BELASTUNGS- UND ERHOLUNGSSTEUERUNG IM HIGH-INTENSITY AUSDAUERTRAINING

Kumulative Dissertation zur Erlangung des akademischen Grades Dr. Sportwiss.

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BELASTUNGS- UND ERHOLUNGSSTEUERUNG IM HIGH-INTENSITY AUSDAUERTRAINING
Von der Fakultät für Sportwissenschaft der Ruhr-Universität Bochum
genehmigte kumulative Dissertation zur Erlangung des
akademischen Grades eines Doktors der Sportwissenschaft.

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Eingereicht am 23. Februar 2016
Disputation am 20. April 2016
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Die der vorliegenden kumulativen Dissertation zugrundeliegenden Publikationen in internationalen Fachzeitschriften mit Review-Verfahren entstanden aus einem gemeinschaftlichen durch das Bundesinstitut für Sportwissenschaft geförderten Forschungsprojekt mit dem Titel: Regenerationsmanagement im Spitzensport (REGman). Die Projektleiter (in alphabetischer Reihenfolge) Prof. Dr. Alexander Ferrauti (Ruhr-Universität Bochum), Prof. Dr. Michael Kellmann (Ruhr-Universität Bochum), Prof. Dr. Tim Meyer (Universität des Saarlandes) und Prof. Dr. Mark Pfeiffer (Johannes Gutenberg-Universität Mainz) waren für die Einwerbung der Förderungsmittel verantwortlich. Zudem waren sie als Koautoren an der Konzeption der Untersuchungen, an der Analyse und Interpretation der Daten sowie an wichtigen Korrekturen bei der Erstellung der Manuskripte beteiligt. Gleiches gilt ebenso für Jaime Fernandez-Fernandez, Jennifer Kappenstein und Christian Raeder, die als Koautoren überdies einen wesentlichen Beitrag bei der Durchführung der Studien leisteten. Ich selbst war sowohl für die Konzeption und Durchführung der Untersuchungen sowie für die Erarbeitung, Analyse und Interpretation der Daten als auch für die Formulierung und Revisionen der Manuskripte verantwortlich.

Thimo Wiewelhove
DANKSAGUNG

Mein besonderer Dank gilt Prof. Dr. Alexander Ferrauti, der mich durch den Besuch seiner Vorlesung im Sommersemester 2008 für die Trainingswissenschaft begeistert und für die wissenschaftliche Arbeit an der Fakultät für Sportwissenschaft der Ruhr-Universität Bochum inspiriert hat. Ebenso danke ich ihm als Betreuer und Doktorvater für die Unterstützung in allen Belangen und seiner konstruktiven Kritik bei der Erstellung dieser Arbeit und der zugrundliegenden Publikationen sowie dem professionellen und gleichermaßen freundschaftlichen Arbeitsklima am Lehrstuhl für Trainingswissenschaft.

Bei Prof. Dr. Billy Sperlich möchte ich mich herzlich für die bereitwillige Erstellung des Zweitgutachtens bedanken. Gleichfalls gebührt großer Dank meinen aktuellen und ehemaligen Kollegen, die als Berater, Co-Autoren und Freunde mit enormer Expertise maßgeblich an der Durchführung der Studien und an der Vorbereitung und Entwicklung dieser Arbeit beteiligt waren. Insbesondere bei Christian Raeder, Dr. Jennifer Kappenstein, Alexander Döweling, Marc Philip Westphal, Dr. Jaime Fernandez-Fernandez, Rauno Álvaro de Paula Simola, Dr. Florian Hanakam, Alexander Ulbricht, Christoph Schneider, Janina Fett und Paul Schaffran möchte ich mich für die große und unschätzbare Hilfe sowie die denkbar beste Zusammenarbeit bedanken.


Von ganzem Herzen danke ich meiner Familie und allen voran meinen Eltern, die mich uneingeschränkt während meiner gesamten schulischen und universitären Ausbildung sowie auf dem Weg bis zum Abschluss dieser Arbeit förderten und unterstützten sowie stets ohne Zweifel und vertrauend an mich glaubten.

Der wohl größte Dank gebührt meiner Freundin Linda dafür, dass sie mich in der ganzen Zeit an vielen Wochenenden und Feiertagen entbehren musste und mich trotz aller Unwägbarkeiten, die ein Promotionsvorhaben mit sich bringt, verständnisvoll, geduldig und motivierend auf meinem Weg begleitete und mir ausnahmslos zur Seite stand.
1 EINLEITUNG


Während die Optimierung der Trainingsqualität seit jeher im Fokus trainingswissenschaftlicher und sportmedizinischer Bemühungen steht, bietet die Erholungsphase im Gesamtgefüge der Trainingssteuerung eine bisher nur unzureichend erforschte Chance, den gestiegenen psychophysischen Belastungen im Leistungssport gerecht zu werden. Praxisleitlinien zur Diagnostik von Ermüdung und Erholtheit sowie zu Anwendungsmöglichkeiten und Wirksamkeit verschiedener Regenerationsmaßnahmen sind bislang nur lückenhaft wissenschaftlich aufbereitet. Konsistente Handlungsempfehlungen könnten jedoch zur Optimierung und Beschleunigung der kompensatorischen Wiederherstellungsvorgänge nach intensiven Belastungen, zur Steigerung der Trainingstoleranz und Trainingsqualität bei hoher Trainingsdichte und zur beschleunigten Wiederherstellung der Wettkampfleistung beitragen (Bishop, Jones, & Woods, 2008).

2 THEORETISCHER HINTERGRUND


Angesichts der insgesamt hohen körperlichen Beanspruchung durch HIT ergibt sich sowohl ein spezifisches akutes Belastungsmuster als auch ein zum Teil enormer kurz- bis mittelfristiger Erholungsbedarf (Howatson & Milak, 2009). Potentielle durch HIT induzierte Belastungs- und Ermüdungsreaktionen werden im folgenden Kapitel ausführlich beschrieben.
THEORETISCHER HINTERGRUND

2.1 Belastungsreaktionen im High-Intensity Ausdauertraining


2.1.1 Akute Belastungsreaktionen

Zumeist wird zwischen einem HIT mit kurz bis lang andauernden hochintensiven, aber meist submaximalen (<100% der maximalen Sauerstoffaufnahme [VO2max]) der Belastungsphasen und einem sprintbasierten HIT mit supramaximalen (>100% der erVO2max) „all-out“-Belastungen unterschieden. Sprintorientierte Trainingsprotokolle werden überdies in ein Wiederholungssprinttraining (3–7 s Sprints mit 60 s oder kürzeren Intervallpausen) und ein Sprintintervalltraining (30 s „all-out“-Belastungen mit 2–4 min Erholungsphasen) differenziert (Buchheit & Laursen, 2013a). Trotz unterschiedlicher Belastungsnormative ergeben sich teils vergleichbare Bewertungen der subjektiv empfundenen Beanspruchungen sowie ähnliche physiologische Anpassungen der entsprechenden Funktionssysteme. Unterschiedlich wirken sich die verschiedenen HIT-Varianten jedoch auf die akuten metabolischen, pulmonalen, kardialen und vaskulären Belastungsreaktionen aus. Diese wurden bereits von einigen Autoren beschrieben, ohne allerdings alle potentiellen Belastungsnormative in ihrer Gesamtheit zu berücksichtigen.

Metabolische Belastungsreaktionen


Bei HIT-Varianten mit lang andauernden Belastungsphasen wird die notwendige Energie jedoch vor allem über anaerob glykolytische Prozesse zur Verfügung gestellt. Als Nebenprodukt wird


**Pulmonale Belastungsreaktionen**

Im Rahmen des HIT gilt eine möglichst lange Ausschöpfung eines hohen Anteils (< 90%) der VO₂max als wichtigstes Kriterium, um die erwünschten physiologischen Anpassungseffekte auszulösen. Die VO₂max wird sowohl durch die pulmonale als auch die kardiovaskulär-metabolische
Kapazität beeinflusst (Meyer & Kindermann, 1999). Daher geht die in der Nähe der VO₂max verbrachte Zeit (t@VO₂max) mit einer Beanspruchung aller beteiligten Mechanismen einher. Folglich reflektiert die t@VO₂max sowohl die pulmonalen als auch die metabolischen, kardialen und vasculären Belastungsreaktionen. Da zur Erreichung der VO₂max ungefähr 100 s benötigt werden (Buchheit & Laursen, 2013a), resultieren HIT-Varianten mit längeren Intervallen (2 – 4 min) im Vergleich zu Protokollen mit kürzeren Intervallen (15 – 30 s) meist in einer deutlich längeren t@VO₂max (Tschakert & Hofmann, 2013). Allerdings weisen Midgley & McNaughton (2006) darauf hin, dass ein adäquates Warm-up sowie eine aktive Intervallpausengestaltung auch bei kurzen Intervallen zu den gewünschten VO₂-Oszillationsraten führt. Da beispielsweise durch ein Wiederholungssprinttraining bei deutlich geringerer VO₂max-Ausschöpfung jedoch ebenfalls Verbesserungen aerob Ausdauerleistungskomponenten (u.a. VO₂max) erreicht werden (Burgomaster, Hughes, Heigenhauser, Bradwell, & Gibala, 2005; Burgomaster et al., 2008; Fernandez-Fernandez, Zimek, Wiewelhove, & Ferrauti, 2012; Ferrari Bravo et al., 2008; Gibala et al., 2006), scheint nicht nur die t@VO₂max, sondern auch die Belastungsintensität adaptationsrelevant zu sein (Tschakert & Hofmann, 2013). In diesem Zusammenhang führten Studien die Verbesserung der VO₂max durch ein Wiederholungssprinttraining auf Anpassungen des muskel-metabolischen Potentials zurück, während HIT-Protokolle mit längeren Intervallen die VO₂max vor allem durch eine verbesserte kardiovaskuläre Kapazität steigerte (Tschakert & Hofmann, 2013).

**Kardiale Belastungsreaktionen**

Entsprechend der VO₂-Kinetik resultieren HIT-Protokolle mit längeren Intervallen in extrem hohen Herzfrequenzen (Hf), während die maximale und durchschnittliche Hf im Verlauf von Varianten mit kürzeren Intervallen niedriger ist. Dabei muss berücksichtigt werden, dass die akuten kardialen Belastungsreaktionen nicht notwendigerweise die muskel-metabolischen Beanspruchungen widerspiegeln. Eine vergleichsweise niedrige Hf während kurzer Intervalle kann mit extrem hohen Blutlaktatkonzentrationen assoziiert sein (Tschakert & Hofmann, 2013).

Es wird vermutet, dass neben der t@ VO₂max und der Belastungsintensität ebenso das Erreichen eines möglichst hohen Schlagvolumens (SV) notwendig ist, um die kardiale Kapazität zu steigern (Cooper, 1997; Daussin et al., 2007; Helgerud et al., 2007; Lepretre, Koralsztein, & Billat, 2004). Ein hierfür optimales HIT-Protokoll, indem sich ein maximales SV ergibt, konnte jedoch bislang nicht identifiziert werden. Dies liegt unter anderem daran, dass das SV nicht nur durch die Belastungsintensität und Belastungsdauer sondern ebenso durch den Trainingsstatus des Athleten, die Körperposition des Sportlers während des Trainings und die individuellen hämodynamischen

**Vaskuläre Belastungsreaktionen**

Auf vaskulärer Ebene resultieren die Belastungsspitzen während eines HIT in mechanischen Stimuli (Scherkräfte in den Blutgefäßen) und/oder Veränderungen des Gefäßtonus, die angiogene Prozesse bewirken (Madsen, Thorup, Overgaard, Bjerre, & Jeppesen, 2015; Rakobowchuk et al., 2008; Rakobowchuk, Stuckey, Millar, Gurr, & Macdonald, 2009). Diese führen zu einer Zunahme der Dehnbarkeit sowie Neubildung von Blutgefäßen und optimieren in der Folge die \( \text{VO}_2 \text{max} \) (Iaia et al., 2009). Unterschiede zwischen verschiedenen HIT-Protokollen in Bezug auf die Beanspruchung des vaskulären Systems wurden jedoch bislang nicht evaluiert.

**2.1.2 Mittelfristige Ermüdungsreaktionen**


HIU mittelfristige primär muskulär bedingte Funktionseinschränkungen (Leveritt & Abernethy, 1999). Diese sind sowohl objektiv messbar als auch subjektiv wahrnehmbar.

**Muskuläre Ermüdung**


Exzentrische Muskelkontraktionen ergeben sich im HIT vor allem aufgrund der hohen Laufgeschwindigkeiten sowie des ständigen Wechsels zwischen Beschleunigungs- und Bremsphasen. Insbesondere während der Bremsarbeit (und vor allem während der Landephasen) treten muskelmechanische Beanspruchungen auf, die zu hohen exzentrischen Muskelspannungen führen und eng mit der Entstehung von Mikrotraumata verbunden sind (Thompson et al., 1999). Die im Vergleich zum klassischen niedrigintensiven und umfangsorientierten Ausdauertraining deutlich erhöhten Laufgeschwindigkeiten erfordern überdies eine gesteigte Ansteuerung schneller Muskelfasern (FT-Fasern). Diese weisen aufgrund ihrer strukturellen Eigenschaften einen höheren Schädigungsgrad als langsame Muskelfasern (ST-Fasern) auf. So wird vermutet, dass ST-Fa-
sern durch breitere Z-Bänder eine stärkere mechanische Bindung zwischen den kontraktilen Einheiten besitzen als FT-Fasern (Weineck, 2004). Folglich ist die Wahrscheinlichkeit von Muskeltraumatisierungen im Rahmen eines HIT gesteigert.


Die durch intensive intervallbasierte Belastungen potentiell induzierten Muskelzellsschädigungen können schließlich in einer bis zu mehrere Tage anhaltenden Minderung der physischen Leistungsfähigkeit resultieren. Dies wird zum Teil durch die strukturellen Schädigungen myofibrillärer Einheiten verursacht, deren Kontraktionskapazitäten infolgedessen reduziert sind (Nédélec et al., 2012). Zugleich führt die erhöhte Durchlässigkeit für Ca\(^{2+}\)-(Calcium-)Ionen (und Signal moleküle) des in Mitleidenschaft gezogenen Sarkolems und/oder sarkoplasmatischen Reticulums zu einer Zunahme der intrazellulären Ca\(^{2+}\)-Konzentration, die in der Folge die Proteolyse (die enzymatische Auflösung) von Strukturproteinen auslöst und den Kraftverlust intensiviert (Toigo, 2014). Konsequenterweise spielt das Auftreten von Mikrotraumata eine entscheidende Rolle bei den nach HIT beobachteten muskulären Ermüdungsphänomenen.

dann schon bis zu drei Tage benötigt (Nédélec et al., 2012). Zwar konnten Pascoe & Gladden (1996) nachweisen, dass die Resyntheserate von Muskelglykogen nach Ausübung hochintensiver Belastungen gesteigert ist, jedoch können Muskelschäden zu einer gleichzeitigen Hemmung der Glykogensynthese führen (Asp, Daugaard, Kristiansen, Kiens, & Richter, 1998; Costill et al., 1990; O’Reilly et al., 1987) und darüber hinaus die belastungsinduzierte Entleerung der Glykogenspeicher forcieren (Byrne et al., 2004; Tee, Bosch, & Lambert, 2007). Da die Entleerung der Muskelglykogenspeicher ebenso entsprechend zunehmender Belastungsintensität dramatisch ansteigt (Gollnick, Piehl, & Saltin, 1974), liegt eine durch HIT induzierte akute bis mittelfristige periphere Ermüdung sowie die damit einhergehenden Leistungseinbußen also möglicherweise ebenfalls in einer energetischen Unterversorgung der Muskelzellen begründet.

Zentrale Ermüdung


Folge zu Müdigkeitserscheinungen und Schläfrigkeit sowie letztlich zu einer zentralbedingten Schwächerung der Muskelaktivität (Ament & Verkerke, 2009).


Ergebnisse diverser Studien deuten jedoch darauf hin, dass sich das zentralnervöse Antriebsverhalten nach intensiven und/oder intermittierenden Belastungen kaum verändert (Bigland-Ritchie, Furbush, & Woods, 1986; Bishop, 2012; Byrne et al., 2004; Taylor, Allen, Butler, & Gandevia, 2000). Dies konnte in vielen Fällen mittels Elektromyographie und transkranialer Magnetstimulation (Bishop, 2012) sowie insbesondere mit Hilfe der Twitch-Interpolationstechnik nachgewiesen werden. Die Twitch-Interpolationstechnik erlaubt eine objektive Messung rekrutierter und nicht-rekrutierter Anteile eines Muskels während einer isometrischen Kontraktion und ermöglicht so eine differenzierte Abschätzung peripherer und zentraler Kraftverluste (Gonschorek, Feistner, Tschernitschek, & Awiszus, 1997; Rutherford, Jones, & Newham, 1986). Dabei wird ein willkürlich unter isometrischen Bedingungen kontrahierender Muskel perkutan mittels Oberflächenelektroden, die entweder am Muskelbauch oder am motorischen Nerv angebracht werden, elektrisch gereizt (Byrne et al., 2004; Gonschorek et al., 1997). Dadurch wird ein zusätzlicher isometrischer Twitch auf die vom Muskel durch Willkürinnervation generierte Kraft gesetzt. Hierbei gilt: Je höher die durch die Elektrostimulation zusätzlich erzeugte Kraft, desto eingeschränkter die Willküraktivierung und desto größer der Einfluss zentraler Ermüdungsmechanismen (Byrne et al., 2004).

Mittels Twitch-Interpolationstechnik vorgelegte Befunde weisen darauf hin, dass vor allem nach exzentrisch akzentuierten Belastungsprotokollen eine Leistungsminderung vorrangig durch peri-
phere Ermüdungsmechanismen verursacht wird (Byrne et al., 2004). Die hierzu publizierten Ergebnisse basieren jedoch überwiegend auf Untersuchungen, die den Einfluss von krafttrainingsorientierten Trainingsprotokollen auf die zentrale und periphere Ermüdung untersuchten. Insofern tragen die Befunde nur eingeschränkt bzw. indirekt bei der Aufklärung der Ursachen für die im Anschluss von HIT beobachteten Leistungseinbußen bei. Eine detaillierte und differenzierte Evaluation HIT-induzierter Belastungs- und Ermüdungsreaktionen ist jedoch bislang nicht erfolgt.

**Problemstellung**


**Tab. 1.** Belastungsprotokolle aus ausgewählten Publikationen zum High-Intensity Interval Training.

<table>
<thead>
<tr>
<th>Autoren</th>
<th>Dauer</th>
<th>Intensität</th>
<th>Pause / Serienpause</th>
<th>Serien x Wdh.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clark et al. (2004)</td>
<td>5 min</td>
<td>85% VO\text{\textsubscript{max}}</td>
<td>60 s</td>
<td>1 x 8</td>
</tr>
<tr>
<td>Leveritt &amp; Abernethy (1999)</td>
<td>5 min</td>
<td>40 – 70% of peak power output</td>
<td>300 s</td>
<td>1 x 5</td>
</tr>
<tr>
<td>Breil, Weber, Koller, Hoppeler, &amp; Vogt (2010)</td>
<td>4 min</td>
<td>90 – 95% HF\text{\textsubscript{max}}</td>
<td>180 s</td>
<td>1 x 4</td>
</tr>
<tr>
<td>Lamberts, Swart, Noakes, &amp; Lambert (2009)</td>
<td>4 min</td>
<td>80% of peak power output</td>
<td>90 s</td>
<td>1 x 8</td>
</tr>
<tr>
<td>Ferrari Bravo et al. (2008)</td>
<td>4 min</td>
<td>90 – 95% HF\text{\textsubscript{max}}</td>
<td>180 s</td>
<td>1 x 4</td>
</tr>
<tr>
<td>Driller, Felt, Gregory, Shing, &amp; Williams (2009)</td>
<td>2,5 min</td>
<td>90% of peak power output</td>
<td>Varierend</td>
<td>1 x 8</td>
</tr>
<tr>
<td>Edge, Bishop, &amp; Goodman (2006)</td>
<td>2 min</td>
<td>120 – 140% of lactate threshold</td>
<td>60 s</td>
<td>1 x 4 – 10</td>
</tr>
<tr>
<td>Laursen, Blanchard, &amp; Jenkins (2002)</td>
<td>1 min</td>
<td>100% of peak power output</td>
<td>120 s</td>
<td>1 x 20</td>
</tr>
<tr>
<td>McKay, Paterson, &amp; Kowalchuk (2009)</td>
<td>1 min</td>
<td>120% VO\text{\textsubscript{max}}</td>
<td>60 s</td>
<td>1 x 8 – 12</td>
</tr>
<tr>
<td>Creer, Ricard, Conlee, Hoyt, &amp; Parcell (2004)</td>
<td>30 s</td>
<td>all out</td>
<td>240 s</td>
<td>4 x 10</td>
</tr>
<tr>
<td>Cicconi-Kolsky et al. (2011)</td>
<td>30 s</td>
<td>all out</td>
<td>150 s</td>
<td>1 x 12</td>
</tr>
<tr>
<td>Dupont, Alkalpo, &amp; Berthoin (2004)</td>
<td>15 s</td>
<td>120% of maximal aerobic speed</td>
<td>15 s</td>
<td>2 x 12 – 15</td>
</tr>
<tr>
<td>Helgerud et al. (2007)</td>
<td>15 s</td>
<td>90 – 95% HF\text{\textsubscript{max}}</td>
<td>15 s</td>
<td>1 x 47</td>
</tr>
<tr>
<td>Fernandez, Zmek, Wiewelhove, &amp; Ferrauti (2012)</td>
<td>5 s</td>
<td>all out</td>
<td>15 s / 8 min</td>
<td>3 x 10</td>
</tr>
<tr>
<td>Ferrari Bravo et al. (2008)</td>
<td>40 m</td>
<td>all out</td>
<td>20 s / 4 min</td>
<td>3 x 6</td>
</tr>
<tr>
<td>Tonnessen, Shallfawi, Haugen, &amp; Enoksen (2011)</td>
<td>40 m</td>
<td>all out</td>
<td>90 s / 10 min</td>
<td>2 – 4 x 4 – 5</td>
</tr>
</tbody>
</table>

VO\text{\textsubscript{max}} = maximale Sauerstoffaufnahme; HF\text{\textsubscript{max}} = maximale Herzfrequenz
2.2 Messung von Erholungsbedarf

In der Literatur werden verschiedene leistungsbezogene, neuromuskuläre, vegetative, laborchemische und psychometrische Parameter zur Überwachung der Trainings- und Wettkampfbeanspruchung sowie zur Erfassung des Regenerationsbedarfs vorgeschlagen. Es folgt zunächst eine Auswahl potentieller in der Trainings- und Forschungspraxis angewendeter Ermüdungsmarker.

2.2.1 Leistungsdiagnostik


2.2.2 Neuromuskuläre Funktionsdiagnostik

Neben der Twitch-Interpolationstechnik (siehe Kap. 2.1.2) wird die Tensiomyographie (TMG) als nichtinvasives Verfahren zur Diagnose neuromuskulärer Ermüdungssymptome vorgeschlagen (Hunter et al., 2012). Die TMG liefert mittels elektrischer Stimulation Aussagen zu kontraktilen Eigenschaften der beanspruchten Muskulatur (Muskelverformung, Kontraktionszeit, Kontraktionsverzögerungszeit, Kontraktionserhaltszeit, Kontraktionserholungszeit) (García-Manso et al., 2012; Rey, Lago-Peñas, & Lago-Ballesteros, 2012). Da so indirekt der Schweregrad mikrotraumatischer Muskelverletzungen quantifiziert werden kann (De Paula Simola et al., 2015; Hunter et al., 2012), wurde das Verfahren bereits in verschiedenen Regenerationsstudien eingesetzt (García-Manso, Rodríguez-Matoso, et al., 2011; García-Manso, Rodríguez-Ruiz, et al., 2011; Rey, Lago-Peñas, Lago-Ballesteros, & Casáis, 2012). Bisherige Untersuchungen deuten darauf hin, dass unter Einhaltung strenger Qualitätskriterien reliable und valide Ergebnisse für die Messung des Regenerationsbedarfs gewonnen werden können (Ditroilo, Smith, Fairweather, & Hunter, 2014). Im Vergleich zu anderen Parametern ist eine routinemäßige Anwendung der TMG insbesondere im sportpraktischen Kontext jedoch weniger praktikabel und ökonomisch.

2.2.3 Vegetative Statusdiagnostik


2.2.4 Labordiagnostik


2.2.5  Subjektive Empfindungsdiagnostik


### Problemstellung

2.3 Erholungssteuerung im High-Intensity Ausdauertraining

Durch hochintensive Belastungen induzierte Ermüdungssymptome können durch zahlreiche regenerationsfördernde Maßnahmen vermeintlich gelindert werden, deren Wirksamkeitsnachweis jedoch nur selten unter wissenschaftlich kontrollierten Bedingungen überzeugend erfolgt ist. Die meisten Verfahren werden im Anschluss an eine Belastung appliziert und dienen folglich der Linderung mittelfristiger Ermüdungsmechanismen, die mehrere Stunden oder Tage anhalten können. Vereinzelt werden Anwendungen (z.B. aktive Erholung, Stretching, Kompressionskleidung, Kälteapplikationen) aber auch während einer Belastung eingesetzt, um akute Ermüdungsvorgänge zu hemmen und so kurzfristig die Qualität der Trainings- oder Wettkampfleistung zu unterstützen. Potentiell erholungsfördernde Maßnahmen sind unter anderem folgende:

- Aktive Erholung (moderate rein aerobe Aktivitäten großer Muskelgruppen)
- Stretching
- Hydrotherapie (z.B. Dampf- oder wechselwarme Kontrastbäder)
- Lichttherapie (z.B. LED-Therapie)
- Ernährung, Nahrungssupplemente und Flüssigkeitszufuhr
- Schlaf, einschließlich intendierter Ruhe- bzw. Tagschlafphasen (z.B. „Powernaps“)
- Psychologische Maßnahmen (z.B. Autogenes Training oder Progressive Muskelrelaxation)
- (Eigen-)Massage, physiotherapeutische Anwendungen und Elektrostimulation
- Kompressionskleidung und gerätegestützte Kompressionsanwendungen
- Wärmeapplikationen (z.B. Sauna oder Infrarottherapie)
- Kälteapplikationen (z.B. Kaltwasserimmersion oder Kryotherapie)


2.3.1 Aktive Erholung

Aktive Erholungsstrategien beinhalten moderate, dynamische und rein aerobe Aktivitäten großer Muskelgruppen wie Jogging, Fahrradfahren, Schwimmen oder sanftes Krafttraining mit dem Ziel der beschleunigten metabolischen und myofibrillären Homöostaseherstellung (Nédélec et al.,

2.3.2 Stretching

2.3.3 Schlaf


2.3.4 Massage

gen. Die inkonsistente Datenlage bezüglich der Wirkungen von Massagen kann dabei u.a. auf die große Auswahl verschiedener Massagetechniken zurückgeführt werden (Nédélec et al., 2013).

2.3.5 Kompressionskleidung

Aktuell ist das Tragen von Kompressionskleidung (insbesondere für die unteren Extremitäten) bei zahlreichen Athleten populär. Sie soll der Verletzungsprophylaxe, Leistungssteigerung und Erholungsförderung dienen (Barnett, 2006a). So wird angenommen, dass Kompressionskleidung über eine gesteigerte Propriozeption sowie eine verringerte Muskeloszillation die Leistung positiv be-
einflusst (Doan et al., 2003). Eindeutige Hinweise auf eine Leistungssteigerung in Training und Wettkampf existieren bislang jedoch nicht (Duffield & Portus, 2007; Duffield et al., 2008; Duffield, Cannon, & King, 2010). Der wissenschaftlich nachgewiesene regenerative Wert von Kompressionsanwendungen ist vergleichbar mit jener der Hydrotherapie: Steigerung des venösen Rück-
stroms, des arteriellen Bluteinstroms sowie des lymphatischen Ausstroms, Vermeidung von Ödembildung, Beschleunigung des Laktatabbaus, Verbesserung von zellulären Reparaturpro-
zessen, Reduktion von Muskelschmerzen und Steigerung des Erholungsempfindens (Davies, Thompson & Cooper, 2009; Nédélec et al., 2013). Doch auch die wissenschaftlichen Nachweise zur regenerativen Wirksamkeit von Kompressionskleidung sind zum Teil äquivok und noch nicht lückenlos aufbereitet (Hausswirth & Mujika, 2013).

2.3.6 Wärmeapplikationen

Das Trockensaunabad ist eine seit langem weit verbreitete und von zahlreichen Athleten regel-
mäßig im Anschluss an Training oder Wettkampf benutzte Wärmeapplikation. Die empfohlene Trockenhitze liegt für einer Anwendungsdauer von 1 – 3 x 5 – 20 min und einer Luftfeuchtigkeit von 15 – 30% zwischen 80 °C und 90 °C (Hausswirth & Mujika, 2013). Zum Teil wurden in Un-
 tersuchungen jedoch ebenso Nutzungsbedingungen mit einer Luftfeuchtigkeit zwischen 3% und 50% definiert sowie Temperaturen von bis zu 110 °C erreicht (Kukkonen-Harjula & Kauppinen, 1988; Paolone, Lanigan, Lewis, & Goldstein, 1980; Shoenfeld, Sohar, Ohry, & Shapiro, 1976). Der während des Saunabads induzierte Anstieg der Körpertemperatur beeinflusst kardiovasku-

2.3.7 Kälteapplikationen


In wissenschaftlichen Untersuchungen konnten muskuläre Beschwerden teilweise gelindert, die Serumkonzentration von CK, Myoglobin oder Entzündungsmarkern gesenkt und die Leistungsfähigkeit erhöht werden (Ascensão, Leite, Rebelo, Magalhães, & Magalhães, 2011; Bailey et al., 2007; Elias, Wyckelsma, Varley, McKenna, & Aughey, 2013; Hausswirth et al., 2011; Ingram, Dawson, Goodman, Wallman, & Beilby, 2009; Minett, Duffield, Kellett, & Portus, 2012; Pointon &

**Problemstellung**

Tab. 2. Übersicht der Wirkungsebenen ausgewählter Regenerationsverfahren mit sportpraktischer Relevanz (modifiziert nach Wiewelhove & Ferrauti, 2016).

<table>
<thead>
<tr>
<th>Wirkungsebene</th>
<th>Parameter</th>
<th>Regenerationsverfahren</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Aktive Erholung</td>
</tr>
<tr>
<td>Energetisch</td>
<td>Laktat-Elimination</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>Substrattransport</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>Glykogen-Gehalt</td>
<td>-</td>
</tr>
<tr>
<td>Zirkulatorisch</td>
<td>Venöser Rückstrom</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>Lokaler Blutfluss</td>
<td>+</td>
</tr>
<tr>
<td>Muskulär</td>
<td>Temperatur</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>Dehnbarkeit</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>DOMS</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>CK</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>Satellitenzellaktivierung</td>
<td>o</td>
</tr>
<tr>
<td>Inflammatorisch</td>
<td>CRP, IL-1, IL-6, TNF α</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>Ödembildung</td>
<td>-</td>
</tr>
<tr>
<td>Endokrinologisch</td>
<td>Cortisol</td>
<td>o</td>
</tr>
<tr>
<td></td>
<td>IGF-1</td>
<td>-</td>
</tr>
<tr>
<td>Neuromuskulär</td>
<td>Aktivierungspotential</td>
<td>o</td>
</tr>
<tr>
<td></td>
<td>Schmerz</td>
<td>+</td>
</tr>
<tr>
<td>Psychovegetativ</td>
<td>Erholungszustand</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>Beanspruchungszustand</td>
<td>o</td>
</tr>
<tr>
<td>Leistungssteigernd</td>
<td>Akut</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>am Folgetag</td>
<td>o</td>
</tr>
</tbody>
</table>

+ = positiver Einfluss im Vergleich zu passiver Regeneration (PR); o = kein Unterschied im Vergleich zu PR; - = negativer Einfluss im Vergleich zu PR; DOMS = Delayed Onset Muscle Soreness; CK = Creatinkinase; Urea = Harnstoff; CRP = C-reaktives Protein; IL = Interleukin; TNF α = Tumornekrosefaktor; IGF = Insulin-like Growth Factor

**Abb. 1.** Gesamtdesign des dreistufigen Arbeitsprogramms. HIT = High-Intensity Interval Training
3.1 Modulübersicht

Die nachstehend beschriebenen Module beziehen sich auf das in Abb. 1 dargestellte Arbeitsprogramm und dokumentieren die zentralen Zielstellungen der Teiluntersuchungen. Die methodische Konzeption der Module zwei und drei erfolgte dabei unter Berücksichtigung der Befunde aus den jeweils vorausgegangenen Stufen.

Modul 1

Modul 2

Modul 3
Um belastungsinduzierte Ermüdungssymptome zu lindern, werden zahlreiche Regenerationsverfahren empfohlen, deren Wirkungen jedoch meist nicht zweifelsfrei geklärt sind. Da HIT jedoch teils mit erheblichem regenerativen Folgebedarf einhergeht, sind effiziente Regenerationsmaßnahmen besonders für diese Belastungsform von großem sportpraktischem Interesse. Ziel des dritten Moduls war es folglich, den Einfluss einer der verbreitetsten Regenerationsinterventionen (d.h. aktive Erholung) auf die durch HIT induzierten Ermüdungerscheinungen zu überprüfen.
4  PUBLIKATIONEN

Die Ergebnisse der aufeinander aufbauenden Untersuchungsabschnitte wurden im Rahmen von drei Beiträgen in internationalen Fachzeitschriften publiziert.

Manuskript 1


Zugriff unter:
http://www.minervamedica.it/en/journals/sports-med-physical-fitness/article.php?cod=R40Y9999N00A150036

Manuskript 2


Zugriff unter:
http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0139801

Manuskript 3


Zugriff unter:
http://journals.humankinetics.com/ijspp-current-issue

4.1 Publikation 1

Acute responses and muscle damage in different high-intensity interval running protocols

*Journal of Sports Medicine and Physical Fitness. 2015. [Epub ahead of print]*

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⁵Institute of Sports Science, Johannes-Gutenberg University, Mainz, Germany
Abstract

Aim

Our study aimed to evaluate the acute responses and exercise-induced muscle damage of five different high-intensity interval training (HIT) protocols adjusted by the maximum velocity obtained in the 30-15 Intermittent Fitness Test (VIFT).

Methods

Sixteen well-trained intermittent sport players (mean ± SD; age, 24.6 ± 2.7 years; VO_{2max}, 58.3 ± 5.9 ml·min·kg^{-1}) participated in five different HIT protocols separated by six days in between (P_{240}: 4×4 min at 80% V_{IFT}; P_{120}: 7×2 min at 85%; P_{30}: 2×10×30 s at 90%; P_{15}: 3×9×15 s at 95%; P_{5}: 4×6×5 s sprints). Blood lactate (La), blood pH, serum creatinkinase (CK), heart rate (HR), session rating of perceived exertion (session-RPE), delayed onset muscle soreness (DOMS) and countermovement jump (CMJ) height were measured.

Results

A significant main effect for protocol (p < 0.05) was found for the acute responses of HR, session-RPE and La with values increasing in longer intervals from P_{15} to P_{120} and P_{240} while blood pH responded inversely. In contrast, P_{5} produced the highest La concentration and blood pH decreases. 24 h post exercise CK, DOMS and the decrease in CMJ height were significantly higher after P_{5} compared to all other protocols (p < 0.05).

Conclusion

HIT protocols of different interval duration and intensity result in varying acute physiological and perceptual demands and exercise-induced muscle damage. Longer intervals with submaximal intensity lead to higher acute cardio-circulatory responses, whereas sprint protocols induce the highest muscle damage and muscle soreness.
**Introduction**

High intensity interval training (HIT) has become a substantial component of modern conditioning, especially in intermittent sports such as team or racket sports. In this regard, several previously published studies have shown positive effects of this training methodology on endurance performance. The rationale behind the use of HIT in various forms is that athletes can enhance cardiorespiratory, metabolic and neuromuscular function, using significantly lower training volumes compared to traditional high-volume low intensity endurance training. HIT involves repeated relatively short intensive work intervals interspersed by active or passive recovery periods. Based on volume, intensity, and expected physiological responses, HIT can be divided into repeated short to long (~15-240 s) bouts of high-yet submaximal-intensity exercises or repeated-sprint training (RST; sprints lasting from ~3-7 s). Submaximal-intensity exercises involve periods of work at 90-100% of maximum oxygen uptake velocity (v\(\dot{V}_O_2\)max), whereas in RST intensities are generally above 100% of v\(\dot{V}_O_2\)max.

In the last few years, manipulating different HIT variables (e.g. intensity and duration of work and relief intervals, number of intervals, number of series) gained scientific interest in regards to find the optimal HIT protocol for performance enhancement. Therefore, to accurately prescribe individual HIT programs, it is necessary to understand the acute responses to HIT when manipulating any of these variables. Additionally, as HIT can be accompanied with high neuromuscular demands, it is also important to understand the exercise-induced muscle damage following different HIT protocols (e.g., to determine the point at which HIT may negatively affect the performance in upcoming competitions). However, there is still a lack of knowledge regarding the detailed physiological reactions to various HIT regimes forced by particular exercise prescriptions, especially by the prescription of work interval intensity.

To set interval intensity, several approaches have been used. However, some of them are unsuitable to adjust intensity during HIT due to physiological and/or practical limitations (e.g. heart rate (HR) based approach). Furthermore, prescribing intensity using the v\(\dot{V}_O_2\)max obtained during laboratory testing protocols, is suitable for long (2-4 min) intervals, but problematic to appropriate individualize training intensities during supramaximal HIT. Thus, athletes having a similar v\(\dot{V}_O_2\)max may have different anaerobic velocity reserves (i.e., reserve of running velocity (AVR) left to the athlete once he has reached his v\(\dot{V}_O_2\)max). During HIT prescribed by the v\(\dot{V}_O_2\)max, athletes with a greater AVR will work at lower percentages of its AVR and will therefore present a lower exercise load compared to athletes with lower AVR. Also the lack of specificity of this mode of assessment (i.e., continuous running) is not reflective of the intermittent nature of team and racket sports, leading to the development of more valid sport-specific tests, like the 30-15 Intermittent Fitness Test (30-15IFT). The 30-15IFT was developed for intermittent exercise
and change of direction (COD) based HIT prescription. Due to the reliability and accuracy of the final running speed obtained in the 30-15IFT (VIFT) for individualizing players training intensity, this protocol is becoming popular when undertaking HIT in intermittent sports.\textsuperscript{18, 19}

To the best of our knowledge, there is a lack of studies showing the acute responses associated with different HIT exercise protocols based on the 30-15IFT. Thus, the aim of this study was to evaluate the acute physiological and perceptual responses as well as the exercise-induced muscle damage among five different HIT protocols adjusted by the maximum velocity obtained in the 30-15IFT (VIFT). We hypothesized that varying HIT variables (i.e., intensity, W/R) at fixed exercise duration will affect the acute responses and exercise-induced muscle damage of HIT.

\section*{Materials and methods}

\subsection*{Study design}

A randomized repeated-measures design was used in this study. All participants attended a familiarization visit to introduce the testing and training procedures and to minimize any learning effect. Preliminary examinations included baseline measures of body composition, an incremental treadmill test to determine VO\textsubscript{2max} and the 30-15IFT\textsuperscript{18} for assessment of intermittent running performance. During the following experimental period athletes participated in five different HIT protocols each separated by six days rest. To eliminate order effects HIT protocol test order was randomly assigned for each subject. For acute responses, blood lactate (La), blood pH, heart rate (HR) and session rating of perceived exertion (session-RPE) were measured. For exercise-induced muscle damage, serum creatinkinase (CK), delayed onset muscle soreness (DOMS) and countermovement jump (CMJ) height were determined. To minimize diurnal variations all tests were conducted at the same time of the day over 5 consecutive weeks in similar environmental conditions (week 1, 22.2°C; week 2, 20.8°C; week 3, 25.5°C; week 4, 20.3°C; week 5, 20.3°C) on a 400 m outdoor field. Participants were instructed to avoid other kinds of moderate-to-hard physical activity and to maintain their normal dietary intake and habitual lifestyle during the experimental period.

\subsection*{Subjects}

Sixteen well-trained male intermittent sports athletes (i.e., tennis, handball, soccer) volunteered to participate in the study (mean ± SD; age 24.6 ± 2.7 years; height 183.1 ± 6.3 cm; body mass 80.0 ± 8.8 kg; VO\textsubscript{2max} 58.3 ± 5.9 ml·min·kg\textsuperscript{-1}). Participants were fully informed about the experimental procedures and were required to give informed consent before any testing took place. The study was approved by the local ethic committee and performed according to the Declaration of Helsinki.
Preliminary Examinations

Preliminary laboratory examinations included anthropometrical measures (height, weight) and a progressive incremental exercise test on a motor driven treadmill (Ergo ELG2, Woodway GmbH; Germany) to determine VO₂max, vVO₂max, HRmax, turn points for lactate (LTP₁, LTP₂) and HR at the lactate turn points (HR LTP₁, HR LTP₂). The treadmill test started with an initial velocity of 8 km·h⁻¹, increasing 2 km·h⁻¹ every 3 min with a constant incline of 0.5% until voluntary exhaustion. VO₂ was continuously analyzed using a breath-by-breath gas collection system (ZAN600USB, Germany). The gas calibration was completed before each test day, and the volume calibration was conducted before each test following the instructions provided by the manufacturer. The highest mean value for 30 s was defined as the VO₂max. Capillary blood samples were taken from hyperemized earlobe during a 30 s break immediately after finishing each velocity level and at the time point of exhaustion and analyzed for La. Blood samples were taken with 20 µl capillaries, hemolyzed in 1-ml microtest tubes and analyzed enzymatic amperometrically by the Biosen S-Line Sport (EKF-Diagnostik, Germany). HR was monitored and recorded at 1 s intervals during the test (RS800CX, Polar Electro, Finland).

On a second preliminary examination day participants completed the 30-15IFT on an outdoor field track. The test consisted of 30 s shuttle runs interspersed with 15 s passive recovery periods. Speed was set at 8 km·h⁻¹ for the first 30 s run and was increased by 0.5 km·h⁻¹ every 45 s stage thereafter. The athletes had to run back and forth between two lines set 40 m apart at a pace dictated by an acoustic signal. The test ended when a player could no longer maintain the imposed running speed or when he was unable to reach a 3 m zone around each line at the moment of the audio signal for three consecutive times. The speed during the last completed stage was defined as maximum performance (VIFT). VIFT was used to calculate the interval intensity of the different HIT protocols as described by Buchheit.

Training Protocols

All protocols were designed with similar total training duration but different work/rest ratios (W/R) and represent the wide spectrum of HIT protocols used in scientific studies and exercise training (work intervals lasting from 240 to 5 s). Exercise mode, number and duration of intervals and rest, intensity (%VIFT) and W/R are shown in Table 1. A standardized continuous 10 min warm-up, consisting of 40 m shuttle runs at 60-70% HRmax followed by four 40 m acceleration sprints, was performed before all HIT sessions. Protocol 240 (P240) and 120 (P120) consisted of 4 min and 2 min straight-line runs, respectively. Protocols 30 (P30) and 15 (P15) were performed as 40 m shuttles, with COD-based 30 s and 15 s intermittent runs. Protocol 5 (P5) consisted of repeated straight
line sprints of 5 s. For $P_5$ athletes were instructed and verbally encouraged during the training to mobilize maximal effort for each 5 s sprint.

**Table 1**  High-intensity training protocol characteristics.

<table>
<thead>
<tr>
<th>Protocol</th>
<th>Exercise mode</th>
<th>Interval and recovery duration</th>
<th>Interval Intensity (%$V_{AT}$)</th>
<th>Recovery Intensity</th>
<th>Work/rest ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>$P_{240}$</td>
<td>Straight-line runs</td>
<td>4 x 4 min $(r = 3$ min$)$</td>
<td>80%</td>
<td>Passiv</td>
<td>2/1</td>
</tr>
<tr>
<td>$P_{120}$</td>
<td>Straight-line runs</td>
<td>7 x 2 min $(r = 2$ min$)$</td>
<td>85%</td>
<td>Passiv</td>
<td>1/1</td>
</tr>
<tr>
<td>$P_{30}$</td>
<td>40m-shuttle runs</td>
<td>2 x 10 x 30 s $(r = 45$ s and R = 3 min$)$</td>
<td>90%</td>
<td>Passiv</td>
<td>1/2</td>
</tr>
<tr>
<td>$P_{15}$</td>
<td>40m-shuttle runs</td>
<td>3 x 9 x 15 s $(r = 30$ s and R = 3 min$)$</td>
<td>95%</td>
<td>Passiv</td>
<td>1/4</td>
</tr>
<tr>
<td>$P_5$</td>
<td>Straight-line sprints</td>
<td>4 x 6 x 5 s $(r = 25$ s and R = 5 min$)$</td>
<td>all out</td>
<td>Passiv</td>
<td>1/12</td>
</tr>
</tbody>
</table>

Abbreviations: $r$ = between-interval recovery duration, R = between-set recovery duration

**Procedures**

**Blood Measures.** Capillary whole-blood samples were taken from the hyperemized earlobe and analyzed for La, pH and CK. La was measured pre-exercise, 3 times during each session after approximately 6, 12 and 18 min (always immediately at the end of a work interval) and at the end of each session. Blood samples were taken with 20 µl capillaries, hemolyzed in 1-ml microtest tubes and analyzed enzymatic amperometrically by the Biosen S-Line Sport (EKF-Diagnostik, Germany). Blood samples for determination of pH were taken with 85 µl capillaries immediately at the end of the last work interval of each session and analyzed by the ABL80 Flex (Radiometer, Denmark). CK was measured before and 24 h after each session. Samples were hemolyzed in 2-ml microtest gel tubes, centrifuged and analyzed by the COBAS INTEGRA® 400 plus (Roche Diagnostics, Germany).

**Heart Rate.** HR was monitored and recorded at 1-s intervals during the HIT sessions (RS800CX, Polar Electro, Finland). From the data the percentage of time spent by the athletes below and above HR LTP₂ as well as average and peak HR was calculated.
Perceptual Measures. The session-RPE method \(^{21}\) was used to calculate the training load for each session. In this method the training intensity was measured using a category-ratio (CR-10) RPE scale \(^{22}\) 30 min after the completion of the HIT session. The training load was then calculated by multiplying the numerical score of the athletes’ perception of effort with the total exercise duration. 24 h after the HIT session athletes were asked to score on a visual analogue scale (VAS) \(^{23}\) the general amount of DOMS. The VAS consisted of a 100 mm line whose endpoints were labeled by “no pain” (left) and “unbearable pain” (right). Subjects had to draw a vertical line at a point on the line that best represented their pain at the time of measurement. The score was the distance in cm from the left border of the scale to the point marked \(^{23}\).

Jump Performance. The CMJ was performed before, 30 min and 24 h after each HIT session. During the CMJ, participants stood on a contact platform (Haynl Elektronik, Germany), placed hands on hips and dropped down to a self-selected level before jumping maximally. Flight time was used to calculate jump height. Each subject performed two maximal CMJ at each measurement time and the mean height was calculated.

Statistical analysis

All data are presented as means and standard deviations (SD) and were tested for normal distribution using the Kolmogorov-Smirnov-Test. A one-way repeated measure ANOVA was performed to examine differences in physiological (HR, La, pH) and perceptual (session-RPE) responses between protocols. Additionally, a repeated measure ANOVA was used to compare exercise-induced muscle damage (CMJ, CK, and DOMS) over time (factor 1: protocol, factor 2: measuring point). When significant main effects were observed, a Bonferroni post-hoc test was performed. \(P < 0.05\) for the \(\alpha\)-error was accepted as level of significance for statistical comparisons. To allow a better interpretation of the results, effect sizes were also calculated (partial eta squared, \(\eta^2_p\)). \(^{24}\) The SPSS statistical software package (version 18, SPSS Inc., Chicago, IL, USA) was used for statistical computation.
Results

Figure 1. Short-term effects of different high-intensity training protocols on heart rate (HR), session-RPE, blood lactate (La) and blood pH. Letters represent significant differences (p<0.05). aSignificantly different from P_{120} and P_{5}; bsignificantly different from all other protocols; csignificantly different from P_{240}, P_{120} and P_{5}; dsignificantly different from P_{120}; esignificantly different from P_{240}, P_{120} and P_{30}; fsignificantly different from P_{240} and P_{120}.

Acute responses

There was a main effect for protocol in mean La (p<0.001, \eta^2_p=0.60). Lower La values were found for P_{15} (p<0.05) compared to all other HIT protocols (Figure 1A). In addition, La during P_{30} was lower compared to P_{120} (p<0.05) and P_{5} (p<0.01), while no differences in mean La concentration were found between P_{240}, P_{120} and P_{5} (p>0.05). Correspondingly a difference between protocols was observed for blood pH (p<0.001, \eta^2_p=0.56). Post hoc analysis revealed lower post training
values in P\textsubscript{240} (p<0.05), P\textsubscript{120} (p<0.01), and P\textsubscript{5} (p<0.001) compared to P\textsubscript{15} as well as in P\textsubscript{120} (p<0.05) and P\textsubscript{5} (p<0.001) compared to P\textsubscript{30} (Figure 1B). The time course for blood lactate concentration is shown in Figure 2. Blood lactate accumulation rises during P\textsubscript{240}, P\textsubscript{120}, P\textsubscript{30} and P\textsubscript{5} while a La steady state was shown for P\textsubscript{15}. Session-RPE showed a main effect for protocol (p<0.001, $\eta^2_p$=0.43), with post hoc analysis revealing lower values in P\textsubscript{15} (p<0.05) compared to all other HIT protocols (Figure 1C). A difference in Session-RPE was also found between P\textsubscript{30} and P\textsubscript{120} (p<0.05), while there was no difference between P\textsubscript{240}, P\textsubscript{120} and P\textsubscript{5} (p>0.05).

Mean and peak HR of the five HIT protocols are shown in Figure 1D. A difference between protocols in mean (p<0.001, $\eta^2_p$=0.58) and peak (p<0.001, $\eta^2_p$=0.68) HR was found. Mean HR was lower in P\textsubscript{5} compared to P\textsubscript{240} (p<0.001), P\textsubscript{120} (p<0.01) and P\textsubscript{30} (p<0.001). The lowest peak HR was observed in P\textsubscript{15} and P\textsubscript{5} (p<0.05), whereas mean and peak HR did not differ between P\textsubscript{240}, P\textsubscript{120} and P\textsubscript{30} (p<0.05). Table 2 shows the percentage of time spent by the athletes below and above HR LTP\textsubscript{2}. In P\textsubscript{30}, P\textsubscript{15} and P\textsubscript{5} athletes spent longer times below HR LTP\textsubscript{2} then above HR LTP\textsubscript{2} (p<0.05), whereas the time at > HR LTP\textsubscript{2} was lower in P\textsubscript{5} compared to P\textsubscript{240}, P\textsubscript{120} and P\textsubscript{30} as well as in P\textsubscript{15} compared to P\textsubscript{120} (p<0.05).
Table 2. Percentage of time spent by the athletes below and above the heart rate at the second lactate turn point (HR LTP\(_2\)) during high-intensity interval training protocols. Data are presented as mean ± SD.

<table>
<thead>
<tr>
<th>Protocol</th>
<th>Below HR LTP(_2)</th>
<th>Above HR LTP(_2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>P(_{240})</td>
<td>65 ± 20</td>
<td>35 ± 25c</td>
</tr>
<tr>
<td>P(_{120})</td>
<td>63 ± 19</td>
<td>37 ± 19c</td>
</tr>
<tr>
<td>P(_{30})</td>
<td>69 ± 22</td>
<td>31 ± 22c</td>
</tr>
<tr>
<td>P(_{15})</td>
<td>82 ± 21</td>
<td>18 ± 21c</td>
</tr>
<tr>
<td>P(_{5})</td>
<td>86 ± 90</td>
<td>14 ± 9c</td>
</tr>
</tbody>
</table>

Letters represent significant differences (p<0.05). Significantly lower than the other HR-category; Significantly different from P\(_{240}\), P\(_{120}\) and P\(_{30}\); Significantly different from P\(_{120}\).

Exercise-induced muscle damage

No main effect for protocol but a main effect for time (CK: p<0.01, \(\eta_p^2=0.57\); CMJ: p<0.05, \(\eta_p^2=0.22\)) and a protocol x time interaction (CK, p<0.01, \(\eta_p^2=0.31\); CMJ, p<0.05, \(\eta_p^2=0.20\)) were also found for CK and CMJ performance (Figure 2A and Table 3). Changes in CMJ performance for P\(_{5}\) were different compared to P\(_{30}\) and P\(_{15}\) (p<0.05). Only P\(_{5}\) provoked a decrease in CMJ performance 30 min and 24 h after training (p<0.05). CK was increased 24 h after P\(_{240}\) and P\(_{5}\) (p<0.05), whereas the increase was greater after P\(_{5}\) compared to the other protocols (p<0.05). DOMS also showed a main effect for protocol (p<0.01, \(\eta_p^2=0.28\)). Muscle soreness was higher in P\(_{5}\) compared to P\(_{30}\) (p<0.01), whereas DOMS did not differ between P\(_{240}\), P\(_{120}\), P\(_{30}\) and P\(_{15}\) (p>0.05) (Figure 3B).

Table 3. Short- and mid-term effects of different high-intensity training protocols on countermovement jump (CMJ) performance. Data are presented as mean ± SD.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Protocol</th>
<th>Pre</th>
<th>Post30min</th>
<th>Post24h</th>
<th>Protocol</th>
<th>Time</th>
<th>Interaction</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>p</td>
<td>(\eta_p^2)</td>
<td>p</td>
<td>(\eta_p^2)</td>
<td>p</td>
<td>(\eta_p^2)</td>
</tr>
<tr>
<td>CMJ (cm)</td>
<td>P(_{240})</td>
<td>38.5 ± 5.7</td>
<td>37.7 ± 5.6</td>
<td>37.2 ± 5.9</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>P(_{120})</td>
<td>39.3 ± 5.1</td>
<td>38.0 ± 5.6</td>
<td>37.7 ± 4.6</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>P(_{30})</td>
<td>37.6 ± 5.4</td>
<td>38.5 ± 4.9</td>
<td>37.6 ± 4.9</td>
<td>0.279</td>
<td>0.085</td>
<td>0.029</td>
</tr>
<tr>
<td></td>
<td>P(_{15})</td>
<td>36.9 ± 6.2</td>
<td>38.8 ± 5.4</td>
<td>37.5 ± 5.2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>P(_{5})</td>
<td>38.7 ± 5.2</td>
<td>36.2 ± 4.8c</td>
<td>35.8 ± 4.6c</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Letters represent significant differences (p<0.05). Significantly different to pre; significant protocol x time interaction. Abbreviations: \(\eta_p^2\), partial eta squared.
BELASTUNGS- UND ERHOLUNGSSTEUERUNG IM HIGH-INTENSITY AUSDAUERTRAINING

Discussion

The aim of this study was to examine acute responses as well as exercise-induced muscle damage of five different HIT protocols, covering the same absolute training duration of approximately 30 min. All HIT protocols induced high acute responses, reflected in different physiological (HR, %HR\text{max}, La, blood pH), perceptual (session-RPE) and performance (CMJ) markers. Moreover, as hypothesized, results demonstrate that altering the structure and characteristics of the HIT protocols significantly alters both, the acute and the post-exercise responses, despite the subjects performing similar exercise duration. Thus, in P\textsubscript{15} acute responses were rather low compared to the other protocols, reflecting that caution is necessary when prescribing HIT, especially if several HIT components are manipulated simultaneously.

Average HR responses during all protocols ranged from 76% to 86% of HR\text{max}, with the time spent above HR LTP\textsubscript{2} being significantly higher in the longer intervals (Table 2). Furthermore, assuming that V\text{ERT} is faster than vVO\textsubscript{2}\text{max}\textsuperscript{19} (i.e., 2.5 ± 0.9 km·h\textsuperscript{-1} in the present study), participants performed the training sessions at intensities ranging from ~90% (P\textsubscript{240}) to ~110% (P\textsubscript{15}) of vVO\textsubscript{2}\text{max} (obtained in the laboratory test) and spent approximately 7 min (P\textsubscript{15}) to 16 min (P\textsubscript{240}) at intensities > 90% of vVO\textsubscript{2}\text{max}. In this regard, several authors suggested that athletes should spend at least several minutes per HIT session at intensities > 90% of vVO\textsubscript{2}\text{max} to stimulate cardiovascular and peripheral adaptations.\textsuperscript{4, 7, 13} In this regard, the amount of high intensity exercise accumulated during
HIT protocols in the current study have been positively related to changes in aerobic fitness when performed regularly.

In all protocols blood lactate levels were quite high and corresponding blood pH was low. Exercise-induced lactate concentrations are also reflected by session-RPE (Figure 1). At a physiological level, HIT provides a simultaneous and a mixed solicitation of the aerobic and anaerobic metabolism. However, especially the anaerobic glycolytic energy contribution and therefore the close relationship between muscle glycogen depletion and muscle fatigue as well as the potentially beneficial effects of lactic acid on the performance of fatigued muscles seems likely to be an important variable in the consideration of prescribing HIT protocols. Interestingly, P240 and P120 induced higher lactate concentrations compared to P30 and P15 (Figure 1). This can be related to the fact that despite the lower interval intensities during P240 (80% VIFT) and P120 (85% VIFT) the running velocity still remains above the maximal lactate steady state velocity. Obviously, despite a lower glycolytic rate in case of a lower running velocity, the total lactate production and peripheral accumulation during the longer intervals exceed those during the shorter but more intensive intervals. This is in agreement with previous HIT research, showing that long intervals at intensities near vVO2max yielding high lactate levels up to 16 mmol·L−1 (i.e., a high rate of anaerobic provision of ATP through glycolysis and therefore an accelerated glycogen depletion) whereas short intervals at the same intensity induce low lactate concentrations.

The highest (P5) and the lowest (P15) lactate concentrations were found in protocols including short interval durations with the highest running velocity (Figure 1). In case of P5 it can be speculated that the insufficient phosphocreatine (PCr) recovery time (25 s) in combination with the high glycolytic rate of the repeated all out sprint protocol is predominantly responsible for the extremely high blood lactate levels. The time-course of PCr recovery occurs exponentially, with an estimated half-life of about 30 s in humans. It can be considered that the 25 s recovery period in P5 resulted in an incomplete PCr restoration between the repeated sprints, leading to increased demands of anaerobic glycolysis to maintain the rate of energy production. The recovery period in P15 (30 s) was comparably to P5 (25 s) yet protocol differences were related to interval intensity and the integration of multiple changes of direction in P15 (Table 1). The introduction of changes of direction has been shown to increase blood lactate, irrespective of the work intensity and duration, with HIT protocols performed at the same intensity showing significantly higher physiological and perceptual responses in shuttle format compared with straight-line running. Consequently, the pronounced difference in the anaerobic glycolytic demands can mainly be attributed to the lower running velocity in P15.

Based on the suggested models for the classification of intermittent exercise by Tschakert and Hofmann, P240, P120, P30 and P5 can be characterized as anaerobic intermittent exercise, due to
the high blood lactate levels and the imbalance between lactate production and elimination (Figure 2). In contrast, \( P_{15} \) is rather characterized as aerobic intermittent exercise due to the lower blood lactate levels and a balance between lactate production and elimination illustrated by a small range of lactate concentrations between the successive sets. HIT that evokes high blood lactate levels may elicit certain benefits, particularly for intermittent sport athletes, such as the improvement of both, anaerobic and aerobic metabolism, La tolerance and \( \text{VO}_{2\text{max}} \). This can probably be explained by a shift of the metabolic pathways from exclusively anaerobic to partially aerobic metabolism caused by high La levels. It has been suggested that elevated \( \text{H}^+ \) concentrations enhance the oxidative mechanisms of energy supply by an inhibition of the glycolytic enzymes phosphorylase and phosphofructokinase and an increase in pyruvate dehydrogenase activity despite high exercise intensities. Therefore, \( P_{240}, P_{120}, P_{30} \) and \( P_5 \) as prescribed in the current study seem to be more adequate to enhance all fitness components due to the higher anaerobic demands (i.e., high blood lactate levels) compared to \( P_{15} \). This is also supported by the calculation of the mean load (\( P_{\text{mean}} \)) as suggested by Tschakert and Hofmann, showing the highest \( P_{\text{mean}} \) in \( P_{240} \) and \( P_{120} \), while the lowest \( P_{\text{mean}} \) was calculated for \( P_{15} \).

When prescribing HIT, the accurate quantification of the physiological impact induced by these sessions is crucial for understanding recovery needs and allowing adequate rest prior to a second training session. In addition to acute responses, inflammation, muscle damage and neuromuscular function after HIT seem to be important variables in order to prescribe appropriate training and rest ratio. Previous studies have reported substantial inflammatory responses to various forms of prolonged, continuous aerobic exercise. However, much less is known about the inflammatory responses to HIT. In this regard, it has been reported that a single bout of HIT increases circulating levels of several inflammatory cytokines and chemokines as well as impairs jumping performance, although the inflammatory response to an acute bout of HIT appears to be substantially lower than that of prolonged, continuous aerobic exercise.

Data of the present study show that CK levels 24 h after HIT were significantly increased only in \( P_{240} \) and \( P_5 \), with \( P_5 \) showing significantly higher values compared to all other protocols (Figure 3A). Increased CK levels especially following \( P_5 \) are in agreement with previous results showing elevated CK and DOMS for even 72 h post exercise after a RST protocol. This could be related to the muscle damage induced as a result of the RST protocol due to considerable accelerations and decelerations along with high eccentric forces generated during sprinting strides. Therefore, this would be an important aspect of RST compared to other HIT formats and has to be taken into account when planning HIT and recovery. Present results also show a significant decline in CMJ height 30 min and 24 h only after \( P_5 \) (Table 3). This seems to be related to the muscle damage caused by this protocol, and is in line with previous research showing reductions in jumping performance after similar HIT protocols.
Two final limitations to our study are, first, that the five different HIT protocols were derived from scientific literature and in detail defined following their usual practical prescription components (e.g., number and duration of intervals and rest, intensity). Therefore, protocols were only matched for total training duration (~30 min) which meets best the usual practical needs. Consequently, it is not clear which of the multiple prescription components in detail were the trigger for those diverse acute responses. However, to the best of our knowledge a standardized and consistent classification model for the comparison of extremely different HIT protocols does not exist yet. Although it has been suggested to match protocols by total energy expenditure or $P_{\text{mean}}$ our pre-investigations showed that in case of all out RST protocols this approach would lead to considerable differences in total training duration (e.g., matching $P_5$ with $P_{240}$ by energy expenditure) which appears to be not useful from a practical point of view (e.g., extreme extension of training duration of $P_5$).

Secondly, the relevance of CK as a marker of muscle damage is questionable since some athletes are non-responders and elevations of CK concentrations are not necessarily a clear indication of muscle fatigue. Also, it has been proposed that myoglobin is a more sensitive measure of muscle damage than CK. However, we were able to show differences in CK concentration over time as well as between HIT protocols, indicating that the determination of CK is sensitive enough for the purpose of our study. In addition, CK is still used widely in science and practice, as CK remains elevated for several days in comparison to other proteins such as myoglobin and its determination is simple.

**Conclusions**

In conclusion results of the present study showed that prescribing HIT protocols based on their acute physiological responses is a complex process and includes the proper management of several training variables as interval intensity and duration, rest intervals between repetitions and sets, exercise modality (e.g., straight running, runs with COD, sprinting) and mean load. Although HIT protocols analyzed in the present study were prescribed using the same absolute training duration, acute responses and exercise-induced muscle damage differ significantly. Coaches and scientists hence are urged to pay attention when defining intermittent exercise, as the prescription of HIT affects the level of acute response and the likely forthcoming adaptations. While the amount of high intensity exercise accumulated during HIT protocols in the current study, especially in $P_{240}$, $P_{120}$, $P_{30}$ and $P_5$, has been positively related to changes in aerobic fitness when performed regularly, it seems important to take differences into consideration the residual muscle damage from HIT sessions. Especially $P_5$ showed elevated CK and DOMS 24 h post exercise. Therefore, this would be an important aspect of RST compared to other HIT formats and has to
be taken into account when planning HIT and recovery. In this context, also the athletes’ training status should be considered since toleration of RST in highly adapted athletes may vary.

Acknowledgement

The present study was initiated and funded by the German Federal Institute of Sport Science. The research was realized within RegMan – Optimization of Training and Competition: Management of Regeneration in Elite Sports (IIA1-081901/12-16). The results of the present study do not constitute endorsement of the product by the authors or the journal.

References


4.2 Publikation 2

Markers for routine assessment of fatigue and recovery in male and female team sport athletes during high-intensity interval training

_PLOS ONE. 2015. 10 (10), 1-17._

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Abstract

Aim

Our study aimed to investigate changes of different markers for routine assessment of fatigue and recovery in response to high-intensity interval training (HIIT).

Methods

22 well-trained male and female team sport athletes (age, 23.0 ± 2.7 years; VO_{2max}, 57.6 ± 8.6 mL·min·kg^{-1}) participated in a six-day running-based HIIT-microcycle with a total of eleven HIIT sessions. Repeated sprint ability (RSA; criterion measure of fatigue and recovery), countermovement jump (CMJ) height, jump efficiency in a multiple rebound jump test (MRJ), 20-m sprint performance, muscle contractile properties, serum concentrations of creatinkinase (CK), c-reactive protein (CRP) and urea as well as perceived muscle soreness (DOMS) were measured pre and post the training program as well as after 72 h of recovery.

Results

Following the microcycle significant changes (p < 0.05) in RSA as well as in CMJ and MRJ performance could be observed, showing a decline (%Δ ± 90% confidence limits, ES = effect size; RSA: -3.8 ± 1.0, ES = -1.51; CMJ: 8.4 ± 2.9, ES = -1.35; MRJ: 17.4 ± 4.5, ES = -1.60) and a return to baseline level (RSA: 2.8 ± 2.6, ES = 0.53; CMJ: 4.1 ± 2.9, ES = 0.68; MRJ: 6.5 ± 4.5, ES = 0.63) after 72 h of recovery. Athletes also demonstrated significant changes (p < 0.05) in muscle contractile properties, CK, and DOMS following the training program and after the recovery period. In contrast, CRP and urea remained unchanged throughout the study. Further analysis revealed that the accuracy of markers for assessment of fatigue and recovery in comparison to RSA derived from a contingency table was insufficient. Multiple regression analysis also showed no correlations between changes in RSA and any of the markers.

Conclusions

Mean changes in measures of neuromuscular function, CK and DOMS are related to HIIT induced fatigue and subsequent recovery. However, low accuracy of a single or combined use of these markers requires the verification of their applicability on an individual basis.
Introduction

High-intensity interval training (HIIT), involving short to long (~5-300 s) intensive work intervals interspersed by active or passive recovery periods, is frequently used in training programs of competitive team sport athletes. This type of intermittent training was shown to improve cardiovascular and metabolic determinants, allowing players to sustain intense phases during the game for longer durations and also to recover from it more rapidly [1, 2]. Additionally, HIIT induces similar adaptations with significant lower training volumes compared to traditional endurance training [3, 4]. This is the main rationale behind its application in team sport conditioning programs, since the complex profile of demands requires that various conditional abilities as well as technical and tactical elements need to be considered and, consequently, the timeframe to improve endurance performance is limited.

However, as a result of high metabolic and neuromuscular demands, HIIT is also accompanied with acute feelings of fatigue [5]. Howatson and Milak [6] have shown that even one single team sport specific HIIT session leads to a significant increase in muscle damage and muscle soreness in the days following the exercise bout. Although effective training programs intend functional overreaching, excessive overload with insufficient recovery should be avoided [7]. If the balance between training stress and recovery is inadequate over a prolonged period, the athlete will experience decreases in performance and a state of overtraining may develop [7]. During in-season training, the challenge for coaches and athletes is to determine the point at which HIIT may negatively affect the performance in upcoming competitions. Therefore, the routine assessment of fatigue and recovery during HIIT is important to improve individual training prescriptions and to ensure competition readiness [8].

Fatigue and recovery is characterized by a combination of several factors involving mechanisms from the central nervous system to the muscle cell itself. In this regard, a change in the players’ specific on-court performance represents the most relevant marker for differentiation between fatigued and recovered athletes. However, the majority of field test recommendations for standardized performance measurements in team sports are physically demanding and induce additional fatigue [9, 10]. Consequently, a variety of other surrogate markers (e.g., subjective, biochemical, neuromuscular, and performance markers) are frequently used in science and practice in order to track the fatigue and recovery process [9]. The daily determination of a wide range of these markers established in endurance sports (e.g., heart rate variability or several markers in the blood), however, seems to be inadequate and difficult to control under the typical team sport surrounding. Therefore, practical parameters that are determined at rest or during low metabolic and neuromuscular demands, without disturbing the training process, are preferred in team sports for the routine assessment of fatigue and recovery [11].
Tools that meet these criteria and that have been proposed in the literature are subjective markers (e.g., delayed onset muscle soreness), neuromuscular performance tests (e.g., jumps), muscle contractile markers (e.g., measured via Tensiomyography) and routine capillary blood parameters (e.g., creatinkinase) [9, 12-14]. However, there is still no consensus regarding the usefulness of these simple tests for the routine assessment of fatigue and recovery in team sport athletes during and after HIIT [7, 9, 11]. Thus, the aim of this study was to investigate the accuracy of the aforementioned markers to reflect changes in fatigue and recovery in response to a six-day HIIT program, designed to induce a temporary functional overload, as well as after 72 h of recovery in male and female team sport athletes. We hypothesized that the training program leads to relevant changes in team sport specific performance and in related variances in markers of fatigue and recovery.

Materials and Methods

Participants

A total of 22 (11 males and 11 females) healthy and well-trained team sport athletes (i.e., soccer, basketball, handball) took part in this study. The baseline physical characteristics of the athletes are shown in Table 1. The mean training frequency of the athletes was 5.7 d·week⁻¹ with a mean training volume of 2.5 h·day⁻¹. After being informed about the exercise protocols and all possible risks associated with participation in the investigation, subjects gave written consents to participate in all procedures. Normal ECG and the absence of cardiovascular, pulmonary and orthopedic diseases were confirmed in a preliminary health examination. Additionally, athletes had to meet two inclusion criteria: minimal performance in the 30-15 Intermittent Fitness Test (30-15IFT) of at least 16 km·h⁻¹ for women or 19 km·h⁻¹ for men and at least five years of specific team sport training experience. Initially, 24 athletes from different regional teams were evaluated for possible participation in the study, of which two failed to fulfill the inclusion criteria. The study was approved by the ethic committee of the Medical Faculty of the Ruhr-University Bochum and performed according to the Declaration of Helsinki.

Table 1. Baseline physical characteristics of the athletes.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Age (yrs)</th>
<th>Height (cm)</th>
<th>Body mass (kg)</th>
<th>Body fat (%)</th>
<th>VO2max (mL·min·kg⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall</td>
<td>23.0 ± 2.7</td>
<td>176.6 ± 7.6</td>
<td>69.5 ± 7.3</td>
<td>17.9 ± 5.8</td>
<td>57.6 ± 8.6</td>
</tr>
<tr>
<td>Male</td>
<td>22.9 ± 1.9</td>
<td>181.6 ± 5.3</td>
<td>73.8 ± 6.4</td>
<td>14.6 ± 3.7</td>
<td>62.9 ± 8.3</td>
</tr>
<tr>
<td>Female</td>
<td>23.0 ± 3.4</td>
<td>171.6 ± 6.0</td>
<td>65.2 ± 5.5</td>
<td>21.1 ± 5.9</td>
<td>52.2 ± 4.8</td>
</tr>
</tbody>
</table>

Parameters are shown as mean ± SD.
Experimental design

A repeated measures study was used to examine the accuracy of markers of fatigue and recovery. The investigation lasted 18 days and was conducted in the athletes’ off-season period during which no additional club training took place. Seven days prior to the HIIT-program all athletes came to the laboratory for a preliminary health examination to exclude contraindications to participation in this study (e.g., cardiovascular, pulmonary, or orthopedic diseases), to obtain data on anthropometrical characteristics and to determine VO2max. After familiarization with performance tests to minimize any learning effect, participants completed the 30-15IFT on a second preliminary examination day followed by four days of rest. Athletes were then examined at baseline (pre), after completing a six-day training program of HIIT (post1), and following a 72 h recovery period (post2), in which no training was allowed (Fig. 1).

On all testing days (pre, post1, post2), repeated sprint ability (RSA) was assessed on a nonmotorized treadmill (NMT), which was defined as an important marker of team sport specific performance and as criterion measure of fatigue and recovery. RSA test has been shown to be closely associated with competitive performance in team sport athletes [15-17] and to be highly reproducible (coefficient of variation (CV) of about 2.5% for velocity) [18, 19]. In addition, prior to the
RSA test, perceived muscle soreness, muscle contractile properties, blood parameters as well as jump and linear sprint performance were determined (in this order) as surrogate markers of fatigue and recovery pre, post₁, and post₂. All measures were taken at the same time of day for each individual on each occasion. Time between jump tests and linear sprint test as well as between linear sprint and RSA test was 30 min and 120 min, respectively. Prior to all performance tests a standardized warm-up was conducted.

Participants were instructed to maintain their normal dietary intake and to refrain from nutritional supplements and alcohol intake during the experimental period. In this regard, athletes were verbally questioned before each testing procedure as well as during the six-day training intervention to ensure that they had adhered to the dietary rules.

**Procedures**

*Incremental treadmill test.* In order to determine VO₂max, a progressive incremental exercise test on a motor driven treadmill (Ergo ELG2, Woodway GmbH, Weil am Rhein, Germany) was used. The treadmill test started with an initial velocity of 8 km·h⁻¹, increasing 2 km·h⁻¹ every 3 min with a constant incline of 0.5% until voluntary exhaustion. VO₂ was continuously analyzed using a breath-by-breath gas collection system (ZAN600USB, nSpire Health GmbH, Oberthulba, Germany). The gas calibration was completed before the test day and the volume calibration was conducted before each test following the instructions provided by the manufacturer. The highest mean value for 30 s was defined as the VO₂max.

*30-15 Intermittent Fitness Test.* The test was conducted outdoors on a tartan track and consisted of 30 s shuttle runs interspersed with 15 s passive recovery periods. Speed was set at 8 km·h⁻¹ for the first 30 s run and was increased by 0.5 km·h⁻¹ every 45 s stage thereafter. The athletes had to run back and forth between two lines set 40 m apart at a pace dictated by an acoustic signal. The test ended when a player could no longer maintain the imposed running speed or when he was unable to reach a 3 m zone around each line at the moment of the audio signal for three consecutive times. The speed of the last completed stage achieved by the participants (VIFT) was used as an inclusion criteria and to calculate the interval intensity of the HIIT protocols applied in the six-day training program as described by Buchheit [20].

*Repeated sprint ability test.* The laboratory RSA test was performed on a Woodway nonmotorized treadmill (NMT) (Force 3.0, Woodway GmbH, Weil am Rhein, Germany) pre, post₁, and post₂. The experimental set-up of the test has previously been described by Oliver et al. [18]. The RSA test consisted of six 4 s maximal sprints from a standing position with 20 s passive recovery between sprints. The peak values attained in each sprint for velocity were recorded and mean peak values for velocity (MV) were calculated. For MV, the intraclass correlation coefficient (ICC)
and the typical error (TE) were previously investigated by our research group and MV was considered to be highly reliable (unpublished results: MV (m·s⁻¹), n = 17, ICC = 0.92, TE = 0.10, CV = 1.5 %).

Jump and linear sprint tests. On each testing day (pre, post₁, and post₂), countermovement jumps (CMJ) and multiple rebound jump tests (MRJ) were performed on a contact platform (Haynl-El-ektronik GmbH, Schönebeck, Germany) with hands placed on hips. For CMJ, participants dropped down to a self-selected level before jumping maximally. Flight time was used to calculate jump height [21]. Each subject performed two maximal CMJ and the mean height was calculated. For MRJ, participants were advised to perform repeated maximum vertical jumps for 15 s with reactive landing phases and ground contact times which should be as short as possible. Flight time and contact time were used to calculate the reactive strength index (RSI) for each jump by dividing the height jumped in meters by the time on the ground in seconds [22]. Based on the RSI, the five best jumps were selected and mean RSI was calculated for further analysis. The 20-m linear sprint was completed outdoors on a tartan track and sprint times were recorded using a wireless double-photocell system (Sportronic, Winnenden-Hertmannsweiler, Germany). Each sprint was initiated without a starting signal and from an individually chosen upright standing position 50 cm behind the first photocell. Participants performed two maximal sprints interspersed by 3 min of passive recovery and the mean sprint time was calculated for further analysis. Previously measured reliability scores for jump and linear sprint tests were regarded as highly reliable (unpublished results: CMJ (cm), n = 38, ICC = 0.92, TE = 1.86, CV = 3.7 %; MRJ (RSI), n = 38, ICC = 0.91, TE = 0.13, CV = 4.0 %; 20-m linear sprint test (s), n = 22, ICC = 0.95, TE = 0.06, CV = 1.8 %).

Muscle contractile markers. For the non-invasive assessment of the contractile properties of knee extensor and flexor muscles, Tensiomyography (TMG) was used under laboratory conditions pre, post₁, and post₂. This technique produces radial displacement of the muscle belly in response to an electrical stimulus (around 100 mA) conducted through the underlying muscle tissue [13, 23]. These displacements are recorded at the skin surface using a spring loaded displacement sensor (TMG-BMC Ltd, Ljubljana, Slovenia). The sensor was positioned perpendicular to the thickest part of the muscle belly, which was established visually and through palpation during a voluntary contraction, and the self-adhesive electrodes were placed symmetrically approximately 5 cm away from the sensor. Once the exact position for the sensor and electrodes was found, it was marked with a dermatological pen and kept constant during the experimental period. Maximal radial muscle belly displacement (Dm) and contraction time between 10 and 90 % Dm (Tc) of the rectus femoris (RF) and biceps femoris (BF) were measured through TMG. Reliability scores for Dm and Tc of the RF and BF were previously examined and considered as reliable (unpublished results: RF Dm (mm), n = 20, ICC = 0.92, TE = 1.00, CV = 9.3 %; RF Tc (ms), n = 20, ICC = 0.94,
Biochemical markers. Venous blood samples were collected on each testing day (pre, post₁, and post₂; between 8 and 10 a.m., and ~2 h after the athletes took a typical breakfast) from an ante-cubital arm vein of the right arm using a 20-gauge disposable Safety-Multifly® needle (Sarstedt AG & Co, Nümbrecht, Germany) while the subject was in a supine position. Samples were collected into 7.5 mL serum gel tubes with clotting activator (Sarstedt AG & Co, Nümbrecht, Germany) and subsequently centrifuged at 3500 rpm for 15 min within 20 min after sampling. The resulting serum was separated from the other compounds, pipetted into micro tubes (Sarstedt AG & Co, Nümbrecht, Germany) and stored at -80 °C. Later, routine techniques (UniCel® DxC 600 Synchron®, Beckmann Coulter GmbH, Krefeld, Germany) were used for analysis of the concentration of creatinkinase (CK), c-reactive protein (CRP), and urea. The diagnostic laboratory used in this study held current quality assurance certification (Referenzinstitut für Bioanalytik, Bonn, Germany).

Subjective marker. Before all tests were performed on each testing day (pre, post₁, and post₂), athletes were asked to score on a visual analogue scale (VAS) the general amount of delayed onset muscle soreness (DOMS). The VAS, which has been shown to be reliable in previous research [24], consisted of a 100 mm line whose endpoints were labeled by “no pain” (left) and “unbearable pain” (right). Subjects had to draw a vertical line at a point on the line that represented their pain at the time of measurement best. The rating resulted from the distance in mm from the left border of the scale to the point marked [14].

Training program

A six-day training intervention was designed to induce a functional overload while remaining tolerable for the athletes. The training program (exercise mode, number and duration of intervals and rest, intensity) consisted of 11 training sessions with an average training duration of 35 min per session (Table 2). To calculate training intensity, participants completed the 30-15 IFT as part of the preliminary examinations. All sessions were completed outdoors on a 400 m tartan track and preceded by a standardized continuous 10 min warm-up, consisting of 40 m shuttle runs at 60-70% HRmax followed by four 40 m acceleration sprints. To ensure that the intended training intensity was maintained by the athletes, all sessions were supervised and individually calculated running distances were controlled. Additionally, training loads were determined by multiplying the numerical score of the athletes’ perception of effort, using a category-ratio RPE scale [25, 26], with the total exercise duration in min. Training loads were kept constant throughout the training period.
Table 2. Six-day high-intensity interval training program.

<table>
<thead>
<tr>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
<th>Day 4</th>
<th>Day 5</th>
<th>Day 6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Straight-line runs</td>
<td>Straight-line runs</td>
<td>Straight-line runs</td>
<td>Straight-line runs</td>
<td>Straight-line runs</td>
<td>Straight-line runs</td>
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<tr>
<td>4 x 4 min</td>
<td>7 x 2 min</td>
<td>4 x 4 min</td>
<td>4 x 4 min</td>
<td>7 x 2 min</td>
<td>4 x 4 min</td>
</tr>
<tr>
<td>a.m. (r = 3 min)</td>
<td>(r = 2 min)</td>
<td>(r = 3 min)</td>
<td>Rest</td>
<td>(r = 3 min)</td>
<td>(r = 2 min)</td>
</tr>
<tr>
<td>80% VIFT</td>
<td>85% VIFT</td>
<td>80% VIFT</td>
<td>80% VIFT</td>
<td>85% VIFT</td>
<td>80% VIFT</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
<th>Day 4</th>
<th>Day 5</th>
<th>Day 6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Straight-line sprints</td>
<td>40m-shuttle runs</td>
<td>Straight-line sprints</td>
<td>40m-shuttle runs</td>
<td>Straight-line sprints</td>
<td>40m-shuttle runs</td>
</tr>
<tr>
<td>4 x 6 x 5 s</td>
<td>2 x 12 x 30 s</td>
<td>4 x 6 x 5 s</td>
<td>2 x 12 x 30 s</td>
<td>4 x 6 x 5 s</td>
<td>2 x 12 x 30 s</td>
</tr>
<tr>
<td>p.m. (r = 25 s; R = 3 min)</td>
<td>(r = 30 s; R = 3 min)</td>
<td>(r = 25 s; R = 3 min)</td>
<td>(r = 30 s; R = 3 min)</td>
<td>(r = 25 s; R = 3 min)</td>
<td>(r = 30 s; R = 3 min)</td>
</tr>
<tr>
<td>all out</td>
<td>90% VIFT</td>
<td>all out</td>
<td>90% VIFT</td>
<td>all out</td>
<td>90% VIFT</td>
</tr>
</tbody>
</table>

VIFT: final running speed obtained in the 30-15 Intermittent Fitness Test; r: passive recovery between intervals; R: passive recovery between series; TL: training load.

Example of training program: [40m-shuttle runs, 2 x 12 x 30 s, 90% VIFT, r = 30 s, R = 3 min] means that the subject had to run two series of 12 intervals at 90% VIFT composed of 30 s passive recovery between intervals and 3 min passive recovery between series.

Example of training load calculation: [Session-RPE (9) x training duration (26 min)] = 234.

Statistical analysis

All statistical analyses were performed by using SPSS (statistical software package version 18, SPSS Inc., Chicago, IL, USA) and Excel 2010 (Microsoft Corp., Redmond, WA, USA). Data are presented as mean ± SD and were tested for normal distribution using the Shapiro-Wilk-Test. Furthermore, 95% confidence interval (CI) is given. A two-factor (time, sex) repeated measure analysis of variance (ANOVA) was used to determine differences among markers of fatigue and recovery between testing days (pre, post1, and post2) as well as between male and female team sport athletes. Bonferroni post-hoc tests were used when the ANOVA main effect was significant. Those markers which were not normally distributed (CK, CRP) were tested using Friedman test. Wilcoxon tests were used when the Friedman test was significant. To allow a better interpretation of the results, the effect size Cohen’s d [27] (defined as [difference between the means]/SD) was calculated for all parameters between testing days. The thresholds for small, moderate, and large effects were 0.20, 0.50, and 0.80, respectively [27].
A 2 x 2 contingency table was used to evaluate the accuracy of the markers for the assessment of fatigue and recovery in comparison to the criterion measure (i.e., RSA). The table was composed of horizontal lines to indicate the presence or absence of fatigue (in accordance with changes in surrogate markers) and vertical lines to indicate the “true” condition of an athlete according to the criterion measure of fatigue. Diagnostic effectiveness (proportion of athletes correctly categorized by the surrogate marker), misclassification rate (proportion of athletes, who were incorrectly classified by the surrogate marker) and Youden’s index (ranges from 0 for a poor accuracy to 1.0 for an excellent accuracy of the surrogate marker) were calculated from the constructed table [28]. Finally, multiple regression analysis was used to assess relationships between changes in surrogate markers and criterion measure of fatigue and recovery. For all statistical analyses, level of significance was set at $p < 0.05$.

Results

No significant time x sex interaction ($p = 0.566$) but a significant main effect for time ($p = 0.010$) was found for RSA test performance. MV was significantly lower following the six-day training intervention (post$_1$: 4.84 ± 0.56 m·s$^{-1}$) than at baseline (pre: 5.02 ± 0.52 m·s$^{-1}$) or after recovery (post$_2$: 4.97 ± 0.56 m·s$^{-1}$). The respective changes were -0.18 ± 0.13 m·s$^{-1}$ ($p = 0.001$; effect size $=-1.51$) from pre to post$_1$ and 0.12 ± 0.26 m·s$^{-1}$ ($p = 0.003$; effect size $=0.53$) from post$_1$ to post$_2$.

Differentiated by sex, markers of fatigue and recovery are illustrated in Fig. 2, Fig. 3, and Fig 4. There were no significant time x sex interactions with respect to any of the determined markers. However, a significant main effect for time was found for CMJ, MRJ, and 20-m sprint performance, as well as for contraction time of the RF and BF, CK, CRP, and DOMS. For CMJ and MRJ performance, a significant decline and a return to baseline level after 72 h of recovery could be observed (Table 3). In addition, athletes demonstrated a significant increase in CK and DOMS following the training program and a significant decrease after the recovery period (Table 3). The HIIT-microcycle also induced a significant increase in 20-m sprint time and contraction time of the RF and BF at post1 compared to baseline values. However, these increases were not reversible between post1 and post2 (Table 3). Dm of the RF and BF, as well as CRP, and urea were not different at post1 and post2 compared to baseline values (Table 3).
Fig. 2. Mean (± SD) countermovement jump (CMJ) height (A), multiple rebound jumps (MRJ) performance (B) and 20-m sprint time (C) in males and females at baseline (pre), after a six-day high-intensity interval training program (post1), and following 72 h of recovery (post2). RSI = reactive strength index. *Significant difference compared to pre ($p < 0.05$). #Significant difference compared to post1 ($p < 0.05$).
Fig. 3. Mean (± SD) of the contraction time (Tc) and maximal radial muscle displacement (Dm) of the rectus femoris (RF) and biceps femoris (BF) in males and females at baseline (pre), after a six-day high-intensity interval training program (post₁), and following 72 h of recovery (post₂). *Significant difference compared to pre (p < 0.05). #Significant difference compared to post₁ (p < 0.05).
Fig. 4. Mean (± SD) of the serum concentration of c-reactive protein (CRP), urea, and creatinkinase (CK) as well as of the rating of delayed onset muscle soreness (DOMS) in males and females at baseline (pre), after a six-day high-intensity interval training program (post₁), and following 72 h of recovery (post₂). *Significant difference compared to pre (p < 0.05). #Significant difference compared to post₁ (p < 0.05).
Table 3. Markers of fatigue and recovery at baseline (pre), after a six-day high-intensity interval training program (post₁), and following 72 h of recovery (post₂) as well as percentage changes of performance and muscle contractile markers between testing days.

<table>
<thead>
<tr>
<th>Performance markers</th>
<th>pre</th>
<th>post₁</th>
<th>post₂</th>
<th>pre-post₁</th>
<th>post₁-post₂</th>
<th>pre-post₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>CMJ (cm)</td>
<td>36.4 ± 6.8 (33.4–36.4)</td>
<td>33.3 ± 6.6* (30.4–36.2)</td>
<td>34.8 ± 6.9* (31.8–37.9)</td>
<td>&lt; 0.001</td>
<td>-8.4 ± 2.9</td>
<td>-3.35 ± 1.3</td>
</tr>
<tr>
<td>MRJ (RSI)</td>
<td>1.79 ± 0.28 (1.66–1.91)</td>
<td>1.48 ± 0.30* (1.34–1.61)</td>
<td>1.60 ± 0.35# (1.44–1.75)</td>
<td>&lt; 0.001</td>
<td>-17.4 ± 4.5</td>
<td>-1.60 ± 0.63</td>
</tr>
<tr>
<td>20m Sprint (s)</td>
<td>3.28 ± 0.24 (3.18–3.39)</td>
<td>3.40 ± 0.30* (3.26–3.53)</td>
<td>3.35 ± 0.24* (3.25–3.46)</td>
<td>&lt; 0.001</td>
<td>3.4 ± 1.8</td>
<td>-0.81 ± 0.37</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Muscle contractile markers</th>
<th>pre</th>
<th>post₁</th>
<th>post₂</th>
<th>pre-post₁</th>
<th>post₁-post₂</th>
<th>pre-post₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>RF Tc (ms)</td>
<td>29.0 ± 3.8 (27.3–30.7)</td>
<td>31.7 ± 4.8* (29.6–33.8)</td>
<td>31.2 ± 4.6* (29.2–33.2)</td>
<td>&lt; 0.002</td>
<td>9.9 ± 5.9</td>
<td>-1.7 ± 4.1</td>
</tr>
<tr>
<td>RF Dm (mm)</td>
<td>8.6 ± 2.1 (7.7–9.6)</td>
<td>8.3 ± 2.2 (7.3–9.3)</td>
<td>8.4 ± 2.1 (7.5–9.4)</td>
<td>&lt; 0.611</td>
<td>-1.7 ± 10.3</td>
<td>2.1 ± 6.2</td>
</tr>
<tr>
<td>BF Tc (ms)</td>
<td>37.7 ± 10.7 (33.0–42.5)</td>
<td>44.9 ± 12.9* (39.2–50.6)</td>
<td>44.5 ± 14.6* (38.0–51.0)</td>
<td>&lt; 0.042</td>
<td>28.7 ± 24.9</td>
<td>-8.1 ± 21.5</td>
</tr>
<tr>
<td>BF Dm (mm)</td>
<td>8.5 ± 3.3 (7.1–10.0)</td>
<td>7.7 ± 2.9 (6.4–8.9)</td>
<td>7.4 ± 3.2 (6.0–8.9)</td>
<td>&lt; 0.097</td>
<td>-6.8 ± 12.2</td>
<td>-3.3 ± 14.1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Biochemical markers</th>
<th>pre</th>
<th>post₁</th>
<th>post₂</th>
<th>pre-post₁</th>
<th>post₁-post₂</th>
<th>pre-post₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>CK (U·L⁻¹)</td>
<td>147 ± 51 (125–170)</td>
<td>1010 ± 887* (617–1403)</td>
<td>269 ± 134# (210–328)</td>
<td>&lt; 0.001</td>
<td>-599 ± 9.6</td>
<td>-0.87</td>
</tr>
<tr>
<td>CRP (mg L⁻¹)</td>
<td>1.52 ± 2.46 (0.33–2.70)</td>
<td>2.23 ± 3.08 (0.74–3.71)</td>
<td>1.27 ± 1.84# (0.38–2.16)</td>
<td>&lt; 0.020</td>
<td>-0.33 ± 0.66</td>
<td>-0.20</td>
</tr>
<tr>
<td>UREA (mg dL⁻¹)</td>
<td>29.6 ± 7.2 (26.1–33.0)</td>
<td>30.9 ± 9.4 (26.4–35.5)</td>
<td>28.2 ± 5.9 (25.4–31.1)</td>
<td>&lt; 0.080</td>
<td>-0.25 ± 0.46</td>
<td>-0.26</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Subjective markers</th>
<th>pre</th>
<th>post₁</th>
<th>post₂</th>
<th>pre-post₁</th>
<th>post₁-post₂</th>
<th>pre-post₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>DOMS (mm)</td>
<td>0.2 ± 0.1 (0.1–0.3)</td>
<td>2.7 ± 1.8* (2.0–3.5)</td>
<td>0.9 ± 1.2# (0.3–1.4)</td>
<td>&lt; 0.001</td>
<td>-1.50 ± 1.44</td>
<td>-0.57</td>
</tr>
</tbody>
</table>

Parameters are shown as mean ± SD (95% confidence interval).

Q: 95% confidence interval; d: Cohen’s d effect size; CMJ: countermovement jump; MRJ: multiple rebound jumps; RSI: reactive strength index; RF: rectus femoris; BF: biceps femoris; Tc: contraction time; Dm: muscle belly displacement; CK: creatine kinase; CRP: C-reactive protein; DOMS: delayed onset muscle soreness.

*Significant difference compared to pre.

#Significant difference compared to post₁.
Diagnostic effectiveness, misclassification rate and Youden’s index for surrogate markers of fatigue and recovery are shown in Table 4. None of the surrogate markers showed sufficient accuracy to discriminate athletes in a fatigued or recovered state in relation to RSA. Multiple regression analysis also revealed no significant correlations (p > 0.05) between changes in RSA and any of the surrogate markers.

Table 4. Accuracy of markers of fatigue and recovery in relation to the criterion measure.

<table>
<thead>
<tr>
<th>Performance markers</th>
<th>Diagnostic effectiveness (%)</th>
<th>Misclassification rate (%)</th>
<th>Youden’s Index</th>
<th>Δpre-post₁</th>
<th>Δpost₁-post₂</th>
<th>Δpre-post₁</th>
<th>Δpre-post₁</th>
<th>Δpost₁-post₂</th>
<th>Δpost₁-post₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>CMJ (cm)</td>
<td>63.6</td>
<td>60.0</td>
<td>36.4</td>
<td>40.0</td>
<td>0.01</td>
<td>0.20</td>
<td></td>
<td></td>
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<tr>
<td>MRJ (RSI)</td>
<td>68.2</td>
<td>60.0</td>
<td>31.8</td>
<td>40.0</td>
<td>0.08</td>
<td>0.20</td>
<td></td>
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<tr>
<td>20-m Sprint (s)</td>
<td>77.3</td>
<td>33.3</td>
<td>22.7</td>
<td>66.7</td>
<td>0.51</td>
<td>0.34</td>
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<tr>
<td>Muscle contractile markers</td>
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<tr>
<td>RF Tc (ms)</td>
<td>68.2</td>
<td>40.0</td>
<td>31.8</td>
<td>60.0</td>
<td>0.38</td>
<td>0.21</td>
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<tr>
<td>RF Dm (mm)</td>
<td>50.0</td>
<td>66.7</td>
<td>50.0</td>
<td>33.3</td>
<td>0.04</td>
<td>0.30</td>
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<tr>
<td>BF Tc (ms)</td>
<td>54.5</td>
<td>40.0</td>
<td>45.5</td>
<td>60.0</td>
<td>0.03</td>
<td>0.23</td>
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<tr>
<td>BF Dm (mm)</td>
<td>50.0</td>
<td>60.0</td>
<td>50.0</td>
<td>40.0</td>
<td>0.11</td>
<td>0.20</td>
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<tr>
<td>Biochemical markers</td>
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<tr>
<td>CK (U·L⁻¹)</td>
<td>50.0</td>
<td>53.3</td>
<td>50.0</td>
<td>46.7</td>
<td>0.11</td>
<td>0.13</td>
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<tr>
<td>CRP (mg·L⁻¹)</td>
<td>31.8</td>
<td>46.7</td>
<td>68.2</td>
<td>53.3</td>
<td>0.07</td>
<td>0.08</td>
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<tr>
<td>UREA (mg·dL⁻¹)</td>
<td>30.0</td>
<td>46.7</td>
<td>70.0</td>
<td>53.3</td>
<td>0.10</td>
<td>0.00</td>
<td></td>
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<tr>
<td>Subjective markers</td>
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<td></td>
</tr>
<tr>
<td>DOMS (mm)</td>
<td>45.5</td>
<td>60.0</td>
<td>54.5</td>
<td>40.0</td>
<td>0.05</td>
<td>0.14</td>
<td></td>
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</tbody>
</table>

CMJ: countermovement jump; MRJ: multiple rebound jumps; RSI: reactive strength index; RF: rectus femoris; BF: biceps femoris; Tc: contraction time; Dm: muscle belly displacement; CK creatinkinase; CRP: C-reactive protein; DOMS: delayed onset muscle soreness.

Discussion

The purpose of the present study was to investigate the accuracy of selected markers to reflect changes in fatigue and recovery in male and female team sport athletes during and after HIIT. The main finding of this study was that a six-day HIIT program induced significant changes in RSA, showing a temporary decline and a return to baseline level after 72 h of recovery. The decrease in RSA indicates that the training program induced a temporary state of fatigue. However, regular RSA testing for a routine assessment of fatigue and recovery may be unduly fatiguing and impractical for most athletes [29]. In this regard, the present study demonstrated that CMJ, MRJ, TMG Tc, CK and DOMS are potential markers of higher practicability and less demanding. This was evident in significant changes in these markers following the training period and after 72 h of recovery. However, due to an insufficient accuracy of these markers in differentiating between fatigued and recovered athletes, their responses to HIIT and their associations
with fatigue and recovery appear to be highly individual. Since changes in markers of fatigue and recovery of males and females tended to be the same, these findings apply equally for both sexes.

Monitoring fatigue and recovery through measures of jump or sprint performance is recently utilized in the team sport environment due to its simplicity of administration, the minimal amount of additional fatigue induced, and its high reproducibility and validity [8, 9]. Therefore, we used the CMJ, the MRJ, and the 20-m linear sprint to monitor changes in the athlete’s neuromuscular function of the lower limbs during the six-day training intervention [22, 30]. In this study jump performance (i.e., jump height and jump efficiency) followed the changes in repeated sprint ability with a decrease in performance after the training period (CMJ: -8.4 ± 6.6%; MRJ: -17.4 ± 10.2%) and an increase of performance following the recovery period (CMJ: 4.9 ± 8.3%; MRJ: 8.5 ± 13.9%). Since the CV of the CMJ and MRJ performance was 3.7% and 4.0% respectively, the magnitude of changes can be considered to be of practical relevance. Linear sprint performance (CV = 1.8 %) also showed a practically relevant decrease following the six-day training program of HIIT (3.4 ± 4.1%), but only tended to increase following the recovery period (-1.1 ± 3.4%).

Failure in the neuromuscular system responsible for altered performance can be explained by a combination of central and peripheral factors involving mechanisms from the central nervous system (e.g., impaired activation or reduced motivation) to exercise-related changes within the muscle fibers itself [9, 31, 32]. However, a decline in performance following exercise-induced fatigue has been demonstrated to be located peripherally (i.e., structural damage of muscle fibers, excitation-contraction coupling failure, redistribution of sarcomere length, impaired metabolism) rather than centrally [33, 34]. Since HIIT has the potential to induce muscle damage [33], it appears that the decreases in vertical jump height, jump efficiency (i.e., reactive strength index), and sprint performance may be related to repeated structural damage and inflammatory response of the muscle fibers caused by the HIIT program [29, 34]. It was shown that when muscle damage was induced through intense exercises, there were prolonged decreases in maximal force, ground reaction force, stretch-reflex sensitivity, muscle joint stiffness regulation and, thus, a reduction in jump and sprint performance [33]. Since the jump performance almost reached baseline levels and sprint performance showed a trend to increase following 72 h of recovery, these findings suggest that the CMJ, MRJ and 20-m sprint test may be potential tools to measure both fatigued and recovered neuromuscular function of team sport athletes following HIIT.

In addition to performance tests, measurements of selected blood markers under standardized conditions are proposed to monitor fatigued and recovered conditions [11]. In the practical team sport surrounding, routine blood parameters such as CK, CRP, and urea collected via capillary blood samples, are popular measures due to the simplicity of sample collection and analysis [7, 8, 12, 35]. In this study, CK reacted to the HIIT program, showing an average elevation of > 1000
U·L\(^{-1}\) after the training period and a decrease to almost baseline levels following the recovery period. However, no changes in CRP und urea could be observed between baseline, post\(_1\), and post\(_2\).

Serum CK activity mirrors the mechanical-muscular strain of the training since CK leak into the plasma from skeletal muscle fibers when they are damaged, including membrane damage and myofibrillar disruptions characterized by myofilament disorganization and loss of Z-disk integrity [9, 11]. Therefore, the elevated CK activity determined at post, appears to support the explanation that damaged muscle fibers were partially responsible for the decline in performance. Similar to the present results, various studies with team sport athletes reported increased CK concentrations following intensified training or competition periods [12, 35-37]. The most likely explanation for the extremely high CK levels measured in this study was the characteristic of HIIT with its accelerations and decelerations as well as the changes of direction leading to high eccentric biomechanical strain on the working muscles, which in turn causes microinjuries of the musculoskeletal system and perceived muscle soreness [12, 33]. In this study, muscle soreness, which was measured subjectively by a VAS, followed the time course of CK activity (Table 3). DOMS increased following the training period and decreased after 72 h of recovery. Therefore, both the objective CK and subjective DOMS measures seemed to have the potential to identify HIIT-induced muscle damage associated with the fatigue and recovery observed in this study’s team sport athletes.

In this context, however, the high variability of measure of CK activity must also be taken into account [8]. Some athletes are non-responders due to a lower permeability of muscle cell membranes and only show small increases in CK activity [11]. Conversely, athletes with high percentages of fast twitch muscle fibers might tend to produce higher CK values [12]. Furthermore, sex could affect the magnitude of CK activity, which is due to a potentially higher CK content of men’s muscle than that of women’s muscle [9, 12, 38]. This assumption is supported by our data, since the mean CK concentration at post, was 64.8% higher in the male compared to female participants (Fig. 3). Therefore, athletes’ individual physical characteristics should be considered when using CK as an indicator of fatigue and recovery. One should also pay attention when solely using DOMS as a marker of fatigue and recovery. Since muscle function is impaired before soreness arises, and functional impairment may also persist when soreness has dissipated, this could lead to problems in an applied environment [33]. If solely the dissipation of muscle soreness is used as a signal to resume regular training, muscle function can be still in a weakened state and the risk of injury would be increased.

Since subsequent muscle damage is also linked to local inflammatory processes [9], the use of CRP may provide important additional information on the athlete’s status. However, despite an
increase in CK activity in this study, no relevant changes in CRP could be determined following the HIIT-program (Table 3). In this context, Singh et al. [39] compared the effects of intermittent running, either with or without body ‘contact’, on muscle damage and inflammatory response. They demonstrated that both ‘contact’ and ‘non-contact’ training resulted in elevated serum CK, while CRP only increased following training with body ‘contact’. Since the addition of tackles to intermittent training further increased muscle damage following exercise, one can speculate that a certain degree of muscle damage requires ‘contact’ to significantly alter serum concentration of CRP. Based on the present results and due to the fact that potential interferences with inflammation are not directly related to muscle damage, it appears that CRP may not be a useful and specific enough marker for monitoring fatigue and recovery following HIIT.

This is also valid for urea, since serum concentrations were not altered at post1 and post2 compared to baseline values. Increased serum concentration of urea is a marker of enhanced protein catabolism and stimulated gluconeogenesis that results from high training volumes and increased energy consumption [12]. Since training volume during the HIIT-period was rather low (35 min per HIIT session; Table 2), no changes in urea and, thus, in the ‘anabolic-catabolic balance’ could be observed. This is in line with the findings by Coutts et al., [35] who reported unaltered urea serum concentrations following intensified training in rugby players.

Recent articles also recommend measures of muscle contractile properties as an effective method for detecting fatigue and recovery in athletes. In this context, TMG was introduced as an involuntary and non-invasive method to measure muscle contractile characteristics (i.e., Tc which is related to the speed of force generation, and Dm, which is representative of muscle tone and contractile force) [13]. Several studies have highlighted its usefulness for practitioners and researchers in detecting muscle damage and its recovery following various forms of exercises (i.e., eccentric exercise, endurance exercise, soccer) [13, 40-42]. For HIIT, the muscles affected most will be the extensor muscles of the knee joint (in the landing and take-off stages) and their antagonist muscles (traction in rear foot and leg recovery) [40]. Therefore, the muscle contractile characteristics of the RF and BF were measured through TMG in this study. Tc observed for both muscles significantly increased after the six-day training program and showed a trend for a decrease between post1 and post2. Dm was unaltered during all testing days.

Decreased Dm and increased Tc have been explained by a reduced efficiency of the excitation-contraction coupling, impairment in membrane conducting properties, and cellular structures destruction (i.e., peripheral fatigue) [42]. In this context, previous studies were able to demonstrate a decline in Dm and an increase in Tc when exercise-induced muscle damage (e.g., elevated CK activity and muscle soreness) was present [13, 42]. Since CK activity and DOMS were increased following the six-day HIIT-period, it can be concluded that Dm measured via TMG cannot be
considered as a useful marker for monitoring fatigue and recovery following HIIT. On the other hand, due to an increase at post$_1$ and a trend for a decrease at post$_2$, Tc of the RF and BF may be a potential marker for monitoring fatigue and recovery.

As highlighted in the previous sections, measures of neuromuscular function, CK and DOMS are potentially useful markers for monitoring of team sport athletes during intensive training cycles. However, in relation to measures of sport-specific performance (i.e., RSA), which is demonstrably the most valid method for the assessment of fatigue and recovery, [8] none of the surrogate markers showed the ability to completely discriminate between fatigued and recovered athletes. Additionally, multiple regression analyses revealed that there were no relationships between changes in RSA and any of the surrogate markers. These findings indicate that responses of markers of fatigue and recovery to a given training stimulus are highly individual and variable, as already emphasized by Nèdèlec et al. [9] and Halson [8]. Additionally, Andersson et al. [37] showed, that the time course of the fatigue and recovery pattern differs significantly between various neuromuscular and biochemical markers. They demonstrated that CMJ performance, CK activity and muscle soreness were still changed 74 h following a football match, whereas sprint performance returned to baseline level already 5 h after the match. This could be a further explanation for the weak relationships between changes of surrogate markers and the criterion measure of fatigue. Consequently, accuracy of a single or combined use of CMJ, MRJ, 20-m sprint test, Tc, CK, and DOMS for the routine assessment of fatigue and recovery and their associations with sport-specific performance needs to be identified in practice for each athlete on an individual and longitudinal basis.

**Study limitations**

First, although high VO$_{2\text{max}}$ values were measured among the participants and most players were members of regional representative teams, the question remains whether the present results can be transferred to professional team sports at the international level. Effects might have been different with a group of high-level athletes. However, we have consciously refrained from recruiting elite players for this standardized research approach due to the reluctance of such populations to deviate from their normal training routine. Second, there was no control group to provide a baseline during the experimental period. In this regard, however, we have stated reliability data to indicate practically relevant changes in markers of fatigue and recovery. Third, the selection of markers that were evaluated in the present study might be considered a further limitation. There are especially some psychological markers (e.g., Recovery-Stress Questionnaire for Athletes [43]) that have been proposed in the literature as instruments to track the fatigue and recovery process and that have not been evaluated in the current investigation. However, the present study
was not designed to analyze the highest possible number of markers of fatigue and recovery, but
to evaluate a well-founded selection of practical tests that can be easily applied in team sports.

Conclusions

The challenge for coaches and athletes is to determine the point at which intensive demands in
training and competition lead to non-functional overreaching and may negatively affect the perfor-
"mance in upcoming competitions [8]. Therefore, routine assessment of fatigue and recovery is
of importance to improve individual training prescription and to ensure competition readiness. To
estimate changes in neuromuscular function following HIIT regardless of sex, this study was able
to show that the power ability and reactive strength (i.e., CMJ, MRJ, 20-m sprint) in the lower
body as well as Tc of the RF and BF are potentially useful markers.

However, in an applied environment, individual athletes respond differently to a given training
stimulus, evidenced by the insufficient accuracy of the markers for monitoring fatigue and recov-
er-y in relation to the criterion measure. Therefore, surrogate markers should be assessed regu-
larly in practice and with enough frequency to give the desired information to the athlete or coach.
In this context, a possible recommendation for professional teams is to provide a fixed installation
of a contact platform at the training ground to incorporate jump performance measurements as a
daily routine. Also subjective assessment of DOMS using a visual analogue scale can be consid-
ered as a potential tool to identify team sport athletes who are susceptible to non-functional over-
load. In addition, CK as a routine blood marker may help to monitor the mechanical-muscular
strain of HIIT. However, neither marker alone, nor specific group of markers significantly corre-
lated with the criterion measure of fatigue. Therefore, a combination of the aforementioned mark-
ers should be used in practice in order to take into consideration all potential mechanisms that
contribute to fatigue.

References

1. Cicioni-Kolsky D, Lorenzen C, Williams MD, Kemp JG. Endurance and sprint benefits of


Sprint interval and traditional endurance training induce similar improvements in peripheral
arterial stiffness and flow-mediated dilation in healthy humans. Am J Physiol Regul Integr


4.3 Publikation 3

Effect of repeated active recovery during a high-intensity interval training shock microcycle on markers of fatigue


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Abstract

Purpose

To investigate the effect of repeated use of active recovery during a four-day shock microcycle with seven high-intensity interval training (HIT) sessions on markers of fatigue.

Methods

Eight elite male junior tennis players (age: 15.1 ± 1.4 years) with an international ranking between 59 and 907 (International Tennis Federation) participated in this study. After each training session, the players completed 15 min of either moderate jogging (active recovery, ACT) or passive recovery (PAS) with a crossover design, which was interrupted by a four-month washout period. Countermovement jump (CMJ) height, serum concentration of creatine kinase (CK), delayed onset muscle soreness (DOMS), and perceived recovery and stress (Short Recovery and Stress Scale) were measured 24 h before and 24 h after the training program.

Results

The HIT shock microcycle induced a large decrease in CMJ performance (ACT: Effect Size [ES] = -1.39, p < 0.05; PAS: ES = -1.42, p < 0.05) and perceived recovery (ACT: ES = -1.79, p < 0.05; PAS: ES = -2.39, p < 0.05), as well as a moderate to large increase in CK levels (ACT: ES = 0.76, p > 0.05; PAS: ES = 0.81, p > 0.05), DOMS (ACT: ES = 2.02, p < 0.05; PAS: ES = 2.17, p < 0.05), and perceived stress (ACT: ES = 1.98, p < 0.05; PAS: ES = 3.06, p < 0.05), compared to the values before the intervention. However, no significant recovery intervention × time interactions or meaningful differences in changes were noted in any of the markers between ACT and PAS.

Conclusions

Repeated use of individualized ACT, consisting of 15 min of moderate jogging, after finishing each training session during a HIT shock microcycle did not affect exercise-induced fatigue.
Introduction

High-intensity interval training (HIT) is frequently used in training programs for team or racket sports to enhance the aerobic fitness of the athletes. The timeframe to improve endurance performance in these sports, however, is limited, since the complex profile of the demands on the athletes requires the integration of various conditional abilities as well as technical and tactical elements into the training. Thus, so-called shock microcycles with up to ten highly concentrated specialized training sessions have been shown to be practically relevant for team or racket sports, since this type of block periodization provides a time-efficient method for improving aerobic capacity.\(^1\) The rationale underlying the use of these training blocks is that potential interference effects of non-compatible workloads can be prevented. In addition, insufficient training stimuli for highly trained athletes can possibly be avoided.\(^2\)

However, because of the high metabolic and neuromuscular demands, HIT is initially accompanied with a disturbance in homeostasis.\(^3\) Even one single HIT session can lead to a significant increase in muscle damage and decrease in performance during the days following the session.\(^4\) Thus, it can be assumed that symptoms of fatigue potentially accumulate during a shock microcycle, which may result in the athlete being unable to train at the predetermined intensity or complete the required load at the next training session.\(^5\) Therefore, post-training recovery is recommended during intense training programs in order to limit the severity of fatigue and/or speed recovery from fatigue.\(^6\) Consequently, appropriate strategies that optimize recovery may help athletes to reduce the decrements in physical performance after HIT, and they may hence benefit from subsequent training and athletic performance.

In daily practice among athletes, active recovery strategies are commonly used immediately after training or competition, with the aim of accelerating the restoration of performance.\(^5,7,8\) Active recovery (ACT) usually consists of aerobic-type whole-body activities (e.g., running, biking, or swimming), performed at loads between 30% and 60% of the individual maximal oxygen consumption and lasting for at least 15 min.\(^7\) Several studies have reported that maintaining submaximal activity after exercise enhances the removal of circulating lactate, due to an increased oxidization of the lactic acid by the skeletal muscles working at low intensities.\(^9,10\) However, even with a severe exercise-induced accumulation of circulating lactate, a clear link between lactic acid and skeletal muscle fatigue is missing.\(^11\) In addition, blood lactate has a half-life of approximately 15 min during passive recovery, and returns to resting levels at 90 min after intensive exercise. This is usually shorter than the timeframe between successive training sessions or competitions in elite team or racket sport athletes.\(^5\) Thus, accelerated lactate removal does not appear to be a valid indicator of the quality of ACT in these kinds of disciplines.
A study by Gill et al.\textsuperscript{12} demonstrated that post-competition ACT enhanced creatine kinase clearance to a greater extent than passive recovery (PAS). Other studies showed that ACT appeared to accelerate the restoration of performance between two HIT sessions,\textsuperscript{6} and provided similar acute relief of muscle soreness, as compared with that observed following massage.\textsuperscript{13} In this context, an analgesic effect and an increase in blood flow are discussed as potential mechanisms related to ACT that contribute to an accelerated recovery. On the other hand, Andersson et al.\textsuperscript{14} found that ACT had no effects on the magnitude of neuromuscular or biochemical changes in response to a soccer match compared to PAS. In addition, ACT seems to impair glycogen synthesis.\textsuperscript{7} Overall, the evidence that ACT enhances recovery is inconsistent and limited. However, investigation of the efficacy of ACT has mainly been based on its effect on the recovery process following a single training session, whereas research on the efficacy of the repeated use of ACT during intense training or competition cycles is lacking. Therefore, the purpose of the current study was to compare the effects of PAS and ACT during a shock microcycle of HIT on markers of performance and fatigue. We hypothesized, first, that the four-day HIT period would lead to an acute reduction of physical capacity as well as an increase in fatigue and soreness. Second, we believed that the use of ACT would promote recovery more effectively than PAS during the training program and thus limit the severity of fatigue.

**Methods**

**Subjects**

Eight competitive male junior tennis players (age: $15.1 \pm 1.4$ years; body mass: $69.0 \pm 7.5$ kg; height: $180 \pm 6.8$ cm) with a national ranking between 1 and 10 (German Tennis Federation) and an international ranking between 59 and 907 (International Tennis Federation) participated in this study. After being informed about the exercise protocols and all the possible risks associated with participation in the investigation, the players and their parents provided written consent to participate in all procedures. Normal electrocardiography findings, as well as the absence of cardiovascular, pulmonary, and orthopedic diseases were confirmed during a preliminary health examination. The study was approved by the ethics committee of the Medical Faculty of the Ruhr-University Bochum and was performed according to the guidelines of the Declaration of Helsinki.

**Experimental Design**

A cross-over study design was used to investigate the effectiveness of ACT during a HIT shock microcycle. Athletes participated in two four-day training periods, which were separated by a four-month washout period. At 72 h prior to the HIT program, all players visited the laboratory for a preliminary health examination, to provide data on anthropometrical characteristics, and to complete the 30-15 Intermittent Fitness Test (30-15IFT). Countermovement jump (CMJ) performance,
creatine kinase (CK) activity, delayed onset muscle soreness (DOMS), as well as perceived recovery and stress were then measured 24 h prior to the microcycle (pre) as well as 24 h after completing the training program (post). During the microcycles and immediately after each training session, players commenced either ACT (shuttle runs at an individually calculated intensity) or PAS (in a seated position) for 15 min. For the assignment to one of two groups, athletes were matched according to their age, peak height velocity, and maximum speed in the 30-15IFT \((V_{IFT})\).

The first group performed ACT during the first HIT period, whereas the other group used PAS. During the second HIT shock microcycle experimental conditions were interchanged.

Food intake was partially standardized throughout the two experimental periods. Each day players had breakfast, lunch and dinner all together and the menu was identical during both microcycles. Furthermore, athletes were instructed to maintain their normal fluid intake and to refrain from nutritional supplements and alcohol intake. In this regard, athletes were verbally questioned daily to ensure that they had adhered to the dietary rules.

**Procedures**

**30-15 Intermittent Fitness Test:** The test was conducted in a multipurpose sports hall on a combined elastic flooring system with a PVC surface, and consisted of 30-s shuttle runs interspersed with 15-s passive recovery periods. The speed was set at 8 km·h\(^{-1}\) for the first 30-s run and was increased by 0.5 km·h\(^{-1}\) at every 45-s stage thereafter. The players had to run back and forth between two lines, set 40 m apart, at a pace dictated by an acoustic signal. The test ended when a player was unable to reach a 3 m zone around each line at the moment of the audio signal for three consecutive times. The speed of the last completed stage that was reached by the athlete \((V_{IFT})\) was used as a criterion to match the intervention groups, to calculate the interval intensity of the HIT protocol, and to define the running intensity for ACT.\(^{15}\)

**Jump Performance:** Following a 5-min standardized warm-up, CMJ were performed on a contact platform (Haynl-Elektronik GmbH, Schönebeck, Germany) with the hands placed on the hips. For CMJ, players dropped down to a self-selected level, before jumping to the maximum height. Flight time was used to calculate jump height. Each subject performed three maximal CMJ, and the mean height was calculated. The previously measured reliability scores for the CMJ test were regarded as highly reliable (unpublished results: CMJ (cm), \(n = 38\), ICC = 0.92, \(TE = 1.86\), CV = 3.7 %).

**Serum concentration of creatine kinase:** Venous blood samples were collected (between 8 am and 10 am) from an antecubital arm vein using a 20-gauge disposable Safety-Multifly® needle (Sarstedt AG & Co, Nümbrecht, Germany). Samples were collected into 7.5 mL serum gel tubes (Sarstedt AG & Co, Nümbrecht, Germany), and subsequently centrifuged at 3500 rpm for 15 min.
The resulting serum was separated from the other compounds, pipetted into micro tubes (Sarstedt AG & Co, Nümbrecht, Germany), and stored at -80°C. Subsequently, routine techniques (UniCel® DxC 600 Synchron®, Beckmann Coulter GmbH, Krefeld, Germany) were used for analysis of the concentration of CK.

**Blood lactate concentration:** Capillary blood samples were obtained from the hyperemized earlobe throughout each training session (i.e., before the first interval of the first series, immediately after the last interval of the final series, and immediately at the end of the recovery intervention); these were analyzed for blood lactate concentration (La). Blood samples were taken with 20-µl capillaries, hemolyzed in 1-ml micro test tubes, and analyzed by using enzymatic amperometry with the Biosen S-Line Sport (EKF-Diagnostik GmbH, Magdeburg, Germany).

**Delayed onset muscle soreness:** Muscle soreness was assessed using a visual analogue scale (VAS). The VAS consisted of a 100 mm line, whose endpoints were labelled as “no pain” (left) and “unbearable pain” (right). Subjects had to draw a vertical line at a point on the line that best represented their pain at the time of the measurement. The score was determined from the distance in mm from the left border of the scale to the point marked.

**Perceived recovery and stress:** Perceived recovery and stress was assessed using the Short Recovery and Stress Scale (SRSS). Athletes were requested to provide responses to eight items on a 0 (does not apply at all) to 6 (fully applies) rating scale. Numbers 1 to 5 on this scale were undefined and were used to delineate the degrees of perceived recovery and stress between the two ends of the scale. The items were “physical performance capability” (PPC), “mental performance capability” (MPC), “emotional balance” (EB), “overall recovery” (OR), “muscular stress” (MS), “lack of activation” (LA), “negative emotional state” (NES), and “overall stress” (OS). Scores for internal consistencies of the SRSS were previously examined among elite athletes and considered to be sufficient (n = 574; α = 0.76).

**Training Program**

During the four-day microcycle, the players completed seven HIT sessions (Figure 1). At each session, the athletes performed three series involving eight intervals, with 20 s passive recovery between the intervals and 6 min passive recovery between each series. Each interval was 15 s in duration and consisted of 20-m shuttle runs at 90% \( V_{IFT} \). All sessions were completed in the same sports hall as the 30-15\( V_{IFT} \), and were preceded by a standardized continuous 10-min warm-up. To ensure that the intended training intensity was maintained by the athletes, all sessions were supervised and the individually calculated running distances were controlled. Finally, the athlete’s perception of the overall difficulty of each training bout was recorded 30 min after the completion of an exercise, using a category-ratio scale.
Recovery Intervention

ACT included 15-min shuttle runs at 40% $V_{\text{IFT}}$ that were started within 5 min after completing each training session (Figure 1). Depending on the individual $V_{\text{IFT}}$ that was reached by the players in the 30-15$V_{\text{IFT}}$, the running speed during ACT ranged from 7.0 to 9.0 km·h$^{-1}$. During ACT, athletes had to run back and forth between two lines at a running pace dictated by an acoustic signal, which was sounded every 15 s. The distance between the lines was calculated based on the $V_{\text{IFT}}$ of the players, and thus indicated the individual running speed. For example, athletes who have reached a speed of 21.5 km·h$^{-1}$ in the 30-15$V_{\text{IFT}}$ had to cover a distance of 36 m every 15 s while the calculated distance for players with a maximal speed of 19.5 km·h$^{-1}$ was 33 m. Running was chosen as a recovery mode based on a survey of national and international elite tennis players conducted before the study, which showed that running was the most frequently used type of ACT for this group of athletes.

Statistical Analysis

Statistical analysis was conducted with the SPSS statistical software package (version 18, SPSS Inc., Chicago, IL, USA) and with a published spreadsheet. Data are presented as mean ± standard deviation and were tested for normal distribution using the Shapiro-Wilk Test. In cases of non-
normal distribution, data were log transformed prior to statistical analysis in order to improve normality and variance homogeneity. A repeated measure analysis of variance with the factors recovery intervention and time was used to determine the differences among markers of fatigue between between ACT and PAS as well as between testing days. Statistical significance was set at $p<0.05$. The magnitude of the changes between testing days as well as the magnitude of differences in changes between ACT and PAS was assessed using the effect size (ES). Threshold values for ES were 0.2 (small), 0.6 (moderate), 1.2 (large), and 2.0 (very large). In addition, confidence intervals (90%) for the between-group differences in changes were estimated, and magnitude-based inferences were made with reference to a smallest worthwhile change, which was calculated as 0.2 multiplied by the between-subject variation of the pre-tests. Quantitative chances of having a harmful, trivial, or beneficial effect of ACT were assessed qualitatively as follows: $<0.5\%$, almost certainly not; $0.5–5\%$, very unlikely; $5–25\%$, unlikely; $25–75\%$, possibly; $75–95\%$, likely; $95–99.5\%$, very likely; and $>99.5\%$, almost certainly. If the chance of a harmful and a beneficial effect were both $>5\%$, the true difference was considered to be unclear.

Figure 2: Mean (± SD) blood lactate concentration throughout each high-intensity interval training session determined pre and post training as well as post recovery intervention. #Significant difference compared to pre training and post recovery ($p < 0.05$). *Significant difference compared to passive recovery ($p < 0.05$).
Results

A significant recovery intervention × time interaction was found for La \((p = 0.017)\) (Figure 2). In both recovery interventions, La was significantly increased immediately after training \((p = 0.001)\), and significantly decreased immediately after the recovery intervention \((p = 0.001)\). La values at rest before exercise \((p = 0.738)\) as well as immediately at the end of the training \((p = 0.188)\) were not different between ACT and PAS. However, La measured immediately at the end of the recovery intervention was significantly lower after ACT compared to PAS \((p = 0.016)\). The players’ ratings of the difficulty of the sessions ranged from 7 to 8 (i.e., very hard) throughout the study, with no differences between recovery interventions \((p = 0.762)\).

The mean pre- and post-training values for the markers of fatigue as well as changes of these values between testing days within recovery interventions, and differences in the changes between PAS and ACT, are presented in Table 1 and Table 2. There was no significant recovery intervention × time interaction in the measures of fatigue. Magnitude-based inferences also showed neither harmful nor beneficial effects on the fatigue markers for ACT as compared to PAS. The results also revealed that the HIT-microcycle induced a significant decrease in CMJ height and a significant increase in DOMS in both recovery interventions, accompanied by a moderate and very large ES, respectively. Moreover, the perceived recovery decreased and perceived stress increased significantly between testing days in both interventions, reflected by large and very large ES for changes in PPC, OR, PS, and OS as well as small and moderate ES for changes in MPC, EB, LA, and NES. The CK activity was not significantly different after the microcycle, compared to the baseline values, in the ACT and PAS intervention. However, a moderate ES for an increase in CK levels between the testing days could be observed in both recovery interventions.

Discussion

This is the first cross-over trial that examined the effects of the repeated use of ACT during a shock microcycle of HIT. The major finding of this investigation was that ACT had no effect on the markers of performance and fatigue following the training program, as compared to PAS. The data suggest that incorporating ACT into a HIT-microcycle seems to be neither beneficial nor detrimental to the recovery process. Furthermore, results show that the four-day training program caused acute changes in measures of fatigue independent of the mode of recovery. These results confirm the findings of other research studies that have demonstrated a significant increase in the symptoms of fatigue following intensified endurance training cycles.\(^{21,22}\)
Table 1. Jump performance, serum concentration of creatine kinase, and delayed onset muscle soreness at 24 h before (pre) and 24 h after (post) a four-day high-intensity shock-microcycle, the changes in the mean values between the pre and post conditions, and the differences in the changes between the recovery interventions (n=8).

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<th>Measure</th>
<th>Group</th>
<th>Pre (Mean ± SD)</th>
<th>Post (Mean ± SD)</th>
<th>Δ Pre-Post (Mean ± SD)</th>
<th>Time</th>
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<th>Δ PAS-ACT (Mean ± 90% CI)</th>
<th>Intervention x Time</th>
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<td></td>
<td></td>
</tr>
<tr>
<td>CMJ (cm)</td>
<td>PAS</td>
<td>40.9 ± 4.4</td>
<td>37.1 ± 4.1*</td>
<td>-3.8 ± 2.9</td>
<td>0.002</td>
<td></td>
<td>-1.42</td>
<td></td>
<td>0.22</td>
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</tr>
<tr>
<td></td>
<td>ACT</td>
<td>40.1 ± 5.6</td>
<td>37.0 ± 6.1*</td>
<td>-3.1 ± 2.4</td>
<td>0.002</td>
<td></td>
<td>-1.39</td>
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<td>0.74</td>
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<tr>
<td>Blood parameter</td>
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<td></td>
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</tr>
<tr>
<td>CK (U·L⁻¹)</td>
<td>PAS</td>
<td>201 ± 32</td>
<td>428 ± 311</td>
<td>226 ± 311</td>
<td>0.060</td>
<td></td>
<td>0.81</td>
<td>112 ± 161</td>
<td>0.846</td>
<td>Unclear</td>
</tr>
<tr>
<td></td>
<td>ACT</td>
<td>182 ± 41</td>
<td>520 ± 291</td>
<td>337 ± 316</td>
<td>0.060</td>
<td></td>
<td>0.76</td>
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<tr>
<td>Muscle soreness</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VAS (mm)</td>
<td>PAS</td>
<td>0.3 ± 0.4</td>
<td>3.3 ± 2.2*</td>
<td>3.0 ± 2.3</td>
<td>0.001</td>
<td></td>
<td>2.17</td>
<td></td>
<td>0.791</td>
<td>0.10</td>
</tr>
<tr>
<td></td>
<td>ACT</td>
<td>0.2 ± 0.6</td>
<td>3.8 ± 2.7*</td>
<td>3.5 ± 2.5</td>
<td>0.001</td>
<td></td>
<td>2.02</td>
<td></td>
<td>0.791</td>
<td>0.10</td>
</tr>
</tbody>
</table>

Abbreviations: PAS, passive recovery; ACT, active recovery; CMJ, countermovement jump; CK, creatine kinase; VAS, visual analogue scale; ES, Effect size; CI, confidence interval.

Small effect (ES=0.2–0.6); moderate effect (ES=0.6–1.2); large effect (ES=1.2–2.0); very large effect (ES>2.0).

*Significantly different compared to pre values (p<0.05).
Table 2. Perceived recovery and stress 24 h before (pre) and 24 h after (post) a four-day high-intensity shock-microcycle, the changes in the mean values between the pre and post conditions, and the differences in the changes between the recovery interventions (n = 8).

<table>
<thead>
<tr>
<th>Measure</th>
<th>Group</th>
<th>Pre</th>
<th>Post</th>
<th>Δ Pre-Post</th>
<th>Time</th>
<th>ES</th>
<th>Δ PAS-ACT</th>
<th>Intervention x Time</th>
<th>ES</th>
<th>Qualitative Inference</th>
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<tr>
<td></td>
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<td>(Mean ± SD)</td>
<td>(Mean ± SD)</td>
<td>(Mean ± SD)</td>
<td>p</td>
<td></td>
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<td></td>
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<td>(Mean ± SD)</td>
<td>(Mean ± SD)</td>
<td>(Mean ± SD)</td>
<td>p</td>
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<td></td>
<td>(Mean ± SD)</td>
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<td>(Mean ± SD)</td>
<td>(Mean ± SD)</td>
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<td>(Mean ± SD)</td>
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<td>(Mean ± SD)</td>
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<td>p</td>
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<td>p</td>
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<td>(Mean ± SD)</td>
<td>(Mean ± SD)</td>
<td>(Mean ± SD)</td>
<td>p</td>
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<td></td>
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<td>(Mean ± SD)</td>
<td>(Mean ± SD)</td>
<td>(Mean ± SD)</td>
<td>p</td>
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<td></td>
<td></td>
<td>(Mean ± SD)</td>
<td>(Mean ± SD)</td>
<td>(Mean ± SD)</td>
<td>p</td>
<td></td>
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<tr>
<td>Short recovery and stress scale</td>
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<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PPC</td>
<td>PAS</td>
<td>5.0 ± 0.8</td>
<td>3.4 ± 0.9*</td>
<td>-1.6 ± 0.5</td>
<td>0.001</td>
<td>-3.36</td>
<td>0.4 ± 0.6</td>
<td>0.285</td>
<td>0.44</td>
<td>Unclear</td>
</tr>
<tr>
<td>ACT</td>
<td>PAS</td>
<td>5.4 ± 0.5</td>
<td>3.4 ± 1.3*</td>
<td>-2.0 ± 1.2</td>
<td>0.017</td>
<td>-1.79</td>
<td>0.4 ± 0.6</td>
<td>0.285</td>
<td>0.44</td>
<td>Unclear</td>
</tr>
<tr>
<td>MPC</td>
<td>ACT</td>
<td>5.6 ± 0.5</td>
<td>4.4 ± 1.3*</td>
<td>-1.2 ± 1.3</td>
<td>0.005</td>
<td>-1.34</td>
<td>0.4 ± 0.6</td>
<td>0.285</td>
<td>0.44</td>
<td>Unclear</td>
</tr>
<tr>
<td>EB</td>
<td>PAS</td>
<td>4.8 ± 0.8</td>
<td>3.3 ± 1.0</td>
<td>-1.5 ± 1.2</td>
<td>0.030</td>
<td>-1.13</td>
<td>0.4 ± 0.6</td>
<td>0.285</td>
<td>0.44</td>
<td>Unclear</td>
</tr>
<tr>
<td>OR</td>
<td>ACT</td>
<td>5.6 ± 0.5</td>
<td>4.6 ± 1.3*</td>
<td>-1.0 ± 1.3</td>
<td>0.001</td>
<td>-1.15</td>
<td>0.4 ± 0.6</td>
<td>0.285</td>
<td>0.44</td>
<td>Unclear</td>
</tr>
<tr>
<td>MS</td>
<td>PAS</td>
<td>5.3 ± 0.7</td>
<td>3.3 ± 1.3*</td>
<td>-2.0 ± 1.2</td>
<td>0.001</td>
<td>-1.39</td>
<td>0.4 ± 0.6</td>
<td>0.285</td>
<td>0.44</td>
<td>Unclear</td>
</tr>
<tr>
<td>LA</td>
<td>ACT</td>
<td>5.6 ± 0.5</td>
<td>4.6 ± 0.9*</td>
<td>-1.0 ± 1.3</td>
<td>0.001</td>
<td>-1.15</td>
<td>0.4 ± 0.6</td>
<td>0.285</td>
<td>0.44</td>
<td>Unclear</td>
</tr>
<tr>
<td>NE</td>
<td>PAS</td>
<td>0.9 ± 0.6</td>
<td>3.5 ± 1.3*</td>
<td>-1.6 ± 1.0</td>
<td>0.001</td>
<td>-1.15</td>
<td>0.4 ± 0.6</td>
<td>0.285</td>
<td>0.44</td>
<td>Unclear</td>
</tr>
<tr>
<td>OS</td>
<td>ACT</td>
<td>0.5 ± 0.8</td>
<td>3.3 ± 1.8*</td>
<td>-2.8 ± 1.5</td>
<td>0.001</td>
<td>-1.98</td>
<td>0.4 ± 0.6</td>
<td>0.285</td>
<td>0.44</td>
<td>Unclear</td>
</tr>
</tbody>
</table>

Abbreviations: PAS, passive recovery; ACT, active recovery; PPC, physical performance capability; MPC, mental performance capability; EB, emotional balance; OR, overall recovery; MS, muscular stress; LA, lack of activation; NES, negative emotional state; OS, overall stress; ES, effect size; CI, confidence interval.

Small effect (ES=0.2–0.6); moderate effect (ES=0.6–1.2); large effect (ES=1.2–2.0); very large effect (ES>2.0).

*Significantly different compared to pre values (p<0.05).
Besides the effect of ACT on the markers of fatigue, the present findings show that the HIT protocol was extremely demanding and produced high La levels of up to 9.6 mmol·L⁻¹. In this regard, the decrease in the accumulated La concentration after HIT was accelerated when training was followed by ACT rather than PAS. This effect is well established but cannot be considered as a valid indicator of the recovery quality of ACT for intermittent sports, as discussed previously. Furthermore, previous studies were able to show that La may be a potent metabolic stimulus for adaptations to training.² Therefore, ACT performed immediately after intensive exercises might reduce the potential adaptations and performance improvements through an accelerated elimination of circulating La.

In this study, the serum concentration of CK (indicative of structural disruptions within myofibers of the involved muscles) was moderately increased after the training period in both recovery interventions (Table 1). It is suggested that ACT promotes recovery from muscle damage and enhances the clearance rate of CK by increasing blood flow as well as favoring changes in blood flow distribution, thus promoting the elimination of muscle-cell debris and nutrient transport to damaged tissues.²³ The results from the current study do not, however, confirm this hypothesis, since there were no differences between the recovery interventions in terms of the activity of CK following the microcycle. This is in consistent with the findings of previous studies¹⁴, ²⁴ that have investigated the effects of low-intensity exercises on biochemical markers of fatigue, and that have shown that ACT did not affect CK clearance, and thus, the recovery of damaged muscle tissues. In contrast, Gill et al.¹² reported an enhanced clearance rate of CK as a result of ACT performed immediately post-exercise, although in this regard it should be noted that a reduced CK activity is not necessarily associated with muscle recovery.²⁵ These inconsistent results can probably be explained by the selection of the type of exercise within ACT. In the study by Gill et al.¹² ACT was performed on a cycle ergometer, whereas in the current study, the intervention involved slow running. In this respect, it is assumed that the use of running as a type of ACT is likely to increase the time before muscle regeneration sets in, as a result of sustained eccentric biomechanical strain on the working muscles.²⁶ Therefore, activities such as cycling or swimming—where the weight is borne by an external element and the eccentric strain is consequently reduced—are probably better suited as an ACT strategy.

The training program also resulted in muscle soreness, as evidenced by the largely increased DOMS 24 h after the last HIT session (Table 1). This is possibly associated with the elevated CK activity found in both recovery interventions, since changes in DOMS and serum concentration of CK are linked to myofibrillar disruptions of muscle fibers and subsequent inflammatory responses. Previous studies have investigated the analgesic effects of ACT on muscle soreness. Andersen et al.¹³ demonstrated that muscle soreness was reduced after 0, 10, 20, and 60 minutes following ACT. Similar results were reported by Zainuddin et al.,²⁷ who showed that muscle soreness was
directly alleviated after ACT. However, the analgesic effect of exercising, as explained by several potential central neural mechanisms (e.g., endorphin release by neurons in the central nervous system), attenuates with the cessation of exercise. In this context, Andersson et al. and Dawson et al. revealed that ACT had no prolonged effects on the recovery pattern of muscle soreness in the days after its application. This is consistent with our results showing that the repeated use of ACT during a four-day HIT shock microcycle was not able to reduce DOMS, as compared to PAS.

CMJ height was largely decreased after the microcycle (PAS: -9.3%; ACT: -7.7%). According to Byrne et al., a decline in CMJ height may result from muscle damage, subsequent inflammatory response, and a reduction in voluntary activation during the performance of maximal exercise due to neural inhibition caused by the presence of muscle soreness; this is supported by the increased CK activity and DOMS measured 24 h after the HIT-microcycle. In this context, the stretch-shortening-cycle (SSC), which has to be recruited in a CMJ test, is strongly implicated with exercise fatigue. It was shown that damaged muscles had a reduced tolerance to impact forces during a SSC due to decreased strength, reflex activity, and initial stiffness. The current study, however, showed that ACT did not positively affect CMJ performance compared to PAS, and thus, did not provide beneficial effects on the post-training recovery (Table 1). This finding corresponds to the elevated CK activity and DOMS, determined after the microcycle in both recovery interventions; moreover, it is consistent with the findings of previous studies, which showed that ACT did not reduce neuromuscular fatigue.

In order to evaluate the different aspects of the perceptions of recovery and stress, the SRSS was developed. Results of the present study show that ratings of PPC, MPC, EB, and OR were significantly decreased, whereas the perceptions of MS, LA, NES and OS were significantly increased in both the ACT and PAS interventions following the training program (Table 2). In addition, the finding that the changes in the ratings of PPC, OR, MS, and OS were large to very large, whereas the changes in ratings of MPC, EB, LA, and NES were small to moderate, indicating that the HIT-microcycle induced physical rather than mental fatigue. The only conceivable explanations with regard to the potential positive effects of ACT on the perceptions of physical stress have been described in the previous sections. However, the finding of no differences between the experimental conditions in the perceptions of recovery and stress following the HIT program supports the conclusion that the repeated use of ACT during an intensive training period does not have beneficial effects on limiting the severity of fatigue.
Practical Application

Players and coaches should be aware that a four-day HIT shock microcycle with a total of seven HIT sessions leads to acute changes in physical and subjective measures. Furthermore, no effects were found between recovery modalities in terms of the mean changes in the markers of fatigue. Thus, athletes and their coaches are advised to focus on other recovery modalities to minimize the severity of fatigue rather than running at low intensities. However, since ACT was not detrimental to the recovery process, individual preferences as well as experiences and beliefs concerning ACT may influence the choice of whether ACT is performed as a recovery method.

Conclusion

The repeated use of submaximal runs for 15 min at 40% $V_{IFT}$ (ACT) during a HIT shock microcycle was unable to limit the severity of exercise-induced fatigue.

References


5 GESAMTDISKUSSION


5.1 Akute Belastungsreaktionen bei fünf verschiedenen Protokollen

P_{240}, P_{120} und P_{5} führten im Trainingsverlauf zur höchsten Blutlaktatakkumulation und stärksten Absenkung des Blut-pH-Wertes, gefolgt von P_{30} sowie P_{15} mit den niedrigsten metabolischen Belastungsreaktionen. Die mittlere und maximale Hf war im Verlauf von P_{240}, P_{120} und P_{30} am höchsten, gefolgt von P_{15} und P_{5} mit den niedrigsten kardialen Belastungsreaktionen. Das subjektive Anstrengungsempfinden spiegelte insbesondere die metabolischen aber auch die kardialen Belastungsreaktionen wider - mit der höchsten Beanspruchung während P_{240}, P_{120} und P_{5}, gefolgt von P_{30} sowie P_{15} mit dem niedrigsten subjektiven Beanspruchsgrad. Andere Untersuchungen erzielten ähnliche Befunde (Astrand et al., 1960; Christensen et al., 1960; Wallner, Simi, Tschakert, & Hofmann, 2014). Sie zeigten, dass HIT-Protokolle mit mehrminütigen Intervallen im Vergleich zu Programmen mit kurzen Belastungsintervallen in signifikant höheren metabolischen und kardialen Belastungsreaktionen resultierten. Ursachen hierfür wurden bereits in Kap 2.1.1 ausführlich beschrieben.

Das Wiederholungssprinttraining stellt hier eine Ausnahme dar. Trotz der geringeren kardialen Beanspruchung sowie der kurzen Belastungsintervalle führte P_{5} zur höchsten Blutlaktatakkumulation und Blut-pH-Wert-Verschiebung. Ähnliches wurde in anderen Untersuchungen beobachtet (Buchheit, Bishop, Haydar, Nakamura, & Ahmaidi, 2010; Buchheit, 2010; Dal Pupo, Detanico, Carminatti, & Santos, 2013; Dawson et al., 1997; Serpiello, McKenna, Stepto, Bishop, & Aughey, 2011). Es ist zu vermuten, dass die Intervallpausendauer bei Wiederholungssprintbelastungen gewöhnlich nicht ausreicht, um das für Sprintleistungen relevante PCr in ausreichender Menge wiederaufzubauen. Der anfallende Energiebedarf muss daher ebenso über anaerob glykolytische Prozesse zur Verfügung gestellt werden. Der Anteil des über die anaerobe Glykogenolyse resynthetisierten ATPs nimmt im Verlauf jedoch ab. So wird im Wiederholungssprinttraining während der Intervallpausen ein Großteil des benötigten ATPs ebenfalls über den oxidativen Metabolismus resynthetisiert (Glaister, 2005; Spencer, Bishop, Dawson, & Goodman, 2005).
5.2 Der Einfluss der Belastungsnormative auf die Belastungsreaktionen


Tab. 3. Steuerungsgrößen im High-Intensity Ausdauertraining.

<table>
<thead>
<tr>
<th>Kernvariablen</th>
<th>Ergänzende Variablen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intensität der Intervalle (z.B. %Hfmax, %(\nu)(\dot{V}O_2)max, %(V_{\dot{E}}), RPE)</td>
<td>Anzahl der Intervalle ([n])</td>
</tr>
<tr>
<td>Dauer der Intervalle ([s] oder [min])</td>
<td>Anzahl der Serien ([n])</td>
</tr>
<tr>
<td>Intensität der Intervallpausen (z.B. %Hfmax, %(\nu)(\dot{V}O_2)max, %(V_{\dot{E}}), RPE)</td>
<td>Intensität der Serienpausen (z.B. %Hfmax, %(\nu)(\dot{V}O_2)max, %(V_{\dot{E}}), RPE)</td>
</tr>
<tr>
<td>Dauer der Intervallpausen ([s] oder [min])</td>
<td>Dauer der Serienpausen ([s] oder [min])</td>
</tr>
<tr>
<td>Mittlere Trainingsbeanspruchung (z.B. Pmean) und Gesamttrainingsdauer</td>
<td>Beanspruchungsform (z.B. Laufen, Fahrradfahren) und Hilfsmittel (z.B. Ropes)</td>
</tr>
</tbody>
</table>

Hfmax = maximale Herzfrequenz; \(\dot{V}O_2\)max = Laufgeschwindigkeit bei Erreichen der maximalen Sauerstoffaufnahme; VIFT = maximale im 30-15 Intermittent Fitness Test erreichte Laufgeschwindigkeit; RPE = Rating of Perceived Exertion / subjektives Belastungsempfinden; Pmean = mittlere Trainingsbeanspruchung (berechnet nach der Formel von Tschakert & Hofmann (2013))

Laut Tschakert & Hofmann (2013) sowie Saltin et al. (1976) sind Intensität und Dauer der Intervalle die gewichtigsten Einflussgrößen auf die akuten metabolischen und kardialen Belastungsreaktionen. Sie zeigten auf: Je länger die Intervalle sind, desto höher ist Blutlaktatakkumulation bzw. Blut-pH-Wert-Absenkung, vorausgesetzt die Intensität der Belastungsphasen liegt oberhalb des individuellen maxLaSS (Beneke, Leithäuser, & Ochentel, 2011; Brooks, 1985; Smekal et al., 2012). Je höher allerdings die Belastungsintensität ist (z.B. „all-out“), desto kürzer kann die Dauer der Intervalle sein, um trotz allem hohe metabolische und kardiale Belastungsreaktionen zu bewirken. Dies konnte teilweise durch die Befunde im ersten Untersuchungsmodul bestätigt werden, da die Beanspruchung während \(P_{240}\), \(P_{120}\) und \(P_5\) insgesamt am höchsten war.


Die im ersten Untersuchungsmodul verglichenen HIT-Protokolle stimmten lediglich in der Gesamttrainingszeit überein. Daher kann im Detail nicht nachvollzogen werden, welche der Belastungsnormative maßgeblich für die unterschiedlichen Belastungsreaktionen verantwortlich waren. In zukünftigen Studien sollten daher die akuten Belastungsreaktionen und mittelfristigen Ermüdungseffekte von HIT-Protokollen evaluiert werden, die sich lediglich in einer der Steuerungsgrößen unterscheiden. Nur so kann der Einfluss der Belastungsnormative detailliert und differenziert erfasst werden.
5.3 Mittelfristige Ermüdungseffekte bei fünf verschiedenen Protokollen


5.4 Diagnostikinventar zur Messung von Erholungsbedarf


Die Kontingenzanalyse mittels Vierfeldertafel ergab für keine der evaluierten Surrogatmarker eine ausreichende Sensitivität bzw. diagnostische Effektivität bei der Erfassung von Ermüdung und


Tab. 4. Praxistaugliches und potentiell sensitives Diagnostikinventar zur Messung von Ermüdung und Erholtheit im High-Intensity Ausdauertraining.

<table>
<thead>
<tr>
<th>Leistungsdiagnostik</th>
<th>Apparaturen</th>
<th>Parameter</th>
<th>Literatur</th>
</tr>
</thead>
<tbody>
<tr>
<td>CMJ</td>
<td>Kontaktplatte oder Kraftmessplatte, Computer mit entsprechender Software</td>
<td>Maximale Sprunghöhe (cm)</td>
<td>Tanner &amp; Gore (2013)</td>
</tr>
<tr>
<td>MRJ</td>
<td>Kontaktplatte oder Kraftmessplatte, Computer mit entsprechender Software</td>
<td>Maximale und mittlere Sprunghöhe (cm), Effizienzkoeffizient des Absprungs (Sprunghöhe / Kontaktdauer)</td>
<td>Girard, Lattier, Micallef, &amp; Millet (2006)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Effizienzkoeffizient des Absprungs (Sprunghöhe / Kontaktdauer)</td>
<td>Voss, Witt, &amp; Werthner (2007)</td>
</tr>
<tr>
<td>Neuromuskuläre Funktionsdiagnostik</td>
<td></td>
<td>Kontraktionszeit [ms]</td>
<td>García-Manso et al. (2012)</td>
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<td></td>
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<td>Garcia-Manso et al. (2012)</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td>Rey, Lago-Perlas, &amp; Lago-Ballesteros (2012)</td>
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<tr>
<td>CK</td>
<td></td>
<td></td>
<td>Meyer &amp; Meister (2011)</td>
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<td></td>
<td></td>
<td></td>
<td>Meeusen et al. (2013)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Meyer &amp; Meister (2011)</td>
</tr>
<tr>
<td>Subjektive Empfindungsdiagnostik</td>
<td></td>
<td></td>
<td>Cleather &amp; Guthrie (2007)</td>
</tr>
<tr>
<td>DOMS</td>
<td></td>
<td></td>
<td>Cleather &amp; Guthrie (2007)</td>
</tr>
</tbody>
</table>

CMJ = Countermovement Jump; MRJ = Multiple Rebound Jumps; TMG = Tensiomyographie; CK = Creatinkinase; DOMS = Delayed Onset Muscle Soreness
5.5 Evidenz der Wirksamkeit von aktiver Erholung


Ferner werden Anpassungen des Energiestoffwechsels durch Laktat reguliert. So steigert Laktat die MCT1-Expression (Monocarboxylat-Transporter) sowie die mitochondriale Biogenese (Hashimoto, Hussien, Oommen, Gohil, & Brooks, 2007; Wahl et al., 2010). In diesem Zusammenhang konnten Wahl et al. (2013) nachweisen, dass eine passive Erholung während eines HIT-Mikrozylus im Vergleich zu einer aktiven Erholung in einer signifikant höheren Steigerung der Ausdauerleistungsfähigkeit resultierte. Sie führten ihre Befunde unter anderem auf stärkere Veränderungen der Säure-Basen-Balance im Rahmen der passiven Erholung zurück. Da eine beschleunigte Laktatelimination in den meisten Sportarten von geringer sportpraktischer Relevanz ist und um das adaptive Potential eines intensiven Ausdauertrainings auszuschöpfen, sollte eine aktive Erholung nicht unmittelbar im Anschluss an ein HIT absolviert werden.

Potentielle regenerationsrelevante Wirkungen von aktiver Erholung wurden bereits ausführlich in Kap. 2.3.1 sowie Kap. 4.3.5 diskutiert. Sie beziehen sich zumeist auf die Veränderung der Blutflussdynamik sowie der analgetischen Wirkung durch extensive körperliche Aktivitäten. Ein Einfluss von aktiver Erholung auf den Reparaturstoffwechsel beschädigter Muskelfaserstrukturen und das subjektive Schmerz- bzw. Erholungsempfinden sowie in der Folge auf die Wiederherstellung der Leistungsfähigkeit konnte im dritten Untersuchungsmodul nicht nachgewiesen werden. Wirkungslos hinsichtlich der Linderung mittelfristiger Ermüdungseffekte war die aktive Erholung auch in diversen anderen Studien (Andersson et al., 2010; Andersson et al., 2008; Andersson, Karlsen, Blomhoff, Raastad, & Kadi, 2010; Bastos et al., 2012; Coffey, Leveritt, & Gill, 2004; Losnegard, Anderssen, Spencer, & Hallen, 2015; Stacey, Gibala, Martin Ginis, & Timmons, 2010; Suzuki et al., 2004; Zainuddin, Sacco, Newton, & Nosaka, 2006). Folglich sollten im Rah-


6 SCHLUSSFOLGERUNG UND PRAXISEMPFEHLUNGEN


Der in der vorliegenden Arbeit nicht nachweisbare Vorteil der aktiven im Vergleich zur passiven Erholung kann vermutlich unter anderem auf die anhaltenden insbesondere exzentrischen muskelmechanischen Belastungen während laufbasierten aktiven Erholungsstrategien zurückgeführt werden. Eine solche Maßnahme ist daher eher nicht zu empfehlen. Positive Auswirkungen einer aktiven Erholung konnten in aktuellen Untersuchungen nur dann nachgewiesen werden, wenn die niedrigintensive Aktivität auf dem Fahrradergometer oder im Wasser absolviert wurde. Radfahren oder Schwimmen sollten in der Sportpraxis daher den Vorzug vor einer laufbasierten Maßnahme erhalten.

ZUSAMMENFASSUNG


In der ersten Studie wurden im Rahmen einer Querschnittsuntersuchung die akuten Belastungs- und mittelfristigen Ermüdungsreaktionen von praxisüblichen, unterschiedlich konzipierten und laufbasierten HIT-Protokollen evaluiert. Die Steuerung der Trainingsintensität erfolgte dabei nach der im 30-15 Intermittent Fitness Test erreichten Maximalgeschwindigkeit (V\textsubscript{IFT}). 16 Athleten aus den Sportspielen (Mittelwert ± SD; Alter, 24,6 ± 2,7 Jahre; VO\textsubscript{2}max, 58,3 ± 5,9 ml/min/kg) absolvierten im Anschluss an eine Voruntersuchung zur Ermittlung von V\textsubscript{IFT} innerhalb von fünf Wochen jeweils einmal wöchentlich in randomisierter Reihenfolge eines von fünf HIT-Programmen (P\textsubscript{240}: 4 × 4 min bei 80% von V\textsubscript{IFT}; P\textsubscript{120}: 7 × 2 min bei 85% von V\textsubscript{IFT}; P\textsubscript{30}: 2 × 10 × 30 bei 90% von V\textsubscript{IFT}; P\textsubscript{15}: 3 × 9 × 15 s bei 95% von V\textsubscript{IFT}; P\textsubscript{5}: 4 × 6 × 5 s Sprints). Zu verschiedenen Messzeitpunkten vor, während und unmittelbar im Anschluss an jede HIT-Einheit sowie nach 24 Stunden wurden folgende Parameter gemessen: Blutlaktatkonzentration, Blut-pH-Wert, Serumaktivität von Creatinkinase (CK), Herzfrequenz, subjektives Belastungsempfinden der Gesamtratingseinheit, empfundener Muskelschmerz sowie Sprungleistung im Countermovement Jump (CMJ).
Bei den akuten Belastungs- und mittelfristigen Ermüdungsreaktionen konnten signifikante Unterschiede (p < 0,05) zwischen den HIT-Protokollen beobachtet werden. Die akute metabolische, kardiale und perceptive Belastung war in Protokollen mit kurzen Intervallen (P_{30} und P_{15}) im Vergleich zu den HIT-Varianten mit langen Intervallen (P_{240} und P_{120}) signifikant niedriger, mit Ausnahme des Sprintprotokolls (P_{5}), das die stärkste Blutlaktatakkumulation sowie Blut-pH-Wert-Abnahme verursachte. Mittelfristige Ermüdungseffekte ergaben sich insbesondere im Anschluss an das Wiederholungssprinttraining, mit signifikant höherem CK-Wert im Blut, Muskelkaterempfinden und Leistungsverlust im Countermovement Jump im Vergleich zu den anderen Protokollen. Aufgrund der höchsten Netto-Trainingszeit bei gleichzeitig hohem metabolischen Stress resultierten P_{240} und P_{120} aber vermutlich in der stärksten Entleerung der intramuskulären Glykogenspeicher.

In der zweiten Untersuchung wurde im Rahmen einer Längsschnittstudie die Sensitivität praxistauglicher Parameter für das Monitoring von Ermüdung und Erholtheit im HIT überprüft. Hierfür absolvierten 22 Athleten aus den Mannschaftssportarten (Mittelwert ± SD; Alter, 23,0 ± 2,7 Jahre; VO_{2max}, 57,6 ± 8,6 ml/min/kg) einen sechstägigen Mikrozyklus (mit insgesamt elf laufbasierten HIT-Einheiten), der in einem temporären funktionellen Overreaching resultieren sollte. Die Steuerung der Trainingsintensität erfolgte dabei ebenfalls nach der V_{IFT}, die im Rahmen einer sportärztlichen Voruntersuchung eine Woche vor Beginn des Mikrozyklus ermittelt wurde. 24 Stunden vor dem Trainingsprogramm sowie 24 und 72 Stunden nach der Belastungsphase wurden folgende Parameter gemessen: Wiederholungssprintfähigkeit auf einem nichtmotorisierten Laufband (sportartspezifische Leistungsfähigkeit und Außenkriterium für die Erfassung von Ermüdung und Erholtheit), Sprungleistung im CMJ, Sprungeffizienz im Multiple Rebound Jumps Test (MRJ), 20-m Linearsprintleistung, Muskelkontraktilität, Konzentration von CK, C-reactives Protein und Harnstoff im Serum sowie empfundener Muskelschmerz.

Der HIT-Mikrozyklus resultierte in einem signifikanten (p < 0,05) sportspielspezifischen Leistungsverlust (%Δ ± 90% Konfidenzlimits, ES = Effektstärke; Wiederholungssprintfähigkeit: -3,8 ± 1,0, ES = -1,51), der innerhalb einer 72-stündigen Erholungsphase wieder abklang (2,8 ± 2,6, ES = 0,53). Vergleichbare Mittelwertsbewegungen zeigte die Sprungleistung (CMJ: -8,4 ± 2,9, ES = -1,35, 4,1 ± 2,9, ES = 0,68), die Sprungeffizienz (MRJ: -17,4 ± 4,5, ES = -1,60, 6,5 ± 4,5, ES = 0,63), die Muskelkontraktilität und die CK-Aktivität sowie das Muskelschmerzempfinden. Weitere Auswertungen mittels multipler Regressionsanalyse sowie Vierfelderkontingenztafel ergaben jedoch, dass keiner der potentiell geeigneten Surrogatmarker ausreichende Sensitivität, Spezifität und diagnostische Effektivität bei der Beurteilung von Ermüdung und Erholtheit aufwies.
In der dritten Untersuchung wurde im Rahmen einer Cross-Over-Studie der Einfluss von aktiver Erholung (AE) auf die durch HIT induzierten Ermüdungsmechanismen untersucht. Hierfür nahmen 8 international gerankte Tennisspieler (Mittelwert ± SD; Alter, 15,1 ± 1,4 Jahre) an zwei viertägigen Mikrozyklen (mit jeweils sieben laufbasierten HIT-Einheiten) teil, die durch eine viermonatige Wash-Out-Phase getrennt waren. In einem der beiden Blöcke erholten sich die Spieler nach jeder HIT-Einheit jeweils aktiv oder passiv. Die Steuerung der Intensität im Training sowie während der AE erfolgte auch in der dritten Studie nach der VIFT, die im Rahmen einer sportärztlichen Voruntersuchung jeweils 72 Stunden vor Beginn der Mikrozyklen ermittelt wurde. 24 Stunden vor dem Trainingsprogramm sowie 24 Stunden nach der Belastungsphase wurden folgende Ermüdungsmarker gemessen: Sprungleistung im CMJ, Serumkonzentration von CK, Muskel- schmerzempfinden sowie subjektiv wahrgenommener Erholungs- und Beanspruchungszustand.

Der HIT-Mikrozyklus induzierte ebenfalls eine bedeutsame Reduktion der Sprungleistung (AE: -3,8 ± 2,9 cm, ES = -1,39, p < 0,05; Passive Erholung [PE]: -3,1 ± 2,4 cm, ES = -1,42, p = 0,05) und des Erholungsempfinden (AE: ES = -1,79, p < 0,05; PE: ES = -2,39, p < 0,05) sowie eine Erhöhung des CK-Levels (AE: ES = 0,76, p > 0,05; PE: ES = 0,81, p > 0,05), Muskelschmerz (AE: ES = 2,02, p < 0,05; PE: ES = 2,17, p < 0,05) und Ermüdungsempfindens (AE: ES = 1,98, p < 0,05; PE: ES = 3,06, p < 0,05). Signifikante Interaktionseffekte bzw. praktisch bedeutsame Unterschiede in den Ermüdungseffekten zwischen AE und PE wurden nicht festgestellt.

7.1 Abstract

High-intensity interval training (HIT), involving short to long (~5-300 s) intensive work intervals interspersed by active or passive recovery periods, is frequently used in training programs of competitive athletes from various disciplines in order to improve (sport-specific) endurance performance. However, as a result of high metabolic and neuromuscular demands, HIT is also accompanied with acute feelings of fatigue. Ensuring that fatigue in HIT is adjusted appropriately is essential for both adaptations to training as well as competition performance. If the balance between appropriate training load and adequate recovery is disrupted, an abnormal training response may occur and a state of overtraining may develop. This applies especially for high-level athletes due to a process of intensifying training and competition loads in many disciplines in recent years. However, research on the acute responses and exercise-induced fatigue of different HIT-protocols that are commonly used in practice as well as on the diagnostic effectiveness of different markers for routine assessment of fatigue and recovery and on the effects of recovery interventions in connection with HIT is still lacking. Therefore, the purpose of the current doctoral thesis was to evaluate evidence-based guidelines for an appropriate managing of training load and recovery in HIT.

The methodology was based on three studies that were build on one another and were carried out in a chronological order. Each investigation was made up of an independent research approach, whereby the study design of part two and three were prepared each by using the findings of the previous sub-studies.

The aim of the first cross-sectional study was to evaluate the acute responses and exercise-induced fatigue of five different HIT-protocols adjusted by the maximum velocity obtained in the 30-15 Intermittent Fitness Test (VIFT). For this purpose, 16 well-trained intermittent sport players (mean ± SD; age, 24.6 ± 2.7 years; VO₂max, 58.3 ± 5.9 ml·min·kg⁻¹) participated in five different running-based HIT-programs separated by six days in between (P240: 4 × 4 min at 80% V_{IFT}; P120: 7 × 2 min at 85% V_{IFT}; P30: 2 × 10 × 30 s at 90% V_{IFT}; P15: 3 × 9 × 15 s at 95%V_{IFT}; P5: 4 × 6 × 5 s sprints). Blood lactate (La) concentration, blood pH, serum concentration of creatinkinase (CK), heart rate (HR), session rating of perceived exertion (session-RPE), delayed onset muscle soreness (DOMS) and countermovement jump (CMJ) height were measured.

A significant main effect for protocol (p < 0.05) was found for the acute responses of HR, session-RPE and La concentration with values increasing in longer intervals from P15 to P120 and P240 while blood pH responded inversely. In contrast, P5 produced the highest La concentration and blood pH decreases. 24 h post exercise serum creatinkinase (CK), delayed onset muscle soreness (DOMS) and the decrease in countermovement jump (CMJ) height were significantly higher.
(p < 0.05) after P_5 compared to all other protocols. Due to the highest work/rest ratio together with the high metabolic stress in P_{240} and P_{120}, these protocols led presumably to the strongest depletion of skeletal muscle glycogen stores.

The second longitudinal study aimed to investigate the diagnostic effectiveness of different markers for routine assessment of fatigue and recovery in response to HIT. For this purpose, 22 well-trained male and female team sport athletes (mean ± SD; age, 23.0 ± 2.7 years; VO_2\text{max}, 57.6 ± 8.6 mL·min·kg^{-1}) participated in a six-day HIT-microcycle with a total of eleven running-based HIT-sessions that were adjusted by the V_{IFT} and designed to induce a temporary functional overload. Repeated sprint ability (RSA; criterion measure of fatigue and recovery), CMJ height, jump efficiency in a multiple rebound jump test (MRJ), 20-m sprint performance, muscle contractile properties, serum concentrations of CK, c-reactive protein (CRP) and urea as well as DOMS were measured pre and post the training program as well as after 72 h of recovery.

Following the microcycle significant changes (p < 0.05) in RSA as well as in CMJ and MRJ performance could be observed, showing a decline (Δ ± 90% confidence limits, ES = effect size; RSA: -3.8 ± 1.0, ES = -1.51; CMJ: 8.4 ± 2.9, ES = -1.35; MRJ: 17.4 ± 4.5, ES = -1.60) and a return to baseline level (RSA: 2.8 ± 2.6, ES = 0.53; CMJ: 4.1 ± 2.9, ES = 0.68; MRJ: 6.5 ± 4.5, ES = 0.63) after 72 h of recovery. Athletes also demonstrated significant changes (p < 0.05) in muscle contractile properties, CK, and DOMS following the training program and after the recovery period. Further analysis revealed that the accuracy of markers for assessment of fatigue and recovery in comparison to RSA derived from a contingency table was insufficient. Multiple regression analysis also showed no correlations between changes in RSA and any of the markers.

The aim the third cross-over study was to examine the effect of a repeated us of active recovery (ACT) on HIT-induced markers of fatigue. For this purpose, eight elite male junior tennis players (mean ± SD; age, 15.1 ± 1.4 years) with an international ranking between 59 and 907 (International Tennis Federation) participated in two four-day HIT-microcycles (each with a total of seven running-based HIT-sessions), which were interrupted by a four-month was-out period. After each training session, the players completed 15 min of either moderate jogging (ACT) or passive recovery (PAS). Running intensity both for HIT-sessions and for ACT was adjusted by the V_{IFT}. CMJ height, serum concentration of CK, DOMS, and perceived recovery and stress (Short Recovery and Stress Scale) were measured 24 h before and 24 h after each training program.

The HIT shock microcycle induced a large decrease in CMJ performance (ACT: -3.8 ± 2.9 cm, ES = -1.39, p < 0.05; PAS: -3.1 ± 2.4 cm, ES = -1.42, p < 0.05) and perceived recovery (ACT: ES = -1.79, p < 0.05; PAS: ES = -2.39, p < 0.05), as well as a moderate to large increase in CK levels (ACT: ES = 0.76, p > 0.05; PAS: ES = 0.81, p > 0.05), DOMS (ACT: ES = 2.02, p < 0.05; PAS:
ES = 2.17, \( p < 0.05 \), and perceived stress (ACT: ES = 1.98, \( p < 0.05 \); PAS: ES = 3.06, \( p < 0.05 \)), compared to the values before the intervention. However, no significant recovery intervention × time interactions or meaningful differences in changes were noted in any of the markers between ACT and PAS.

HIT protocols of different interval duration and intensity result in varying acute physiological and perceptual demands. Longer intervals lead to higher acute cardio-circulatory and metabolic responses, whereas sprint protocols induce the highest state of fatigue. Mean changes in measures of neuromuscular function (i.e., CMJ and MRJ performance as well as muscle contractile properties), CK and DOMS are related to HIT induced fatigue and subsequent recovery. However, low accuracy of a single or combined use of these markers requires the verification of their applicability on an individual basis. A repeated use of ACT during a HIT shock microcycle did not affect exercise-induced fatigue. Thus, athletes and their coaches are advised to focus on other recovery modalities to minimize the severity of fatigue after HIT. However, since ACT was not detrimental to the recovery process, individual preferences as well as experiences and beliefs concerning ACT may influence the choice of whether ACT is performed as a recovery method.
LITERATURVERZEICHNIS


ABBILDUNGSVERZEICHNIS

Arbeitsprogramm und Zielstellung

Abb. 1. Gesamtdesign des dreistufigen Arbeitsprogramms. HIT = High-Intensity Interval Training

Publikation 1

Figure 1. Short-term effects of different high-intensity training protocols on heart rate (HR), session-RPE, blood lactate (La) and blood pH. Letters represent significant differences (p<0.05). aSignificantly different from P_{120} and P_{5}; bsignificantly different from all other protocols; csignificantly different from P_{240}, P_{120} and P_{5}; dsignificantly different from P_{120};esignificantly different from P_{240}, P_{120} and P_{30}; fsignificantly different from P_{240} and P_{120}

Figure 2. Changes in blood lactate concentration during different high-intensity training protocols measured pre-exercise (0), after approximately 6 (1), 12 (2) and 18 (3) min (always immediately at the end of a work interval) and at the end (4) of each session. Blood lactate concentrations during exercise were significantly different from pre-exercise values in all protocols (p<0.05). Letters represent significant increases in blood lactate concentrations during exercise (p<0.05)

Figure 3. Mid-term effects of different high-intensity training protocols on serum creatinkinase (CK) and delayed onset muscle soreness (DOMS). Letters represent significant differences (p<0.05). aSignificantly different to baseline; bSignificant protocol x time interaction; csignificantly different from P_{30}

Publikation 2

Figure 1. Study design. 30-15_{IFT} = 30-15 Intermittent Fitness Test, HIIT = high-intensity interval training

Figure 2. Mean (± SD) countermovement jump (CMJ) height (A), multiple rebound jumps (MRJ) performance (B) and 20-m sprint time (C) in males and females at baseline (pre), after a six-day high-intensity interval training program (post_1), and following 72 h of recovery (post_2). RSI = reactive strength index. *Significant difference compared to pre (p < 0.05). #Significant difference compared to post_1 (p < 0.05)
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MPC = Mental Performance Capability / Mentale Leistungsfähigkeit
MRJ = Multiple Rebound Jumps
MS = Muscular Stress / Muskuläre Beanspruchung
MV = Mean Peak Velocity / Mittlere Maximalgeschwindigkeit
NES = Negative Emotional State / Emotionale Unausgeglichenheit
NMT = Nonmotorized Treadmill / Nichtmotorisiertes Laufband
$O_2$ = Sauerstoff
OR = Overall Recovery / Allgemeiner Erholungszustand
OS = Overall Stress / Allgemeiner Beanspruchungszustand
PAS/PE = Passive Recovery / Passive Erholung
PCr = Phosphokreatin
$P_{mean}$ = Mittlere Belastung
POMS = Profile of Mood States
PPC = Physical Performance Capability / Körperliche Leistungsfähigkeit
rMSSD = Root Mean Square of the Successive Differences
$r$ = Intervallpausendauer
R = Serienpausendauer
RF = Rectus Femoris
RPE = Rating of Perceived Exertion / Subjektives Belastungsempfinden
RSA = Repeated Sprint Ability / Wiederholungssprintfähigkeit
RSI = Reactive Strength Index / Reaktivkraftindex
RST = Repeated Sprint Training / Wiederholungssprinttraining
SD = Standard Deviation / Standardabweichung
SSC = Stretch-Shortening-Cycle / Dehnungsverkürzungszyklus
ST-Faser = Slow-Twitch-Fasern
SV = Schlagvolumen
t@$V_{O_2}$max = An der maximalen Sauerstoffaufnahme verbrachte Belastungszeit
Tc = Contraction Time / Kontraktionszeit
TE = Typical Error / Standardfehler
TMG = Tensiomyographie
TNF $\alpha$ = Tumornekrosefaktor Alpha
TQR = Total Quality Recovery
Urea = Harnstoff
VAS = Visual Analog Scale / Visuell-analoge Skala
$V_{IFT}$ = Maximale im 30-15 Intermittent Fitness Test erreichte Laufgeschwindigkeit
$V_{O_2}$ = Sauerstoffaufnahme
$V_{O_2}$max = Maximale Sauerstoffaufnahme
<table>
<thead>
<tr>
<th>Abkürzung</th>
<th>Beschreibung</th>
</tr>
</thead>
<tbody>
<tr>
<td>vVO₂max</td>
<td>Laufgeschwindigkeit bei Erreichen der maximalen Sauerstoffaufnahme</td>
</tr>
<tr>
<td>W/R</td>
<td>Work/Rest Ratios / Belastungs-Erholungs-Verhältnis</td>
</tr>
</tbody>
</table>
ANHANG

RESEARCH ARTICLE
Markers for Routine Assessment of Fatigue and Recovery in Male and Female Team Sport Athletes during High-Intensity Interval Training
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Abstract

Aim
Our study aimed to investigate changes of different markers for routine assessment of fatigue and recovery in response to high-intensity interval training (HIIT).

Methods
22 well-trained male and female team sport athletes (age, 23.0 ± 2.7 years; VO₂max, 57.6 ± 8.6 mL·min⁻¹·kg⁻¹) participated in a six-day running-based HIIT-microcycle with a total of eleven HIIT sessions. Repeated sprint ability (RSA; criterion measure of fatigue and recovery), countermovement jump (CMJ) height, jump efficiency in a multiple rebound jump test (MRJ), 20-m sprint performance, muscle contractile properties, serum concentrations of creatine kinase (CK), c-reactive protein (CRP) and urea as well as perceived muscle soreness (DOMS) were measured pre and post the training program as well as after 72 h of recovery.

Results
Following the microcycle significant changes (p < 0.05) in RSA as well as in CMJ and MRJ performance could be observed, showing a decline (%Δ ± 90% confidence limits, ES = effect size; RSA: -3.8 ± 1.0, ES = -1.51; CMJ: 8.4 ± 2.9, ES = -1.35; MRJ: 17.4 ± 4.5, ES = -1.60) and a return to baseline level (RSA: 2.6 ± 2.6, ES = 0.53; CMJ: 4.1 ± 2.9, ES = 0.68; MRJ: 6.5 ± 4.5, ES = 0.63) after 72 h of recovery. Athletes also demonstrated significant changes (p < 0.05) in muscle contractile properties, CK, and DOMS following the training program and after the recovery period. In contrast, CRP and urea remained unchanged throughout the study. Further analysis revealed that the accuracy of markers for assessment of fatigue and recovery in comparison to RSA derived from a contingency table was
Conclusions

Mean changes in measures of neuromuscular function, CK and DOMS are related to HIIT induced fatigue and subsequent recovery. However, low accuracy of a single or combined use of these markers requires the verification of their applicability on an individual basis.

Introduction

High-intensity interval training (HIIT), involving short to long (5-300 s) intensive work intervals interspersed by active or passive recovery periods, is frequently used in training programs of competitive team sport athletes. This type of intermittent training was shown to improve cardiovascular and metabolic determinants, allowing players to sustain intense phases during the game for longer durations and also to recover from it more rapidly [1, 2]. Additionally, HIIT induces similar adaptations with significant lower training volumes compared to traditional endurance training [3, 4]. This is the main rationale behind its application in team sport conditioning programs, since the complex profile of demands requires that various conditional abilities as well as technical and tactical elements need to be considered and, consequently, the timeframe to improve endurance performance is limited.

However, as a result of high metabolic and neuromuscular demands, HIIT is also accompanied with acute feelings of fatigue [5]. Howatson and Milak [6] have shown that even one single team sport specific HIIT session leads to a significant increase in muscle damage and muscle soreness in the days following the exercise bout. Although effective training programs intend functional overreaching, excessive overload with insufficient recovery should be avoided [7]. If the balance between training stress and recovery is inadequate over a prolonged period, the athlete will experience decreases in performance and a state of overtraining may develop [7]. During in-season training, the challenge for coaches and athletes is to determine the point at which HIIT may negatively affect the performance in upcoming competitions. Therefore, the routine assessment of fatigue and recovery during HIIT is important to improve individual training prescriptions and to ensure competition readiness [8].

Fatigue and recovery is characterized by a combination of several factors involving mechanisms from the central nervous system to the muscle cell itself. In this regard, a change in the players’ specific on-court performance represents the most relevant marker for differentiation between fatigued and recovered athletes. However, the majority of field test recommendations for standardized performance measurements in team sports are physically demanding and induce additional fatigue [9, 10]. Consequently, a variety of other surrogate markers (e.g., subjective, biochemical, neuromuscular, and performance markers) are frequently used in science and practice in order to track the fatigue and recovery process [9]. The daily determination of a wide range of these markers established in endurance sports (e.g., heart rate variability or several markers in the blood), however, seems to be inadequate and difficult to control under the typical team sport surrounding. Therefore, practical parameters that are determined at rest or during low metabolic and neuromuscular demands, without disturbing the training process, are preferred in team sports for the routine assessment of fatigue and recovery [11].

Tools that meet these criteria and that have been proposed in the literature are subjective markers (e.g., delayed onset muscle soreness), neuromuscular performance tests (e.g., jumps),
Table 1. Baseline physical characteristics of the athletes.

<table>
<thead>
<tr>
<th></th>
<th>Age (yrs)</th>
<th>Height (cm)</th>
<th>Body mass (kg)</th>
<th>Body fat (%)</th>
<th>VO2max (mL min kg⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall (n = 22)</td>
<td>23±2.7</td>
<td>176.6±7.6</td>
<td>69.5±7.3</td>
<td>17.9±0.8</td>
<td>57.6±8.6</td>
</tr>
<tr>
<td>Male (n = 11)</td>
<td>22.9±1.9</td>
<td>181.6±5.3</td>
<td>73.8±6.4</td>
<td>14.6±3.7</td>
<td>62.8±8.3</td>
</tr>
<tr>
<td>Female (n = 11)</td>
<td>23.0±3.4</td>
<td>171.6±6.0</td>
<td>65.2±5.5</td>
<td>21.1±5.9</td>
<td>52.2±4.8</td>
</tr>
</tbody>
</table>

Parameters are shown as mean ± SD.

doi:10.1371/journal.pone.0139801.t001

Assessment of Fatigue in High-Intensity Training

Materials and Methods

Participants

A total of 22 (11 males and 11 females) healthy and well-trained team sport athletes (i.e., soccer, basketball, handball) took part in this study. The baseline physical characteristics of the athletes are shown in Table 1. The mean training frequency of the athletes was 5.7 ± 1.9 days week⁻¹ with a mean training volume of 2.5 ± 0.8 h day⁻¹. After being informed about the exercise protocols and all possible risks associated with participation in the investigation, subjects gave written consents to participate in all procedures. Normal ECG and the absence of cardiovascular, pulmonary and orthopedic diseases were confirmed in a preliminary health examination. Additionally, athletes had to meet two inclusion criteria: minimal performance in the 30–15 Intermittent Fitness Test (30–15IFT) of at least 16 km h⁻¹ for women or 19 km h⁻¹ for men and at least five years of specific team sport training experience. Initially, 24 athletes from different regional teams were evaluated for possible participation in the study, of which two failed to fulfill the inclusion criteria. The study was approved by the ethic committee of the Medical Faculty of the Ruhr-University Bochum and performed according to the Declaration of Helsinki.

Experimental design

A repeated measures study was used to examine the accuracy of markers of fatigue and recovery. The investigation lasted 18 days and was conducted in the athletes’ off-season period during which no additional club training took place. Seven days prior to the HIIT program all athletes came to the laboratory for a preliminary health examination to exclude contraindications to participation in this study (e.g., cardiovascular, pulmonary, or orthopedic diseases), to obtain data on anthropometrical characteristics and to determine VO2max. After familiarization with performance tests to minimize any learning effect, participants completed the 30–15IFT on a second preliminary examination day followed by four days of rest. Athletes were then examined at baseline (pre), after completing a six-day training program of HIIT (post1), and following a 72 h recovery period (post2), in which no training was allowed (Fig 1).

On all testing days (pre, post1, post2), repeated sprint ability (RSA) was assessed on a non-motorized treadmill (NMT), which was defined as an important marker of team sport specific
performance and as criterion measure of fatigue and recovery. RSA test has been shown to be closely associated with competitive performance in team sport athletes [15–17] and to be highly reproducible (coefficient of variation (CV) of about 2.5% for velocity) [18, 19]. In addition, prior to the RSA test, perceived muscle soreness, muscle contractile properties, blood parameters as well as jump and linear sprint performance were determined (in this order) as surrogate markers of fatigue and recovery pre, post1, and post2. All measures were taken at the same time of day for each individual on each occasion. Time between jump tests and linear sprint test as well as between linear sprint and RSA test was 30 min and 120 min, respectively. Prior to all performance tests a standardized warm-up was conducted.

Participants were instructed to maintain their normal dietary intake and to refrain from nutritional supplements and alcohol intake during the experimental period. In this regard, athletes were verbally questioned before each testing procedure as well as during the six-day training intervention to ensure that they had adhered to the dietary rules.

**Procedures**

**Incremental treadmill test.** In order to determine VO2max, a progressive incremental exercise test on a motor driven treadmill (Ergo ELG2, Woodway GmbH, Weil am Rhein, Germany) was used. The treadmill test started with an initial velocity of 8 km·h⁻¹, increasing 2 km·h⁻¹ every 3 min with a constant incline of 0.5% until voluntary exhaustion. VO2 was continuously analyzed using a breath-by-breath gas collection system (ZAN600USB, nSpire Health GmbH, Oberthalba, Germany). The gas calibration was completed before the test day and the volume calibration was conducted before each test following the instructions provided by the manufacturer. The highest mean value for 30 s was defined as the VO2max.

**30–15 intermittent fitness test.** The test was conducted outdoors on a tartan track and consisted of 30 s shuttle runs interspersed with 15 s passive recovery periods. Speed was set at 8 km·h⁻¹ for the first 30 s run and was increased by 0.5 km·h⁻¹ every 45 s stage thereafter. The athletes had to run back and forth between two lines set 40 m apart at a pace dictated by an acoustic signal. The test ended when a player could no longer maintain the imposed running speed or when he was unable to reach a 3 m zone around each line at the moment of the audio signal for three consecutive times. The speed of the last completed stage achieved by the participants (V[30]) was used as an inclusion criteria and to calculate the interval intensity of the HIT protocols applied in the six-day training program as described by Buchheit [20].

**Repeated sprint ability test.** The laboratory RSA test was performed on a Woodway non-motorized treadmill (NMT) (Force 3.0, Woodway GmbH, Weil am Rhein, Germany) pre,
post1, and post2. The experimental set-up of the test has previously been described by Oliver et al. [18]. The RSA test consisted of six 4 s maximal sprints from a standing position with 20 s passive recovery between sprints. The peak values attained in each sprint for velocity were recorded and mean peak values for velocity (MV) were calculated. For MV, the intraclass correlation coefficient (ICC) and the typical error (TE) were previously investigated by our research group and MV was considered to be highly reliable (unpublished results: MV (m·s⁻¹), n = 17, ICC = 0.92, TE = 0.10, CV = 1.5%).

**Jump and linear sprint tests.** On each testing day (pre, post1, and post2), countermovement jumps (CMJ) and multiple rebound jump tests (MRJ) were performed on a contact platform (Haynal-Elektronik GmbH, Schönebeck, Germany) with hands placed on hips. For CMJ, participants dropped down to a self-selected level before jumping maximally. Flight time was used to calculate jump height [21]. Each subject performed two maximal CMJ and the mean height was calculated. For MRJ, participants were advised to perform repeated maximum vertical jumps for 15 s with reactive landing phases and ground contact times which should be as short as possible. Flight time and contact time were used to calculate the reactive strength index (RSI) for each jump by dividing the height jumped in meters by the time on the ground in seconds [22]. Based on the RSI, the five best jumps were selected and mean RSI was calculated for further analysis. The 20-m linear sprint was completed outdoors on a tartan track and sprint times were recorded using a wireless double photocell system (Sportronic, Winningen-Hertmannsweiler, Germany). Each sprint was initiated without a starting signal and from an individually chosen upright standing position 50 cm behind the first photocell. Participants performed two maximal sprints interspersed by 3 min of passive recovery and the mean sprint time was calculated for further analysis. Previously measured reliability scores for jump and linear sprint tests were regarded as highly reliable (unpublished results: CMJ (cm), n = 38, ICC = 0.92, TE = 1.86, CV = 3.7%; MRJ (cm), n = 38, ICC = 0.91, TE = 0.13, CV = 4.0%; 20-m linear sprint test (s), n = 22, ICC = 0.95, TE = 0.06, CV = 1.8%).

**Muscle contractile markers.** For the non-invasive assessment of the contractile properties of knee extensor and flexor muscles, Tensiometryography (TMG) was used under laboratory conditions pre, post1, and post2. This technique produces a radial displacement of the muscle belly in response to an electrical stimulus (around 100 mA) conducted through the underlying muscle tissue [13,23]. These displacements are recorded at the skin surface using a spring loaded displacement sensor (TMG-RMC Ltd, Ljubljana, Slovenia). The sensor was positioned perpendicular to the thickest part of the muscle belly, which was established visually and through palpation during a voluntary contraction, and the self-adhesive electrodes were placed symmetrically approximately 5 cm away from the sensor. Once the exact position for the sensor and electrodes was found, it was marked with a dermatological pen and kept constant during the experimental period. Maximal radial muscle belly displacement (Dm) and contraction time between 10 and 90% Dm (Tc) of the rectus femoris (RF) and biceps femoris (BF) were measured through TMG. Reliability scores for Dm and Tc of the RF and BF were previously examined and considered as reliable (unpublished results: RF Dm (mm), n = 20, ICC = 0.92, TE = 1.00, CV = 9.3%; RF Tc (ms), n = 20, ICC = 0.94, TE = 1.90, CV = 4.9%; BF Dm (mm), n = 20, ICC = 0.95, TE = 0.90, CV = 10.4%; BF Tc (ms), n = 20, ICC = 0.91, TE = 5.60, CV = 8.7%).

**Biochemical markers.** Venous blood samples were collected on each testing day (pre, post1, and post2) between 8 and 10 a.m., and ~2 h after the athletes took a typical breakfast) from an antecubital arm vein of the right arm using a 20-gauge disposable Safety-Multiject needle (Sarstedt AG & Co, Nümbrecht, Germany) while the subject was in a supine position. Samples were collected into 7.5 mL serum gel tubes with clotting activator (Sarstedt AG & Co, Nümbrecht, Germany) and subsequently centrifuged at 3500 rpm for 15 min within 20 min.
after sampling. The resulting serum was separated from the other compounds, pipetted into micro tubes (Sarstedt AG & Co, Nümbrecht, Germany) and stored at –80°C. Later, routine techniques (UniCel DxC 600 Synchrone®, Beckmann Coulter GmbH, Krefeld, Germany) were used for analysis of the concentration of creatinkinase (CK), c-reactive protein (CRP), and urea. The diagnostic laboratory used in this study held current quality assurance certification (Referenzzentrum für Bioanalytik, Bonn, Germany).

**Subjective marker.** Before all tests were performed on each testing day (pre, post1, and post2), athletes were asked to score on a visual analogue scale (VAS) the general amount of delayed onset muscle soreness (DOMS). The VAS, which has been shown to be reliable in previous research [24], consisted of a 100 mm line whose endpoints were labeled “no pain” (left) and “unbearable pain” (right). Subjects had to draw a vertical line at a point on the line that represented their pain at the time of measurement best. The rating resulted from the distance in mm from the left border of the scale to the point marked [14].

### Training program

A six-day training intervention was designed to induce a functional overload while remaining tolerable for the athletes. The training program (exercise mode, number and duration of intervals and rest, intensity) consisted of 11 training sessions with an average training duration of 35 min per session (Table 2). To calculate training intensity, participants completed the 30–15 protocol as part of the preliminary examinations. All sessions were completed outdoors on a 400 m tartan track and preceded by a standardized 10 min warm-up, consisting of 40 m shuttle runs at 60–70% of $V_{\text{max}}$, followed by four 40 m acceleration sprints. To ensure that the intended training intensity was maintained by the athletes, all sessions were supervised and individually calculated running distances were controlled. Additionally, training loads were determined by multiplying the numerical score of the athletes’ perception of effort, using a category-ratio RPE scale [25,26], with the total exercise duration in min. Training loads were kept constant throughout the training period.

### Statistical analysis

All statistical analyses were performed by using SPSS (statistical software package version 18, SPSS Inc., Chicago, IL, USA) and Excel 2010 (Microsoft Corp., Redmond, WA, USA). Data are

<table>
<thead>
<tr>
<th>Table 2. Six-day high-intensity interval training program.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Day 1</strong></td>
</tr>
<tr>
<td>-----------</td>
</tr>
<tr>
<td><strong>a.m.</strong></td>
</tr>
<tr>
<td><strong>r = 3 min</strong></td>
</tr>
<tr>
<td><strong>p.m.</strong></td>
</tr>
<tr>
<td><strong>r = 25 s; R = 5 min</strong></td>
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</tbody>
</table>

$V_{\text{max}}$: final running speed obtained in the 30–15 intermittent Fitness Test; r: passive recovery between intervals; R: passive recovery between series; TL: training load.

Example of training program: [40m-shuttle runs, 2 x 12 x 30 s, 90% $V_{\text{max}}$; r = 30 s; R = 3 min] means that the subject had to run two series of 12 intervals at 90% $V_{\text{max}}$ composed of 30 s passive recovery between intervals and 3 min passive recovery between series.

Example of training load calculation: [(session-RPE) (9) x training duration (26 min)] = 254.
presented as mean ± SD and were tested for normal distribution using the Shapiro-Wilk Test. Furthermore, 95% confidence interval (CI) is given. A two-factor (time, sex) repeated measure analysis of variance (ANOVA) was used to determine differences among markers of fatigue and recovery between testing days (pre, post1, and post2) as well as between male and female team sport athletes. Bonferroni post-hoc tests were used when the ANOVA main effect was significant. Those markers which were not normally distributed (CK, CRP) were tested using Friedman test. Wilcoxon tests were used when the Friedman test was significant. To allow a better interpretation of the results, the effect size Cohen’s d [27] (defined as [difference between the means]/[SD]) was calculated for all parameters between testing days. The thresholds for small, moderate, and large effects were 0.20, 0.50, and 0.80, respectively [27].

A 2 x 2 contingency table was used to evaluate the accuracy of the markers for the assessment of fatigue and recovery in comparison to the criterion measure (i.e., RSA). The table was composed of horizontal lines to indicate the presence or absence of fatigue (in accordance with changes in surrogate markers) and vertical lines to indicate the “true” condition of an athlete according to the criterion measure of fatigue. Diagnostic effectiveness (proportion of athletes correctly categorized by the surrogate marker), misclassification rate (proportion of athletes, who were incorrectly classified by the surrogate marker) and Youden’s index (ranges from 0 for a poor accuracy to 1.0 for an excellent accuracy of the surrogate marker) were calculated from the constructed table [28]. Finally, multiple regression analysis was used to assess relationships between changes in surrogate markers and criterion measure of fatigue and recovery. For all statistical analyses, level of significance was set at p < 0.05.

Results
No significant time x sex interaction (p = 0.566) but a significant main effect for time (p = 0.010) was found for RSA test performance. MV was significantly lower following the six-day training intervention (post1: 4.84 ± 0.56 m s⁻¹) than at baseline (pre: 5.02 ± 0.32 m s⁻¹) or after recovery (post2: 4.97 ± 0.56 m s⁻¹). The respective changes were -0.18 ± 0.13 m s⁻¹ (p = 0.001; effect size = -1.51) from pre to post1, and 0.12 ± 0.26 m s⁻¹ (p = 0.003; effect size = 0.53) from post1 to post2. Differentiated by sex, markers of fatigue and recovery are illustrated in Fig2, Fig 3 and Fig 4. There were no significant time x sex interactions with respect to any of the determined markers. However, a significant main effect for time was found for CMJ, MJR, and 20-m sprint performance, as well as for contraction time of the RF and BF. CK, CRP, and DOMS. For CMJ and MJR performance, a significant decline and a return to baseline level after 72 h of recovery could be observed (Table 3). In addition, athletes demonstrated a significant increase in CK and DOMS following the training program and a significant decrease after the recovery period (Table 3). The HIIT-microcycle also induced a significant increase in 20-m sprint time and contraction time of the RF and BF at post, compared to baseline values. However, these increases were not reversible between post1 and post2 (Table 3). Dm of the RF and BF, as well as CRP, and urea were not different at post1 and post2 compared to baseline values (Table 3).

Diagnostic effectiveness, misclassification rate and Youden’s index for surrogate markers of fatigue and recovery are shown in Table 4. None of the surrogate markers showed sufficient accuracy to discriminate athletes in a fatigued or recovered state in relation to RSA. Multiple regression analysis also revealed no significant correlations (p > 0.05) between changes in RSA and any of the surrogate markers.

Discussion
The purpose of the present study was to investigate the accuracy of selected markers to reflect changes in fatigue and recovery in male and female team sport athletes during and after HIIT.
Fig 2. Mean (± SD) countermovement jump (CMJ) height (A), multiple rebound jumps (MRJ) performance (B) and 20-m sprint time (C) in males and females at baseline (pre), after a six-day high-intensity interval training program (post), and following 72 h of recovery (postL). RSI = reactive strength index. *Significant difference compared to pre \( (p < 0.05) \). †Significant difference compared to post, \( (p < 0.05) \).
The main finding of this study was that a six-day HIIT program induced significant changes in RSA, showing a temporary decline and a return to baseline level after 72 h of recovery. The decrease in RSA indicates that the training program induced a temporary state of fatigue. However, regular RSA testing for a routine assessment of fatigue and recovery may be unduly fatiguing and impractical for most athletes [29]. In this regard, the present study demonstrated that CMJ, MRI, TMG Tc, CK and DOMS are potential markers of higher practicability and less demanding. This was evident in significant changes in these markers following the training period and after 72 h of recovery. However, due to an insufficient accuracy of these markers in
differentiating between fatigued and recovered athletes, their responses to HIIT and their associations with fatigue and recovery appear to be highly individual. Since changes in markers of fatigue and recovery of males and females tended to be the same, these findings apply equally for both sexes.

Monitoring fatigue and recovery through measures of jump or sprint performance is recently utilized in the team sport environment due to its simplicity of administration, the minimal amount of additional fatigue induced, and its high reproducibility and validity [8, 9]. Therefore, we used the CMJ, the MJR, and the 20-m linear sprint to monitor changes in the
Table 3. Markers of fatigue and recovery at baseline (pre), after a six-day high-intensity interval training program (post1), and following 72 h of recovery (post2) as well as percentage changes of performance and muscle contractile markers between testing days.

<table>
<thead>
<tr>
<th>Markers</th>
<th>Time</th>
<th>pre</th>
<th>post1</th>
<th>post2</th>
<th>pre-post1</th>
<th>post1-post2</th>
<th>pre-post2</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Performance markers</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CMJ (cm)</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>36.4±6.8</td>
<td>33.3±6.0*</td>
<td>35.4±6.0</td>
<td>&lt;0.001</td>
<td>-5.4±3.9</td>
<td>-1.35</td>
</tr>
<tr>
<td>MRJ (RIS)</td>
<td></td>
<td>1.79±0.28</td>
<td>1.66±0.91*</td>
<td>1.46±0.36</td>
<td>&lt;0.001</td>
<td>-1.7±4.5</td>
<td>-1.60</td>
</tr>
<tr>
<td>20 m Sprint (s)</td>
<td></td>
<td>3.28±0.24</td>
<td>3.18±3.35*</td>
<td>3.40±0.36</td>
<td>&lt;0.001</td>
<td>3.4±1.8</td>
<td>-0.81</td>
</tr>
<tr>
<td><strong>Muscle contractile markers</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>RF Tc (ms)</td>
<td></td>
<td>29.0±3.8</td>
<td>27.3±3.0</td>
<td>31.7±4.8</td>
<td>&lt;0.002</td>
<td>9.9±5.9</td>
<td>-0.72</td>
</tr>
<tr>
<td>F Dm (mm)</td>
<td></td>
<td>8.6±2.1</td>
<td>7.7±5.9</td>
<td>8.3±2.2</td>
<td>&lt;0.011</td>
<td>-1.7±10.3</td>
<td>-0.17</td>
</tr>
<tr>
<td>BF Tc (ms)</td>
<td></td>
<td>37.1±10.7</td>
<td>29.0±4.5</td>
<td>44.9±12.9</td>
<td>&lt;0.042</td>
<td>28.7±24.9</td>
<td>-0.46</td>
</tr>
<tr>
<td>BF Dm (mm)</td>
<td></td>
<td>8.5±3.3</td>
<td>7.1±5.0</td>
<td>7.7±2.9</td>
<td>&lt;0.037</td>
<td>-6.8±12.2</td>
<td>-0.39</td>
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<tr>
<td><strong>Biochemical markers</strong></td>
<td></td>
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<tr>
<td>CK (U L⁻¹)</td>
<td></td>
<td>147±61</td>
<td>125±70</td>
<td>161±140</td>
<td>&lt;0.001</td>
<td>-9.9±9.9</td>
<td>-0.99</td>
</tr>
<tr>
<td>CRP (mg L⁻¹)</td>
<td></td>
<td>1.5±2.6</td>
<td>2.0±2.7</td>
<td>2.2±3.0</td>
<td>&lt;0.029</td>
<td>-0.3±3.0</td>
<td>-0.68</td>
</tr>
<tr>
<td>UREA (mg dl⁻¹)</td>
<td></td>
<td>29.6±7.2</td>
<td>26.1±30.0</td>
<td>30.9±9.4</td>
<td>&lt;0.080</td>
<td>-2.6±5.9</td>
<td>-0.05</td>
</tr>
<tr>
<td><strong>Subjective markers</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DOMS (mm)</td>
<td></td>
<td>0.2±0.1</td>
<td>0.1±0.3</td>
<td>2.7±5.8</td>
<td>&lt;0.001</td>
<td>-1.5±1.4</td>
<td>-1.44</td>
</tr>
</tbody>
</table>

Parameters are shown as mean ± SD (95% confidence interval). CI: 95% confidence interval; D: Cohen d effect size; CMJ: countermovement jump; MRJ: multiple rebound jumps; RSI: reactive strength index; RF: rectus femoris; Tc: contraction time; Dm: muscle belly displacement; CK: creatinokinase; CRP: C-reactive protein; DOMS: delayed onset muscle soreness.

*Significant difference compared to pre.
#Significant difference compared to post1.

doi:10.1371/journal.pone.0133890.003
Table 4. Accuracy of markers of fatigue and recovery in relation to the criterion measure.

<table>
<thead>
<tr>
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<th>Diagnostic effectiveness (%)</th>
<th>Miscategorization rate (%)</th>
<th>Youden’s index</th>
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<tr>
<td></td>
<td>Δpre-post,1</td>
<td>Δpost,-post,2</td>
<td>Δpre-post,1</td>
</tr>
<tr>
<td>Performance markers</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>CMJ (cm)</td>
<td>63.6</td>
<td>60.0</td>
<td>36.4</td>
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<tr>
<td>MPJ (RSTI)</td>
<td>68.2</td>
<td>60.0</td>
<td>31.8</td>
</tr>
<tr>
<td>20-m Sprint (s)</td>
<td>77.3</td>
<td>33.3</td>
<td>22.7</td>
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<td>Muscle contractile markers</td>
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<tr>
<td>RF Tc (ms)</td>
<td>68.2</td>
<td>40.0</td>
<td>31.8</td>
</tr>
<tr>
<td>RF Dm (mm)</td>
<td>50.0</td>
<td>66.7</td>
<td>50.0</td>
</tr>
<tr>
<td>BF Tc (ms)</td>
<td>54.5</td>
<td>40.0</td>
<td>45.5</td>
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<tr>
<td>BF Dm (mm)</td>
<td>50.0</td>
<td>60.0</td>
<td>50.0</td>
</tr>
<tr>
<td>Biochemical markers</td>
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<td></td>
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<tr>
<td>CK (U·L⁻¹)</td>
<td>50.0</td>
<td>53.3</td>
<td>50.0</td>
</tr>
<tr>
<td>CRP (mg·L⁻¹)</td>
<td>31.8</td>
<td>46.7</td>
<td>68.2</td>
</tr>
<tr>
<td>UREA (mg·dL⁻¹)</td>
<td>30.0</td>
<td>46.7</td>
<td>70.0</td>
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<tr>
<td>Subjective markers</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DOMS (mm)</td>
<td>45.6</td>
<td>60.0</td>
<td>54.5</td>
</tr>
</tbody>
</table>

CMJ: countermovement jump; MPJ: multiple rebound jumps; RSTI: reactive strength index; RF: rectus femoris; BF: biceps femoris; Tc: contraction time; Dm: muscle belly displacement; CK creatine kinase; CRP: C-reactive protein; DOMS: delayed onset muscle soreness.

doi:10.1371/journal.pone.0139801.t004

athlete’s neuromuscular function of the lower limbs during the six-day training intervention [22,30]. In this study jump performance (i.e., jump height and jump efficiency) followed the changes in repeated sprint ability with a decrease in performance after the training period (CMJ: -8.4 ± 6.6% MRJ: -17.4 ± 10.2%) and an increase of performance following the recovery period (CMJ: 4.9 ± 8.3%; MRJ: 8.5 ± 13.9%). Since the CV of the CMJ and MRJ performance was 3.7% and 4.0% respectively, the magnitude of changes can be considered to be of practical relevance. Linear sprint performance (CV = 1.8%) also showed a practically relevant decrease following the six-day training program of HIIT (3.4 ± 4.1%), but only tended to increase following the recovery period (-1.1 ± 3.4%).

Failure in the neuromuscular system responsible for altered performance can be explained by a combination of central and peripheral factors involving mechanisms from the central nervous system (e.g., impaired activation or reduced motivation) to exercise-related changes within the muscle fibers itself [9, 31, 32]. However, a decline in performance following exercise-induced fatigue has been demonstrated to be located peripherally (i.e., structural damage of muscle fibers, excitation-contraction coupling failure, redistribution of sarcomere length, impaired metabolism) rather than centrally [33, 34]. Since HIIT has the potential to induce muscle damage [33], it appears that the decreases in vertical jump height, jump efficiency (i.e., reactive strength index), and sprint performance may be related to repeated structural damage and inflammatory response of the muscle fibers caused by the HIIT program [29, 34]. It was shown that when muscle damage was induced through intense exercises, there were prolonged decreases in maximal force, ground reaction force, stretch–reflex sensitivity, muscle joint stiffness regulation and, thus, a reduction in jump and sprint performance [33]. Since the jump performance almost reached baseline levels and sprint performance showed a trend to increase following 72 h of recovery, these findings suggest that the CMJ, MRJ and 20-m sprint test may be potential tools to measure both fatigued and recovered neuromuscular function of team sport athletes following HIIT.
In addition to performance tests, measurements of selected blood markers under standardized conditions are proposed to monitor fatigued and recovered conditions [11]. In the practical team sport surrounding, routine blood parameters such as CK, CRP, and urea collected via capillary blood samples, are popular measures due to the simplicity of sample collection and analysis [7, 8, 12, 35]. In this study, CK reacted to the HIIT program, showing an average elevation of >1000 U/L after the training period and a decrease to almost baseline levels following the recovery period. However, no changes in CRP und urea could be observed between baseline, post1, and post2.

Serum CK activity mirrors the mechanical-muscular strain of the training since CK leak into the plasma from skeletal muscle fibers when they are damaged, including membrane damage and myofibrillar disruptions characterized by myofilament disorganization and loss of Z-disk integrity [9, 11]. Therefore, the elevated CK activity determined at post reveals the explanation that damaged muscle fibers were partially responsible for the decline in performance. Similar to the present results, various studies with team sport athletes reported increased CK concentrations following intensified training or competition periods [12, 35–37]. The most likely explanation for the extremely high CK levels measured in this study was the characteristic of HIIT with its accelerations and decelerations as well as the changes of direction leading to high eccentric biomechanical strain on the working muscles, which in turn causes microinjuries of the musculoskeletal system and perceived muscle soreness [12, 33]. In this study, muscle soreness, which was measured subjectively by a VAS, followed the time course of CK activity (Table 3). DOMS increased following the training period and decreased after 72 h of recovery. Therefore, both the objective CK and subjective DOMS measures seemed to have the potential to identify HIIT-induced muscle damage associated with the fatigue and recovery observed in this study’s team sport athletes.

In this context, however, the high variability of measure of CK activity must also be taken into account [8]. Some athletes are non-responders due to a lower permeability of muscle cell membranes and only show small increases in CK activity [11]. Conversely, athletes with high percentages of fast twitch muscle fibers might tend to produce higher CK values [12]. Furthermore, sex could affect the magnitude of CK activity, which is due to a potentially higher CK content of men’s muscle than that of women’s muscle [9, 12, 30]. This assumption is supported by our data, since the mean CK concentration at post1 was 64.8% higher in the male compared to female participants (Fig 3). Therefore, athletes’ individual physical characteristics should be considered when using CK as an indicator of fatigue and recovery. One should also pay attention when solely using DOMS as a marker of fatigue and recovery. Since muscle function is impaired before soreness arises, and functional impairment may also persist when soreness has dissipated, this could lead to problems in an applied environment [33]. If solely the dissipation of muscle soreness is used as a signal to resume regular training, muscle function can be still in a weakened state and the risk of injury would be increased.

Since subsequent muscle damage is also linked to local inflammatory processes [9], the use of CRP may provide important additional information on the athlete’s status. However, despite an increase in CK activity in this study, no relevant changes in CRP could be determined following the HIIT-program (Table 3). In this context, Singh et al. [39] compared the effects of intermittent running, either with or without body ‘contact’, on muscle damage and inflammatory response. They demonstrated that both ‘contact’ and ‘non-contact’ training resulted in elevated serum CK, while CRP only increased following training with body ‘contact’. Since the addition of tackling to intermittent training further increased muscle damage following exercise, one can speculate that a certain degree of muscle damage requires ‘contact’ to significantly alter serum concentration of CRP. Based on the present results and due to the fact that potential interferences with inflammation are not directly related to muscle damage, it appears that...
CRP may not be a useful and specific enough marker for monitoring fatigue and recovery following HIIT.

This is also valid for urea, since serum concentrations were not altered at post1 and post2 compared to baseline values. Increased serum concentration of urea is a marker of enhanced protein catabolism and stimulated gluconeogenesis that results from high training volumes and increased energy consumption [12]. Since training volume during the HIIT-period was rather low (35 min per HIIT session; Table 2), no changes in urea and, thus, in the ‘anabolic-catabolic balance’ could be observed. This is in line with the findings by Coutts et al., [35] who reported unaltered urea serum concentrations following intensified training in rugby players.

Recent articles also recommend measures of muscle contractile properties as an effective method for detecting fatigue and recovery in athletes. In this context, TMG was introduced as an involuntary and non-invasive method to measure muscle contractile characteristics (i.e., Tc which is related to the speed of force generation, and Dm, which is representative of muscle tone and contractile force) [13]. Several studies have highlighted its usefulness for practitioners and researchers in detecting muscle damage and its recovery following various forms of exercises (i.e., eccentric exercise, endurance exercise, soccer) [13, 40–42]. For HIIT, the muscles affected most will be the extensor muscles of the knee joint (in the landing and take-off stages) and their antagonist muscles (traction in rear foot and leg recovery) [40]. Therefore, the muscle contractile characteristics of the RF and BF were measured through TMG in this study. Tc observed for both muscles significantly increased after the six-day training program and showed a trend for a decrease between post1 and post2. Dm was unaltered during all testing days.

Decreased Dm and increased Tc have been explained by a reduced efficiency of the excitation-contraction coupling, impairment in membrane conducting properties, and cellular structures destruction (i.e., peripheral fatigue) [43]. In this context, previous studies were able to demonstrate a decline in Dm and an increase in Tc when exercise-induced muscle damage (e.g., elevated CK activity and muscle soreness) was present [13, 42]. Since CK activity and DOMS were increased following the six-day HIIT-period, it can be concluded that Dm measured via TMG cannot be considered as a useful marker for monitoring fatigue and recovery following HIIT. On the other hand, due to an increase at post1 and a trend for a decrease at post2, Tc of the RF and BF may be a potential marker for monitoring fatigue and recovery.

As highlighted in the previous sections, measures of neuromuscular function, CK and DOMS are potentially useful markers for monitoring of team sport athletes during intensive training cycles. However, in relation to measures of sport-specific performance (i.e., RSA), which is demonstrably the most valid method for the assessment of fatigue and recovery, [8] none of the surrogate markers showed the ability to completely discriminate between fatigued and recovered athletes. Additionally, multiple regression analyses revealed that there were no relationships between changes in RSA and any of the surrogate markers. These findings indicate that responses of markers of fatigue and recovery to a given training stimulus are highly individual and variable, as already emphasized by Nickel et al. [9] and Halson [8]. Additionally, Andersson et al. [37] showed that the time course of the fatigue and recovery pattern differs significantly between various neuromuscular and biochemical markers. They demonstrated that CMJ performance, CK activity and muscle soreness were still changed 74 h following a football match, whereas sprint performance returned to baseline level already 5 h after the match. This could be a further explanation for the weak relationships between changes of surrogate markers and the criterion measure of fatigue. Consequently, accuracy of a single or combined use of CMJ, MRI, 20-m sprint test, Tc, CK, and DOMS for the routine assessment of fatigue and recovery and their associations with sport-specific performance needs to be identified in practice for each athlete on an individual and longitudinal basis.
Study limitations

First, although high VO\textsubscript{2max} values were measured among the participants and most players were members of regional representative teams, the question remains whether the present results can be transferred to professional team sports at the international level. Effects might have been different with a group of high-level athletes. However, we have consciously refrained from recruiting elite players for this standardized research approach due to the reluctance of such populations to deviate from their normal training routine. Second, there was no control group to provide a baseline during the experimental period. In this regard, however, we have stated reliability data to indicate practically relevant changes in markers of fatigue and recovery. Third, the selection of markers that were evaluated in the present study might be considered a further limitation. There are especially some psychological markers (e.g., Recovery-Stress Questionnaire for Athletes [43]) that have been proposed in the literature as instruments to track the fatigue and recovery process and that have not been evaluated in the current investigation. However, the present study was not designed to analyze the highest possible number of markers of fatigue and recovery, but to evaluate a well-founded selection of practical tests that can be easily applied in team sports.

Conclusions

The challenge for coaches and athletes is to determine the point at which intensive demands in training and competition lead to non-functional overreaching and may negatively affect the performance in upcoming competitions [8]. Therefore, routine assessment of fatigue and recovery is of importance to improve individual training prescription and to ensure competition readiness. To estimate changes in neuromuscular function following HIIT regardless of sex, this study was able to show that the power ability and reactive strength (i.e., CML, MRL, 20-m sprint) in the lower body as well as Tc of the RF and BF are potentially useful markers.

However, in an applied environment, individual athletes respond differently to a given training stimulus, evidenced by the insufficient accuracy of the markers for monitoring fatigue and recovery in relation to the criterion measure. Therefore, surrogate markers should be assessed regularly in practice and with enough frequency to give the desired information to the athlete or coach. In this context, a possible recommendation for professional teams is to provide a fixed installation of a contact platform at the training ground to incorporate jump performance measurements as a daily routine. Also subjective assessment of DOMS using a visual analogue scale can be considered as a potential tool to identify team sport athletes who are susceptible to non-functional overload. In addition, CK as a routine blood marker may help to monitor the mechanical-muscular strain of HIIT. However, neither marker alone, nor specific group of markers significantly correlated with the criterion measure of fatigue. Therefore, a combination of the aforementioned markers should be used in practice in order to take into consideration all potential mechanisms that contribute to fatigue.

Supporting Information

S1 Data Set.
(XLSX)

Author Contributions

Conceived and designed the experiments: TW CR TM MK MP AF. Performed the experiments: TW CR AF. Analyzed the data: TW CR AF. Contributed reagents/materials/analysis tools: TW CR TM MK MP AF. Wrote the paper: TW CR TM MK MP AF.
References


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