Additionally, CK elevated from pre to post 24h in a similar way in all loading conditions, but without any statistical difference between training conditions, even if this study

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15/10/2023 05:31:37 EEST - 167.114.118.212

included heavy loads, up to 80% of BM. Regarding performance, CMJ height decreased from immediately post- in all conditions but 24h post, CMJ performance return to the initial levels. Similar to this, speed performance at 20-m sprint decreased immediately post-training only in 0% and 80% loading, while at 24h, sprint times had returned to pre levels. Moreover, mean power decreased during knee flexion at 24h post-training, but only in 20% load.

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

3. METHODS

3.1. Participants

According to a preliminary power analysis (a probability error of 0.05, and a statistical power of 90%), a total sample size of 8 - 10 participants was considered appropriate in order to detect statistically meaningful changes between groups. Thus, ten healthy male and female track and field and football athletes, aged 18-30 years old, volunteered to participate in the study. Inclusion criteria were a) experience in sprint-training for at least one year, b) no musculoskeletal injuries for at least six months prior to the study, c) no use of ergogenic supplements or other drugs for at least one month prior to the study, d) no participation at exercise with eccentric component for at least three days prior to the study, e) no alcohol and energy drinks consumption before each experimental trial. All participants signed an informed consent form and they informed about all the benefits and risks of the study. Written informed consent to participate in the study was provided by all volunteers after they were informed about all potential risks, discomforts, and benefits of the study. The procedures were in accordance with the 1975 Declaration of Helsinki, as revised in 2013, and approval was received from the bioethics committee of the Department of Physical Education and Sport Science, University of Thessaly (17159/2-8/9-12-2020). The study is registered at ClinicalTrials.gov (ID: NCT04766411).

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

3.2. Study design

The study used a randomized, four-trial, cross-over, repeated measures design (Figure 1). During the first week (1st, 2nd visits), the participants signed an informed consent form and were informed about all the benefits and risks of the study. In addition, they filled in and signed a medical history questionnaire. Participants were then instructed by a dietitian how to record a 7-days diet recall to ensure that they would not consume to greater extent nutrients that may affect EIMD and fatigue (e.g. antioxidants, amino acids, etc.) and to ensure that the energy intake during the trials would be the same. Additionally, baseline assessment of anthropometric characteristics, body composition, and aerobic capacity (VO_{2max}) were performed. During the 3rd visit, the participants performed in a randomized order, one of the four experimental protocols: a) Unresisted sprint-acceleration training (UNR), b) resisted sprint-acceleration training with an external load of 10% of BM (R10), c) Resisted sprint training with an external load 20% of BM (R20), and d) Control trial (CT).

Prior to each experimental protocol, assessment of 10-m and 30-m sprint-time and average speed at 10m and 30m sprint, DOMS of the knee extensors (KE) and flexors (KF) of both dominant (DL) and non-dominant limb (NDL), blood lactate, CK, peak concentric and eccentric isokinetic torque, as well as countermovement jump (CMJ) performance was performed. Additionally, blood lactate and DOMS were also assessed post each trial, while DOMS and all the remaining of the above indices were further assessed at 24h, 48h and 72h after the end of the trial. During the 7th-10th, 11th-13th, and 14th-16th visits, participants performed the same procedures for the remaining experimental protocols.

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

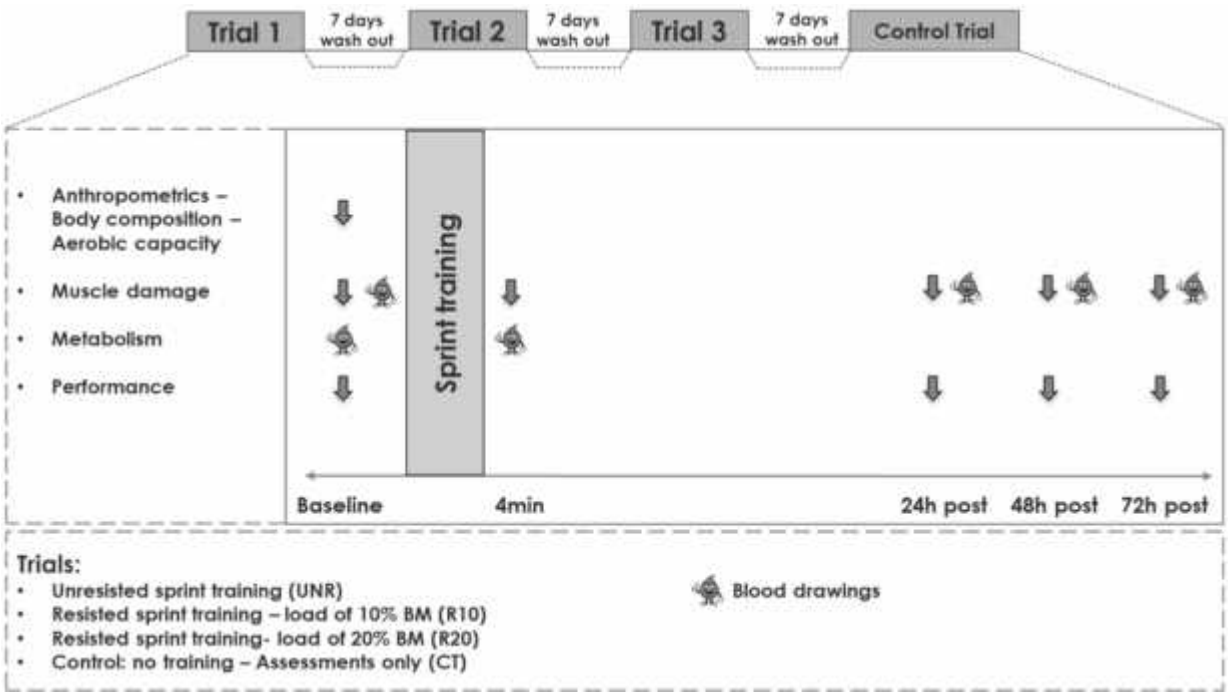

Figure 1. Study design.

3.3. Exercise protocols

Each protocol consisted of 2 sets of 3 x 20-m and 1 set of 3 x 30-m sprints (total distance of 210m). For the control trial, participants performed all the measurements that were included in the experimental trials without performing any exercise protocol. In all of the experimental protocols, a 3-min rest period between sets was applied. Prior to the exercise protocol, the individuals performed a warm-up of a total duration of 30-35 min consisting of 10-min submaximal running and stretching exercises, followed by a sprint-

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

specific warm-up (sprint drills, two to three submaximal 30-40-m accelerations and another two maximal 10-20-m accelerations).

Before each exercise trial, there was an extensive explanation of the training contents. In order to avoid a repeated-bout effect, muscle damage markers, performance and neuromuscular fatigue indicators, was measured before each trial. Additionally, a 7-days wash out period was applied between trials. Testing and blood sampling sessions were performed at the same time of day for all the trials.

3.4. Blood sampling

Participants were asked not to engage in any intense physical activity and not to consume alcohol or caffeine products for at least 72h before reporting to the laboratory. Blood samples (20 mL) were drawn before the exercise, immediately post, 24, 48 and 72h post-exercise from a forearm vein with participants in a seated position. Plasma was prepared by centrifugation (1370g, 4°C, 10 min) from blood samples collected into tubes containing ethylenediaminetetraacetic acid (EDTA) to measure CK activity. Plasma samples stored at −80 °C and were thawed only once, before the analysis.

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

3.5. Evaluation of anthropometric characteristics, body composition and aerobic capacity

Body mass was measured to the nearest 0.05kg (Stadiometer 208; Seca, Birmingham, UK) while being lightly dressed and barefoot, and standing height was measured to the nearest 0.1cm (Stadiometer 208; Seca, Birmingham, UK), according to a previous study (Poulios 2018). Body Mass Index (BMI) was calculated from the equation BMI (kg/m^2) = body weight (kg)/height^2 (m).

Body composition assessment of participants was performed using a dual beam X-ray absorptiometry machine (DXA, Lunar DPXNT) and its corresponding software (Encore 2007, General Electric Company, Madison, WI, USA). A full-body assessment was performed, with participants placed in a supine position with their body aligned, knees extended, and arms parallel and next to the body. The variables extracted were the amount of muscle mass, fat mass and lean body mass, percentage (%) of body fat as well as bone mass and bone mineral density. All measurements were made with the machine fully calibrated.

Aerobic capacity was measured via maximum rate of oxygen consumption (VO_{2max}) using an automatic gas exchange analyzer (Vmax Encore 29, BEBJO296, Yorba Linda, CA, USA), during a graded exercise protocol on a treadmill (Stex 8025 T, Korea), according to a previous study (Poulios 2018). The initial speed was set at 10 km/h and increased by 1km/h every 2min, with 0% slope throughout the measurement. The heart rate (HR) was recorded continuously during the measurement using a Polar HR monitor (Polar H10

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15/10/2023 05:31:37 EEST - 167.114.118.212

oscilloscope). The procedure was terminated when participants met at least three of the following criteria: (i) appearance of a plateau in the oxygen uptake curve despite increasing speed, (ii) respiratory quotient >1.10, (iii) HR close to age-predicted HR_{max}, (iv) participant exhaustion (Medicine 2013). To calculate the volumes of oxygen and carbon dioxide, an open spirometry system was used, with the possibility of recording and analyzing the gases every 30 sec (breath by breath). Before each measurement, the analyzer was calibrated with commercially available precision gases (16% O_2, 4% CO_2, 80% N_2).

3.6. Performance evaluation

Running speed of 10-m and 30-m sprints were measured on a track and field stadium using light cells (Chronojump system). CMJ was performed on a force platform to assess jump height, ground reaction force, peak and mean power, vertical stiffness and peak rate of force development. The peak concentric, and eccentric isokinetic torque of the extensors (KE), knee flexors (KF) of the DL and NDL was evaluated on an isokinetic dynamometer at 60°/sec (Cybex, HUMAC NORM 360, Ronkonkoma, NY) at $60^{o}\cdot Cyb^{-1}$ as previously described (Poulios 2018).

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

3.7. Muscle damage evaluation

Delayed Onset Muscle Damage (DOMS), of knee extensors and knee flexors of both lower extremities was determined during palpation of the muscle belly and the distal region after performing three repetitions of a full squat, and each participant rated perceived soreness on a scale ranging from 1 (no soreness) to 10 (very sore).

CK was measured using a Clinical Chemistry Analyzer Z1145 (Zafiropoulos Diagnostica, Greece) with commercially available kits (Zafiropoulos, Greece).

3.8. Metabolism evaluation

Capillary blood for lactate measurements was collected at rest (pre) as well as 4 minutes after the end of each training protocol, and lactate was estimated with a hand portal lactate analyzer (Lactate Plus, Nova biomedical, USA).

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15/10/2023 05:31:37 EEST - 167.114.118.212

4. STATISTICAL ANALYSIS

The normality of the sample distribution was examined with a Shapiro Wilk test. CK and DOMS were analyzed with non-parametric tests. Friedman and Wilcoxon Signed Rand tests were performed for within trials analysis, and Kruskal-Wallis and Mann Whitney tests were applied for between trials comparisons. All the remaining dependent variables were analyzed using a two-way ANOVA [trial (unresisted sprint training, 10% BW resisted sprint training, 20% BW resisted sprint training, Control trial) × time (pre-training, 24h, 48h, 72h)] with repeated measures on time to examine possible differences on recovery after the three sprint training protocols and control trial. Statistical significance was set at $p < 0.05$. Effect Sizes (ESs) and confidence intervals (CIs) were calculated using the Hedge g method, corrected for bias. Accordingly, ES was interpreted as none, small, medium-sized, and large for values 0.00 to 0.19, 0.20 to 0.49, 0.50 to 0.79, and ≥0.8, respectively. Statistical analyses were performed with SPSS, version PASW 18.0 (SPSS Inc., Chicago, Ill.). The results are presented as Mean (Standard Deviation) [M (SD)].

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

5. RESULTS

5.1. Baseline characteristics of the participants

Baseline characteristics of the participants are presented at Table 1.

Table 1. Baseline characteristics of the participants.

	MEAN (SD)
Age (years)	20.6 (2.42)
Body mass (kg)	68.56 (8.41)
Height (cm)	1.77 (0.08)
BMI (kg/m^2)	22.04 (2.13)
Body fat (%)	18.01 (9.21)
Fat mass (g)	11528.5 (6506.48)
Lean mass (g)	52258.1 (10172.44)
Fat free mass (g)	55319.5 (10565.41)
BMC (g)	3.26 (0.38)
BMD (g/cm^2)	1.33 (0.06)
VO_{2max} (ml/kg/min)	50.94 (5.28)

BMI: Body mass index; BMC: Bone mineral content; BMD: Bone mineral density; VO_{2max}: Maximal oxygen consumption

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15/10/2023 05:31:37 EEST - 167.114.118.212

5.2. Lactate

Changes in lactate concentration during recovery are depicted in Figure 2. Two-way ANOVA repeated measures revealed main effect of time [$F_{(1.000,36.000)} = 183.774$, $\eta^2=0.836$, $p<0.001$], main effect of trial [$F_{(3, 36)} =17.554$, $p>0.001$], as well as time by trial interaction [$F_{(3.000, 36.000)} = 22.966$, $p<0.001$]. More specific, lactate concentration increased from pre to post following UNR [Pre: 1.210 (0.19); Post: 6.650 (2.35); p = 0.000; ES = -3.13; CI: -4.43 to -1.82], R10 trial [Pre: 1.280 (0.32); Post: 5.560 (1.26); p = 0.000; ES = -4.46; CI=-6.10 to -2.82], and R20 trial [Pre: 1.220 (0.29); Post: 7.260 (2.73); p = 0.000; ES: -2.98; C I= -4.25 to -1.71] trial, while no changes in lactate concentration were observed in CT. Additionally, compared to CT, lactate concentration post training was higher in UNR trial (ES = -3.11; CI = -4.42 to -1.81), in R10 trial (ES = -4.61; CI = -6.29 to -2.93), and in R2O trial (ES = -2.29; CI = -4.26 to -1.71). Lactate concentration post training was similar between sprint training trials.

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15/10/2023 05:31:37 EEST - 167.114.118.212

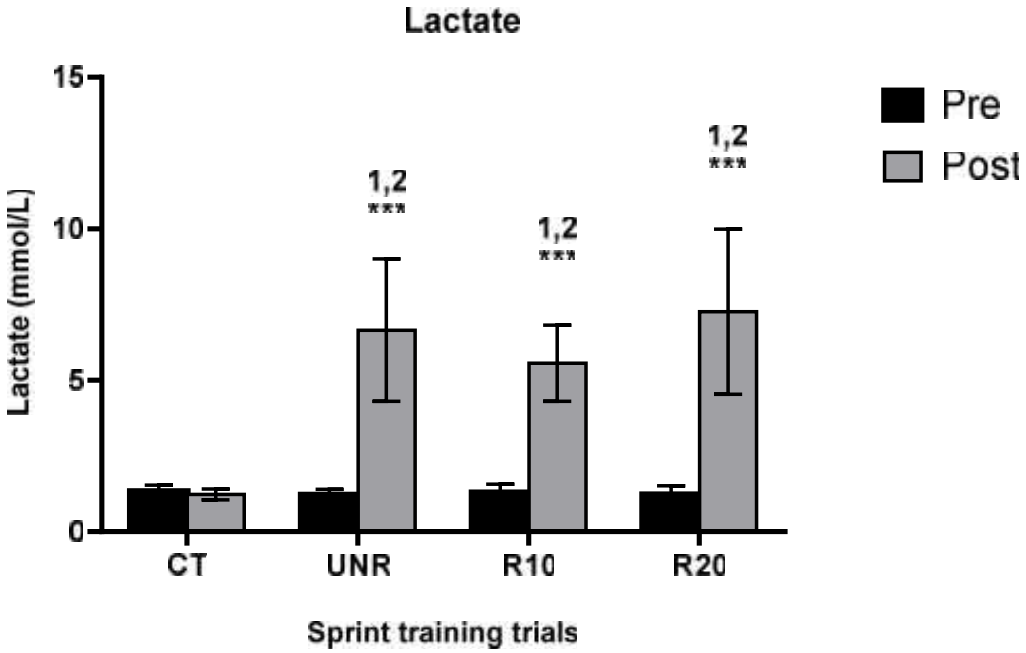

Figure 2. Changes in lactate during recovery following control trial (CT) and sprint training with unresisted sprints (UNR), and resisted sprints with 10% BM (R10) and 20% BM (R20) external load. [1]Different compared to pre in the same trial. [2]Different compared to CT. ***p<0.001.

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

5.3. Creatine kinase

Changes in CK are depicted at Figure 3. For within trials analysis, non-parametric Friedman and Wilcoxon Signed Rank tests revealed that, in UNR trial CK concentration increased by 62% at 24h [Mean (SD): 250.65 (173.90); z = -2.599; p = 0.009; ES: -0.66; CI: -1.56 to 0.24], 53% at 48h [237.37 (154.44); z = -2.293; p = 0.022; ES: -0.62; CI: -1.52 to 0.28] and 35% at 72h [208.71 (114.97); p = 0.009; z = -2.601; ES: -0.49; CI: -1.38 to 0.40] post-training compared to pre-training [155.2 (90.86)] levels. In R10, CK concentration significantly increased by 54% at 24h [251.45 (134.09); z = -2.803; p = 0.005; ES: -0.70; CI: -1.60 to 0.21], 46% at 48h [240.05 (82.52); z = -2.395; p = 0.017; ES: -0.77; CI: -1.68 to 0.14] and 33% at 72h [218.05 (102.56); z = -2.703; p = 0.007; ES: -0.50; CI: -1.39 to 0.39] post-training compared to pre-training [163.7 (105.95)] levels. In R20 trial, CK concentration significantly increased by 32% at 24h [Mean (SD): 236.45 (132.84); z = -2.402; p = 0.016; ES: -0.46; CI: -1.35 to 0.43], and 81% at 48h [322.55 (303.22); z = -2.395; p = 0.017; ES: -0.61; CI: -1.50 to 0.29)] post-training compared to pre-training [179.00 (105.69)] levels. CK was similar between trials throughout the study.

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

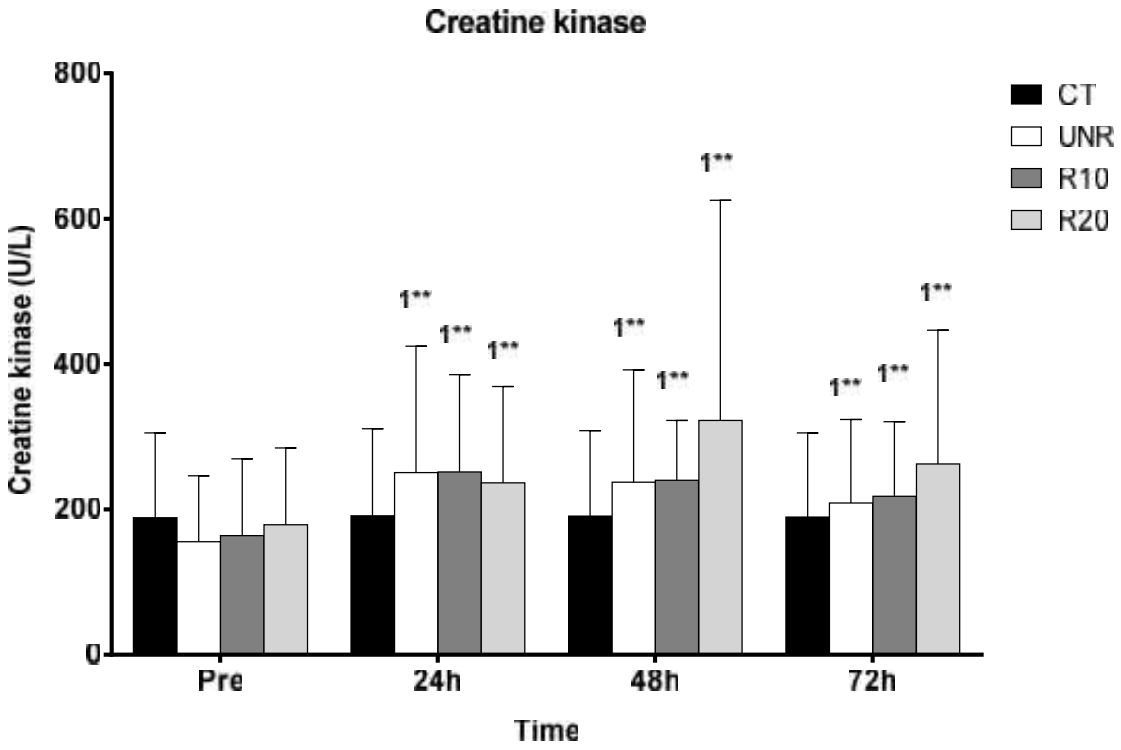

Figure 3. Changes in creatine kinase during recovery following control trial (CT) and sprint training with unresisted sprints (UNR), and resisted sprints with 10% BM (R10) and 20% BM (R20) external load. [1]Different compared to pre in the same trial. **p<0.001.

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

5.4. Delayed onset of muscle soreness

5.4.1. Muscle origin of the dominant and non-dominant limb

Changes in DOMS in muscle origin of DL and NDL are presented at Table 2. For within trials analysis, non-parametric Friedman and Wilcoxon Signed Rank tests revealed that, in UNR trial DOMS of the KE of DL increased at 48h (z = -2.000, p = 0.046; ES: -1.05; CI: -1.98 to -0.11) and 72h (z = -2.449, p = 0.014; ES: -1. 57; CI: -2.58 to 0.57) post-training compared to pre-training. In R20 trial, DOMS increased at 24h (z = = -2.070, p = 0.038; ES: -1.21; CI: -2.16 to -0.26), and 48h (z = = -2.060, p = 0.039; ES: -1.13; CI: -2.08 to -0.19) post-training compared to pre-training. Additionally, Kruskal-Wallis and Mann Whitney tests revealed that, compared to CT, DOMS at KE of the DL was higher during recovery in UNR [(24h: z = -2.163, p = 0.031; ES: -0.85; CI: -1.77 to 0.07) (72h: z = -2.854, p = 0.004; ES: -1.57; CI: -2.58 to -0.57)], R10 [(post: z = -2.169, p = 0.030; ES: -1.00; CI: -1.93 to -0.07) (72h: z = -2.169, p = 0.030; ES: -1.00; CI: -1.93 to -0.07)] and R20 [(24h: z = -2.492, p = 0.013; ES: -1.21; CI: -2.16 to -0.26) (48h: z = -1.968, p = 0.049; ES: -0.66; CI: -1.13 to -0.19)]. DOMS for KE of the DL was similar during recovery between the sprint training trials.

For the KE of NDL, in UNR DOMS increased at 24h (z = -2.271, p = 0.023; ES: -1.46; CI: -2.45 to -0.47), post-training compared to pre-training. In R10, DOMS increased immediately post (z = -2.236, p = 0.025; ES: -1.29; CI: -2.25 to -0.32), at 24h (z = -2.401, p = 0.016; ES: -1.70; CI: -2.72 to -0.68), 48h (z = -2.232, p = 0.026; ES: -1.43; CI: -2.41 to -0.44) post-training compared to pre-training. In R20, DOMS increased at 24h (z = -2.271, p = 0.023; ES: -1.46; CI: -2.45 to -0.47) post-training compared to pre-training.

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

Additionally, Kruskal-Wallis and Mann Whitney tests revealed that, compared to CT, DOMS at the KE of the NDL was higher during recovery in UNR (24h: z = -2.814, p = 0.002; ES: -1.45; CI: -2.44 to -0.47), R10 [(post: z = -2.517, p = 0.012; ES: -1.29; CI: -2.25 to -0.32) (24h: z = -3.117, p = 0.002; ES: -1.70; CI: -2.72 to -0.68) (48h: z = -2.062, p = 0.039; ES: -1.43; CI: -2.41 to -0.44) (72h: z = -2.169, p = 0.030; ES: -1.02; CI: -1.95 to -0.09)] and R20 (24h: z = -2.814, p = 0.005; ES: -1.46; CI: -2.45 to -0.47). DOMS for the KE of the NDL was similar during recovery between the sprint training trials.

For the KF of the DL, in UNR trial DOMS increased at 24h (z = -2.121, p = 0.034; ES: -1.00; CI: -1.93 to -0.07) and 48h (z = -2.121, p = 0.034; ES: -1.22; CI: -2.17 to -0.26) post-training compared to pre-training. In R20 trial, DOMS increased at 48h (z = -2.000, p = 0.046; ES: -1.05; CI: -1.98 to -0.11), and 72h (z = -2.236, p = 0.025; ES: -1.29; CI: -2.25 to -0.32) post-training. Additionally, compared to CT, DOMS at the KF of the DL was higher during recovery in UNR [(24h: z = -2.500, p = 0.012; ES: -1.00; CI: -1.93 to -0.07) (48h: z = -2.500, p = 0.012; ES: -1.22; CI: -2.17 to -0.26)], R10 [(24h: z = -2.169, p = 0.030; ES: -1.00; CI: -1.93 to -0.07) (48h: z = -2.166, p = 0.030; ES: -1.15; CI: -2.10 to -0.21) (72h: z = -2.500, p = 0.012; ES: -1.29; CI: -2.25 to -0.32)], and R20 (48h: z = -2.179, p = 0.029; ES: -1.05; CI: -1.98 to -0.11) (72h: z = -2.517, p = 0.012; ES: -1.29; CI: -2.25 to -0.32)]. DOMS for the KF of the DL was similar during recovery between the sprint training trials.

For the KF of the NDL, in UNR trial DOMS increased at 24h (z = -2.460, p = 0.014; ES: -1.78; CI: -2.82 to -0.75), and 48h (z = -2.121, p = 0.034; ES: -1.22; CI: -2.17 to -0.26) post-training compared to pre-training. In R10 trial, DOMS increased immediately post (z = -2.236, p = 0.025; ES: -1.29; CI: -2.25 to -0.32), at 24h (z = -2.070, p = 0.038; ES: -1.22; CI:

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

-2.18 to -0.27), 48h (z = -2.000, p = 0.046; ES: -1.05; CI: -1.98 to -0.11) and 72h (z = -2.070, p = 0.038; ES: -1.21; CI: -2.16 to -0.26) post-training compared to pre-training. In R20 trial, DOMS increased immediately post (z = -2.236, p = 0.025; ES: -1.29; CI: -2.25 to -0.32), at 48h (z = -2.000, p = 0.046; ES: -1.05; CI: -1.98 to -0.11) and 72h (z = -2.000, p = 0.046; ES: -1.05; CI: -1.98 to -0.11) post-training compared to pre-training. Additionally, compared to CT, DOMS at the KF of the NDL was higher during recovery in UNR [(24h: z = -3.139, p = 0.002; ES: -1.78; CI: -2.82 to -0.75) (48h: z = -2.500, p = 0.012; ES: -1.22; CI: -2.17 to -0.26)], R10 [(immediately post: z = -2.517, p = 0.012; ES: -1.29; CI: -2.25 to -0.32) (24h: z = -2.492, p = 0.013; ES: -1.22; CI: -2.18 to -0.27) (48h: z = -2.179, p = 0.029; ES: -1.05 CI: -1.98 to -0.11) (72h: z = -2.492, p = 0.013; ES: -1.05; CI: -1.98 to -0.11), and R20 [(immediately post: z = -2.517, p = 0.012; ES: -1.29; CI: -2.25 to -0.32) (48h: z = -2.179, p = 0.029; ES: -1.05; CI: -1.98 to -0.11) (72h: z = -2.179, p = 0.029; ES: -1.05; CI: -1.98 to -0.11). DOMS for the KF of the NDL was similar during recovery between the sprint training trials.

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

Table 2. Changes in DOMS at muscle origin during recovery.

	Baseline	Post	24h	48h	72h
DOMS of the knee extensors of the dominant limb					
Control trial	1 (0.0)	1 (0.0)	1 (0.0)	1 (0.0)	1 (0.0)
Unresisted trial	1 (0.0)	1.6 (1.27)[1*]	2.9 (2.33)[1*, 2*]	2.7 (1.89)[1*, 2*]	2.1 (1.85)
10% BM resisted trial	1 (0.0)	2.8 (1.55)[1*,2*]	3.8 (2.82)[1*, 2**]	3.6 (2.55)[1*, 2*]	2.6 (2.17)[2*]
20% BM resisted trial	1 (0.0)	1.9 (1.45)	2.8 (2.57)[1*, 2*]	2.2 (1.55)[1*, 2*]	1.6 (1.27)
DOMS of the knee extensors of the non-dominant limb					
Control trial	1 (0.0)	1.0 (0.0)	1.0 (0.0)	1.0 (0.0)	1.0 (0.67)
Unresisted trial	1 (0.0)	1.9 (1.45)	3.5 (2.12)[1*, 2**]	2.2 (1.55)[1*, 2*]	1.3 (0.95)[1*]
10% BM resisted trial	1 (0.0)	2.2 (1.55)[2*]	4.2 (2.35)[1*, 2***]	3.7 (2.06)[1*, 2**]	2.9 (2.13)[1*, 2*]
20% BM resisted trial	1 (0.0)	2.2 (1.55)[1*, 2*]	2.6 (2.17)[2*]	3.3 (1.70)[1*, 2**]	1.9 (1.45)[1*]
DOMS of the knee flexors of the dominant limb					
Control trial	1 (0.0)	1 (0.0)	1 (0.0)	1 (0.0)	1 (0.0)
Unresisted trial	1 (0.0)	1.6 (1.27)	4.0 (3.20)[1, 2*]	2.9 (2.13)[1, 2*]	2.1 (1.85)
10% BM resisted trial	1 (0.0)	2.7 (1.90)[1, 2*]	3.3 (2.67)[1, 2*]	3.6 (2.72)[1, 2*]	2.2 (1.55)
20% BM resisted trial	1 (0.0)	2.5 (1.58)[1, 2*]	2.8 (2.57)[2*]	2.4 (1.90)[1, 2*]	2.2 (1.55)[1*]
DOMS of the knee flexors of the non-dominant limb					
Control trial	1 (0.0)	1 (0.0)	1 (0.0)	1 (0.0)	1.0 (0.0)
Unresisted trial	1.0 (0.0)	1.6 (1.27)	3.3 (2.83)[1*]	2.9 (2.13)[1*]	2.1 (1.85)[1*]
10% BM resisted trial	1.0 (0.0)	2.2 (1.55)	3.6 (2.55)[1*]	3.6 (2.37)[1*]	2.4 (1.90)[1*]
20% BM resisted trial	1.0 (0.0)	2.5 (1.58)[1*]	2.9 (2.13)	2.7 (1.90)	2.5 (1.58)[1*]

DOMS: Delayed onset of muscle soreness; KE: Knee extensors; KF: Knee flexors; DL: Dominant limb; NDL: Non-dominant limb. [1]Different from baseline; [2]Different from Control trial; *p<0.05; **p<0.01; ***p<0.001.

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

5.4.2. Muscle belly of the dominant and non-dominant limb

Changes in DOMS in muscle belly of DL and NDL are presented at Table 3. For within trials analysis, non-parametric Friedman and Wilcoxon Signed Rank tests revealed that, in UNR trial DOMS of the KE of DL increased at 24h (z = -2.121, p = 0.034; ES: -1.10; CI: -2.05 to -0.16), and 48h (z = -2.121, p = 0.034; ES: -1.22; CI: -2.17 to -0.26) post-training compared to pre-training. In R10 trial, DOMS increased immediately post (z = -2.499, p = 0.014; ES: -1.57; CI: -2.58 to -0.57), at 24h (z = -2.032, p = 0.026; ES: -1.34; CI: -2.32 to -0.37), and 48h (z = -2.032, p = 0.026; ES: -1.38; CI: -2.36 to -0.41) compared to pre-training. In R20 trial, DOMS increased at 48h (z = -2.000, p = 0.046; ES: -1.05; CI: -1.98 to -0.11) post-training compared to pre-training. Additionally, Kruskal-Wallis and Mann Whitney tests revealed that, compared to CT, DOMS at RF of the DL was higher during recovery in UNR [(24h: z = -2.500, p = 0.012; ES: -1.10; CI: -2.05 to -0.16) (48h: z = -2.500, p = 0.012; ES: -1.22; CI: -2.17 to -0.26)], R10 [(post: z = -2.854, p = 0.004; ES: -1.57; CI: -2.58 to -0.57) (24h: z = -2.804, p = 0.005; ES: -1.34; CI: -2.32 to -0.37) (48h: z = -2.804, p = 0.005; ES: -1.38; CI: -2.36 to -0.41) (72h: z = -2.166, p = 0.030; ES: -1.00; CI: -1.93 to -0.07)] and R20 [(24h: z = -2.164, p = 0.030; ES: -0.95; CI: -1.87 to -0.02) (48h: z = -2.179, p = 0.029; ES: -1.05; CI: -1.98 to -0.11)]. DOMS for RF of the DL was similar during recovery between the sprint training trials.

For KE of NDL, in UNR DOMS increased at 24h (z = -2.530, p = 0.011; ES: -1.60; CI: -2.60 to -0.59), and 48h (z = -2.000, p = 0.046; ES: -1.05; CI: -1.98 to -0.11) post-training compared to pre-training. In R10, DOMS increased immediately post (z = -2.000, p = 0.046; ES: -1.05; CI: -1.98 to -0.11), at 24h (z = -2.640, p = 0.008; ES: -1.84; CI: -2.89 to -0.80), 48h

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

(z = -2.428, p = 0.015; ES: -1.78; CI: -2.81 to -0.74), and 72h (z = -2.070, p = 0.038; ES: -1.21; CI: -2.16 to -0.26) post-training compared to pre-training. In R20, DOMS increased immediately post (z = -2.000, p = 0.046; ES: -1.05; CI: -1.98 to -0.11), and 48h (z = -2.530, p = 0.011; ES: -1.83; CI: -2.88 to -0.79) post-training compared to pre-training. Additionally, compared to CT, DOMS at KE of the NDL was higher during recovery in UNR [(24h: z = -3.162, p = 0.002; ES: -1.60; CI: -2.60 to -0.59) (48h: z = -2.179, p = 0.029; ES: -1.05; CI: -1.98 to -0.11)], R10 [(post: z = -2.179, p = 0.029; ES: -1.05; CI: -1.98 to -0.11) (24h: z = -3.473, p = 0.001; ES: -1.84; CI: -2.89 to -0.80) (48h: z = -3.127, p = 0.002; ES: -1.78; CI: -2.81 to -0.74) (72h: z = -2.492, p = 0.013; ES: -1.21; CI: -2.16 to -0.26)] and R20 [(immediately post: z = -2.179, p = 0.029; ES: -1.05; CI: -1.98 to -0.11) (24h: z = -2.166, p = 0.030; ES: -1.00; CI: -1.93 to -0.07) (48h: z = -3.162, p = 0.002; ES: -1.83; CI: -2.88 to -0.79)]. DOMS for RF of the NDL was similar during recovery between the sprint training trials.

For KF of the DL, in UNR trial DOMS increased at 24h (z = -2.226, p = 0.026; ES: -1.27; CI: -2.23 to -0.31) and 48h (z = -2.070, p = 0.038; ES: -1.21; CI: -2.16 to -0.26) post-training compared to pre-training. In R10 trial, DOMS increased immediately post (z = -2000, p = 0.046; ES: -1.22; CI: -2.17 to -0.26), 24h (z = -2070, p = 0.038; ES: -1.17; CI: -2.11 to -0.22) and 48h (z = -2.070, p = 0.038; ES: -1.29; CI: -2.26 to -0.33) post-training. In R20 trial, DOMS increased and immediately post (z = -2.236, p = 0.025; ES: -1.29; CI: -2.25 to -0.32), and 72h (z = -2000, p = 0.046; ES: -1.05; CI: -1.98 to -0.11) post-training. Additionally, compared to CT, DOMS at KF of the DL was higher up to 48h during recovery in UNR [(24h: z = -2.802, p = 0.005; ES: -1.327 CI: -2.23 to -0.31) (48h: z = -2.492, p = 0.013; ES: -1.21; CI: -2.16 to -0.26)], R10 [(post: z = -2.500, p = 0.012; ES: -1.22; CI: -2.17 to -0.26) (24h: z = -

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

2.487, p = 0.013; ES: -1.17; CI: -2.11 to -0.22) (48h: z = -2.490, p = 0.013; ES: -1.29; CI: -2.26 to -0.33)], and R20 (post: z = -2.517, p = 0.012; ES: -1.29; CI: -2.25 to -0.32) (24h: z = -1.164, p = 0.030; ES: -0.95; CI: -1.87 to -0.02) (48h: z = -1.169, p = 0.030; ES: -1.00; CI: -1.93 to -0.07)]. DOMS for BF of the DL was similar during recovery between the sprint training trials.

For KF of the NDL, in UNR trial DOMS increased at 24h (z = -2.070, p = 0.038; ES: -1.10; CI: -2.04 to -0.16), and 48h (z = -2.070, p = 0.038; ES: -1.21; CI: -2.16 to -0.26), compared to pre-training. In R10 trial, DOMS increased immediately post (z = -2.000, p = 0.046; ES: -1.05; CI: -1.98 to -0.11), at 24h (z = -2.232, p = 0.026; ES: -1.38; CI: -2.36 to -0.41), and 48h (z = -2.271, p = 0.023; ES: -1.49; CI: -2.48 to -0.50) post-training compared to pre-training. In R20 trial, DOMS increased immediately post (z = -2.236, p = 0.025; ES: -1.29; CI: -2.25 to -0.32), at 24h (z = -2.070, p = 0.038; ES: -1.21; CI: -2.16 to -0.26), 48h (z = -2.121, p = 0.034; ES: -1.21; CI: -2.17 to -0.26) and 72h (z = -2.236, p = 0.025; ES: -1.29; CI: -2.25 to -0.32) post-training compared to pre-training. Additionally, compared to CT, DOMS at KF of the NDL was higher during recovery in UNR [(24h: z = -2.492, p = 0.013; ES: -1.10; CI: -2.04 to -0.16) (48h: z = -2.492, p = 0.013; ES: -1.21; CI: -2.16 to -0.26)], R10 [(immediately post: z = -2.179, p = 0.029; ES: -1.05; CI: -1.98 to -0.11) (24h: z = -2.804, p = 0.005; ES: -1.38; CI: -2.36 to -0.41) (48h: z = -2.814, p = 0.005; ES: -1.49; CI: -2.48 to -0.50) (72h: z = -2.169, p = 0.030)], and R20 [(immediately post: z = -2.517, p = 0.012; ES: -1.29; CI: -2.25 to -0.32) (24h: z = -2.492; p = 0.013; ES: -1.21; CI: -2.16 to -0.26) (48h: z = -2.500; p = 0.012; ES: -1.22; CI: -2.17 to -0.26) (72h: z = -2.517, p = 0.012)]. DOMS for BF of the NDL was similar during recovery between the sprint training trials.

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

Table 3. Changes in DOMS at muscle belly during recovery.

	Baseline	post	24h	48h	72h
DOMS of the knee extensors of the dominant limb					
Control trial	1 (0.0)	1 (0.0)	1 (0.0)	1 (0.0)	1 (0.0)
Unresisted trial	1 (0.0)	1.6 (1.27)[1*]	2.9 (2.33)[1*, 2*]	2.7 (1.89)[1*, 2*]	2.1 (1.85)
10% BM resisted trial	1 (0.0)	2.8 (1.55)[1*,2*]	3.8 (2.82)[1*, 2**]	3.6 (2.55)[1*, 2*]	2.6 (2.17)[2*]
20% BM resisted trial	1 (0.0)	1.9 (1.45)	2.8 (2.57)[1*, 2*]	2.2 (1.55)[1*, 2*]	1.6 (1.27)
DOMS of the knee extensors of the non-dominant limb					
Control trial	1 (0.0)	1.0 (0.0)	1.0 (0.0)	1.0 (0.0)	1.0 (0.67)
Unresisted trial	1 (0.0)	1.9 (1.45)	3.5 (2.12)[1*, 2**]	2.2 (1.55)[1*, 2*]	1.3 (0.95)[1*]
10% BM resisted trial	1 (0.0)	2.2 (1.55)[2*]	4.2 (2.35)[1*, 2***]	3.7 (2.06)[1*, 2**]	2.9 (2.13)[1*, 2*]
20% BM resisted trial	1 (0.0)	2.2 (1.55)[1*, 2*]	2.6 (2.17)[2*]	3.3 (1.70)[1*, 2**]	1.9 (1.45)[1*]
DOMS of the knee flexors of the dominant limb					
Control trial	1 (0.0)	1 (0.0)	1 (0.0)	1 (0.0)	1 (0.0)
Unresisted trial	1 (0.0)	1.6 (1.27)	4.0 (3.20)[1, 2*]	2.9 (2.13)[1, 2*]	2.1 (1.85)
10% BM resisted trial	1 (0.0)	2.7 (1.90)[1, 2*]	3.3 (2.67)[1, 2*]	3.6 (2.72)[1, 2*]	2.2 (1.55)
20% BM resisted trial	1 (0.0)	2.5 (1.58)[1, 2*]	2.8 (2.57)[2*]	2.4 (1.90)[1, 2*]	2.2 (1.55)[1*]
DOMS of the knee flexors of the non-dominant limb					
Control trial	1 (0.0)	1 (0.0)	1 (0.0)	1 (0.0)	1.0 (0.0)
Unresisted trial	1.0 (0.0)	1.6 (1.27)	3.3 (2.83)[1*]	2.9 (2.13)[1*]	2.1 (1.85)[1*]
10% BM resisted trial	1.0 (0.0)	2.2 (1.55)	3.6 (2.55)[1*]	3.6 (2.37)[1*]	2.4 (1.90)[1*]
20% BM resisted trial	1.0 (0.0)	2.5 (1.58)[1*]	2.9 (2.13)	2.7 (1.90)	2.5 (1.58)[1*]

DOMS: Delayed onset of muscle soreness; KE: Knee extensors; KF: Knee flexors; DL: Dominant limb; NDL: Non-dominant limb. [1]Different from baseline; [2]Different from Control trial; *p<0.05; **p<0.01; ***p<0.001.

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15/10/2023 05:31:37 EEST - 167.114.118.212

5.4.3. Muscle insertion of dominant limb and non-dominant limb

Changes in DOMS in muscle insertion of DL and NDL are presented at Table 4. For within trials analysis, non-parametric Friedman and Wilcoxon Signed Rank tests revealed that, in R10 trial, DOMS increased immediately post (z = -2.236; p = 0.025; ES: -1.29; CI: -2.25 to -0.32), compared to pre-training. No changes were observed during recovery in control, UNR, and R20 trials. Additionally, Kruskal-Wallis and Mann Whitney tests revealed that, compared to CT, DOMS at RF of the DL was higher immediately post only in R10 trial (post: z = -2.517, p = 0.012; ES: -1.29; CI: -2.25 to -0.32). DOMS for RF of the DL was similar during recovery between control, UNR, and R20 trials.

For KE of NDL, in R10, DOMS increased immediately post post-training (z = -2.236, p = 0.025; ES: -1.29; CI: -2.25 to -0.32) compared to pre-training. In R20, DOMS increased immediately post post-training (z = -2.000, p = 0.046; ES: -1.05; CI: -1.98 to -0.11) compared to pre-training. Additionally, compared to CT, DOMS at KE of the NDL was higher during recovery in R10 (immediately post: z = -2.517, p = 0.012; ES: -1.29; CI: -2.25 to -0.32) and R20 (immediately post: z = -2.179, p = 0.029; ES: -1.05; CI: -1.98 to -0.11). DOMS for RF of the NDL was similar during recovery between the sprint training trials.

For KF of the DL, DOMS increased at 24h (z = -2.070, p = 0.038; ES: -1.21; CI: -2.16 to -0.26) post-training compared to pre-training only in UNR trial. Additionally, compared to CT, DOMS at KF of the DL was higher up to 48h during recovery in UNR (24h: z = -2.492, p = 0.013; ES: -1.21; CI: -2.16 to -0.26), and R10 (post: z = -2.169, p = 0.030; ES: -1.00; CI: -

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

1.93 to -0.07). DOMS for BF of the DL was higher in R10 trial compared to UNR immediately post-training (z = -2.169, p = 0.030; ES: -1.00; CI: -1.93 to -0.07).

For KF of the NDL, in R10 trial, DOMS increased at 48h (z = -2.333, p = 0.020; ES: -1.48; CI: -2.47 to -0.49) post-training compared to pre-training. In R20 trial, DOMS increased immediately post training (z = -2.000, p = 0.046; ES: -1.05; CI: -1.98 to -0.11) compared to pre-training. Additionally, compared to CT, DOMS at KF of the NDL was higher during recovery in UNR (24h: z = -2.166, p = 0.030; ES: -1.00; CI: -1.93 to -0.07), R10 [(24h: z = -2.169, p = 0.030; ES: -1.02; CI: -1.95 to -0.09) (48h: z = -2.828, p = 0.005; ES: -1.48; CI: -2.47 to -0.49)], and R20 [(immediately post: z = -2.179, p = 0.029; ES: -1.05; CI: -1.98 to -0.11) (24h: z = -2.169, p = 0.030; ES: -1.00; CI: -1.93 to -0.07)]. DOMS for BF of the NDL was similar during recovery between the sprint training trials.

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

Table 4. Changes in DOMS at muscle insertion during recovery.

	Baseline	post	24h	48h	72h
DOMS of the knee extensors of the dominant limb					
Control trial	1.0 (0.0)	1.0 (0.0)	1.0 (0.0)	1.0 (0.0)	1.0 (0.0)
Unresisted trial	1.0 (0.0)	1.9 (1.45	1.8 (1.75)	1.8 (1.75)	1.9 (1.45)
10% BM resisted trial	1.0 (0.0)	2.5 (1.58)^{1*,2**}	1.8 (1.75)	2.1 (1.85)	1.3 (0.95)
20% BM resisted trial	1.0 (0.0)	1.3 (0.95)	2.3 (2.16)	1.8 (1.75)	1.6 (1.27)
DOMS of the knee extensors of the non-dominant limb					
Control trial	1.0 (0.0)	1.0 (0.0)	1.0 (0.0)	1.0 (0.0)	1.0 (0.0)
Unresisted trial	1.0 (0.0)	1.6 (1.27)	2.0 (2.31)	1.8 (1.75)	1.0 (0.00)
10% BM resisted trial	1.0 (0.0)	2.5 (1.58)^{1**,2**}	1.9 (1.45)	2.0 (2.11)	1.6 (1.27)
20% BM resisted trial	1.0 (0.0)	2.2 (1.55)^{1*,2**}	2.1 (1.85)	1.8 (1.75)	1.9 (1.45)
DOMS of the knee flexors of the dominant limb					
Control trial	1.0 (0.0)	1.0 (0.0)	1.0 (0.0)	1.0 (0.0)	1.0 (0.0)
Unresisted trial	1.0 (0.0)	1.0 (0.0)^{2**}	2.9 (2.13)^{1**,2**}	2.1 (2.28)^{2**}	1.5 (1.58)
10% BM resisted trial	1.0 (0.0)	2.4 (1.90)^{2**}	2.5 (2.59)^{2***}	1.9 (1.73)^{2**}	1.6 (1.27)
20% BM resisted trial	1.0 (0.0)	1.3 (0.95)	1.8 (1.75)	1.9 (1.45)	1.9 (1.45)
DOMS of the knee flexors of the non-dominant limb					
Control trial	1.0 (0.0)	1.0 (0.0)	1.0 (0.0)	1.0 (0.0)	1.0 (0.0)
Unresisted trial	1.0 (0.0)	1.6 (1.27)	2.6 (2.17)^{2**}	2.3 (2.16)	1.6 (1.27)
10% BM resisted trial	1.0 (0.0)	1.9 (1.45)^{1**}	2.8 (2.39)^{2**}	3.0 (1.83)^{2**}	1.9 (1.45)
20% BM resisted trial	1.0 (0.0)	2.2 (1.55)^{1**,2*}	2.4 (1.90)^{2**}	1.6 (1.27)	1.9 (1.45)

DOMS: Delayed onset of muscle soreness; KE: Knee extensors; KF: Knee flexors; DL: Dominant limb; NDL: Non-dominant limb. [1]Different from baseline; [2]Different from Control trial; *p<0.05; **p<0.01; ***p<0.001.

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

5.5. Performance

5.5.1. Sprinting performance

Changes in sprinting performance following sprint training are presented in Figures 4 and 5. Two-way ANOVA repeated measures revealed time by trial interaction for 10m-sprint time [F(7.113,85.361) = 2.061, p = 0.056], average speed at 10m-sprint [(7.156,85.846) = 2.137, p = 0.047], and 30m sprint-time [F(7.113,85.361) = 1.957, p = 0.051]. Main effect of time existed for 30m sprint-time [F(3,108) = 4.756, p = 0.004], and average speed at 30m [F= (3,108) =7.184, p = 0.000]. More specific, 10m sprint-time increased at 48h post-training [1.851 (0.16); p = 0.009; ES = -0.62; CI = -1.52 to 0.28)] compared to pre-training [1.765 (0.11)] (Figure 2A) and average speed at 10m decreased [19.581 (1.67); p = 0.016; ES = 0.59; CI = -0.31 to 1.48)] compared to pre-training [20.462 (1.20)] only in R20 trial (Figure 2B). 30m-sprint time increased at 24h [(4.349 (0.36); p = 0.012; ES = -0.39; CI = -1.28 to 0.49), 48h [(4.402 (0.44); p = 0.001; ES = -0.55; CI = -1.44 to 0.35), and 72h [(4.333 (0.41); p = 0.054; ES = -0.33; CI = -1.21 to 0.56) compared to pre-training [4.205 (0.342)] only in R20 trial (Figure 5A), while average speed at 30m decreased in all trials at 24h [(CT: 25.817 (1.85); p = 0.033; ES = 0.03; CI = -0.85 to 1.18) (UNR: 25.795 (1.74); p = 0.033; ES = 0.07; CI = -0.81 to 0.94) (R10: 25.817 (1.44); p = 0.033; ES = 0.22; CI = -0.66 to 1.09) (R20: 25.223 (1.92); p = 0.033; ES = 0.30; CI = -0.58 to 1.18)] and 48h [CT: 25.896 (1.83); p = 0.011; ES = -0.01; CI = -0.89 to 0.87, UNR: 25.645 (1.79); ES = 0.14; p = 0.011; CI = -0.74 to 1.02, R10: 25.146 (1.89); p = 0.011; ES = 0.21; CI = -0.67 to 1.09, R20: 24.912 (2.36); p = 0.011; ES = 0.40; CI = -0.48 to 1.29], compared to pre-

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

training [CT: 25.876 (1.93), Unresisted: 25.922 (2.18), R10: 25.581 (2.02), R20: 25.829 (1.969)] (Figure 5B). Sprint indices were similar between trials throughout the study.

5.5.2. Isokinetic strength performance

Changes in isokinetic strength during recovery are presented in Table 5. Two-way ANOVA repeated measures revealed main effect of time for eccentric strength of the KE for the NDL [$F_{(3,84)} = 4.209$, $p<0.05$]. More specific, eccentric strength of the KE for the NDL decreased in all trials at 24h (Control: p = 0.039, ES = -0.16, CI = -1.04 to 0.72; Unresisted: p = 0.039, ES = 0.31, CI = -0.57 to 1.20; R20: p = 0.039, ES = 0.67, CI = -0.23 to 1.57; R20: p = 0.039, ES = 0.14, CI = -0.74 to 1.01) compared to pre-training. Sprint training did not affect the isokinetic strength of the KE of the DL, as well as the isokinetic strength of KF of the DL and NDL. Isokinetic strength of both limbs was similar between training trials throughout the study.

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

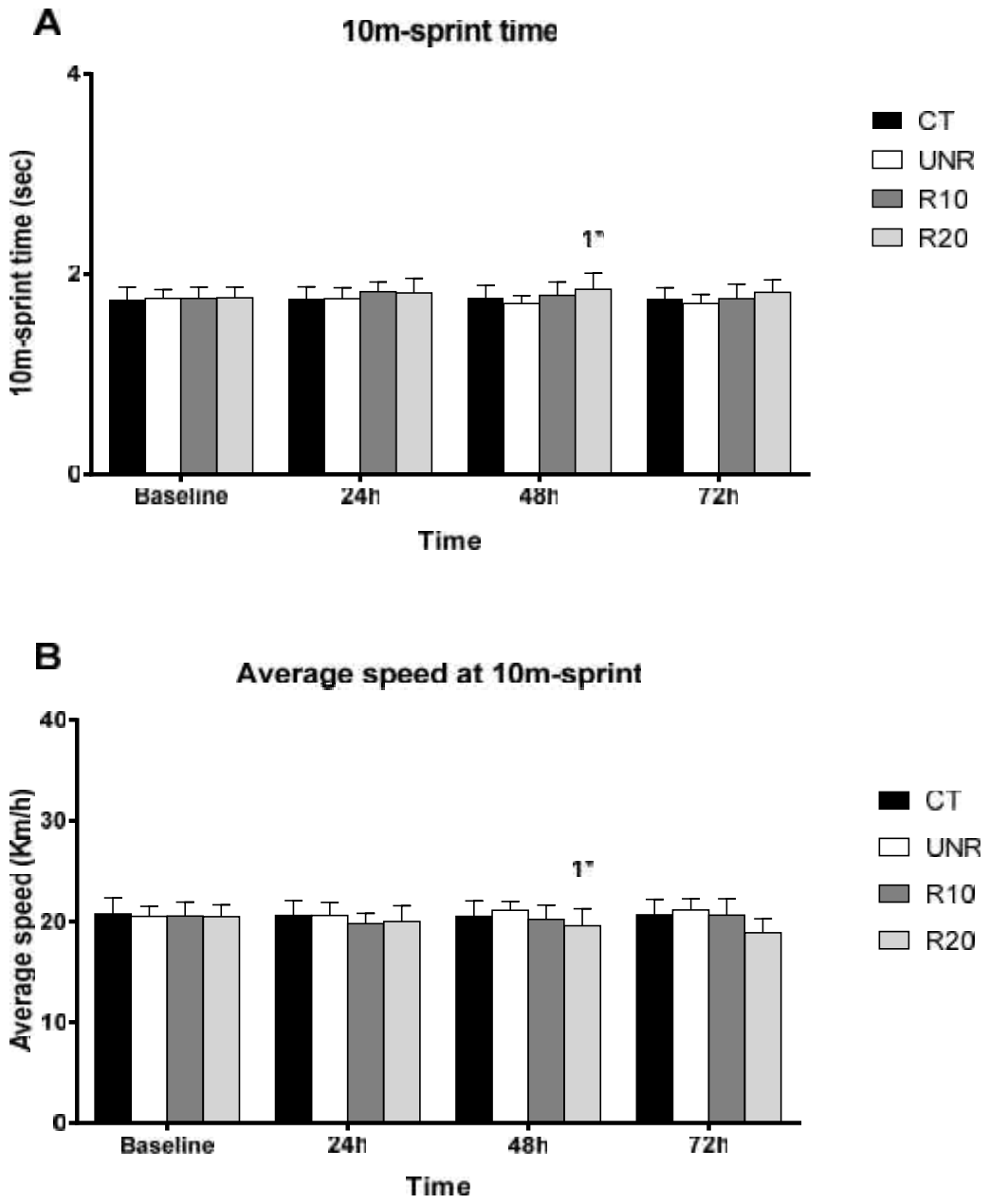

Figure 4. Changes in **10m-sprint time (A) and 10m average speed (B)** during recovery following control trial (CT) and sprint training with unresisted sprints (UNR), and resisted sprints with 10% BM (R10) and 20% BM (R20) external load. [1]Different compared to pre in the same trial. *p<0.05.

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

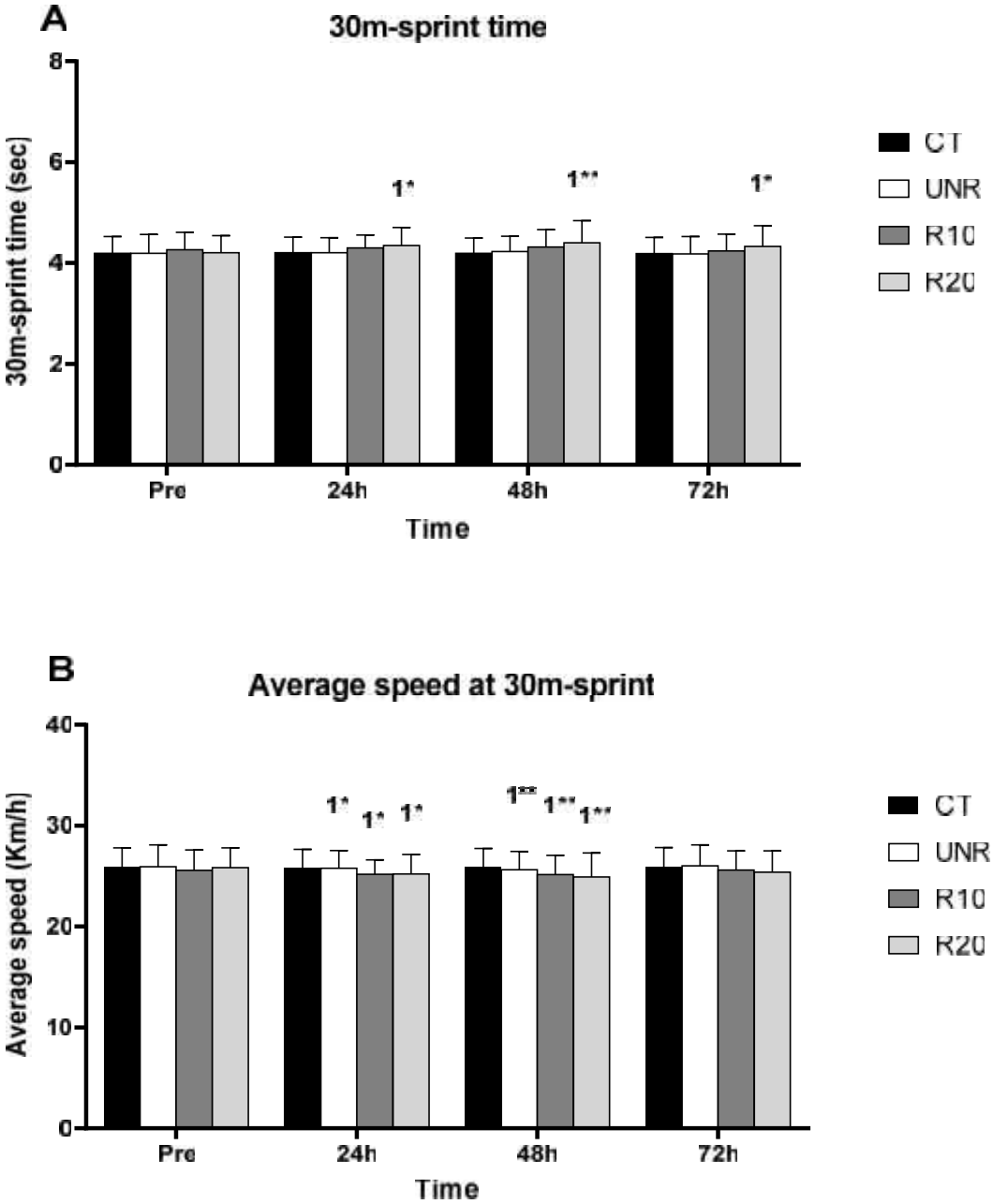

Figure 5. Changes in **30m-sprint time (A) and 30m average speed (B)** during recovery following control trial (CT) and sprint training with unresisted sprints (UNR), and resisted sprints with 10% BM (R10) and 20% BM (R20) external load. [1]Different compared to pre in the same trial. *p<0.05. **p<0.01.

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

Table 5. Changes in isokinetic strength during recovery.

	Pre	24h	48h	72h
Knee extensors, concentric peak torque of the dominant limb (N·m)				
Control	191.25 (35.864)	185.50 (38.954)	197.19 (38.941)	187.19 (30.112)
Unresisted	195.00 (37.459)	201.00 (38.023)	191.50 (26.044)	191.00 (35.006)
10% BM Resisted	199.13 (38.327	194.50 (38.589)	198.00 (52.769)	198.25 (41.091)
20% BM Resisted	186.75 (36.323)	188.50 (44.766)	190.90 (40.757)	186.50 (35.521)
Knee extensors, concentric peak torque of the non-dominant limb (N·m)				
Control	191.25 (38.651)	186.5 (36.66)	195 (44.30)	187.38 (37.63)
Unresisted	200 (40.967)	197 (34.16)	197.25 (38.47)	194.25 (36.42)
10% BM Resisted	200.13 (45.980)	197.5 (37.35)	189.63 (38.92)	202 (49.47)
20% BM Resisted	194.75 (48.163)	197.75 (51.65)	182.88 (50.47)	185.88 (32.44)
Knee flexors, concentric peak torque of the dominant limb (N·m)				
Control	116.63 (20.70)	113.13 (21.25)	118.63 (23.20)	116.88 (16.97)
Unresisted	115.25 (19.98)	115 (22.64)	118.88 (15.58)	110.25 (20.72)
10% BM Resisted	117.88 (19.32)	114.5 (21.90)	120.38 (18.18)	120.5 (25.77)

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

20% BM Resisted	116.38 (19.49)	118.63 (26.30)	109.63 (16.43)	111.63 (15.31)
Knee flexors, concentric peak torque of the non-dominant limb (N·m)				
Control	118.5 (22.48)	119.38 (21.41)	119.5 (24.06)	115.25 (21.33)
Unresisted	123.13 (28.63)	113.88 (19.62)	114.5 (23.04)	114.5 (13.52)
10% BM Resisted	122.88 (28.43)	119.63 (21.14)	114.18 (22.92)	118.63 (26.19)
20% BM Resisted	119.0 (27.77)	120.38 (29.42)	108.38 (22.07)	115.75 (20.40)
Knee extensors, eccentric peak torque of the dominant limb (N·m)				
Control	254 (40.16)	246.13 (44.56)	249.13 (48.59)	241.38 (44.87)
Unresisted	268.75 (61.97)	257.38 (55.66)	259.25 (49.92)	246.38 (63.03)
10% BM Resisted	274.38 (61.20)	255.63 (48.95)	260.25 (86.73)	254.25 (61.74)
20% BM Resisted	251.13(44.91)	259.25 (52.44)	244 (35.64)	250.63 (43.21)
Knee extensors, eccentric peak torque of the non-dominant limb (N·m)				
Control	247.38 (42.54)	244.88 (41.25)1*	244.38 (48.0)	241.88 (48.48)
Unresisted	265.88 (53.56)	249 (49.34)1*	251.25 (56.83)	246.13 (60.86)
10% BM Resisted	273.38 (72.09)	229.25 (51.84)1*	248.63 (69.46)	248.75 (55.72)
20% BM Resisted	261.5 (55.67)	253.25 (60.78)1*	236.5 (54.26)	251.38 (47.64)

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

Knee flexors, eccentric peak torque of the dominant limb (N·m)				
Control	146.75 (28.74)	140.75 (28.60)	145.88 (34.16)	134.25 (30.40)
Unresisted	143.5 (26.15)	134.50 (28.74)	137.5 (25.18)	136.5 (27.58)
10% BM Resisted	139.5 (24.87)	129.75 (28.68)	139.38 (32.13)	133.38 (29.88)
20% BM Resisted	142.16 (25.38)	139.13 (32.80)	132.75 (25.41)	132.88 (24.85)
Knee flexors, eccentric peak torque of the non-dominant limb (N·m)				
Control	147.25 (31.65)	149.88 (32.44)	146.75 (32.31)	144.38 (31.92)
Unresisted	148.38 (36.95)	140.75 (25.38)	141.5 (29.78)	141.88 (29.69)
10% BM Resisted	150 (37.18)	144 (30.94)	151.5 (28.43)	147.5 (34)
20% BM Resisted	150.5 (32.23)	154.88 (40.60)	139.38 (43.54)	148 (36.31)

[1]Different compared to pre-training. *$p<0.05$.

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

5.5.3. Countermovement jump performance

Changes in countermovement jump parameters are presented at Table 6. Two-way ANOVA repeated measures did not reveal significant time by trial interaction, or main effect of time or trial for any of the measured variables of CMJ performance in any of the sprint training trials.

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

Table 6. Changes in countermovement jump performance during recovery.

	Baseline	24h	48h	72h
Jump height (m)				
Control trial	0.342 (0.07)	0.336 (0.06)	0.341 (0.07)	0.335 (0.06)
Unresisted	0.354 (0.07)	0.344 (0.07)	0.334 (0.07)	0.336 (0.08)
10% BM resisted	0.355 (0.08)	0.351 (0.07)	0.350 (0.08)	0.346 (0.07)
20% BM resisted	0.346 (0.08)	0.351 (0.07)	0.350 (0.08)	0.349 (0.07)
Peak Ground Reaction Force (N)				
Control trial	990.965 (209.97)	981.379 (179.4)	1003.076 (215.88)	1025.034 (187.41)
Unresisted	1010.373 (242.87)	1000.694 (265.46)	1003.796 (284.73)	1006.820 (247.39)
10% BM resisted	980.438 (188.41)	976.192 (198.83)	1008.223 (217.75)	1018.804 (254.15)
20% BM resisted	1018.195 (268.56)	1037.991 (273.64)	988.633 (243.07)	986.959 (276.55)
Concentric Peak Power (W)				
Control trial	51.730 (9.51)	51.501 (9.22)	51.846 (9.73)	51.500 (8.70)
Unresisted	53.602 (10.35)	52.786 (8.37)	51.639 (9.77)	51.816 (10.76)
10% BM resisted	54.048 (7.18)	52.261 (9.06)	53.118 (8.92)	53.722 (7.91)
20% BM resisted	52.502 (8.59)	52.524 (10.41)	52.695 (9.58)	52.950 (10.06)

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

Eccentric Peak Power (W)				
Control trial	-18.609 (4.20)	-18.026 (4.05)	-19.230 (4.91)	-17.9680(4.82)
Unresisted	-18.092 (4.02)	-16.991 (4.03)	-17.417 (5.96)	-18.521 (5.20)
10% BM resisted	-17.373 (4.52)	-17.965 (3.94)	-18.043 (3.26)	-17.351 (5.20)
20% BM resisted	-18.876 (5.37)	-20.822 (4.49)	-18.592 (4.65)	-19.472 (4.70)
Concentric Mean Power (W)				
Control trial	28.492 (5.08)	27.859 (4.40)	28.237 (5.03)	27.800 (4.69)
Unresisted	29.351 (5.48)	28.593 (3.91)	27.663 (5.65)	28.113 (5.90)
10% BM resisted	29.734 (3.63)	28.709 (5.05)	28.926 (4.25)	29.289 (4.25)
20% BM resisted	29.269 (4.66)	29.095 (5.92)	28.799 (4.93)	28.160 (5.21)
Eccentric Mean Power (W)				
Control trial	-6.852 (0.92)	-6.593 (0.64)	-6.879 (0.77)	-6.608 (0.99)
Unresisted	-6.680 (0.80)	-6.247 (1.11)	-6.396 (1.45)	-6.544 (1.03)
10% BM resisted	-6.172 (1.35)	-6.848 (0.88)	-6.642 (0.98)	-6.205 (1.69)
20% BM resisted	-6.566 (1.39)	-7.042 (0.90)	-6.688 (0.98)	-6.847 (0.79)
Eccentric Average Rate of Force Development ($N \cdot sec^{-1}$)				
Control trial	5.050 (1.58)	4.468(1.29)	5.072 (1.98)	4.775 (1.74)
Unresisted	4.522(1.31)	4.493 (1.72)	4.130 (1.95)	4.617 (1.79)
10% BM resisted	4.496(1.68)	5.045 (1.10)	4.643 (1.78)	5.373 (3.23)
20% BM resisted	5.515 (2.48)	4.424 (2.07)	4.417 (1.80)	4.777 (1.76)

Institutional Repository - Library & Information Centre - University of Thessaly
15/10/2023 05:31:37 EEST - 167.114.118.212

Eccentric Mean Power – Minimum Ground Reaction Force (W)				
Control trial	-10.583 (2.02)	-10.107 (2.28)	-10.673 (2.04)	-10.134 (2.19)
Unresisted	-9.782 (1.82)	-9.431 (2.09)	-9.595 (2.03)	-9.888 (1.97)
10% BM resisted	-9.299 (2.81)	-10.095 (1.42)	-10.667 (1.72)	-9.631 (3.16)
20% BM resisted	-10.298 (3.15)	-10.292 (1.79)	-9.580 (2.11)	-10.283 (1.62)
Eccentric Mean Power – Minimum Velocity (W)				
Control trial	-13.669 (2.96)	-13.111 (2.75)	-13.854 (3.19)	-13.154 (3.54)
Unresisted	-13.249 (2.70)	-12.313 (3.53)	-12.697 (4.13)	-13.501 (3.50)
10% BM resisted	-12.316 (3.99)	-13.325 (2.76)	-13.161 (2.65)	-12.453 (4.66)
20% BM resisted	-13.355 (4.50)	-14.702 (3.67)	-13.328 (3.74)	-14.194 (3.50)
Reactive Strength Index – Modified				
Control trial	.488 (0.12)	.475 (0.10)	.482 (0.12)	.462 (0.09)
Unresisted	.502 (0.12)	.475 (0.11)	.460 (0.14)	.469 (0.13)
10% BM resisted	.480 (0.10)	.481 (0.10)	.489 (0.10)	.479 (0.12)
20% BM resisted	.479 (0.13)	.501 (0.14)	.480 (0.14)	.494 (0.14)
Maximum Velocity ($m \cdot sec^{-1}$)				
Control trial	2.697 (0.26)	2.673 (0.24)	2.687 (0.28)	2.667 (0.25)
Unresisted	2.741 (0.27)	2.731 (0.18)	2.664 (0.28)	2.677 (0.31)
10% BM resisted	2.795 (0.21)	2.730 (0.26)	2.731 (0.26)	2.770 (0.18)
20% BM resisted	2.768(0.21)	2.731 (0.27)	2.760 (0.22)	2.744 (0.24)

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15/10/2023 05:31:37 EEST - 167.114.118.212

Minimum Velocity (m·sec^{-1})				
Control trial	-1.328 (0.19)	-1.294 (0.18)	-1.333 (0.20)	-1.287 (0.22)
Unresisted	-1.301 (0.18)	-1.251 (0.22)	-1.261 (0.29)	-1.306 (0.22)
10% BM resisted	-1.242 (0.23)	-1.308 (0.19	-1.297 (0.17)	-1.261 (0.27)
20% BM resisted	-1.298 (0.23)	-1.389 (0.19)	-1.308 (0.21)	-1.354 (0.18)
Vertical Ground Reaction Force – Velocity Zero (N)				
Control trial	889.189 (193.31)	843.126 (166.33)	886.568 (226.42)	858.525 (239.91)
Unresisted	889.950 (195.05)	861.657 (188.26)	821.971 (246.70)	889.510 (262.98)
10% BM resisted	887.970 (186.89)	885.829 (169.24)	896.210 (197.87)	882.665 (261.10)
20% BM resisted	957.954 (257.18)	958.064 (273.77	903.701 (258.60)	887.900 (243.83)
Minimum Vertical Ground Reaction Force (N)				
Control trial	-546.570 (92.95)	-522.094 (111.60)	-527.842 (108.28)	-510.564 (115.29)
Unresisted	-536.287 (67.95)	-517.814 (92.12)	-503.986 (132.94)	-528.336 (77.92)
10% BM resisted	-516.600 (104.32)	-523.930 (83.56)	-533.947 (123.79)	-539.606 (85.61)
20% BM resisted	-541.527 (109.19)	-582.736 (82.41)	-550.064 (96.79)	-578.377 (91.30)
Vertical Displacement (m)				
Control trial	-.315 (0.05)	-.308 (0.05)	-.321 (0.05)	-.317 (0.06)
Unresisted	-.314 (0.05)	-.301 (0.07)	-.315 (0.05)	-.312 (0.07)
10% BM resisted	-.284 (0.08)	-.329 (0.05)	-.311 (0.06)	-.298 (0.06)
20% BM resisted	-.293 (0.09)	-.325 (0.05)	-.318 (0.06)	-.311 (0.06)

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15/10/2023 05:31:37 EEST - 167.114.118.212

Vertical Stiffness ($N \cdot m^{-1}$)				
Control trial	46.195 (10.15)	46.719 (9.53	44.242 (11.49)	47.234 (10.73)
Unresisted	47.262 (12.96)	50.890 (18.26)	48.494 (19.46)	47.709 (14.99)
10% BM resisted	54.317 (19.92)	43.307 (10.06)	47.509 (13.72)	47.648 (14.08)
20% BM resisted	54.620 (22.01)	46.000 (11.02)	45.569 (11.13)	45.709 (12.11)

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6. DISCUSSION

The present study, examined for the first time the recovery kinetics of muscle damage and performance indices following different sprint-acceleration training protocols for up to 72h post-training, under both unresisted and resisted sprints, against a control trial. The main findings were that a) acute unresisted and resisted sprint-acceleration training increase lactate concentration post-training similarly, b) acute unresisted and resisted sprint-acceleration training induce EIMD, which may persist for 72h post-training, c) sprint performance is deteriorated during recovery only following resisted sprints with 20% BM, d) isokinetic eccentric peak torque of the KE may be compromised for 24h following unresisted and resisted sprint-acceleration training, and e) performance at CMJ is not affected following sprint-acceleration training with external loads up to 20% BM.

Lactate concentration usually rises during and after acute high-intensity exercise due to the upregulated metabolic processes taking place inside the muscle to compensate for the increased energy demands. The elevated blood lactate levels indicate that glycolysis is highly activated, and the magnitude of lactate rise depends on the intensity of exercise, with higher intensity leading to higher lactate concentration (Fiorenza et al., 2019). Thus, blood lactate may rise from 1 mmol·L-1 at rest to 20 mmol·L-1 after maximum exercise (Mougios 2019). Energy demands during a short maximal sprint (≤ 10 sec), are mainly derived from the breakdown of stored muscle phosphagens adenosine triphosphate and phosphocreatine, and anaerobic glycolysis.(Balsom, Seger, Sjödin, &

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15/10/2023 05:31:37 EEST - 167.114.118.212

Ekblom, 1992; Bogdanis, Nevill, Lakomy, & Boobis, 1998). However, repeated short sprints during the same training session increases lactate levels (Balsom et al., 1992; Bogdanis et al., 1998). Elevated blood lactate concentration is accompanied by high levels of H+ and pH decrease, which to some degree, may contribute to fatigue and muscle function impairment (Balsom et al., 1992; Bogdanis et al., 1998; Proske & Allen, 2005). In our study the acceleration sprint training protocols consisted of maximum linear sprints of 20 m and 30 m, under external loading of either 0% BW, 10% BW, or 20% BW. All sprint training trials resulted in increased lactate concentration compared to CT, and although lactate responses were not significantly different between trials, R20 provoked the highest rise (6-fold), followed by UNR (5.5-fold), and R10 (4.3-fold) trial. As far as we know, only one other study (Bachero-Mena et al., 2020) assessed the acute physiological responses after linear sprint acceleration training for up to 24h. In that study, external loads up to 80% BW were incorporated, and the authors reported similar lactate responses between the external loads of 0% and 20%, but significantly higher lactate rise under the external loads of 60% BW and 80% BW. The above findings indicate that linear sprint training induces lactate elevation in a load-dependent manner. (Bachero-Mena et al., 2020), also reported higher values of fatigue index during the sprint-training protocol as the external load increased. Unfortunately, in our study, fatigue index during the sprint-training sessions, as well as performance immediately post-training were not evaluated, and this comprises a limitation, as we could not estimate whether the extent of lactate elevation after the different sprint-training protocols was associated with different extend of fatigue, or whether the extend of fatigue during training were

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15/10/2023 05:31:37 EEST - 167.114.118.212

associated with performance reduction during recovery the following days. Yet, the magnitude of lactate elevation after resisted linear sprint acceleration training seems to be lower compared to other sprint-training modalities, such as, maximal speed training (M. Johnston et al., 2015), repeated sprint training (Howatson & Milak, 2009; Keane et al., 2015) speed endurance (Kritikos et al., 2021; Tzatzakis et al., 2019)or combined plyometric and resistance training (Doma, Burt, & Connor, 2021). The different characteristics of such training protocols such as total distance covered and resting periods, induce different metabolic demands, thus leading to greater lactate response.

CK is an indirect indicator of EIMD (Pedersen 2019; Clarkson 2019). CK is a muscle protein, which, under situations of muscle injury,(Howatson & Milak, 2009; Keane et al., 2015), increased serum CK levels can be observed (Noakes, 1987). CK activity usually peaks at 24h – 48h post EIMD, and depending on the extend of the injury, it recovers after several days post-exercise (Barros, Alves, Zogaib, & Conforti, 2017; Machado et al., 2012; Siegel, Silverman, & Lopez, 1980),(P. M. Clarkson, Kearns, Rouzier, Rubin, & Thompson, 2006; Deli et al., 2017; Jamurtas et al., 2005; Papanikolaou et al., 2021). In our study, CK activity increased after all sprint-training trials, while remained unchanged under CT. The greatest rise (1.8-fold) was observed at R20 trial at 48h, followed by UNR (1.6-fold) and R10 (1.5-fold) trials at 24h. These findings agree with those of Bachero-Mena et al. who also reported similar CK responses 24h post training between 0% BM and 20% BM loads, but also between 0% BM and the heavier loads up to 80% BM. Considering the CK rise in both our study and that of Bachero-Mena et al., it seems that CK changes are of rather moderate magnitude, compared to those reported following other speed-related training

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15/10/2023 05:31:37 EEST - 167.114.118.212

modalities such as maximal speed (M. Johnston et al., 2015), repeated-sprints (Howatson & Milak, 2009; Keane et al., 2015), speed endurance (Tzatzakis et al., 2019), plyometric (Chatzinikolaou et al., 2010), or combined plyometric and resistance (Doma et al., 2021) training protocols. As with metabolic response, the different characteristics of such training protocols may lead to different extend of EIMD. More specific, in such studies, greater total distance is usually covered during training, resting periods between repetitions are shorter, and decelerations and/or changes of direction with a greater eccentric component are implemented, thus predisposing to greater exercise-induced muscle distraction.

It is worth noting that, CK activity presents high interindividual variability, such that some individuals are found to have high levels of serum CK compared to other individuals of similar characteristics both at rest and when exposed to the same exercise protocol even when other non-modifiable factors such as gender, age, and training status are accounted for (Baird et al., 2012). Especially regarding CK response after exercise, some athletes tend to be “low responders”, while others tend to be “high responders” (Brancaccio et al., 2007). Thus, an issue has been raised on the validity of CK activity to reflecting EIMD (Baird et al., 2012). However, increased levels of CK may lead to a further injury and prolonged fatigue, while persistent increased CK levels may indicate subclinical disorders (Baird et al., 2012; Brancaccio et al., 2007)thus, it is imperative to monitor the changes of the enzyme due to intense exercise. Consistent with the above phenomenon, interindividual variability was also evident in our study. Resting CK activity ranged from 84 to 426 U/L, and the variability could be explained by the fact that the participants were

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15/10/2023 05:31:37 EEST - 167.114.118.212

both male and female athletes, and probably by the different training level. During recovery from sprint training, various changes in CK activity were also observed amongst participants.

DOMS is a typical symptom of EIMD and usually increases after several hours and may persist for several days following the end of exercise (Deli et al., 2017; Jamurtas et al., 2005; Papanikolaou et al., 2021; Tzatzakis et al., 2019). In our study, DOMS increased after all acceleration sprint-training trials in both KE and KF, of both DL and NDL under R20 trial, and for KE of the NDL under R10 trial. Additionally, DOMS kinetics mimicked those of CK during recovery, such that peak changes for both indices were recorded at 24 – 48 h post-training. As far as we know, no other studies estimated DOMS after linear acceleration sprint-training, thus no direct comparison can be made. Nevertheless, DOMS magnitude and recovery kinetics in our study under both unresisted and resisted trials, were similar with that recorded after maximal-speed training (M. Johnston et al., 2015), repeated-sprints (Howatson & Milak, 2009; Keane et al., 2015), speed endurance (Kritikos et al., 2021; Tzatzakis et al., 2019) and plyometric training (Chatzinikolaou et al., 2010) protocols.

EIMD may also lead to performance deterioration, the following days after training (Deli et al., 2017; Jamurtas, 2018; Papanikolaou et al., 2021; Tzatzakis et al., 2019). In our study, sprint performance was affected by linear sprint acceleration training, however only following R20 trial. More specific, sprint performance at 10m declined at 48h, while sprint performance at 30m at 24h, 48h, and 72h. This finding suggests that external loads greater than 10% BM are needed to affect sprint performance in both early acceleration

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15/10/2023 05:31:37 EEST - 167.114.118.212

and late acceleration phase. In Bacchero-Mena study (Bachero-Mena et al., 2020),reduced sprint performance under the external loads of 0% BM and 80% BM was reported immediately post-training, which contrary to our results, recovered completely at 24h post-training in all training loads. However, in that study, the training protocol included only 20 m-sprints and no 30 m-sprints, the testing distance of 20 m instead of 30 m was used, and sprint performance recovery was measured up to 24 h and no longer, and these differences in the experimental design may account for the different results between the two studies. Deterioration of sprint performance has also been reported following other speed-related training modalities such as repeated-sprints (), speed-endurance (Tzatzakis et al., 2019), plyometric (Chatzinikolaou et al., 2010) or combined plyometric and resistance (Doma et al., 2021) training protocols.

Strength is another important element of training, especially in speed athletes, in order to maximize power and maximal speed. In case that a sprint training is followed by a strength training session the next days, it could probably cause a reduction in strength performance, due to EIMD. In the present study, eccentric peak torque of the KE of the NDL decreased at 24h post following all trials. As far as we know, no other study has examined the recovery kinetics of eccentric isokinetic strength after linear sprint-training. However, eccentric torque of the KF has been significantly correlated with horizontal force production and EMG activity during sprint acceleration, while impairment of KF function has been related with lower sprint acceleration performance (Morin et al., 2012). Thus, the reduction of eccentric torque observed in our study may partially justify the deterioration of sprint performance, at least following the R20 trial. Bachero-Mena

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15/10/2023 05:31:37 EEST - 167.114.118.212

(Bachero-Mena et al., 2020) examined the concentric isokinetic strength of KE and KF of the DL and reported a decrement of mean power of the KF at 24 h post-training under the 20% BM trial. Considering that all types of muscle actions are incorporated in sprinting, a reduced eccentric or concentric strength and/or a compromised muscle function of either the KE or KF, may lead to deteriorated sprint performance the following days. Isokinetic strength reduction has also been reported following other speed-related training modalities, however, the impairment after linear acceleration sprint training seems to be of lower magnitude compared to repeated-sprints (Howatson & Milak, 2009; Keane et al., 2015)), speed-endurance (Tzatzakis et al., 2019) and plyometric training (Chatzinikolaou et al., 2010)) protocols.

Countermovement jump-related indices didn't show any significant changes in any of the sprint trials during recovery, regardless the reduction in sprint performance following R20 trial. Similarly, Bachero Mena et al. (Bachero-Mena et al., 2020) also did not report CMJ performance decline even under higher external loads than those used in our study. The deterioration of sprint performance, indicates that sprint acceleration training induces neuromuscular fatigue (Fiorenza et al., 2019; Tzatzakis et al., 2019), that impairs maximal power during sprint lasting for 72h. Neuromuscular fatigue may be central-type associated to changes in neural drive, motor unit recruitment and/or firing frequency, or peripheral-type related to changes in muscles' contractile properties and metabolic perturbations such as depleted energy substrates and accumulation of metabolic by-products (Fiorenza et al., 2019). Previous research on other sprint-related protocols report a more pronounced central fatigue (Tzatzakis et al., 2019), but also a combination

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15/10/2023 05:31:37 EEST - 167.114.118.212

of central and peripheral fatigue (Thomas et al., 2018). On the other hand, powerful actions like jumping critically depend on rapid muscle force generation by the KE, which seems to remain relatively unchanged under fatiguing conditions (Thorlund, Aagaard, & Madsen, 2009), which may partly explain the absence of CMJ performance deterioration following sprint-acceleration training (Bachero-Mena et al., 2020), or the smaller and more short-lived deterioration of CMJ compared to sprinting performance following other sprint-related training modalities (Tzatzakis et al., 2019).

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15/10/2023 05:31:37 EEST - 167.114.118.212

7. CONCLUSIONS

Acceleration sprint training with both unresisted and resisted sprints may induce EIMD which is accompanied by a deterioration in sprinting performance and muscle function the following days. Higher loads induce greater metabolic demands, EIMD symptoms and performance decline and this needs to be considered by coaches for effectively designing the training programs of their athletes to optimize athletic performance and minimize injury risk. We suggest that short acceleration sprints and jumps may be repeated 48h after unresisted and 10%BW-resisted sprint-training, while more than 72h of recovery are needed after 20% BM-resisted sprint-training.

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APPENDIX 1

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ΠΑΝΕΠΙΣΤΗΜΙΟ ΘΕΣΣΑΛΙΑΣ
ΤΜΗΜΑ ΕΠΙΣΤΗΜΗΣ ΦΥΣΙΚΗΣ ΑΓΩΓΗΣ ΚΑΙ ΑΘΛΗΤΙΣΜΟΥ

Εσωτερική Επιτροπή Δεοντολογίας

Τρίκαλα: 9/12/2020

Αριθμ, Πρωτοκ:1715

Βεβαίωση έγκρισης της πρότασης για διεξαγωγή Έρευνας με τίτλο: Η κινητική της αποκατάστασης έπειτα από διαφορετικά πρωτόκολλα προπόνησης ταχύτητας: Επιταχύνσεις με ή χωρίς φορτίο.

Επιστημονικώς υπεύθυνη / επιβλέπουσα: Δελή Χαρίκλεια
Ιδιότητα: Επίκουρη καθηγήτρια
Ίδρυμα: Πανεπιστήμιο Θεσσαλίας
Τμήμα: Επιστήμης φυσικής αγωγής και αθλητισμού

Κύριος ερευνητής-τρια / φοιτητής-τρια: Λιάκου Χριστίνα
Πρόγραμμα Σπουδών: ΠΜΣ ''Άσκηση και Υγεία: Αξιολόγηση και Συνταγογράφηση.''
Ίδρυμα: Πανεπιστήμιο Θεσσαλίας
Τμήμα: Επιστήμης φυσικής αγωγής και αθλητισμού

Η προτεινόμενη έρευνα θα είναι: Μεταπτυχιακή Διατριβή

Τηλ. επικοινωνίας: 6946312447
Email επικοινωνίας: xr_liakou@yahoo.gr

Η Εσωτερική Επιτροπή Δεοντολογίας του Τ.Ε.Φ.Α.Α., Πανεπιστημίου Θεσσαλίας μετά την υπ. Αριθμ. 2-8/9-12-2020 συνεδρίασή της εγκρίνει τη διεξαγωγή της προτεινόμενης έρευνας.

Ο Πρόεδρος της
Εσωτερικής Επιτροπής
Δεοντολογίας – ΤΕΦΑΑ

Τσιόκανος Αθανάσιος

Καθηγητής

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Appendix 2

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Appendix 2A: Candidate's responsibilities throughout the study

During the PhD program the candidate had the following duties:

- Completion of the courses required for the accomplishment of 90 ECTS
- Submission of the required documents to receive ethics approval for the study
- Implementation of the study
- Perform the data analysis as well as the relevant power and statistical analysis
- Writing the PhD thesis and public defense
- Participation in relevant seminars and conferences

Appendix 2B: Skills acquired during the MSc program

Through the present study the candidate has also been actively involved in other research projects and clinical trials during the MSc program, and gained considerable experience in the fields of molecular exercise physiology and exercise biochemistry reflected by the following acquired skills:

- Assessment of body composition using advanced techniques such as dual energy x-ray absorptiometry - DXA
- Evaluation of physical performance
- Evaluation of hematological, biochemical, and redox status indices

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Appendix 2C: Academic activities during the MSc program

Articles in Peer-reviewed Scientific Journals

1. **Christina A. Liakou,** Ioannis G. Fatouros, Athanasios Poulios, Themistoklis Tsatalas, Evangeli Karampina, Despoina Kaloudi, Anastasia Rosvoglou, Panagiotis Tsimeas, Anna Kamperi, Niki Syrou, Athanasios Gatsas, Konstantinos Papanikolaou, Dimitrios Draganidis, Panagiotis Tsaklis, Giannis Giakas, Athanasios Z. Jamurtas, Chariklia K. Deli (2022). Recovery Kinetics Following Sprint Training: Resisted Versus Unresisted Sprints. International Journal of Sports Physiology and Performance (Under review).

Presentations in Scientific Conferences with peer review

1. **Liakou C.A.,** Kaloudi D., Karabina E., Rosvoglou A., Kamperi A., Poulios A., Sirou N., Gatsas A., Karanika P., Tsimeas P., Papanikolaou K., Draganidis D., Tsatalas T., Tsaklis P., Giakas G., Jamurtas A.Z., Fatouros I.G., Deli C.K. Recovery kinetics following different sprint training protocols: resisted versus unresisted sprints. 10th Conference of Biochemistry and Physiology of Exercise, 21-23 October 2022, Athens, Greece.
2. Kamperi A., **Liakou C.A.,** Gatsas A., Poulios A., Rosvoglou A., Syrou N., Draganidis D., Papagianni M., Papanikolaou K., Tsimeas P., Jamurtas A.Z., Fatouros I.G., Deli C.K. The effect of biological maturation on metabolism, neuromuscular fatigue, exercise–induced muscle damage, and performance after acute plyometric training. 10th Conference of Biochemistry and Physiology of Exercise, 21-23 October 2022, Athens, Greece.

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3. Kaloudi D., **Liakou C.A.,** Karabina E., Gatsas A., Kamperi A., Rosvoglou A., Poulios A., Syrou N., Tsimeas P., Papanikolaou K., Draganidis D., Tsatalas T., Giakas G., Tsaklis P., Jamurtas A.Z., Fatouros I.G., Deli C.K. Recovery kinetics following different power training protocols. 10th Conference of Biochemistry and Physiology of Exercise, 21-23 October 2022, Athens, Greece.
4. A. Rosvoglou, A. Poulios, D. Draganidis, K. Papanikolaou, C.K. Deli, N. Syrou, A. Pappas, P. Tsimeas, **C. Liakou, D.** Vlissaris, A.Z. Jamurtas, I.G. Fatouros. The effects of eccentric exercise volume on the recovery kinetics and muscle damage manifestations. 10th Conference of Biochemistry and Physiology of Exercise, 21-23 October 2022, Athens, Greece.

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