BELASTUNGS- UND ERHOLUNGSSTEUERUNG IM HIGH-INTENSITY AUSDAUERTRAINING

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BELASTUNGS- UND ERHOLUNGSSTEUERUNG IM HIGH-INTENSITY AUSDAUERTRAINING

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INHALTSVERZEICHNIS

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1 EINLEITUNG

Die Leistungsentwicklung im Sport steht in ständiger Wechselwirkung mit den durch Trainings- und Wettkampfaktivitäten ausgelösten Ermüdungs- und Erholungsprozessen. So resultiert eine sportliche Belastung stets in einer reversiblen (im Sinne der Superkompensation beabsichtigten) Abnahme der Leistungsfähigkeit, die in Abhängigkeit von Belastungsumfang und Belastungsintensität erst nach einigen Minuten, Stunden oder Tagen abklingt (Barnett, 2006a). Bleiben notwendige Erholungszeiten bei der Trainings- und Wettkampfplanung unberücksichtigt, nimmt das Risiko für chronische Überlastungsreaktionen zu (Meeusen et al., 2013). Dies betrifft insbesondere den Spitzensport, da Trainingsumfang, Trainingsintensität und Trainingsdichte sowie Wettkampfhäufigkeit und Leistungsdichte in vielen Disziplinen in den letzten Jahren deutlich angestiegen sind.

Während die Optimierung der Trainingsqualität seit jeher im Fokus trainingswissenschaftlicher und sportmedizinischer Bemühen 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).

Besondere Aufmerksamkeit gebührt in diesem Kontext dem hochintensiven Intervalltraining, das inzwischen sowohl im internationalen als auch im deutschen Sprachraum häufig als High-Intensity Ausdauertraining oder High-Intensity Interval Training (HIT) bezeichnet wird. Einerseits ist das HIT eine in zahlreichen Sportarten etablierte und vielfach angewandte Trainingsmethode zur Verbesserung allgemeiner und sportartspezifischer Ausdauerleistungskomponenten, deren Belastungsreaktionen aufgrund der verschiedenen Gestaltungsmöglichkeiten jedoch stark variieren können (Tschakert & Hofmann, 2013). Andererseits repräsentiert das HIT aufgrund der hohen Trainingsintensität eine Belastungsform mit meist erheblichem regenerativen Folgebedarf (Byrne, Twist, & Eston, 2004). Ziel der vorliegenden kumulativen Dissertation ist es folglich, evidenzgestützte und für die leistungssportliche Praxis anwendbare Handlungsempfehlungen zur Belastungs- und Erholungssteuerung im High-Intensity Ausdauertraining zu erarbeiten. Dies ist trotz des hohen Erholungsbedarfs nach HIT bislang nicht zufriedenstellend erfolgt. Entsprechende Fragestellungen wurden auf der Basis eines mehrstufigen Untersuchungsdesigns analysiert und die Ergebnisse im Rahmen von drei Beiträgen in internationalen Fachzeitschriften publiziert.

2 THEORETISCHER HINTERGRUND

High-Intensity Ausdauertraining wird bereits seit Beginn des 20. Jahrhunderts in vielen ausdauerdeterminierten Sportarten angewandt. Insbesondere Athleten aus leichtathletischen Disziplinen wie dem Mittel- und Langstreckenlauf verhalf diese Variante des Intervalltrainings zu außergewöhnlichen Wettkampfleistungen (Tschakert & Hofmann, 2013). So gelten die Siege von Emil Zàtopek im 5.000- und 10.000-Meter-Lauf sowie im Marathonlauf bei den Olympischen Spielen 1952 in Helsinki bis heute als legendär. Der aus der Tschechoslowakei stammende Weltrekordler absolvierte während der Wettkampfvorbereitung dabei täglich bis zu 100 × 400-Meter-Läufe (Billat, 2001). Infolge der umfangreichen wissenschaftlichen Evaluation von physiologischen Reaktionen und Adaptationen auf ein hochintensives Ausdauertraining erlangte diese Trainingsmethode in den vergangenen zwei Jahrzehnten speziell in den Spielsportarten viel Aufmerksamkeit (Tschakert & Hofmann, 2013). Dies liegt darin begründet, dass wesentliche Vorteile des HIT gegenüber klassischen Ausdauertrainingskonzepten bei der Verbesserung der sportspielspezifischen Ausdauerleistungsfähigkeit identifiziert werden konnten.

Eine Vielzahl von Studien hat gezeigt, dass HIT im Vergleich zum traditionellen umfangsorientierten Ausdauertraining bei deutlich reduziertem Trainingsumfang zu ähnlichen Anpassungseffekten führt (Burgomaster et al., 2008; Faude, Schnittker, Schulte-Zurhausen, Müller, & Meyer, 2013; Gibala et al., 2006; Rakobowchuk et al., 2008). Angesichts der komplexen Beanspruchungsprofile verschiedener Spielsportarten ergibt sich daraus insbesondere für leistungs- und wettkampforientierte Athleten ein bedeutsamer Nutzen. Die Verbesserung von Kraft, Schnelligkeit und Beweglichkeit sowie koordinativ-technischer und taktischer Fähigkeiten stellen hier wichtige konkurrierende Trainingsinhalte dar. Durch die günstigere Aufwand-Nutzen-Relation des HIT kann folglich die für die Steigerung der Ausdauerleistungsfähigkeit übrig bleibende Trainingszeit ökonomischer genutzt werden. Ferner ermöglicht die intermittierende Belastungsstruktur beim HIT sowie die damit verbundenen spezifischen Adaptationen eine für intervallbasierte Sportarten valide Wettkampfvorbereitung (Iaia, Rampinini, & Bangsbo, 2009). So versetzt HIT Sportspieler langfristig in die Lage, intensive Aktivitäten während des Wettkampfs über einen längeren Zeitraum aufrechtzuerhalten und zwischen Belastungsspitzen schneller zu regenerieren (Cicioni-Kolsky, Lorenzen, Williams, & Kemp, 2011).

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.

2.1 Belastungsreaktionen im High-Intensity Ausdauertraining

HIT ist durch hochintensive intermittierende Belastungsphasen gekennzeichnet, die zwischen einigen Sekunden und mehreren Minuten andauern und in Abhängigkeit ihrer Dauer unterschiedlich oft wiederholt werden. Die Belastungsphasen werden durch aktive oder passive Erholungsphasen unterbrochen, woraus sich das für ein HIT charakteristische Intervallmuster ergibt (Buchheit & Laursen, 2013a). Dabei kann die Belastung als Lauf- oder Sprintaktivität mit oder ohne Richtungswechsel oder unter Einbau sportartspezifischer Bewegungsmuster absolviert werden (McMillan, Helgerud, Macdonald, & Hoff, 2005).

2.1.1 Akute Belastungsreaktionen

Zumeist wird zwischen einem HIT mit kurz bis lang andauernden hochintensiven, aber meist submaximalen (<100% der maximalen Sauerstoffaufnahme [$\dot{V}O_2max$]) der Belastungsphasen und einem sprintbasierten HIT mit supramaximalen (>100% der er $\dot{V}O_2max$) „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

Während der eher kurzen Belastungsphasen einer intensiven intermittierenden körperlichen Arbeit reichen die Respiration sowie der Sauerstofftransport zur Arbeitsmuskulatur nicht aus, um den O_2-Bedarf zu decken. Dies liegt an der zeitverzögerten Antwort von $\dot{V}O_2$ und resultiert in einer ATP-Resynthese über intrazellulär gespeicherten Sauerstoff und/oder anaerobe Stoffwechselwege. Somit spielen Oxymyoglobin als intrazellulärer O_2-Speicher sowie Phosphokreatin (PCr) als energiereiches Phosphat beim HIT eine wichtige Rolle, da sie insbesondere in den ersten Sekunden der Belastungsintervalle die Energieversorgung der Arbeitsmuskulatur gewährleisten. Zudem werden die intrazellulären Sauerstoff- und PCr-Speicher in den Erholungsphasen rasch wieder aufgefüllt (Tschakert & Hofmann, 2013).

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

hierbei Laktat (Milchsäure) gebildet, das aufgrund des Konzentrationsgefälles im Verlauf in den Blutkreislauf diffundiert. In diesem Zusammenhang konnten Studien nachweisen, dass Intervalle mit einer Dauer von 2 – 4 min im Vergleich zu kürzeren Intervallen zu deutlich höheren Blutlaktatkonzentrationen (bis zu 17,0 mmol/l) führen (Astrand, Astrand, Christensen, & Hedman, 1960; Christensen, Hedman, & Saltin, 1960). Die damit vermehrt in Lösung gehenden H^+-Ionen beeinflussen in der Folge den pH-Wert und somit den Säure-Basen-Status des Blutes. Die Laktatbildungsrate ist abhängig von der Belastungsintensität, während die Menge des produzierten Laktats das Produkt von Belastungsintensität und Belastungsdauer darstellt. Liegt die Belastungsintensität längerer Intervalle über dem maximalen Laktat-Steady-State (maxLaSS), führt dies im Trainingsverlauf zu einer Akkumulation der Blutlaktatkonzentration. Kürzere Intervalle (30 s) mit „all-out"-Belastungen (z.B. Sprints) können aufgrund der extrem hohen Intensität ebenfalls eine Blutlaktatakkumulation verursachen. Bei kürzeren submaximalen Intervallen wird die Energieversorgung jedoch vorrangig über das intrazellulär gespeicherte O_2 und PCr gewährleistet, sodass Laktat lediglich in vergleichsweise kleinen Mengen produziert wird. Trotz der kurzen Intervallpausen reicht die Erholungszeit hierbei aus, um ein Gleichgewicht zwischen Laktatbildung und Laktatelimination herzustellen (Tschakert & Hofmann, 2013).

Intensive Belastungen im Bereich eines Laktat-Steady-State können länger toleriert werden. Infolgedessen sind höhere Trainingsumfänge realisierbar. Andererseits sind hohe Blutlaktatkonzentrationen Auslöser spezifischer und für viele Athleten relevanter Adaptationen (z.B. verbesserte Laktattoleranz). Zudem wird vermutet, dass die Laktatakkumulation zu einer Inhibierung der mit dem Enzym Phosphofructokinase in Verbindung stehenden Stoffwechselmechanismen führt und in der Folge eine Feedback-Hemmung anaerob-glykolytischer Prozesse verursacht (McCartney et al., 1986; Parolin et al., 1999). Dadurch wird der Organismus gezwungen, das notwendige ATP aerob zu resynthetisieren. Dies könnte eine Ursache für die im Rahmen von HIT beobachteten Anpassungen aerober Ausdauerleistungskomponenten sein (Tschakert & Hofmann, 2013). So dokumentierten Stepto, Martin, Fallon & Hawley (2001) während eines HIT mit 5-minütigen Belastungsintervallen eine Kohlenhydratoxidationsrate von 340 μmol/kg/min bei deutlich geringerer jedoch im Belastungsverlauf ansteigender Fettoxidationsrate (16 – 25 μmol/kg/min). Dies spricht für eine ansteigende Aktivität aerober Stoffwechselprozesse im Trainingsverlauf, was entweder auf einen Abfall der Belastungsintensität und/oder auf eine Hemmung anaerober Mechanismen zurückgeführt werden kann.

Pulmonale Belastungsreaktionen

Im Rahmen des HIT gilt eine möglichst lange Ausschöpfung eines hohen Anteils (< 90%) der $\dot{V}O_2$max als wichtigstes Kriterium, um die erwünschten physiologischen Anpassungseffekte auszulösen. Die $\dot{V}O_2$max wird sowohl durch die pulmonale als auch die kardiovaskulär-metabolische

Kapazität beeinflusst (Meyer & Kindermann, 1999). Daher geht die in der Nähe der $\dot{V}O_2$max verbrachte Zeit (t@$\dot{V}O_2$max) mit einer Beanspruchung aller beteiligten Mechanismen einher. Folglich reflektiert die t@$\dot{V}O_2$max sowohl die pulmonalen als auch die metabolischen, kardialen und vaskulären Belastungsreaktionen.

Da zur Erreichung der $\dot{V}O_2$max 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@$\dot{V}O_2$max (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 $\dot{V}O_2$-Oszillationsraten führt. Da beispielsweise durch ein Wiederholungssprinttraining bei deutlich geringerer $\dot{V}O_2$max-Ausschöpfung jedoch ebenfalls Verbesserungen aerober Ausdauerleistungskomponenten (u.a. $\dot{V}O_2$max) 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@$\dot{V}O_2$max, sondern auch die Belastungsintensität adaptationsrelevant zu sein (Tschakert & Hofmann, 2013). In diesem Zusammenhang führten Studien die Verbesserung der $\dot{V}O_2$max durch ein Wiederholungssprinttraining auf Anpassungen des muskel-metabolischen Potentials zurück, während HIT-Protokolle mit längeren Intervallen die $\dot{V}O_2$max vor allem durch eine verbesserte kardiovaskuläre Kapazität steigerte (Tschakert & Hofmann, 2013).

Kardiale Belastungsreaktionen

Entsprechend der $\dot{V}O_2$-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@ $\dot{V}O_2$max 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

Verhaltensmuster bestimmt wird (Buchheit & Laursen, 2013a). Zudem ist der Einfluss von unterschiedlichen HIT-Protokollen auf das SV bislang nur selten evaluiert worden.

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 $\dot{V}O_2$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

Die in Abhängigkeit der gewählten Belastungsnormative auftretenden Ermüdungsphänomene weisen zunächst unterschiedliche zeitliche Dimensionen auf und können sowohl einzelne Muskelgruppen als auch den Gesamtorganismus betreffen. Die kurzfristigste Form der Ermüdung ereignet sich bereits während jeder einzelnen Muskelkontraktion. So führt beispielsweise die Abstoß- bzw. Stützphase während des Laufens zu einer unmittelbaren Ermüdung der beanspruchten Muskulatur, indem Adenosintriphosphat verbraucht wird und während der Schwungphase resynthetisiert werden muss (Bishop et al., 2008). Ferner kann zwischen akuten und mittelfristigen Formen der Ermüdung unterschieden werden. Erstere sind Ermüdungsmechanismen im direkten Anschluss an eine akute muskuläre Belastung und wurden bereits in zahlreichen Publikationen ausführlich diskutiert. Sie führen während HIT dazu, dass die Leistung infolge zu kurzer Intervallpausen im Verlauf einer Trainingseinheit kontinuierlich abnimmt und die absolute Trainingsintensität nicht aufrechterhalten werden kann. Letztere können noch Tage oder Wochen nach einer HIT-Einheit festgestellt werden und die Leistungsbereitschaft während anschließender Trainings- oder Wettkampfbelastungen beeinflussen (Bishop et al., 2008; Hollmann & Strüder, 2009). Sie werden im Verlauf dieser Arbeit detaillierter beschrieben.

Neben der Differenzierung nach zeitlichen Merkmalen wird zwischen zentraler (Gehirn und Rückenmark) und peripherer (Muskeln) Ermüdung unterschieden (Ament & Verkerke, 2009). Periphere Ermüdung kann demzufolge als Unfähigkeit der beanspruchten Muskulatur zur Aufrechterhaltung einer gegebenen Leistung verstanden werden. Zentrale Faktoren beziehen sich auf die Ermüdung des zentralen Nervensystems (Hollmann & Strüder, 2009). Beide Formen der Ermüdung sind meist eng miteinander gekoppelt und beeinflussen sich gegenseitig (Weineck, 2004). Während die zentrale Ermüdung vor allem als Folge wiederholt ausgeführter Bewegungsabläufe mit hoher koordinativer Beanspruchung auftritt (Marées, 2003), ergeben sich im Anschluss an ein

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

Muskuläre Ermüdung

Indikatoren für eine mittelfristige muskuläre Ermüdung wurden von Thompson, Nicholas & Williams (1999) im Anschluss an ein HIT untersucht. Innerhalb der ersten 48 Stunden nach Belastungsende beobachteten sie eine signifikante Erhöhung der Creatinkinase- (CK) und Aspartat-Aminotransferase-Aktivität im Serum sowie des Schmerzempfindens in der beanspruchten Muskulatur. Diese Symptome waren mit denen vergleichbar, die nach ungewohnten mehrstündigen oder exzentrischen muskulären Belastungen gemessen wurden. Ähnliche Befunde legten Howatson & Milak (2009) vor. Sie zeigten, dass der CK-Wert im Blut sowie das Gefühl von Muskelschmerz nach einem einmalig absolvierten Intervalltraining gesteigert und die Leistungsfähigkeit für 72 Stunden herabgesetzt war.

Ursache für die Freisetzung muskelspezifischer Enzyme ins Blut sowie eines gesteigerten Schmerzempfindens und den damit zusammenhängenden Leistungseinbußen sind mikroskopisch kleine Muskelverletzungen (sog. Mikrotraumata) (Toigo, 2014). Diese werden speziell durch intensive dynamisch-negative (exzentrische) Muskelbeanspruchungen ausgelöst, da die hierbei auftretenden intramuskulären Kräfte meist um ein Vielfaches über denen bei statischer oder dynamisch-positiver (konzentrischer) Muskelarbeit liegen (Hollmann & Strüder, 2009). Der Schädigungsmechanismus kann dabei folgendermaßen erklärt werden: Bei exzentrischer Arbeit kommt es auf beiden Seiten der Z-Scheiben zu entgegengesetzten Zugbewegungen, da die kontraktilen Einheiten der Muskelzelle trotz der hohen äußeren Dehnungskräfte versuchen, ihre Kontraktionsarbeit zu verrichten und den Muskel bzw. das Sarkomer zu verkürzen. Die Folge sind Mikrotraumatisierungen bindegewebiger und filamentärer Muskelfaserstrukturen (Weineck, 2004).

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 gesteigerte 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.

Mikrotraumata sind dadurch charakterisiert, dass das myofibrilläre Strukturmuster durch Aufquellungen, Verbreiterungen und vielfältige mikroskopische Einrisse der Z-Scheiben verloren geht (Hollmann & Strüder, 2009). Durch die damit einhergehende Beschädigung des myofibrillären Cytoskeletts nimmt die Durchlässigkeit des Sarkolemms und anderer myofibrillärer Strukturen für intrazelluläre Proteine zu (Toigo, 2014). Diese diffundieren im Verlauf aus der Interzellularflüssigkeit ins Blut und helfen somit bei der Quantifizierung muskelspezifischer Ermüdungsphänomene (Ahonen, 2008; Nédélec et al., 2012). Die mechanisch bedingten Verletzungen sind in der Folge mit Entzündungsprozessen (Einwanderung von Entzündungszellen wie Makrophagen und Neutrophile) verbunden, welche die Gewebeverletzungen weiter verstärken. Zudem setzen die Entzündungszellen chemische Mediatoren wie Bradykinine und Prostaglandine frei, welche an die extrazellulären Schmerzrezeptoren binden und infolgedessen an der Schmerzempfindung beteiligt sind (Toigo, 2014). Das Gefühl von Muskelschmerz-, -schwellung und -steifigkeit manifestiert sich dabei gewöhnlich erst einige Stunden nach Belastungsende und kann durch reflektorische Verspannungen noch verstärkt werden (Hollmann & Strüder, 2009; Toigo, 2014).

Die durch intensive intervallbasierte Belastungen potentiell induzierten Muskelzellschädigungen 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 Signalmoleküle) des in Mitleidenschaft gezogenen Sarkolemms und/oder sarkoplasmatischen Retikulums 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.

Von ebenfalls wesentlicher Bedeutung ist die im Rahmen von HIT induzierte partielle Entleerung der Muskelglykogenspeicher infolge der hohen energetischen Flussrate sowie der Aktivierung der anaeroben Glykolyse. So konnten MacDougall, Ward & Sutton (1977) im Rahmen einer einmalig absolvierten hochintensiven und intervallbasierten Belastung eine Verringerung der Glykogenkonzentration von 28% beobachten. Die Auffüllung der Speicher benötigte dabei insgesamt 24 Stunden. Im unmittelbaren Anschluss an ein Fußballmatch sind die Glykogenspeicher in Abhängigkeit von der Anzahl der absolvierten hochintensiven Belastungsphasen sogar teils nahezu aufgebraucht (Bangsbo, Iaia & Krustrup, 2007). Für die komplette Auffüllung der Speicher werden

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 Muskelzellschädigungen zu einer gleichzeitigen Hemmung der Glykogenresynthese 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

Der Ursprung peripherer bzw. muskulärer Ermüdungserscheinungen liegt außerhalb des zentralen Nervensystems und führt im Verlauf durch Prozesse distal der neuromuskulären Synapsen zu einem Leistungsverlust (Ament & Verkerke, 2009). Zentrale Ermüdung bezieht sich sowohl auf das zentrale als auch das periphere Nervensystem und kann durch eine Abnahme des neuronalen Antriebs ebenso die Leistungsbereitschaft vermindern (Meeusen, Watson, Hasegawa, Roelands, & Piacentini, 2006).

So wird beispielsweise die Tryptophanüberschwemmung des Gehirns als Ursache für eine zentralbedingte Ermüdung diskutiert. Hierbei wird verstärkt Tryptophan durch Decarboxylierung zu Serotonin synthetisiert, dass als wichtiger Neurotransmitter und in bestimmten Gebieten des Gehirns angereichert unter anderem an der Schlafsteuerung und Förderung von Müdigkeit beteiligt ist (Hollmann & Strüder, 2009). Der Anstieg des Tryptophanspiegels im Gehirn wird dabei von zwei Prozessen ausgelöst, die speziell während langandauernder Ausdauerbelastungen eintreten. Einerseits verbraucht die Skelettmuskulatur unter Arbeitsbedingungen vermehrt verzweigtkettige Aminosäuren (BCAAs). Hierdurch verringert sich die Konzentration von BCAAs im Blut. Da BCAAs über den gleichen Carrier wie Tryptophan ins Gehirn gelangen, wächst infolge einer Veränderung des Tryptophan-BCAAs-Verhältnisses im Blut zugunsten des Tryptophans die Wahrscheinlichkeit, dass Tryptophan an der Blut-Hirn-Schranke einen Carrier zum Transport ins Gehirn findet (Ament & Verkerke, 2009; Newsholme & Blomstrand, 1995). Andererseits resultieren hochvolumige Ausdauerbelastungen in einer Zunahme der freien Fettsäuren (FFS) im Blut. Die erhöhte arterielle FFS-Konzentration lässt wiederum den Spiegel an ungebundenem Tryptophan ansteigen. Da lediglich freies Tryptophan über die Blut-Hirn-Schranke in das Gehirn gelangt, wächst ebenso durch den Anstieg der arteriellen FFS-Konzentration die Wahrscheinlichkeit einer Tryptophananschwemmung im Gehirn (Ament & Verkerke, 2009; Hollmann & Strüder, 2009). Die durch diese beiden Prozesse induzierte Erhöhung des Serotoninspiegels führt in der

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

Die durch muskuläre Belastungen induzierten Mikrotraumata gehen gewöhnlich mit einer gesteigerten Schmerzwahrnehmung einher (sog. Muskelkater). Die Muskelkatersymptomatik tritt dabei erst mehrere Stunden nach Belastungsende auf, erreicht seinen Höhepunkt nach etwa 2-3 Tagen und hält im Extremfall bis zu einer Woche an (Clarkson & Hubal, 2002; Weineck, 2004). Typischerweise entwickelt sich Muskelkater im Anschluss an ungewohnt intensive, aber insbesondere nach exzentrischen Muskelbeanspruchungen (Hollmann & Strüder, 2009; Weineck, 2004). Folglich hat auch HIT das Potential, Muskelkater zu verursachen. Dies konnte von mehreren Studien belegt werden, in denen eine signifikante Erhöhung der Schmerzwahrnehmung im Anschluss an ein HIT beobachtet wurde (Howatson & Milak, 2009; Thompson et al., 1999). Zwar besteht lediglich ein schwacher zeitlicher Zusammenhang zwischen dem Auftreten von Muskelkater und histologischen Befunden von Muskelzellschädigungen (Byrne et al., 2004). Jedoch kann das Gefühl von Muskelschmerz-, -schwellung und -steifigkeit in einer zentralnervösen Hemmung des neuromuskulären Aktivierungsverhaltens resultieren, infolgedessen die physische Leistungsfähigkeit gemindert ist (Byrne et al., 2004).

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 transkranieller Magnetstimulation (Bishop, 2012) sowie insbesondere mit Hilfe der Twitch-Interpolationstechnik nachgewiesen werden. Die Twitch-Interpolationstechnik erlaubt eine objektive Messung rekrutierter und nichtrekrutierter Anteile eines Muskels während einer isometrischen Kontraktion und ermöglicht so eine differenzierte Abschätzung peripher und zentral verursachter 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 Willkürinnvervation 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

Akute und mittelfristige Belastungs- und Ermüdungsreaktionen wurden unter Berücksichtigung aller potentiellen Belastungsnormative nur lückenhaft evaluiert. Vor allem die variablen Gestaltungsmöglichkeiten eines HIT sind dabei für die Belastungs- und Erholungssteuerung problematisch. Der Vergleich aktueller Publikationen macht deutlich, dass sich die untersuchten Trainingsprotokolle in der Wahl der Belastungsnormative teils erheblich voneinander unterscheiden (Tab. 1). Folglich variieren die akuten Belastungsreaktionen in Abhängigkeit der gewählten HIT-Protokolle (Tschakert & Hofmann, 2013) und somit gleichermaßen die akuten und mittelfristigen Ermüdungsreaktionen. Ziel des ersten Untersuchungsmoduls der vorliegenden kumulativen Dissertation war es demnach, akute Belastungsreaktionen sowie akute und mittelfristige Ermüdungs- bzw. Erholungseffekte im Rahmen unterschiedlicher HIT-Protokolle, die das breite Spektrum von Gestaltungsmöglichkeiten eines HIT abbildeten, zu evaluieren.

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

Autoren	Dauer	Intensität	Pause / Serienpause	Serien x Wdhl.
Clark et al. (2004)	*5 min*	85% VO_2max	60 s	1 x 8
Leveritt & Abernethy (1999)	*5 min*	40 – 70% of peak power output	300 s	1 x 5
Breil, Weber, Koller, Hoppeler, & Vogt (2010)	*4 min*	90 – 95% HFmax	180 s	1 x 4
Lamberts, Swart, Noakes, & Lambert (2009)	*4 min*	80% of peak power output	90 s	1 x 8
Ferrari Bravo et al. (2008)	*4 min*	90 – 95% HFmax	180 s	1 x 4
Driller, Fell, Gregory, Shing, & Williams (2009)	*2,5 min*	90% of peak power output	Variierend	1 x 8
Edge, Bishop, & Goodman (2006)	*2 min*	120 – 140% of lactate threshold	60 s	1 x 4 – 10
Laursen, Blanchard, & Jenkins (2002)	*1 min*	100% of peak power output	120 s	1 x 20
McKay, Paterson, & Kowalchuk (2009)	*1 min*	120% VO_2max	60 s	1 x 8 – 12
Creer, Ricard, Conlee, Hoyt, & Parcell (2004)	*30 s*	all out	240 s	4 x 10
Cicioni-Kolsky et al. (2011)	*30 s*	all out	150 s	1 x 12
Dupont, Akakpo, & Berthoin (2004)	*15 s*	120% of maximal aerobic speed	15 s	2 x 12 – 15
Helgerud et al. (2007)	*15 s*	90 – 95% HFmax	15 s	1 x 47
Fernandez, Zimek, Wiewelhove, & Ferrauti (2012)	*5 s*	all out	15 s / 8 min	3 x 10
Ferrari Bravo et al. (2008)	*40 m*	all out	20 s / 4 min	3 x 6
Tonnessen, Shalfawi, Haugen, & Enoksen (2011)	*40 m*	all out	90 s / 10 min	2 – 4 x 4 – 5

VO_2max = maximale Sauerstoffaufnahme; Hfmax = 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

Wichtigstes Außenkriterium bei der Erfassung von Ermüdung und Erholtheit ist die aktuell abrufbare sportartspezifische Leistungsfähigkeit. Die valideste Beurteilung des Ermüdungsstatus des Athleten liefern somit sportmotorische (Feld-)Tests, die Veränderungen spezifischer Leistungskomponenten mit ausreichender Sensitivität für relevante intraindividuelle Leistungsdifferenzen abbilden. Die Auswahl eines leistungsdiagnostischen Tests oder einer Testbatterie erfolgt dabei unter Berücksichtigung der sportart- oder belastungsspezifischen Besonderheiten. Insbesondere im Rahmen komplexer Belastungssituationen (z.B. Spielsportarten) können diese jedoch nicht immer vollständig abgebildet werden.

Aus einer Vielzahl an sportmotorischen Tests kann die Sprungkraftdiagnostik hervorgehoben werden, da diese durchweg für die Athleten wenig belastend ist und weder mit dem Trainings- noch mit dem Erholungsprozess interferiert. Zudem wird die Leistung in den Sprungtests (mit Ausnahme des Squat Jumps) in besonderem Maße durch den Dehnungs-Verkürzung-Zyklus beeinflusst (Nédélec et al., 2012), dessen Effizienz wiederum eng mit peripheren Ermüdungseffekten zusammenhängt (Nicol, Avela, & Komi, 2006). Vor allem der Countermovement Jump (CMJ) wird in zahlreichen Regenerationsstudien zur Messung von Ermüdung und Erholtheit verwendet (Halson, 2014). Aber auch andere Sprungtests besitzen Praxisrelevanz. So liefern Tiefsprünge (als singulärer Drop-Jump oder in Form von Prellsprung-Serien) gegenüber dem CMJ durch die Erfassung der Bodenkontaktzeit und der generierten Sprunghöhe bzw. deren Verrechnung zusätzliche Informationen über die Qualität der neuromuskulären Ansteuerung und der Umsetzung in Leistung (Ball & Zanetti, 2012).

Neben der Sprungkraftdiagnostik werden ebenfalls Linearsprinttests, Sprintserien (Wiederholungssprinttests), Agilitytests, Maximalkrafttests oder ausdauerorientierte submaximale und maximale Feldtests zur Evaluation des Regenerationsbedarfs verwendet (Nédélec et al., 2012). Sie sind jedoch zum Teil deutlich intensiver als Sprungkrafttests und resultieren dadurch in einer potentiell zusätzlichen Akkumulation von Ermüdungssymptomen. Zudem sind sie auch aus praktischen Gründen nicht immer für die routinemäßige Erfassung von Ermüdung und Erholtheit geeignet (Meeusen et al., 2013).

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, Kontraktionserhaltungszeit, 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, 2013; Šimunič, 2012; Tous-Fajardo et al., 2010; Zagar & Krizaj, 2005). 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

Die bei einer Aufzeichnung der Herzaktivität abgeleiteten Parameter liefern nichtinvasive Informationen zur Aktivität des kardial- autonomen Nervensystems. Neben der Dokumentation von Ruhe-, submaximalen und Erholungsherzfrequenzen ist die Messung der Herzfrequenzvariabilität (HRV) ein bekanntes Verfahren zur Erfassung von Ermüdung und Erholtheit (Buchheit, 2014). Die HRV bildet dabei die Interaktion zwischen dem sympathischen und parasympathischen Nervensystem ab (Aubert, Seps, & Beckers, 2003; Rajendra Acharya, Paul Joseph, Kannathal, Lim, & Suri, 2006). Körperliche Belastungen induzieren über eine Reihe von Reflexen eine akute Verschiebung der autonomen Balance zugunsten einer Sympathikusdominanz. Daher kann die zeitlich engmaschige Bestimmung der efferenten Sympathikus- bzw. Vagusaktivierung oder Inhibierung über Einzelwerte oder gleitende Mittelwerte und mehrtägige Trends wichtige Informationen liefern, um einen unerwünschten Übergang in ein nicht-funktionales Overreaching zu vermeiden (Hottenrott, Hoos, & Esperer, 2006; Naranjo & Cruz, 2015; Plews, Laursen, Kilding, & Buchheit, 2012, 2013). Von den traditionellen HRV-Indizes (methodische Zeitbereichs- und Spektralindizes sowie physiologische Indizes, die die instante, zirkadiane oder Gesamt-HRV erfassen (Hottenrott et al., 2006)), wird der zeitanalytische Parameter rMSSD (Root Mean Square of the Sucessive Differences) u.a. aufgrund seiner hohen Reliabilität und Stabilität sowie seiner vergleichsweise unkomplizierten Aggregierung am ehesten für die Messung von Regenerationsbedarf empfohlen (Plews, 2014).

Mehr noch als die TMG erfordern HRV-basierte Analysen eine hohe Expertise des Untersuchers. Zudem ist die längerfristige Evaluation individueller HRV-Verhaltensmuster erforderlich, um relevante Differenzen valide abzubilden. Die hierfür notwendigen engmaschigen und meist langfristigen HRV-Messungen gehen dabei zu Lasten der Athletencompliance. Überdies weist die HRV im Vergleich zu anderen Parametern angesichts der zeitaufwendigen statistischen Ableitung bzw. Aggregierung von Sekundärparametern aus den Rohdaten für den sportpraktischen Kontext eine nur geringe Praktikabilität und Testökonomie auf. Insbesondere Mannschaftssportarten sind hiervon betroffen.

2.2.4 Labordiagnostik

In der Literatur werden eine Vielzahl von biochemischen, hormonellen und immunologischen Laborwerten zur Überwachung des Ermüdungsstatus von Athleten empfohlen (Halson, 2014), die entweder aus dem venösen und Kapillarblut, dem Speichel oder dem Urin gewonnen werden können. Dabei wird zwischen Parametern unterschieden, die auf eine muskuläre Überbeanspruchung mit einhergehender Muskeldestruktion (u.a. Creatinkinase und Myoglobin) (Nédélec et al., 2012) sowie die darauffolgenden Entzündungskaskaden hinweisen (u.a. verschiedene Zytokine und Chemokine wie beispielsweise TNF-alpha, IL-1, IL-6 oder IL-8) (Zwetsloot, John, Lawrence, Battista, & Shanely, 2014) und solchen, die eher einen überhöhten energetischen Umsatz und eine daraus resultierende katabole Stoffwechsellage (u.a. Harnstoff, Testosteron, Cortisol, Glutamin und Glutamat sowie vereinzelte Quotienten) signalisieren (Meyer, Kellmann, Pfeiffer, & Ferrauti, 2013). Die Sensitivität der Parameter ist dabei abhängig von der Beanspruchungscharakteristik der Belastung. So reagieren stoffwechselbezogene Parameter meist auf Veränderungen von Trainingsumfängen, während eine erhöhte Aktivität von Indikatoren für Muskelzellschädigungen insbesondere nach hochintensiven muskulären Beanspruchungen beobachtet werden können (Meyer et al., 2013).

Die Labordiagnostik besitzt aufgrund des hohen Standardisierungsgrad bei der Materialgewinnung und der Analytik sowie den regelmäßigen Qualitätskontrollen der Laboratorien den Vorteil einer guten Reproduzierbarkeit und weitgehend gewährleisteten Objektivität (Meyer et al., 2013). Abgesehen von einigen Routineparametern wie Creatinkinase oder Harnstoff ist die Bestimmung vieler empfohlener Marker, die nicht in der medizinischen Routine gemessen werden, jedoch entsprechend teuer (Meyer et al., 2013). Zudem ist für viele Parameter eine venöse Blutentnahme notwendig. Diese ist einerseits aufgrund des organisatorischen Aufwands vor allem in der Sportpraxis nicht immer realisierbar und kann andererseits zugunsten der Athletencompliance nicht gleichermaßen engmaschig durchgeführt werden wie beispielsweise die subjektive Empfindungsdiagnostik (vgl. Kap. 2.2.5). Außerdem sollte auch hier die Verlässlichkeit und Trennschärfe einzelner Laborwerte im Rahmen einer individuellen Profibildung evaluiert werden.

2.2.5 Subjektive Empfindungsdiagnostik

Neben zahlreichen anderen Verfahren haben sich das Profile of Mood States (POMS) (McNair, Lorr, & Droppelman, 1981) und der Erholungs-Belastungs-Fragebogen für Sportler (EBF-Sport) (Kellmann & Kallus, 2001) international etabliert. Beide Tools sind jedoch für eine engmaschige, tägliche Erhebung aufgrund der zahlreichen Items zu komplex und gehen zu Lasten der Athletencompliance. Aufgrund der einfachen Erfassung und der hohen Praxisrelevanz hat sich daher die Quantifizierung von Ermüdung und Erholtheit mittels Total Quality Recovery Skala (TQR-Skala) (Kenttä & Hassmén, 1998) sowie der Messung von Muskelschmerz auf einer visuell-analogen (Visual Analog Scale) (Cleather & Guthrie, 2007), numerischen (0-10 Numeric Pain Rating Scale) (Tiidus, 2008) oder verbalen (Verbal Pain Intensity Scale) (Tiidus, 2008) Skala bewährt. Zur Schließung der Lücke zwischen aufwendigen Komplexverfahren und eindimensionalen Kurzskalen wurde das Akutmaß (AEB) (Hitzschke et al., im Druck) und die Kurzskala (KEB) (Kellmann, Kölling, & Hitzschke, im Druck) zur Erfassung von Erholung und Beanspruchung entwickelt. Letztere besteht aus acht Items (vier Items zur Messung des empfunden Erholungszustandes und vier Items zur Bestimmung des empfundenen Beanspruchungszustandes) mit einer jeweils mehrstufigen Likert-Skala von null (trifft gar nicht zu) bis sechs (trifft voll zu).

Fragebogen- oder skalenbasierte Tests zur Erfassung von Regenerationsbedarf besitzen eine hohe Praktikabilität und erfassen spezifische Ermüdungskomponenten mit genügender Validität. Die gute Testökonomie wird jedoch teilweise durch die fehlende Objektivität überschattet. So repräsentiert die „soziale Erwünschtheit“ insbesondere im Hochleistungssport einen entscheidenden Störfaktor. Wenn beispielsweise Einsatzzeiten in den Mannschaftssportarten mit der Auszahlung von Prämien verknüpft ist und Spieler die Nichtberücksichtigung bei einem diagnostizierten Ermüdungszustand fürchten, könnte dies den Athleten zu falschen Angaben verleiten.

Problemstellung

Ein belastungsübergreifender oder belastungsspezifischer Goldstandard mit den in der Literatur empfohlenen Parametern konnte sich in der Ermüdungsdiagnostik bislang nicht etablieren. Dies liegt mitunter an den komplexen, interindividuell differierenden und in Abhängigkeit der Belastungsstruktur variierenden psychophysischen Ermüdungsmustern. Zudem wurde die Sensitivität und Genauigkeit verschiedener Parameter bei der Überwachung der Trainings- und Wettkampfbeanspruchung sowie der Erfassung des Regenerationsbedarfs insbesondere im Rahmen spezifischer Belastungssituationen bisher nur selten systematisch untersucht. Folglich wurde im zweiten Untersuchungsmodul der vorliegenden kumulativen Dissertation die Sensitivität potenziell relevanter Parameter für die Diagnose von Ermüdung und Erholtheit im Rahmen eines HIT evaluiert. Die Ergebnisse des ersten Untersuchungsmoduls dienten dabei der empirisch begründeten Festlegung eines HIT-Programms mit hohem regenerativem Folgebedarf.

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)

Eine Umfrage in der ersten französischen Fußballiga zeigte eine weit verbreitete Anwendung von Kaltwassertherapie, aktiver Erholung, Massage, Stretching und Kompressionskleidung (Nédélec et al., 2012). Die Befragung national und international agierender Tennisspieler ergab, dass Stretching, Schlaf, Nahrungs- und Flüssigkeitsaufnahme, aktive Erholung, Massage und Kälteapplikationen in abfallender Reihung die am häufigsten frequentierten Regenerationsverfahren sind (Wiewelhove, Reader & Ferrauti, 2015). Im Folgenden wird auf solche Verfahren vertieft eingegangen, die sich in der Sportpraxis vor allem nach intensiven Belastungen großer Beliebtheit erfreuen. Ausgenommen bleibt der Bereich der Ernährung.

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.,

2013). Die wissenschaftliche Evidenz zur Wirksamkeit aktiver Erholung ist jedoch nicht überzeugend gegeben. Unbestritten ist eine kurzfristig raschere Laktatelimination und pH-Wert Regulation (Barnett, 2006a). Dies scheint jedoch im Kontext vieler Disziplinen kein relevanter Regenerationseffekt zu sein (Bishop et al., 2008). Vielmehr verspricht man sich durch den erhöhten lokalen Blutfluss einen schnelleren Abtransport von Abfallprodukten des Reparaturstoffwechsels geschädigter Muskelfaserstrukturen sowie eine verbesserte Nährstoffversorgung des in Mitleidenschaft gezogenen Gewebes (Hausswirth & Mujika, 2013). Zudem könnte sich die analgetische Wirkung sanfter körperlicher Betätigungen positiv auf das im Rahmen von mikroskopischen Muskelzellschädigungen auftretende Schmerzempfinden auswirken (Andersen et al., 2013). Beide Aspekte konnten bislang jedoch nicht eindeutig nachgewiesen werden. Darüber hinaus wurden auch negative Effekte aktiver Erholungsstrategien aufgedeckt. So führt beispielsweise der anhaltend gesteigerte Energieumsatz zu einer Verlangsamung der Wiedereinlagerung von Muskelglykogen (Fairchild et al., 2003).

2.3.2 Stretching

Vorrangiges Ziel von Stretchingmethoden ist die Verbesserung der sportmotorischen Beweglichkeit im Trainingsprozess durch eine in Abhängigkeit von den Gelenkstrukturen gesteigerte Dehnfähigkeit. Bezogen auf die Regeneration konnten bisher jedoch keine potentiell erholungsfördernden Mechanismen identifiziert werden (Lambert & van Wyk, 2009; Nédélec et al., 2013). Dies mag auch der Grund sein, warum regenerative Wirkungen durch Stretching bislang nur von wenigen Studien überprüft und nicht überzeugend nachgewiesen wurden (Hausswirth & Mujika, 2013). In einer Studie von Robey, Dawson, Goodman & Beilby (2009) hatte Stretching keinen Einfluss auf den empfundenen Muskelschmerz sowie die Wiederherstellung der Leistungsfähigkeit im Anschluss an eine intensive Belastung. Ähnliches dokumentierten Cheung, Hume & Maxwell (2003), Connolly, Sayers & Mchugh (2003), Gulick, Kimura, Sitler, Paolone & Kelly (1996) sowie Mika, Mika, Fernhall & Unnithan (2007). Sie zeigten, dass Stretching die Symptome mikroskopischer Muskelverletzungen nicht lindert. Im Einzelfall berichten Athleten jedoch von einem gesteigerten Wohlbefinden. Dies könnte durch die Annahme, das Dehnprogramme zu einer Herabsetzung der Ruhespannung führt, erklärt werden. So vermutet Wiemeyer (2003), dass insbesondere statisches Dehnen aufgrund reduzierter afferenter und efferenter Zuflüsse zur Formatio reticularis zu allgemeinen Desaktivierungsprozessen und letztlich zu einem psychophysischen Entspannungszustand führt. Allerdings kann Stretching auch kontrainduziert sein. So können passive Dehntechniken belastungsinduzierte Mikrotraumata verstärken und vor Trainings- oder Wettkampfaktivitäten die (Schnell-)Kraftleistung reduzieren (Hausswirth & Mujika, 2013; Nédélec et al., 2013).

2.3.3 Schlaf

Neurophysiologische Theorien nehmen an, dass Schlaf dabei hilft, die neuronale und metabolische Beanspruchung der Wachphase zu kompensieren (Frank, 2006). Schlaf geht mit verringertem Atem- und Herzschlagrhythmus sowie reduzierter zerebraler Durchblutung und stark erhöhter Sekretion von Wachstumshormonen einher, sodass eine physiologische Restitution ermöglicht wird (Åkerstedt & Nilsson, 2003). Der für Athleten notwendige Schlafumfang differiert dabei interindividuell erheblich und folgt einer negativen U-Funktion (Hausswirth & Mujika, 2013). Für alle Sportler gilt: zu geringer oder zu hoher Schlafumfang sowie defizitäre Schlafqualität gehen mit Störungen der zentralnervösen Aktivierung, Verringerung des Muskelglykogengehalts, verstärktem Schmerzempfinden, erhöhter Infektanfälligkeit, reduziertem Adaptationspotential sowie unerwünschten Leistungseinbußen und inflammatorischen Reaktionen einher (Cohen, Doyle, Alper, Janicki-Deverts, & Turner, 2009; Haack & Mullington, 2005; Hausswirth & Mujika, 2013; Irwin et al., 2008; Irwin, Wang, Campomayor, Collado-Hidalgo, & Cole, 2006; Nédélec et al., 2013; Skein, Duffield, Edge, Short, & Mündel, 2011).

Externe (z.B. Temperatur, Lärm, Helligkeit) und interne (z.B. Muskelschmerzen, Wettkampfnervosität, Hyperhydration) Faktoren wirken auf die homöostatische und zirkadiane Schlafregulation und können Schlafqualität und -quantität negativ beeinflussen (Fischer, Nagai, & Teixeira, 2008). Kommt es infolgedessen zu Störungen der Schlafroutine können sog. Powernaps einem Schlafdefizit entgegenwirken und die Erholung begünstigen (Nédélec et al., 2013). Powernaps sind kurze Schlafperioden außerhalb der nächtlichen Schlafphase, die die allgemeine mentale und physische Leistungsfähigkeit unterstützen (Hausswirth & Mujika, 2013). Zudem fördern Sie Konzentrations- und Reaktionsfähigkeit, die während eines Schlafmangelzustands reduziert sind (Waterhouse, Atkinson, Edwards, & Reilly, 2007). Je länger allerdings ein solcher Powernap andauert, desto länger braucht der Athlet, um die Schlaftrunkenheit nach solch einer Schlafphase zu überwinden (Hausswirth & Mujika, 2013). Ein Powernap sollte daher nicht unmittelbar vor einer Trainings- oder Wettkampfbelastung stattfinden.

2.3.4 Massage

Massagen sind in vielen Disziplinen seit mehreren Dekaden weit verbreitet. Der zentrale Wirkungsmechanismus soll in der mechanischen Erwärmung und der lokal gesteigerten Muskeldurchblutung liegen. Experimentelle Überprüfungen ergaben jedoch zumeist keine nennenswerten Veränderungen der Muskeldurchblutung, Dehnbarkeit und Kraft in der Vor- und Nachbereitung von Training und Wettkampf (Shoemaker, Tiidus, & Mader, 1997; Tiidus & Shoemaker, 1995; Weerapong, Hume, & Kolt, 2005; Wiktorsson-Moller, Oberg, Ekstrand, & Gillquist, 1983; Wiltshire et al., 2010). Demgegenüber wird in einigen Studien von einer Verbesserung von Gemütslage

und Wohlbefinden sowie einer gesteigerten Entspannung berichtet. (Farr, Nottle, Nosaka, & Sacco, 2002; Hemmings, Smith, Graydon, & Dyson, 2000; Moyer, Rounds, & Hannum, 2004; Zainuddin, Newton, Sacco, & Nosaka, 2005). Ergo scheinen Massagen lediglich in der Lage zu sein, den subjektiv empfundenen Erholungszustand positiv zu beeinflussen. Dennoch bezweifeln Poppendieck et al. (2016) in ihrer Übersichtsarbeit, dass die bislang nachgewiesenen Effekte die weit verbreitete Anwendung von Massagen im Kontext der Regenerationssteuerung rechtfertigen. 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 beeinflusst (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 Rückstroms, des arteriellen Bluteinstroms sowie des lymphatischen Ausstroms, Vermeidung von Ödembildung, Beschleunigung des Laktatabbaus, Verbesserung von zellulären Reparaturprozessen, 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 regelmäßig 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 Untersuchungen 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-

läre, pulmonale und neuromuskuläre Mechanismen sowie inflammatorische, hormonelle und immunologische Reaktionen (Hausswirth & Mujika, 2013). Über deren Einfluss auf regenerative Prozesse existieren bislang jedoch kaum wissenschaftlich relevante Informationen. Vereinzelt wird von einer Reduktion von Bewegungsbeschwerden des aktiven und passiven Bewegungsapparates sowie von einer Minderung empfundener Muskelschmerzen berichtet (Hannuksela & Ellahham, 2001; Kukkonen-Harjula & Kauppinen, 2006). Auch nutzen Athleten das Saunabad als Entspannungsverfahren und zur anschließenden Steigerung der Schlafqualität (Hausswirth & Mujika, 2013). Dehydrierung oder zentralnervöse Deaktivierung durch die Hitzeexposition können sich jedoch auch leistungsmindernd auswirken (Gutiérrez, Mesa, Ruiz, Chirosa, & Castillo, 2003), sodass eine Saunaanwendung nur in ausreichendem Abstand zum Training oder Wettkampf empfohlen wird.

2.3.7 Kälteapplikationen

Sportpraktische Kälteanwendungen erfolgen zumeist durch Kaltwasserimmersion (KWI) oder Kryotherapie. Empfohlene bzw. evaluierte Immersionsprotokolle variieren in Dauer, Wassertemperatur und Eintauchtiefe zwischen Intervallanwendungen von beispielsweise 5 x 1 min bei einer Wassertemperatur von 5 – 8 °C und Daueranwendungen von 10 – 20 min bei einer Wassertemperatur von 10 – 15 °C (Poppendieck, Faude, Wegmann, & Meyer, 2013). Der gesteigerte hydrostatische Druck verspricht u.a. einen verbesserten venösen Rückstrom sowie eine abschwellende Wirkung (Hausswirth & Mujika, 2013). Die eigentlichen Regenerationseffekte von KWI werden allerdings vorrangig auf die Wassertemperatur zurückgeführt (Nédélec et al., 2013). Wie bei anderen kryotherapeutischen Applikationen (z.B. Eisbeutel, Eismassage, Eisspray oder Kältekammer) vermindert die Temperatursenkung den Blutfluss zu den Extremitäten und bewirkt in der Folge eine Umleitung des Blutstroms von der Peripherie zum Körperkern und damit ebenfalls einen erhöhten venösen Rückstrom sowie eine verbesserte Herzeffizienz (Lambert & van Wyk, 2009; Nédélec et al., 2013). Zudem wird angenommen, dass die durch Kälte ausgelöste Vasokonstriktion inflammatorische Prozesse hemmt (Hausswirth & Mujika, 2013), während eine kurzfristige analgetische Wirkung infolge reduzierter Nevenleitgeschwindigkeit, Muskelspindelaktivität, spinalmotorischer Reflextätigkeit und Spastizität als gesichert gilt (Meeusen & Lievens, 1986; Nédélec et al., 2013).

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 &

Duffield, 2012; Rowsell, Coutts, Reaburn, & Hill-Haas, 2009, 2011). In gleicher Weise dokumentierten jedoch auch zahlreiche Untersuchungen entweder einen Placebo-Effekt, überhaupt keine Wirkungen oder sogar einen negativen Einfluss durch KWI Anwendungen auf die Erholung und Leistungsbereitschaft (z.B. Reduktion der Glykogen-Resyntheserate, akuter Leistungsabfall infolge reduzierter Muskeltemperatur oder subkutane Schwellungen aufgrund einer erhöhten Permeabilität von Lymphgefäßen) (Broatch, Petersen, & Bishop, 2014; Costello, Algar, & Donnelly, 2012; Delextrat, Calleja-González, Hippocrate, & Clarke, 2012; Hausswirth & Mujika, 2013; R Meeusen & Lievens, 1986; Nédélec et al., 2013; Paddon-Jones & Quigley, 1997; Tseng et al., 2013). Angesichts der unterschiedlichen publizierten Immersionsprotokolle sowie der heterogenen Datenlage zu KWI und anderen kryotherapeutischen Anwendungen gilt eine Beschleunigung der Regeneration durch (Immersions-)Kälte bislang ebenfalls als nicht gesichert.

Problemstellung

Regenerationsverfahren dienen der beschleunigten Wiederherstellung der psychophysischen Leistungsfähigkeit nach intensiven Belastungen. Das Leistungsniveau kann insbesondere nach einem HIT für bis zu mehrere Tage reduziert sein. Die Evidenz zur Wirksamkeit populärer Erholungsmaßnahmen ist jedoch aufgrund lückenhafter, inkonsistenter und heterogener Befundlage nicht eindeutig geklärt. So können bislang keine regenerativen Verfahren uneingeschränkt zur Linderung der durch HIT induzierten Ermüdungsreaktionen empfohlen werden. Folglich wurde im dritten Untersuchungsmodul der vorliegenden kumulativen Dissertation der Einfluss eines der verbreitetsten Regenerationsinterventionen (d.h. aktive Erholung) auf die durch einen mehrtätigen praxisorientierten HIT-Mikrozyklus induzierten Ermüdungserscheinungen untersucht. Hierfür wurde das im zweiten Untersuchungsmodul evaluierte Diagnostikinventar verwendet. Zwar wurde in der Literatur schon mehrfach auf mögliche adaptationsmindernde Effekte durch eine langfristige Anwendung aktiver Erholungsmaßnahmen hingewiesen. Dennoch wurde Im dritten Modul zunächst nur der regenerative Wert von aktiver Erholung überprüft.

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

Wirkungsebene	Parameter	Regenerationsverfahren						
		Aktive Erholung	Stretching	Schlaf	Massage	Kompression	Wärme	Kälte
Energetisch	Laktat-Elimination	+	o	o	o	o	o	o
	Substrattransport	+	o	o	+	+	o	+
	Glykogen-Gehalt	-	o	+	o	o	o	o
Zirkulatorisch	Venöser Rückstrom	+	o	o	+	+	o	+
	Lokaler Blutfluss	+	o	o	+	-	-	-
Muskulär	Temperatur	+	o	o	+	+	+	-
	Dehnbarkeit	+	+	o	+	o	+	-
	DOMS	o	-	o	+	+	+	+
	CK	-	-	o	-	+	o	o
	Satellitenzellaktivierung	o	o	+	o	o	o	o
Inflammatorisch	CRP, IL-1, IL-6, TNF α	-	o	+	o	o	o	+
	Ödembildung	-	-	o	+	+	-	+
Endokrinologisch	Cortisol	o	o	+	o	o	-	+
	IGF-1	-	o	+	o	o	o	-
Neuromuskulär	Aktivierungspotential	o	-	o	o	o	-	-
	Schmerz	+	o	o	+	+	+	+
Psychovegetativ	Erholungszustand	-	o	+	+	+	+	+
	Beanspruchungszustand	o	o	+	+	+	o	+
Leistungssteigernd	Akut	+	-	+	+	o	-	-
	am Folgetag	o	o	+	o	o	o	o

+ = 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

3 ARBEITSPROGRAMM UND ZIELSTELLUNG

Das Arbeitsprogramm bestand aus drei Modulen, die inhaltlich und chronologisch aufeinander aufbauten (Abb. 1). Jedes Modul beinhaltete dabei einen in sich geschlossenen Forschungsansatz mit entsprechenden Zielstellungen. Die Ergebnisse der Untersuchungen wurden im Rahmen von drei Beiträgen in internationalen Peer-Review-Fachzeitschriften publiziert.

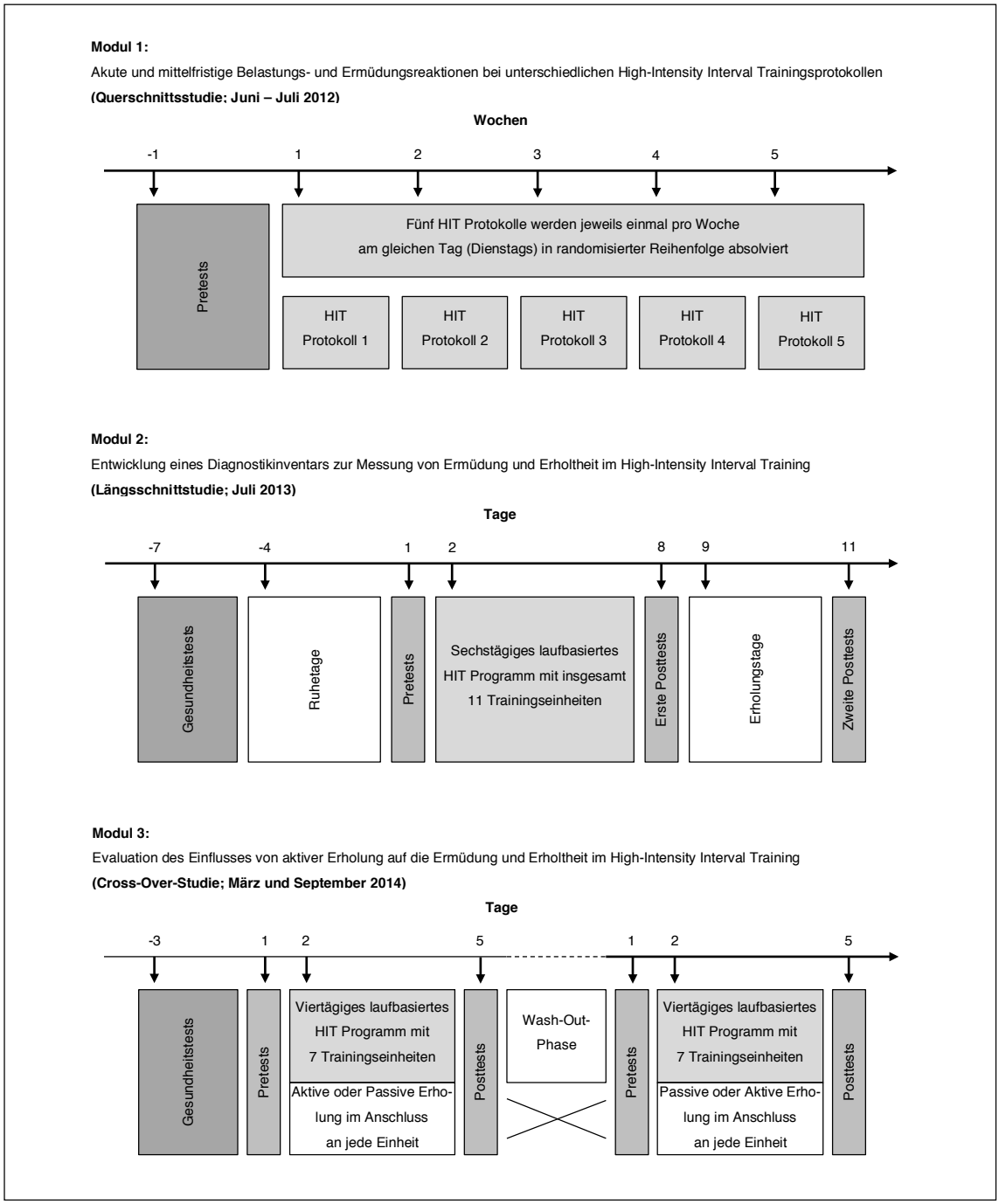

Abb. 1. Gesamtdesign des dreistufigen Arbeitsprogramms. HIT = High-Intensity Interval Training

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

Der positive Einfluss eines hochintensiven Intervalltrainings auf allgemeine und spezifische Ausdauerleistungskomponenten wurde bereits in zahlreichen Längsschnittstudien belegt. Infolgedessen konnte sich diese Trainingsmethode über leichtathletische Ausdauerdisziplinen hinaus auch in anderen Sportarten und speziell in den Spielsportarten etablieren. Akute Belastungsreaktionen sowie akute und mittelfristige Ermüdungs- bzw. Erholungseffekte wurden jedoch bislang unter Berücksichtigung der unzähligen Gestaltungsmöglichkeiten eines HIT nicht konsistent klassifiziert. Ziel des *ersten* Moduls war es folglich, akute und mittelfristige neuromuskuläre, biochemische und perzeptive Belastungs- und Ermüdungsreaktionen im Rahmen unterschiedlicher HIT-Protokolle zu quantifizieren. Ferner bestand das Ziel darin, Belastungsnormative eines HIT mit hohem regenerativem Folgebedarf für das Modul 2 empirisch begründet festzulegen.

Modul 2

In der Literatur werden eine Vielzahl von Parametern für die Diagnostik von Ermüdung und Erholtheit vorgeschlagen. Insbesondere im Rahmen spezifischer Belastungssituationen (z.B. HIT) sind die empfohlenen Marker jedoch bislang kaum systematisch auf die erforderliche Sensitivität für das Monitoring von Belastungs- und Ermüdungsreaktionen evaluiert worden. Ziel des *zweiten* Moduls war es folglich, die Sensitivität für die Sportpraxis relevanter Parameter für die Messung von Ermüdung und Erholtheit im Rahmen eines HIT zu überprüfen und geeignete Marker zu definieren. Ebenso bestand das Ziel darin, ein Diagnostikinventar zu entwickeln, mithilfe dessen die Wirksamkeit der in Modul 3 untersuchten Regenerationsmaßnahme überprüft werden sollte.

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 Ermüdungserscheinungen zu überprüfen.

4 PUBLIKATIONEN[1]

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

Manuskript 1

Wiewelhove, T., Fernandez-Fernandez, J., Raeder, C., Kappenstein, J., Meyer, T., Kellmann, M., Pfeiffer, M. & Ferrauti, A. (2015). Acute responses and muscle damage in different high-intensity interval running protocols. *J Sports Med Phys Fitness*, Epub ahead of print.

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

Manuskript 2

Wiewelhove, T., Raeder, C., Meyer, T., Kellmann, M., Pfeiffer, M. & Ferrauti, A. (2015). Markers for routine assessment of fatigue and recovery in male and female team sport athletes during high-intensity interval training. *PLoS One*, *10* (10), 1-17.

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

Manuskript 3

Wiewelhove, T., Raeder, C., Meyer, T., Kellmann, M., Pfeiffer, M. & Ferrauti, A. (2016). Effect of repeated active recovery during a high-intensity interval training shock microcycle on markers of fatigue. *Int J Sports Physiol Perform*, Epub ahead of print.

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

[1] Für die Dissertation wurde die Gestaltung der Manuskripte an Schrift, Schriftgröße und Zeilenabstand der vorliegenden Arbeit angepasst. Alle anderen Formatierungen sowie die inhaltliche Aufbereitung sind mit den zur Veröffentlichung eingereichten Manuskripten identisch und entsprechen insofern den Vorgaben der Fachzeitschriften. Die Originalpublikation des zweiten Manuskripts ist im Zeitschriftenlayout dieser Arbeit angehängt. Manuskript 1 und Manuskript 3 sind bislang noch nicht in der Originalfassung erschienen und können daher nicht im Anhang aufgeführt werden.

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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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 (V_{IFT}).

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 [1]. In this regard, several previously published studies have shown positive effects of this training methodology on endurance performance.[2-6] The rationale behind the use of HIT in various forms is, that athletes can enhance cardiorespiratory, metabolic and neuromuscular function, using significant lower training volumes compared to traditional high-volume low intensity endurance training.[7-12] HIT involves repeated relatively short intensive work intervals interspersed by active or passive recovery periods.[1] 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).[7, 13] Submaximal-intensity exercises involve periods of work at 90-100% of maximum oxygen uptake velocity ($v\dot{V}O_{2max}$), whereas in RST intensities are generally above 100% of $v\dot{V}O_{2max}$.[14]

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.[7] Therefore, to accurately prescribe individual HIT programs, it is necessary to understand the acute responses to HIT when manipulating any of these variables.[1, 15] Additionally, as HIT can be accompanied with high neuromuscular demands,[16] 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.[1]

To set interval intensity, several approaches have been used.[7] 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).[7] Furthermore, prescribing intensity using the $v\dot{V}O_{2max}$ obtained during laboratory testing protocols, is suitable for long (2-4 min) intervals, but problematic to appropriate individualize training intensities during supramaximal HIT.[7] Thus, athletes having a similar $v\dot{V}O_{2max}$ may have different anaerobic velocity reserves (i.e., reserve of running velocity (AVR) left to the athlete once he has reached his $v\dot{V}O_{2max}$). During HIT prescribed by the $v\dot{V}O_{2max}$, 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.[17] 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 ($30\text{-}15_{IFT}$).[1, 7, 17, 18] The $30\text{-}15_{IFT}$ 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 30-15$_{IFT}$ (V_{IFT}) for individualizing players training intensity, this protocol is becoming popular when undertaking HIT in intermittent sports.[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 30-15$_{IFT}$. 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 30-15$_{IFT}$ (V_{IFT}). 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.

Materials and methods

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 $\dot{V}O_{2max}$ and the 30-15$_{IFT}$ [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.

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; $\dot{V}O_{2max}$ 58.3 ± 5.9 ml·min·kg^{-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 $\dot{V}O_{2max}$, $v\dot{V}O_{2max}$, HR_{max}, turn points for lactate (LTP_1, LTP_2) and HR at the lactate turn points (HR LTP_1, HR LTP_2).[20] The treadmill test started with an initial velocity of 8 $km \cdot h^{-1}$, increasing 2 $km \cdot h^{-1}$ every 3 min with a constant incline of 0.5% until voluntary exhaustion. $\dot{V}O_2$ 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 $\dot{V}O_{2max.}$ 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 $30\text{-}15_{IFT}$ [18] 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 $km \cdot h^{-1}$ for the first 30 s run and was increased by 0.5 $km \cdot h^{-1}$ 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 (V_{IFT}).[18] V_{IFT} was used to calculate the interval intensity of the different HIT protocols as described by Buchheit.[17]

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 ($\%V_{IFT}$) and W/R are shown in Table 1. A standardized continuous 10 min warm-up, consisting of 40 m shuttle runs at 60-70% HR_{max} followed by four 40 m acceleration sprints, was performed before all HIT sessions. Protocol 240 (P_{240}) and 120 (P_{120}) consisted of 4 min and 2 min straight-line runs, respectively. Protocols 30 (P_{30}) and 15 (P_{15}) were performed as 40 m shuttles, with COD-based 30 s and 15 s intermittent runs. Protocol 5 (P_5) 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.

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

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 LTP_2 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 α-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, η^2_p).[24] The SPSS statistical software package (version 18, SPSS Inc., Chicago, IL, USA) was used for statistical computation.

Results

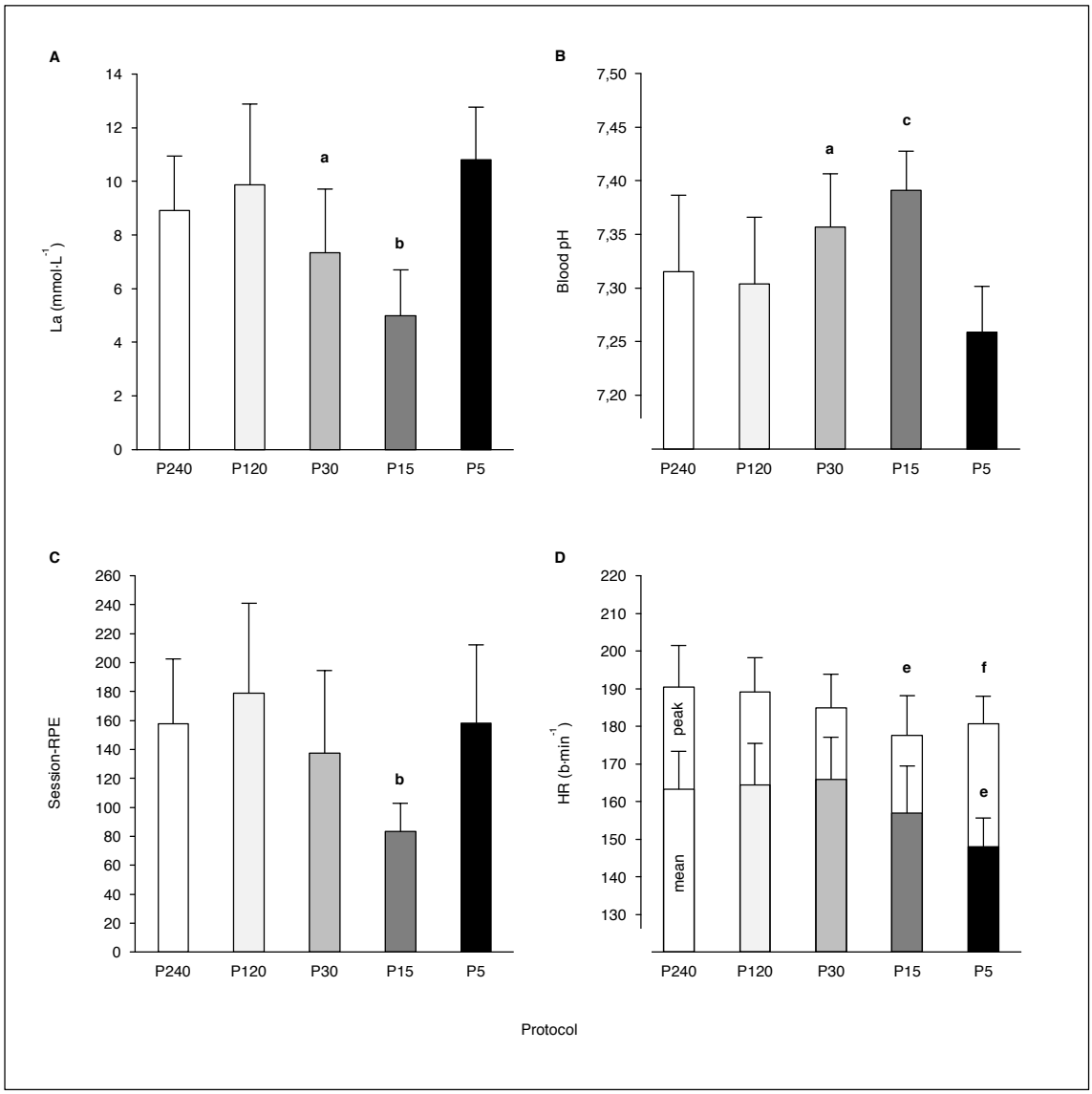

Figure 1. Short-term effects of different high-intensity training protocols on heart rate (HR), session-RPE, blood lactate (La) and blood pH. Letters represent significant differences (p<0.05). [a]Significantly different from P_{120} and P_5; [b]significantly different from all other protocols; [c]significantly different from P_{240}, P_{120} and P_5; [d]significantly different from P_{120}; [e]significantly different from P_{240}, P_{120} and P_{30}; [f]significantly different from P_{240} and P_{120}.

Acute responses

There was a main effect for protocol in mean La (p<0.001, η^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, η^2_p=0.56). Post hoc analysis revealed lower post training

values in P_{240} (p<0.05), P_{120} (p<0.01), and P_5 (p<0.001) compared to P_{15} as well as in P_{120} (p<0.05) and P_5 (p<0.001) compared to P_{30} (Figure 1B). The time course for blood lactate concentration is shown in Figure 2. Blood lactate accumulation rises during P_{240}, P_{120}, P_{30} and P_5 while a La steady state was shown for P_{15}. Session-RPE showed a main effect for protocol (p<0.001, η^2_p=0.43), with post hoc analysis revealing lower values in P_{15} (p<0.05) compared to all other HIT protocols (Figure 1C). A difference in Session-RPE was also found between P_{30} and P_{120} (p<0.05), while there was no difference between P_{240}, P_{120} and P_5 (p>0.05).

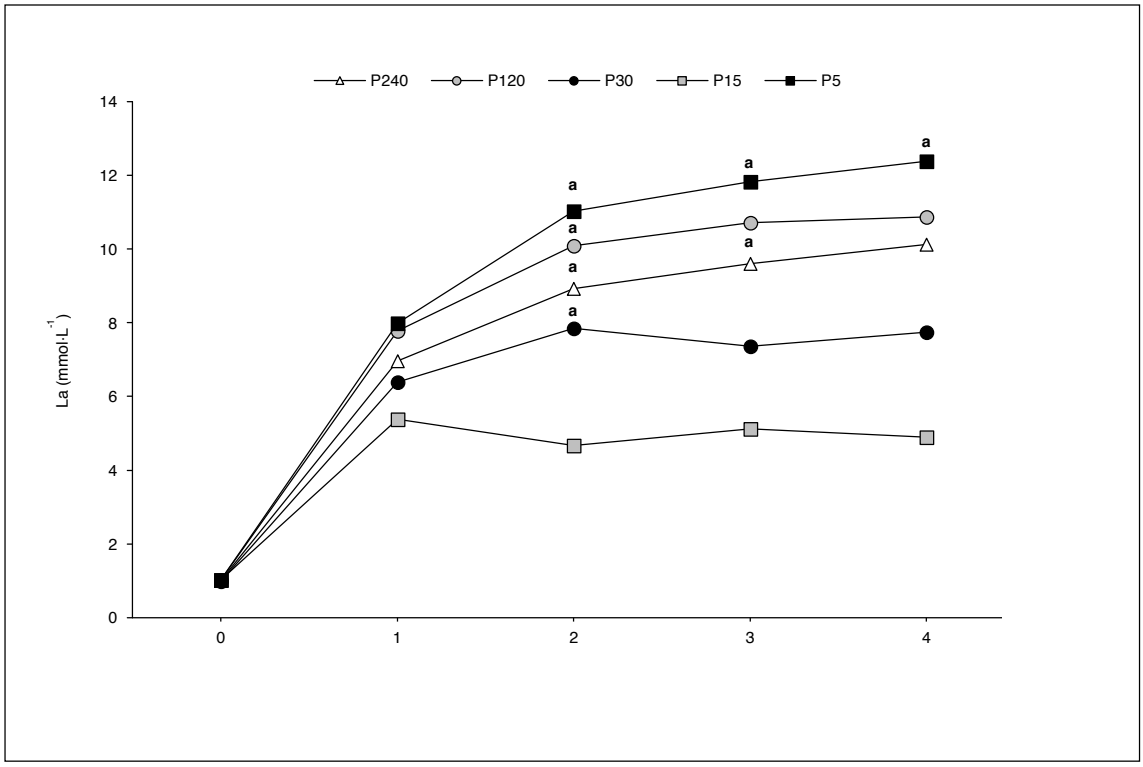

Figure 2. Changes in blood lactate concentration during different high-intensity training protocols measured pre-exercise (0), after approximately 6 (1), 12 (2) and 18 (3) min (always immediately at the end of a work interval) and at the end (4) of each session. Blood lactate concentrations during exercise 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).

Mean and peak HR of the five HIT protocols are shown in Figure 1D. A difference between protocols in mean (p<0.001, η^2_p=0.58) and peak (p<0.001, η^2_p=0.68) HR was found. Mean HR was lower in P_5 compared to P_{240} (p<0.001), P_{120} (p<0.01) and P_{30} (p<0.001). The lowest peak HR was observed in P_{15} and P_5 (p<0.05), whereas mean and peak HR did not differ between P_{240}, P_{120} and P_{30} (p<0.05). Table 2 shows the percentage of time spent by the athletes below and above HR LTP_2. In P_{30}, P_{15} and P_5 athletes spent longer times below HR LTP2 then above HR LTP2 (p<0.05), whereas the time at > HR LTP2 was lower in P_5 compared to P_{240}, P_{120} and P_{30} as well as in P_{15} compared to P_{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.

	Below HR LTP_2	Above HR LTP_2
Protocol	Percentage of time (%)	
P_{240}	65 ± 20	35 ± 25c
P_{120}	63 ± 19	37 ± 19c
P_{30}	69 ± 22	31 ± 22[ac]
P_{15}	82 ± 21	18 ± 21[ac]
P_5	86 ± 90	14 ± 9[abc]

Letters represent significant differences (p<0.05). [a]Significantly lower than the other HR-category; [b]Significantly different from P_{240}, P_{120} and P_{30}; [c]Significantly different from P_{120}.

Exercise-induced muscle damage

No main effect for protocol but a main effect for time (CK: $p<0.01$, $\eta^2_p=0.57$; CMJ: $p<0.05$, $\eta^2_p=0.22$) and a protocol × time interaction (CK, $p<0.01$, $\eta^2_p=0.31$; CMJ, $p<0.05$, $\eta^2_p=0.20$) 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 ($p<0.01$, $\eta^2_p=0.28$). 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.

					Protocol		Time		Interaction	
Variable	Protocol	Pre	Post30min	Post24h	*p*	η^2_p	*p*	η^2_p	*p*	η^2_p
CMJ (cm)	P_{240}	38.5 ± 5.7	37.7 ± 5.6	37.2 ± 5.9	0.279	0.085	0.029	0.223	0.016	0.195
	P_{120}	39.3 ± 5.1	38.0 ± 5.6	37.7 ± 4.6						
	P_{30}	37.6 ± 5.4	38.5 ± 4.9	37.6 ± 4.9						
	P_{15}	36.9 ± 6.2	38.8 ± 5.4	37.5 ± 5.2						
	P_5	38.7 ± 5.2	36.2 ± 4.8[ab]	35.8 ± 4.6[ab]						

Letters represent significant differences (p<0.05). [a]Significantly different to pre; [b]significant protocol x time interaction. Abbreviations: η^2_p, partial eta squared.

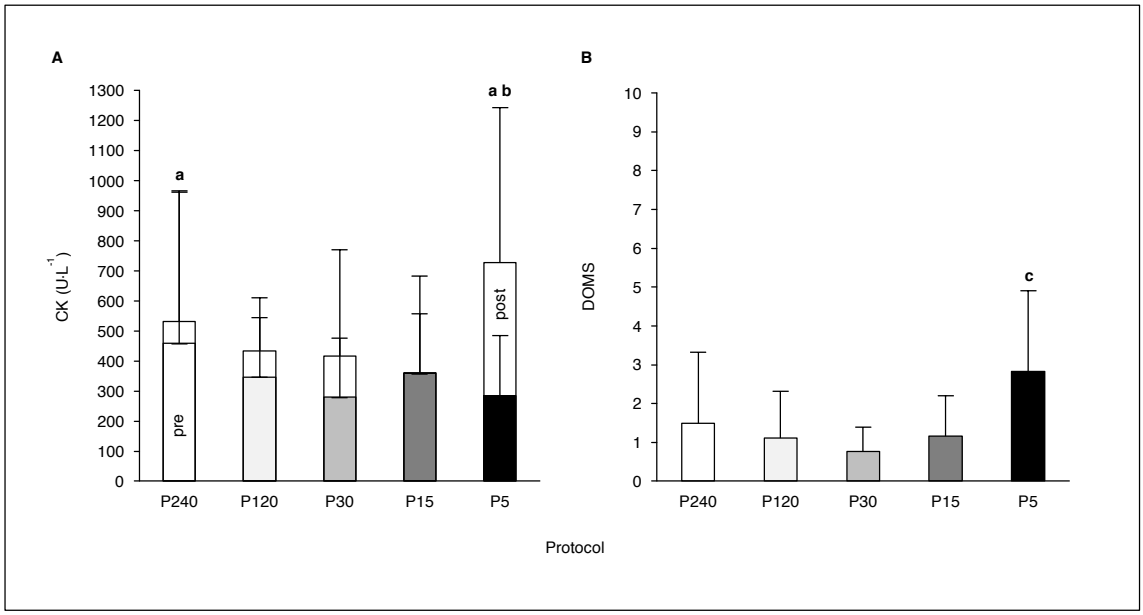

Figure 3. Mid-term effects of different high-intensity training protocols on serum creatinkinase (CK) and delayed onset muscle soreness (DOMS). Letters represent significant differences (p<0.05). [a]Significantly different to baseline; [b]significant protocol x time interaction; [c]significantly different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

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_{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_{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_{max}, with the time spent above HR LTP_2 being significantly higher in the longer intervals (Table 2). Furthermore, assuming that V_{IFT} is faster than vVO_{2max} [19] (i.e., 2.5 ± 0.9 km·h^{-1} in the present study), participants performed the training sessions at intensities ranging from ~90% (P_{240}) to ~110% (P_{15}) of vVO_{2max} (obtained in the laboratory test) and spent approximately 7 min (P_{15}) to 16 min (P_{240}) at intensities > 90% of vVO_{2max}. In this regard, several authors suggested that athletes should spend at least several minutes per HIT session at intensities > 90% of vVO_{2max} to stimulate cardiovascular and peripheral adaptations.[4, 7, 13] In this regard, the amount of high intensity exercise accumulated during

HIT protocols in the current study has 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.[7] 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.[1, 25, 26] Interestingly, P_{240} and P_{120} induced higher lactate concentrations compared to P_{30} and P_{15} (Figure 1). This can be related to the fact that despite the lower interval intensities during P_{240} (80% V_{IFT}) and P_{120} (85% V_{IFT}) the running velocity still remains above the maximal lactate steady state velocity.[1] 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 the $v\dot{V}O_{2max}$ yielding high lactate levels up to 16 $mmol \cdot L^{-1}$ (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.[1, 25]

The highest (P_5) and the lowest (P_{15}) lactate concentrations were found in protocols including short interval durations with the highest running velocity (Figure 1). In case of P_5 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.[27] It can be considered that the 25 s recovery period in P_5 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 P_{15} (30 s) was comparably to P_5 (25 s) yet protocol differences were related to interval intensity and the integration of multiple changes of direction in P_{15} (Table 1). The introduction of changes of direction has been shown to increase blood lactate, irrespective of the work intensity and duration, [13] with HIT protocols performed at the same intensity showing significantly higher physiological and perceptual responses in shuttle format compared with straight-line running.[28] Consequently, the pronounced difference in the anaerobic glycolytic demands can mainly be attributed to the lower running velocity in P_{15}.

Based on the suggested models for the classification of intermittent exercise by Tschakert and Hofmann,[1] P_{240}, P_{120}, P_{30} and P_5 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 $\dot{V}O_{2max}$.[1] 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 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.[29] 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_{mean}) as suggested by Tschakert and Hofmann,[1] showing the highest P_{mean} in P_{240} and P_{120}, while the lowest P_{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.[8] Previous studies have reported substantial inflammatory responses to various forms of prolonged, continuous aerobic exercise.[30-32] 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 [33] as well as impairs jumping performance, [13] although the inflammatory response to an acute bout of HIT appears to be substantially lower than that of prolonged, continuous aerobic exercise.[34]

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.[35] 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.[36] 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.[37]

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_{mean} [1] 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.[38] Also, it has been proposed that myoglobin is a more sensitive measure of muscle damage than CK.[39] 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.[40]

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.[1, 7] 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.

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4.2 Publikation 2

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; $\dot{V}O_{2max}$, 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 (30-15$_{IFT}$) 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.

		Age (yrs)	Height (cm)	Body mass (kg)	Body fat (%)	$\dot{V}O_{2max}$ (mL·min·kg^{-1})
Overall	(n = 22)	23.0 ± 2.7	176.6 ± 7.6	69.5 ± 7.3	17.9 ± 5.8	57.6 ± 8.6
Male	(n = 11)	22.9 ± 1.9	181.6 ± 5.3	73.8 ± 6.4	14.6 ± 3.7	62.9 ± 8.3
Female	(n = 11)	23.0 ± 3.4	171.6 ± 6.0	65.2 ± 5.5	21.1 ± 5.9	52.2 ± 4.8

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 $\dot{V}O_{2max}$. After familiarization with performance tests to minimize any learning effect, participants completed the 30-15_{IFT} 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 ($post_1$), and following a 72 h recovery period ($post_2$), in which no training was allowed (Fig. 1).

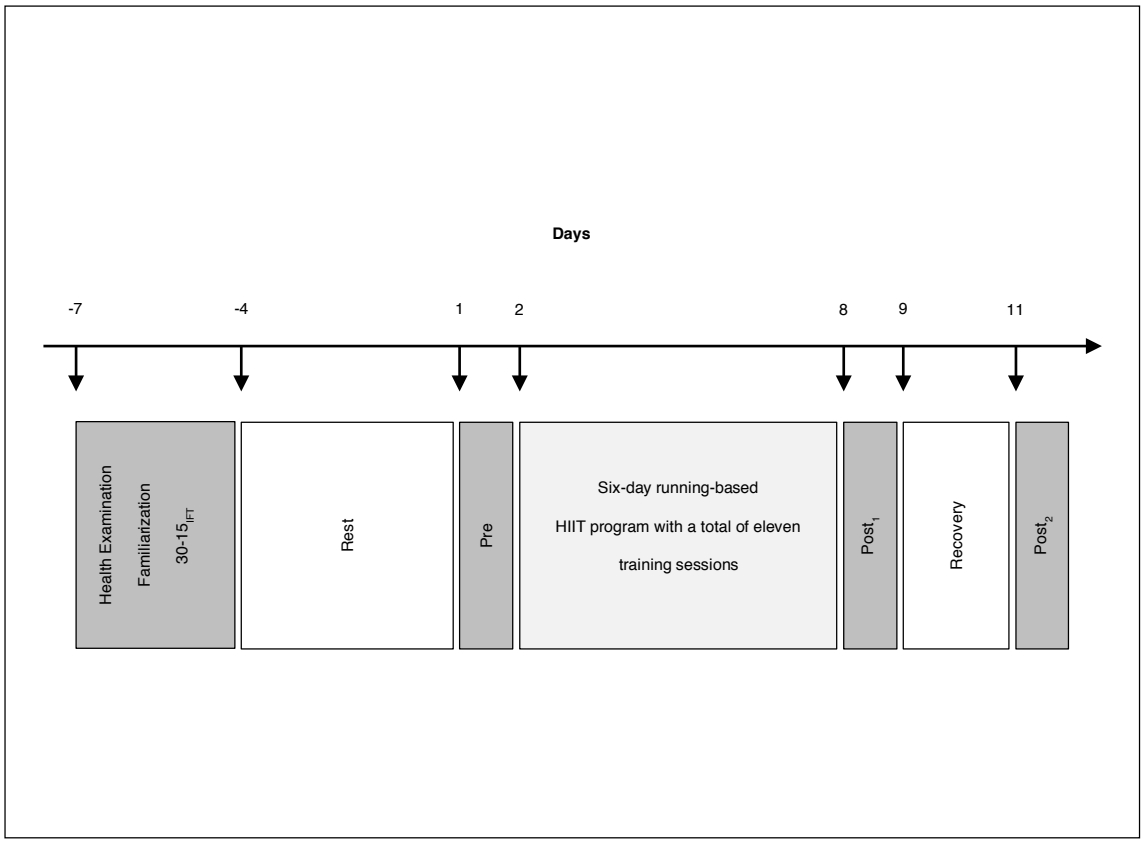

Fig. 1. Study design. 30-15_{IFT} = 30-15 Intermittent Fitness Test, HIIT = high-intensity interval training.

On all testing days (pre, $post_1$, $post_2$), 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, $post_1$, and $post_2$. 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 $\dot{V}O_{2max}$, 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 $km \cdot h^{-1}$, increasing 2 $km \cdot h^{-1}$ every 3 min with a constant incline of 0.5% until voluntary exhaustion. $\dot{V}O_2$ 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 $\dot{V}O_{2max.}$

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 $km \cdot h^{-1}$ for the first 30 s run and was increased by 0.5 $km \cdot h^{-1}$ 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_{IFT}) 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, $post_1$, and $post_2$. 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 ($m \cdot s^{-1}$), n = 17, ICC = 0.92, TE = 0.10, CV = 1.5 %).

Jump and linear sprint tests. On each testing day (pre, $post_1$, and $post_2$), countermovement jumps (CMJ) and multiple rebound jump tests (MRJ) were performed on a contact platform (Haynl-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, 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, $post_1$, and $post_2$. 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,

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, $post_1$, and $post_2$; 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-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, $post_1$, and $post_2$), 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 $30\text{-}15_{IFT}$ 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% HR_{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.

Table 2. Six-day high-intensity interval training program.

	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6
	Straight-line runs	Straight-line runs	Straight-line runs		Straight-line runs	Straight-line runs
	4 x 4 min	7 x 2 min	4 x 4 min		4 x 4 min	7 x 2 min
a.m.	(r = 3 min)	(r = 2 min)	(r = 3 min)	Rest	(r = 3 min)	(r = 2 min)
	80% V_{IFT}	85% V_{IFT}	80% V_{IFT}		80% V_{IFT}	85% V_{IFT}
	TL: 231 ± 59	TL: 236 ± 48	TL: 210 ± 63		TL: 217 ± 71	TL: 256 ± 56
	Straight-line sprints	40m-shuttle runs	Straight-line sprints	40m-shuttle runs	Straight-line sprints	40m-shuttle runs
	4 x 6 x 5 s	2 x 12 x 30 s	4 x 6 x 5 s	2 x 12 x 30 s	4 x 6 x 5 s	2 x 12 x 30 s
p.m.	(r = 25 s; R = 5 min)	(r = 30 s; R = 3 min)	(r = 25 s; R = 5 min)	(r = 30 s; R = 3 min)	(r = 25 s; R = 5 min)	(r = 30 s; R = 3 min)
	all out	90% V_{IFT}	all out	90% V_{IFT}	all out	90% V_{IFT}
	TL: 207 ± 64	TL: 270 ± 67	TL: 225 ± 67	TL: 257 ± 59	TL: 232 ± 68	TL: 290 ± 56

V_{IFT}: 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_{IFT}, r = 30 s, R = 3 min] means that the subject had to run two series of 12 intervals at 90% V_{IFT} 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, $post_1$, and $post_2$) 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 $m \cdot s^{-1}$) than at baseline (pre: 5.02 ± 0.52 $m \cdot s^{-1}$) or after recovery ($post_2$: 4.97 ± 0.56 $m \cdot s^{-1}$). The respective changes were -0.18 ± 0.13 $m \cdot s^{-1}$ ($p = 0.001$; effect size = -1.51) from pre to $post_1$ and 0.12 ± 0.26 $m \cdot s^{-1}$ ($p = 0.003$; 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).

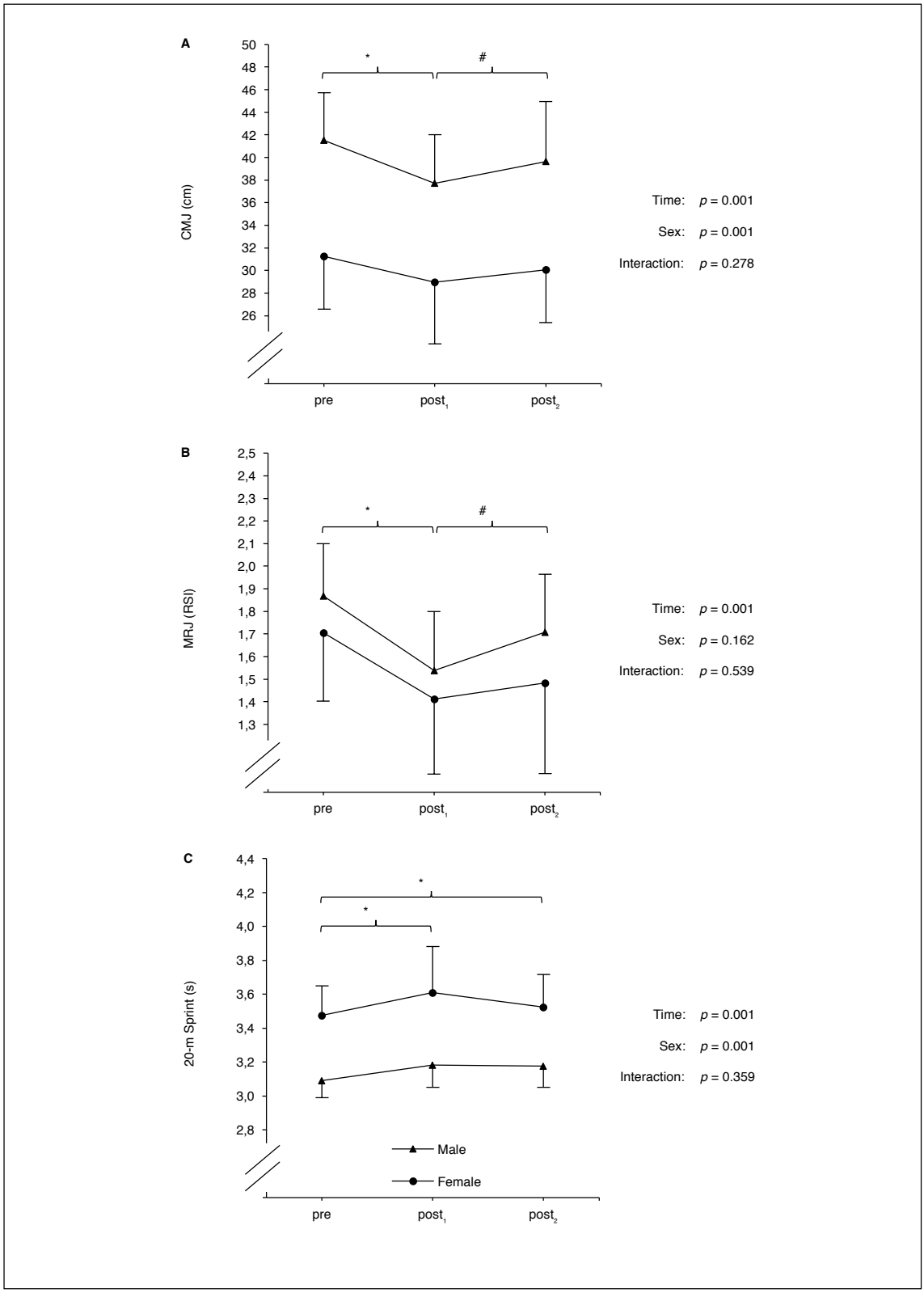

Fig. 2. Mean (± SD) countermovement jump (CMJ) height (A), multiple rebound jumps (MRJ) performance (B) and 20-m sprint time (C) in males and females at baseline (pre), after a six-day high-intensity interval training program ($post_1$), and following 72 h of recovery ($post_2$). RSI = reactive strength index. *Significant difference compared to pre ($p < 0.05$). #Significant difference compared to $post_1$ ($p < 0.05$).

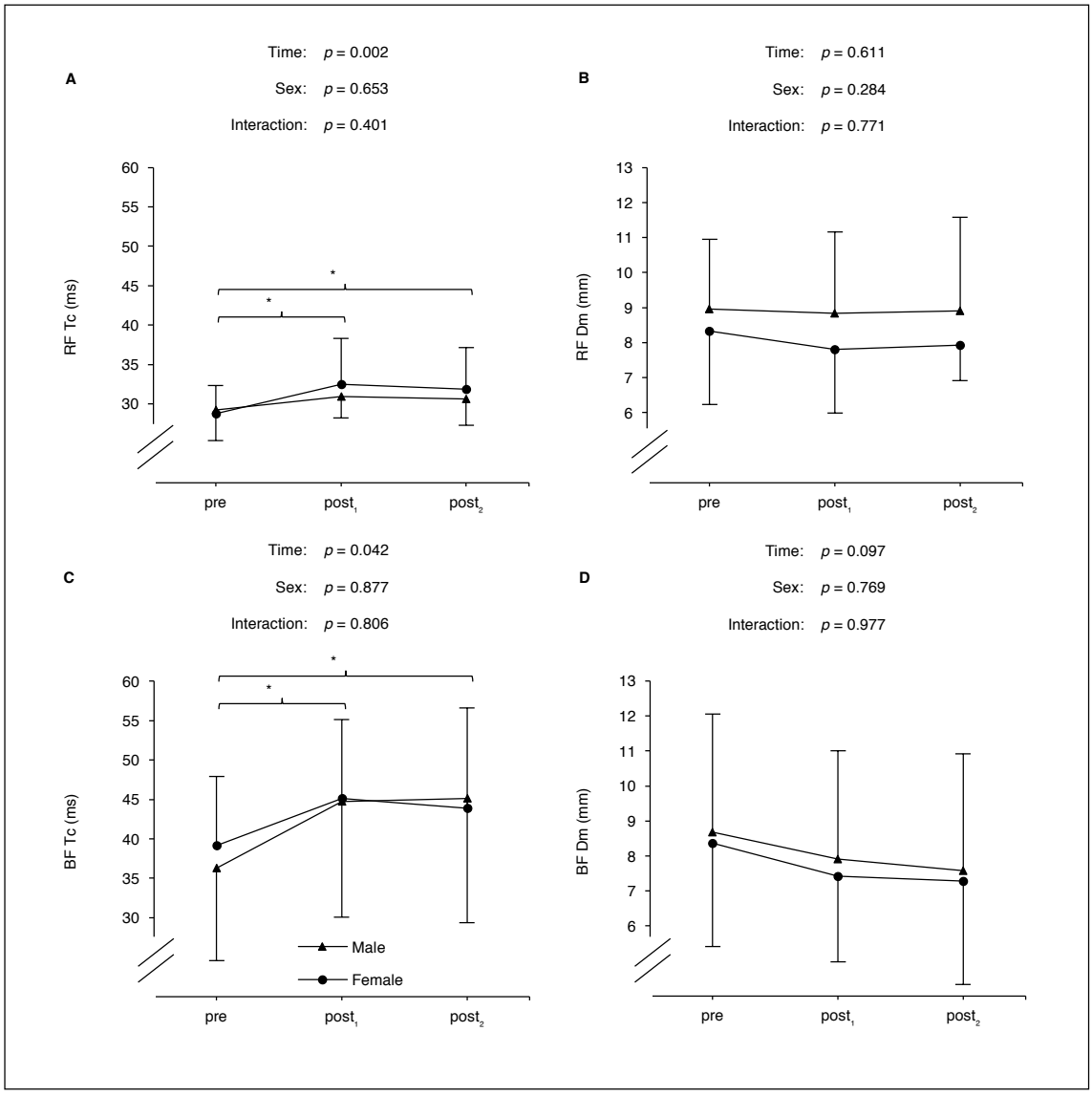

Fig. 3. Mean (± SD) of the contraction time (Tc) and maximal radial muscle displacement (Dm) of the rectus femoris (RF) and biceps femoris (BF) in males and females at baseline (pre), after a six-day high-intensity interval training program ($post_1$), and following 72 h of recovery ($post_2$). *Significant difference compared to pre ($p < 0.05$). #Significant difference compared to $post_1$ ($p < 0.05$).

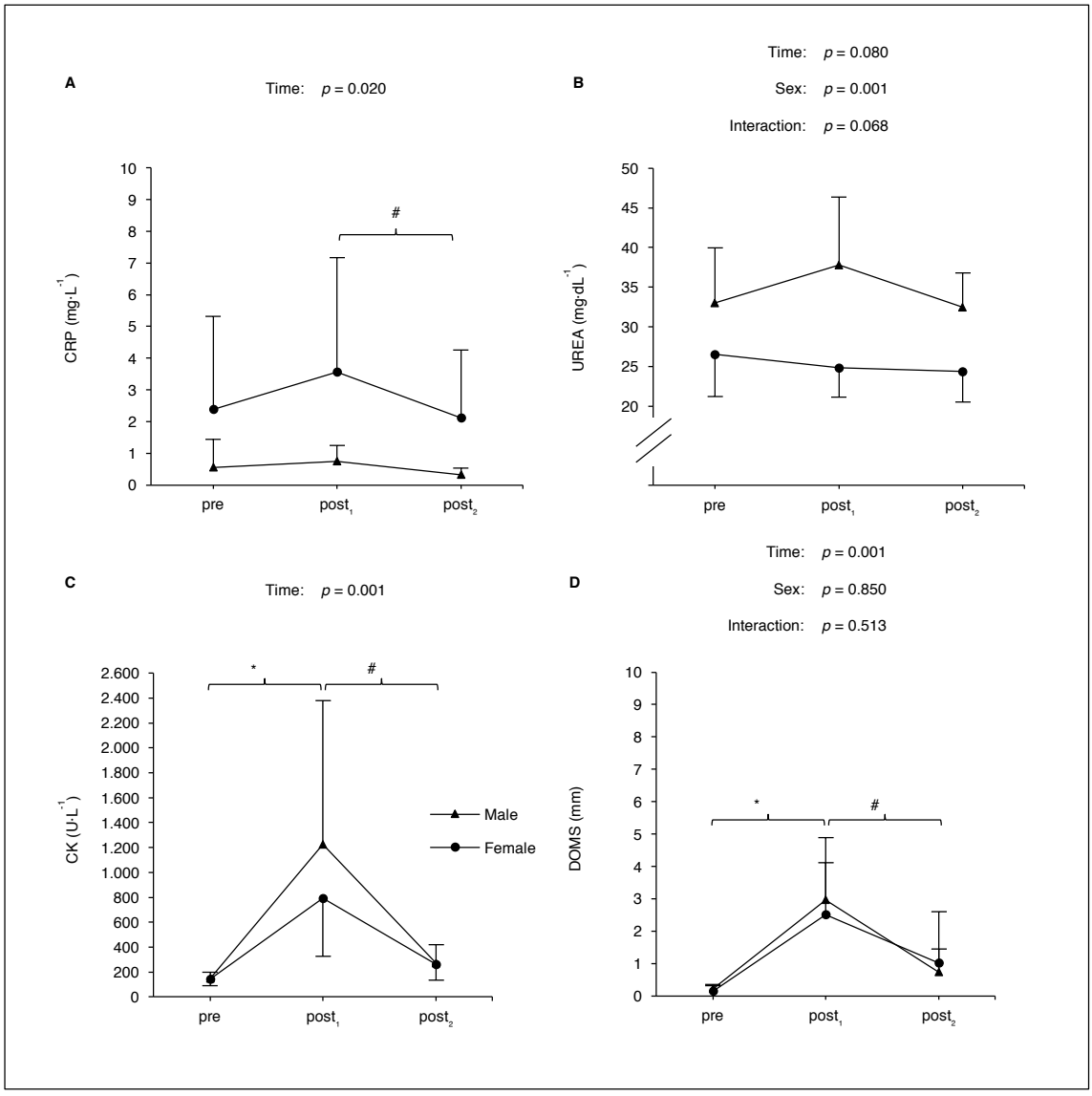

Fig. 4. Mean (± SD) of the serum concentration of c-reactive protein (CRP), urea, and creatinkinase (CK) as well as of the rating of delayed onset muscle soreness (DOMS) in males and females at baseline (pre), after a six-day high-intensity interval training program ($post_1$), and following 72 h of recovery ($post_2$). *Significant difference compared to pre ($p < 0.05$). #Significant difference compared to $post_1$ ($p < 0.05$).

Table 3. Markers of fatigue and recovery at baseline (pre), after a six-day high-intensity interval training program ($post_1$), and following 72 h of recovery ($post_2$) as well as percentage changes of performance and muscle contractile markers between testing days.

							Time	pre-$post_1$		$post_1$-$post_2$		pre-$post_2$	
	pre		$post_1$		$post_2$		*p*	%Δ ± CI	*d*	%Δ ± CI	*d*	%Δ ± CI	*d*
Performance markers													
CMJ (cm)	36.4 ± 6.8	(33.4–39.4)	33.3 ± 6.6*	(30.4–36.2)	34.8 ± 6.9#	(31.8–37.9)	< 0.001	-8.4 ± 2.9	-1.35	4.1 ± 2.9	-0.68	-4.3 ± 2.2	-0.79
MRJ (RSI)	1.79 ± 0.28	(1.66–1.91)	1.48 ± 0.30*	(1.34–1.61)	1.60 ± 0.35#	(1.44–1.75)	< 0.001	-17.4 ± 4.5	-1.60	6.5 ± 4.5	-0.63	-10.9 ± 5.3	-0.91
20-m Sprint (s)	3.28 ± 0.24	(3.18–3.39)	3.40 ± 0.30*	(3.26–3.53)	3.35 ± 0.24*	(3.25–3.46)	< 0.001	3.4 ± 1.8	-0.81	-1.2 ± 1.6	-0.37	2.2 ± 3.2	-0.65
Muscle contractile markers													
RF Tc (ms)	29.0 ± 3.8	(27.3–30.7)	31.7 ± 4.8*	(29.6–33.8)	31.2 ± 4.6*	(29.2–33.2)	< 0.002	9.9 ± 5.9	-0.72	-1.7 ± 4.1	-0.17	8.2 ± 6.0	-0.58
RF Dm (mm)	8.6 ± 2.1	(7.7–9.6)	8.3 ± 2.2	(7.3–9.3)	8.4 ± 2.1	(7.5–9.4)	< 0.611	-1.7 ± 10.3	-0.17	2.1 ± 6.2	-0.10	0.4 ± 10.4	-0.11
BF Tc (ms)	37.7 ± 10.7	(33.0–42.5)	44.9 ± 12.9*	(39.2–50.6)	44.5 ± 14.6*	(38.0–51.0)	< 0.042	28.7 ± 24.9	-0.46	-8.1 ± 21.5	-0.03	20.5 ± 18.9	-0.59
BF Dm (mm)	8.5 ± 3.3	(7.1–10.0)	7.7 ± 2.9	(6.4–8.9)	7.4 ± 3.2	(6.0–8.9)	< 0.097	-6.8 ± 12.2	-0.39	-3.3 ± 14.1	-0.10	-10.1 ± 15.0	-0.46
Biochemical markers													
CK ($U \cdot L^{-1}$)	147 ± 51	(125–170)	1010 ± 887*	(617–1403)	269 ± 134*#	(210–328)	< 0.001		-0.99		-0.95		-0.87
CRP ($mg \cdot L^{-1}$)	1.52 ± 2.46	(0.33–2.70)	2.23 ± 3.08	(0.74–3.71)	1.27 ± 1.84#	(0.38–2.16)	< 0.020		-0.33		-0.66		-0.20
UREA ($mg \cdot dL^{-1}$)	29.6 ± 7.2	(26.1–33.0)	30.9 ± 9.4	(26.4–35.5)	28.2 ± 5.9	(25.4–31.1)	< 0.080		-0.25		-0.46		-0.26
Subjective markers													
DOMS (mm)	0.2 ± 0.1	(0.1–0.3)	2.7 ± 1.8*	(2.0–3.5)	0.9 ± 1.2#	(0.3–1.4)	< 0.001		-1.50		-1.44		-0.57

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

CI: 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 creatinkinase; CRP: C-reactive protein; DOMS: delayed onset muscle soreness.

*Significant difference compared to pre.

#Significant difference compared to $post_1$.

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.

	Diagnostic effectiveness (%)		**Misclassification rate (%)**		**Youden`s Index**	
	Δpre-post$_1$	**Δpost$_1$-post$_2$**	**Δpre-post$_1$**	**Δpost$_1$-post$_2$**	**Δpre-post$_1$**	**Δpost$_1$-post$_2$**
Performance markers						
CMJ (cm)	63.6	60.0	36.4	40.0	0.01	0.20
MRJ (RSI)	68.2	60.0	31.8	40.0	0.08	0.20
20-m Sprint (s)	77.3	33.3	22.7	66.7	0.51	0.34
Muscle contractile markers						
RF Tc (ms)	68.2	40.0	31.8	60.0	0.38	0.21
RF Dm (mm)	50.0	66.7	50.0	33.3	0.04	0.30
BF Tc (ms)	54.5	40.0	45.5	60.0	0.03	0.23
BF Dm (mm)	50.0	60.0	50.0	40.0	0.11	0.20
Biochemical markers						
CK ($U \cdot L^{-1}$)	50.0	53.3	50.0	46.7	0.11	0.13
CRP ($mg \cdot L^{-1}$)	31.8	46.7	68.2	53.3	0.07	0.08
UREA ($mg \cdot dL^{-1}$)	30.0	46.7	70.0	53.3	0.10	0.00
Subjective markers						
DOMS (mm)	45.5	60.0	54.5	40.0	0.05	0.14

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

$U \cdot L^{-1}$ 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 $post_1$ 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 $post_1$ 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 $post_1$ and $post_2$ 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 $post_1$ and $post_2$. 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 $\dot{V}O_{2max}$ 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., 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 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.

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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.[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,[6] and provided similar acute relief of muscle soreness, as compared with that observed following massage.[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.[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.[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 ± 1.4 years; body mass: 69.0 ± 7.5 kg; height: 180 ± 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 ($30\text{-}15_{IFT}$). 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 30-15$_{IFT}$ (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 ear-lobe 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-*µ*l 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.[16]

Perceived recovery and stress: Perceived recovery and stress was assessed using the Short Recovery and Stress Scale (SRSS).[17] 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 ($n = 574$; $\alpha = 0.76$).

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$_{IFT}$, 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.[18]

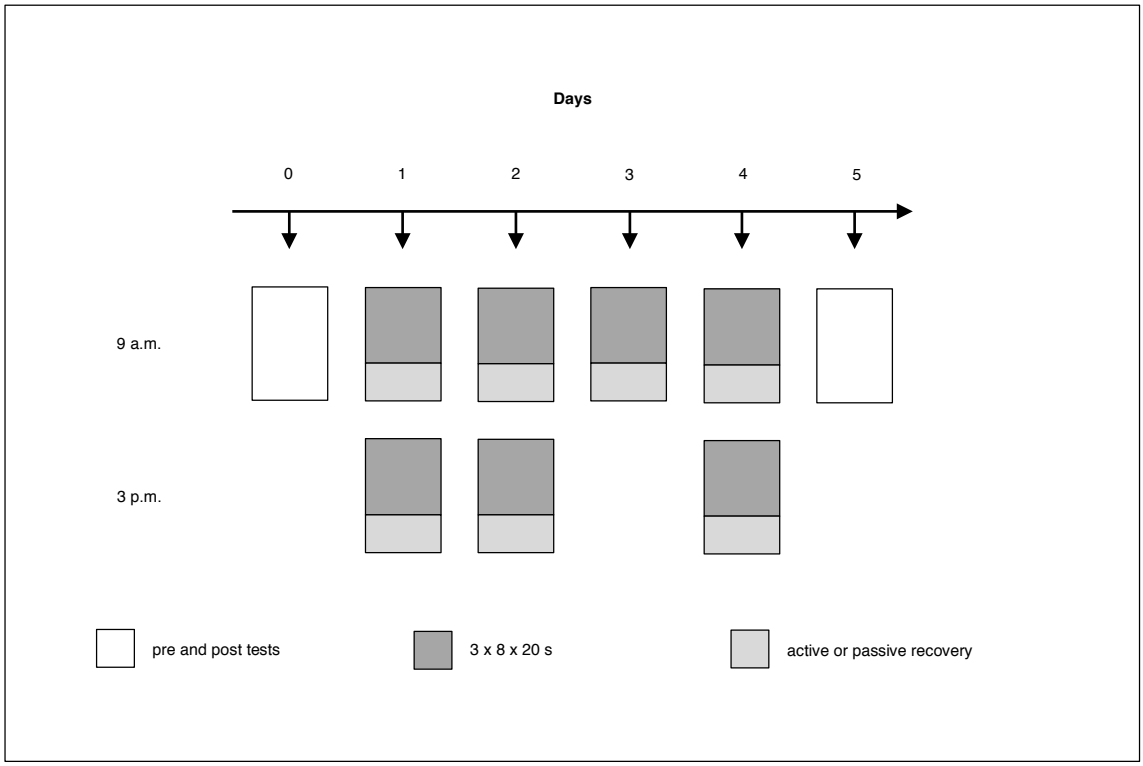

Figure 1: Arrangement of the seven high-intensity interval training sessions during the four-day shock microcycle.

Recovery Intervention

ACT included 15-min shuttle runs at 40% V_{IFT} that were started within 5 min after completing each training session (Figure 1). Depending on the individual V_{IFT} that was reached by the players in the 30-15$_{IFT}$, 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_{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$_{IFT}$ 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.[19] 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).[20] 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.[20] If the chance of a harmful and a beneficial effect were both > 5%, the true difference was considered to be unclear.

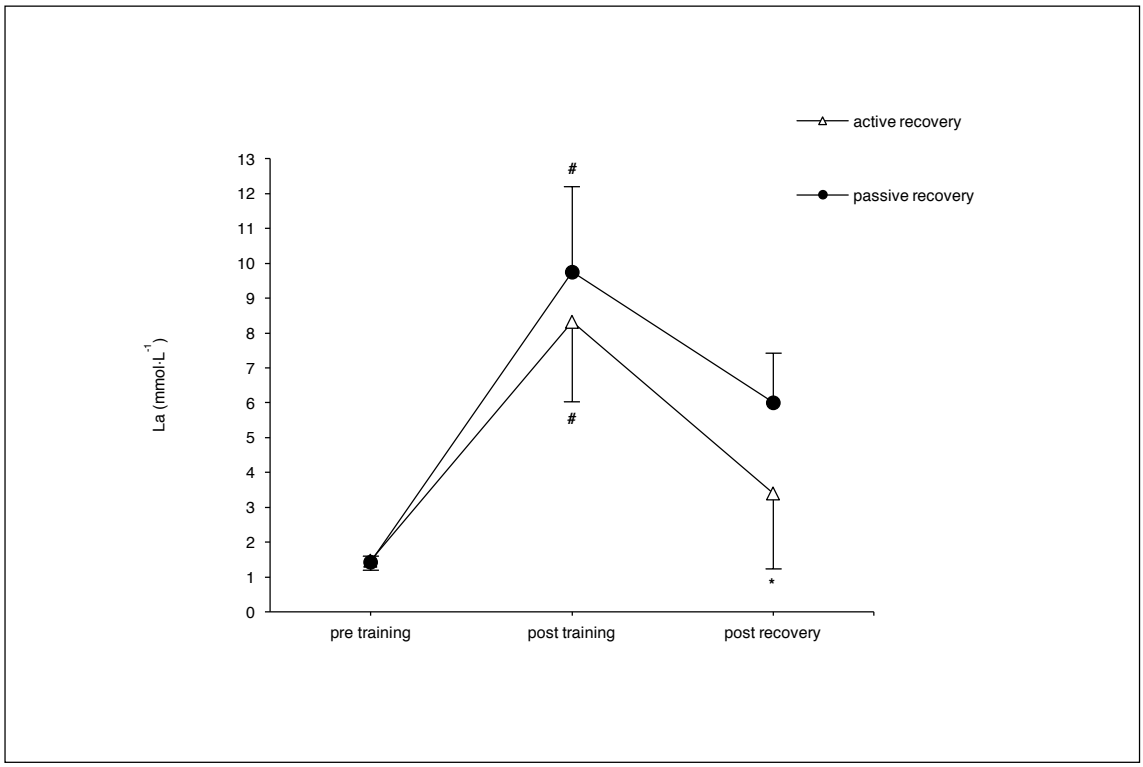

Figure 2: Mean (± SD) blood lactate concentration throughout each high-intensity interval training session determined pre and post training as well as post recovery intervention. #Significant difference compared to pre training and post recovery ($p < 0.05$). *Significant difference compared to passive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

				Group changes			Differences in group changes			
Measure	**Group**	**Pre**	**Post**	**ΔPre-Post**	**Time**	**ES**	**ΔPAS-ACT**	**Intervention x Time**	**ES**	**Qualitative Inference**
		(Mean ± SD)	(Mean ± SD)	(Mean ± SD)	*p*		(Mean ± 90% CI)	*p*		
Jump performance										
CMJ (cm)	PAS	40.9 ± 4.4	37.1 ± 4.1*	-3.8 ± 2.9	0.002	-1.42	0.7 ± 2.4	0.578	0.22	Unclear
	ACT	40.1 ± 5.6	37.0 ± 6.1*	-3.1 ± 2.4		-1.39				
Blood parameter										
CK ($U \cdot L^{-1}$)	PAS	201 ± 32	428 ± 311	226 ± 311	0.060	0.81	112 ± 161	0.846	0.74	Unclear
	ACT	182 ± 41	520 ± 291	337 ± 316		0.76				
Muscle soreness										
VAS (mm)	PAS	0.3 ± 0.4	3.3 ± 2.2*	3.0 ± 2.3	0.001	2.17	0.5 ± 2.2	0.791	0.10	Unclear
	ACT	0.2 ± 0.6	3.8 ± 2.7*	3.5 ± 2.5		2.02				

Abbreviations: PAS, passive recovery; ACT, active recovery; CMJ, countermovement jump; CK, creatinkinase; 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).

				Group changes			Differences in group changes			
Measure	**Group**	**Pre**	**Post**	**ΔPre-Post**	**Time**	**ES**	**ΔPAS-ACT**	**Intervention x Time**	**ES**	**Qualitative Inference**
		(Mean ± SD)	(Mean ± SD)	(Mean ± SD)	*p*		(Mean ± 90% CI)	*p*		
Short recovery and stress scale										
PPC	PAS	5.0 ± 0.8	3.4 ± 0.9*	-1.6 ± 0.5	0.001	-3.36	0.4 ± 0.6	0.285	0.44	Unclear
	ACT	5.4 ± 0.5	3.4 ± 1.3*	-2.0 ± 1.2		-1.79				
MPC	PAS	5.5 ± 0.8	4.0 ± 0.8*	-1.5 ± 1.2	0.006	-1.34	0.1 ± 0.9	0.802	0.10	Unclear
	ACT	5.8 ± 0.5	4.4 ± 1.3*	-1.4 ± 1.3		-1.13				
EB	PAS	4.8 ± 0.7	4.3 ± 1.0	-0.5 ± 1.2	0.048	-0.45	0.5 ± 0.8	0.275	0.45	Unclear
	ACT	5.6 ± 0.5	4.6 ± 0.9*	-1.0 ± 0.9		-1.15				
OR	PAS	4.9 ± 1.0	2.5 ± 0.9*	-2.4 ± 1.1	0.001	-2.39	0.4 ± 0.9	0.442	0.31	Unclear
	ACT	5.3 ± 0.7	3.3 ± 1.2*	-2.0 ± 1.2		-1.79				
MS	PAS	0.8 ± 0.7	3.8 ± 1.2*	3.0 ± 1.1^{D}	0.001	3.00	0.0 ± 1.2	1.000	0.00	Unclear
	ACT	0.5 ± 0.8	3.5 ± 1.7*	3.0 ± 1.6^{D}		2.00				
LA	PAS	0.5 ± 0.8	1.9 ± 1.5*	1.4 ± 1.3^{B}	0.007	1.13	0.3 ± 0.9	0.676	0.19	Unclear
	ACT	0.3 ± 0.5	1.4 ± 0.9*	1.1 ± 1.0^{B}		1.21				
NES	PAS	0.6 ± 0.7	1.9 ± 1.6*	1.3 ± 1.4^{B}	0.018	0.96	0.1 ± 0.7	0.732	0.13	Unclear
	ACT	0.0 ± 0.0	1.1 ± 1.0*	1.1 ± 1.0^{B}		1.21				
OS	PAS	0.9 ± 0.6	3.5 ± 1.3*	2.6 ± 0.9^{D}	0.001	3.06	0.1 ± 0.7	0.732	0.13	Unclear
	ACT	0.5 ± 0.8	3.3 ± 1.8*	2.8 ± 1.5^{D}		1.98				

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 studies[14, 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),[28] attenuates with the cessation of exercise.[29] In this context, Andersson et al.[14] and Dawson et al.[30] 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.,[31] 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.[31] 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.[14,32]

In order to evaluate the different aspects of the perceptions of recovery and stress, the SRSS was developed.[17] 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.

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5 GESAMTDISKUSSION

Die vorliegende Dissertation befasst sich mit der Belastungs- und Erholungssteuerung im HIT. In den drei Untersuchungsmodulen wurden die akuten Belastungs- und mittelfristigen Ermüdungsreaktionen im Rahmen von fünf praxisüblichen und unterschiedlich gestalteten HIT-Protokollen quantifiziert, die Sensitivität von praxistauglichen Parametern zur Diagnostik von Ermüdung und Erholtheit im HIT evaluiert sowie der Einfluss von aktiver Erholung auf die durch HIT induzierten Ermüdungseffekte überprüft. Die Ergebnisse der Teilstudien wurden in drei Artikeln vorgestellt und jeweils diskutiert. An dieser Stelle schließt die Arbeit mit einer kurzen übergeordneten Darstellung und Diskussion der Ergebnisse und deren Bedeutung für die Sportpraxis.

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 Beanspruchungsgrad. 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

Klassisches hochvolumiges Ausdauertraining nach der Dauermethode ist durch die Belastungsintensität und –Dauer charakterisiert. Intervallbasiertes Ausdauertraining kann hingegen auf diverse Art und Weise variiert werden (Saltin, Essén, & Pedersen, 1976). Bei der Trainingssteuerung im HIT müssen daher verschiedene Belastungsnormative (Tab. 3) berücksichtigt werden. Hieraus entstanden in der Trainingspraxis sowie in wissenschaftlichen Studien eine Vielzahl an unterschiedlichen HIT-Varianten (Tab. 1). Obwohl sie zu ähnlichen Anpassungen aerober und anaerober Leistungskomponenten führen, unterscheiden sich die akuten Belastungsreaktionen zwischen unterschiedlichen HIT-Protokollen teils erheblich. Für eine adressatengerechte Belastungs- und Erholungssteuerung sollte jedoch der Einfluss der einzelnen Steuerungsgrößen auf die akuten Belastungsreaktionen bekannt sein. Modelle zur Klassifizierung verschiedener HIT-Protokolle unter Berücksichtigung der physiologischen Belastungsreaktionen wurden unter anderem von Buchheit & Laursen (2013) sowie Tschakert & Hofmann (2013) vorgeschlagen. Sie berücksichtigen jedoch bislang nicht alle Belastungsnormative. Dies liegt mitunter daran, dass der potentielle Einfluss auf die Trainingswirkung bisher nicht von allen Variablen zufriedenstellend evaluiert worden ist.

Tab. 3. *Steuerungsgrößen im High-Intensity Ausdauertraining.*

Kernvariablen	Ergänzende Variablen
Intensität der Intervalle (z.B. %Hfmax, %vVO_2max, %V_{IFT}, RPE)	Anzahl der Intervalle ([n])
Dauer der Intervalle ([s] oder [min])	Anzahl der Serien ([n])
Intensität der Intervallpausen (z.B. %Hfmax, %vVO_2max, %V_{IFT}, RPE)	Intensität der Serienpausen (z.B. %Hfmax, %vVO_2max, %V_{IFT}, RPE)
Dauer der Intervallpausen ([s] oder [min])	Dauer der Serienpausen ([s] oder [min])
Mittlere Trainingsbeanspruchung (z.B. Pmean) und Gesamttrainingsdauer	Beanspruchungsform (z.B. Laufen, Fahrradfahren) und Hilfsmittel (z.B. Ropes)

Hfmax = maximale Herzfrequenz; vVO_2max = 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.

Der Vergleich von P_{15} mit P_5 signalisiert, dass auch die Dauer der Intervallpausen die Laktatkinetik sowie den Säure-Basen-Status beeinflusst. In beiden Protokollen lag die Laufgeschwindigkeit deutlich oberhalb des maxLaSS. Die Dauer der Intervallpausen in P_{15} reichte aber aus, um das anfallende Laktat zu eliminieren und ein LaSS zu erreichen. Gosselin, Kozlowski, DeVinney-Boymel, & Hambridge (2012) zeigten, dass bei gleicher Intensität und Dauer der Belastungsintervalle das Trainingsprotokoll mit den kürzeren Intervallpausen in einer stärkeren Laktatanhäufung und einer höheren durchschnittlichen Hf resultierte. Auch Glaister (2005) schlussfolgert in seiner Übersichtsarbeit, dass die metabolische Antwort während intermittierender Arbeit gleichermaßen durch die Pausendauer determiniert wird. Im dritten Untersuchungsmodul führte beispielsweise ein Protokoll mit einer 15-sekündigen Intervalldauer und gleicher Intervallintensität bei einer kürzeren Pausendauer zu deutlich höheren Blutlaktatwerten (9,6 mmol/l). Folglich bestimmt auch die Dauer der Erholungsphasen die akuten Belastungsreaktionen.

Mehrere Untersuchungen konnten zeigen, dass auch die Intensität der Intervall- und Serienpausen in Abhängigkeit der Pausendauer die akuten Belastungsreaktionen beeinflussen (Abderrahmane et al., 2013; Abderrahman et al., 2013b; Buchheit et al., 2009; Dupont, Blondel, & Berthoin, 2003; Dupont, Moalla, Guinhouya, Ahmaidi, & Berthoin, 2004; Wahl, Mathes, Achtzehn, Bloch, & Mester, 2014; Wahl, Mathes, et al., 2013; Wahl, Zinner, et al., 2013). Zusammenfassend zeigten die Studien, dass eine aktive Erholung während kürzerer Intervall- und Serienpausen in höheren metabolischen und hormonellen Belastungsreaktionen resultieren, während gleiches für eine passive Erholung während längerer Intervall- und Serienpausen gilt. Dies konnte in der vorliegenden Arbeit nicht überprüft werden, da sich die Athleten zwischen den Belastungsphasen der fünf miteinander verglichenen HIT-Protokolle stets passiv erholten. Aufgrund der vielen Publikationen zu diesem Thema sei aber an dieser Stelle auf die Bedeutung der Pausenintensität für die durch HIT induzierten akuten Belastungsreaktionen hingewiesen.

Trotz der kurzen Dauer der Belastungsintervalle waren die akuten metabolischen und kardialen Belastungsreaktionen während P_{30} im Vergleich zu P_{15} deutlich stärker. Zusätzlich zu den zuvor beschriebenen Einflussgrößen liegen die Protokollunterschiede hier womöglich in der Integration von Richtungswechseln begründet. Bei vergleichbarer Intervallintensität resultieren sportspielspezifische Richtungswechselläufe im Vergleich zu Linearläufen in höheren Laktatbildungsraten sowie stärkeren physiologischen und perzeptiven Belastungsreaktionen (Buchheit, Haydar, Hader, Ufland, & Ahmaidi, 2011; Buchheit & Laursen, 2013b; Dal Pupo et al., 2013; Hader, Mendez-Villanueva, Ahmaidi, Williams, & Buchheit, 2014). Zwar absolvierten die Athleten sowohl in P_{30} als auch in P_{15} Shuttleläufe über 40 m. Die Anzahl der Richtungswechsel war während P_{30} aber im Mittel insgesamt mehr als doppelt so hoch. Daher sollte der Einfluss von Richtungswechseln auf die akuten Belastungsreaktionen bei der Planung von HIT-Protokollen ebenfalls berücksichtigt werden.

Die durch ein HIT induzierten akuten Belastungsreaktionen differieren zwischen unterschiedlichen Trainingsprotokollen zum Teil erheblich und werden insbesondere durch die Dauer und Intensität der Intervalle beeinflusst. Aber auch die Dauer und Intensität der Intervall- bzw. Serienpausen sowie die Beanspruchungsform spielen eine wichtige Rolle bei der Belastungssteuerung. Während P_{15} aufgrund des erreichten LaSS höhere Trainingsumfänge gestattet, führen insbesondere P_{240}, P_{120} und P_5 zu den gewünschten metabolischen und kardialen Beanspruchungen, die mit chronischen und HIT-spezifischen Adaptationen assoziiert werden. Bei kürzeren Pausenzeiten in P_{30} und P_{15} würden jedoch auch hier womöglich deutlich höhere akute Belastungsreaktionen erreicht. Zusammenfassend können die Aussagen zu den Belastungsreaktionen im HIT von Tschakert & Hofmann (2013) sowie Saltin et al. (1976) folgendermaßen ergänzt werden: Je länger die Belastungsphasen und kürzer die Erholungsphasen sind und je mehr Richtungswechsel in das Training integriert werden, desto höher ist die potentielle Blutlaktatakkumulation bzw. Blut-pH-Wert-Absenkung, vorausgesetzt die Intensität der Belastungsphasen liegt oberhalb des individuellen maxLaSS. Neben der Belastungsintensität werden die akuten Belastungsreaktionen dabei auch durch die Pausenintensität determiniert.

Die Bestimmung des Anstrengungsempfindens bei körperlicher Arbeit gilt schon lange als Standard (Löllgen, 2004). Die Validität der Session-RPE-Methode zur Bewertung der Beanspruchung wurde bereits für unterschiedliche Belastungssituationen bestätigt (Foster, Mikat & Porcari, 2006; Alexiou & Coutts, 2008; Coutts, Murphy, Pine, Reaburn, & Impellizzeri, 2003; Lupo, Capranica, & Tessitore, 2014; Wallace, Slattery, & Coutts, 2009). So besteht meist eine enge Korrelation des PRE-Wertes zu physiologischen Parametern während einer Belastung (u.a. Hf, Laktatwerte, Sauerstoffaufnahme, Atemfrequenz)(Gaudino et al., 2015; Green et al., 2006; Impellizzeri, Rampinini, Coutts, Sassi, & Marcora, 2004; Löllgen, 2004; Perandini, Siqueira-Pereira, Okuno, Soares-Caldeira, & Nakamura, 2012). In der vorliegenden Arbeit spiegelte das subjektive Belastungsempfinden insbesondere die metabolischen Stoffwechselreaktionen in den fünf HIT-Protokollen wider. Die höchsten RPE-Werte wurden während P_{240}, P_{120} und P_5 angegeben, während P_{15} als am wenigsten anstrengend eingeschätzt wurde. Demzufolge ist die Session-RPE-Methode ein praktikables Tool, um speziell die metabolischen Belastungsreaktionen während eines HIT zu quantifizieren.

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

P_5 führte innerhalb der ersten 24 Stunden nach Belastungsende zur höchsten CK-Aktivität im Serum sowie zum stärksten Schmerzempfinden in der beanspruchten Muskulatur. Auch die Sprungleistung war ausschließlich nach P_5 reduziert. Lediglich P_{240} verursachte ebenfalls eine Erhöhung des CK-Werts im Blut innerhalb der ersten 24 Stunden im Anschluss an die HIT-Einheit. Ähnliche muskuläre Ermüdungserscheinungen wurden auch in weiteren Untersuchungen vor allem nach einem Wiederholungssprinttraining aber auch im Anschluss an andere HIT-Varianten beobachtet (Howatson & Milak, 2009; Keane, Salicki, Goodall, Thomas, & Howatson, 2015; Thompson et al., 1999). Aufgrund der höchsten Netto-Trainingszeit und Blutlaktatakkumulation resultierten P_{240} und P_{120} zudem vermutlich in der stärksten Muskelglykogenspeicherentleerung.

Hauptunterschied zwischen P_5 und den anderen HIT-Protokollen ist die extrem hohe Laufgeschwindigkeit sowie der ständige Wechsel zwischen Beschleunigungs- und Bremsphasen. Wie in Kap 2.1.2 ausführlich beschrieben, führt dies zu hohen exzentrischen Muskelspannungen und letztlich in den beobachteten neuromuskulären Funktionseinschränkungen. Der geringere, aber dennoch signifikante Anstieg der CK-Konzentration im Blut im Anschluss an P_{240} kann durch die höhere Netto-Belastungsdauer (16 min) im Vergleich zu den anderen HIT-Protokollen erklärt werden. So weisen Fallon, Sivyer, Sivyer, & Dare (1999) sowie Noakes (1987) darauf hin, dass ein Anstieg muskelspezifischer Enzyme im Blut neben der Belastungsintensität vorrangig durch die Belastungsdauer induziert wird. Diverse Studien konnten zeigen, dass hochvolumige Ausdauerbelastungen trotz niedriger Laufgeschwindigkeiten mit einer erhöhten CK-Aktivität sowie neuromuskulären Ermüdungserscheinungen einhergehen (García-Manso, Rodríguez-Ruiz, et al., 2011; Neubauer, König, & Wagner, 2008; Noakes & Carter, 1976; Nokes, Kotzenberg, McArthur & Dykman, 2983; Rama et al., 1994).

Hauptsächlich P_5 induzierte nach bereits einmaliger Verabreichung mittelfristige primär muskulär bedingte Funktionseinschränkungen. Diese haben das Potential, die Leistungsfähigkeit für einige Tage zu reduzieren (Keane et al., 2015). Da in der Praxis häufig mehrere HIT-Einheiten während eines Trainingsblocks in kurzer Zeit absolviert werden, sollte der regenerative Folgebedarf im Rahmen eines Wiederholungssprinttrainings unbedingt bei der Trainingsplanung berücksichtigt werden. Ein Wiederholungssprinttraining unmittelbar vor einer Wettkampfbelastung ist beispielsweise kontrainduziert. Aber auch HIT-Protokolle mit niedrigeren Laufgeschwindigkeiten können aufgrund der längeren Netto-Belastungsdauer mit Muskelzellschädigungen, Muskelschmerzen und Leistungseinbußen einhergehen. Ferner verursachen die längeren Netto-Belastungszeiten von P_{240} und P_{120} bei gleichzeitig hohem metabolischen Stress eine womöglich größere Entleerung der intramuskulären Glykogenspeicher im Vergleich zu P_5. Eine adäquate Kohlenhydratzufuhr sollte daher insbesondere nach HIT-Protokollen mit längeren Intervallen erfolgen.

5.4 Diagnostikinventar zur Messung von Erholungsbedarf

Für die Evaluation der Sensitivität praxistauglicher Parameter zur Diagnostik von Ermüdung und Erholtheit im HIT absolvierten insgesamt 22 Athleten aus den Sportspielen ein sechstägiges HIT-Programm. Die Auswahl der Trainingsprotokolle erfolgte anhand der Befunde aus dem ersten Untersuchungsmodul mit dem Ziel, einen hohen regenerativen Folgebedarf zu provozieren. Das Trainingsprogramm resultierte in einer reversiblen Abnahme der sportspielspezifischen Leistungsfähigkeit, die innerhalb 72 Stunden nach dem Mikrozyklus wieder abklang. Ein ähnliches Muster zeigten eine Auswahl von leistungsdiagnostischen, neuromuskulären, laborchemischen und subjektiven Surrogatmarkern. Demnach sind Sprungleistung, Muskelkontraktilität, CK-Aktivität im Serum sowie das subjektiv empfundene Muskelschmerzempfinden potentiell praktikable Tools für die Messung des Erholungsbedarfs nach HIT. Eine detailliertere Auswertung der Daten mittels multipler Regressionsanalyse sowie Vierfelderkontingenztafel offenbarte jedoch, dass keiner der potentiell geeigneten Surrogatmarker ausreichende Sensitivität, Spezifität und diagnostische Effektivität aufweist. Unter zunehmender Ermüdung bzw. mangelnder Erholung leidet letztlich die sportartspezifische Leistungsfähigkeit (Meyer et al., 2013). Die Ergebnisse des zweiten Untersuchungsmoduls, die sowohl für die männlichen als auch die weiblichen Sportler gelten, entstammen daher der Annahme, dass die aktuell abrufbare sportspielspezifische Leistungsfähigkeit wichtigstes Außenkriterium bei der Erfassung von Ermüdung und Erholtheit für die vorliegende Stichprobe ist.

In der Sportpraxis sollten geeignete Indikatoren zur Diagnostik von Ermüdung und Erholtheit eine individuelle Bewertung des Regenerationsbedarfs erlauben (Meyer et al., 2013). Studien, die aufzeigen, dass eine Auswahl bestimmter Parameter in einer Gruppenstatistik zwar im Mittel der experimentell induzierten Ermüdung folgen, sind für eine individuelle Statusdiagnostik jedoch nicht zwangsläufig geeignet (Meyer et al., 2013). So hat sich im zweiten Untersuchungsmodul beispielsweise die CK-Aktivität im Serum im Mittel mit statistischer Signifikanz zwischen den Messzeitpunkten in die zu erwartenden Richtungen bewegt. Jedoch ergab die Analyse mittels Kontingenztafel, dass die Reduktion der sportartspezifischen Leistungsfähigkeit im Anschluss an das sechstägige HIT-Programm lediglich bei 9 von 15 Athleten durch einen Anstieg des CK-Wertes im Blut abgebildet wurde. Wenn also in Einzelfällen in den Surrogatmarkern ein gegenläufiger Trend verzeichnet wird, obwohl von einer Ermüdung auszugehen ist, reicht die diagnostische Effektivität des Parameters für ein leistungssportliches Setting nicht aus (Meyer et al., 2013). Will man jedoch die Wirksamkeit einer Erholungsstrategie im Rahmen von HIT wissenschaftlich nachweisen, sind Mittelwertsstatistiken bzw. Mittelwertsbewegungen durchaus brauchbar.

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

Erholtheit. Auch die Regressionsanalyse konnte keine signifikanten Zusammenhänge zwischen den Veränderungen der spezifischen Leistungsfähigkeit und den leistungsdiagnostischen, neuromuskulären, laborchemischen und subjektiven Parametern ermitteln. Diese Ergebnisse zeigen, dass belastungsinduzierte Veränderungen von Ermüdungsmarkern hochindividuell sind. Auf inter- und intraindividuell variierende Ermüdungsreaktionen wurde bereits von anderen Autoren hingewiesen (Halson, 2014; Nédélec et al., 2012).

Die niedrigen Zusammenhänge zwischen einerseits der Indikatorleistung und den Surrogatmarkern sowie andererseits zwischen den Surrogatmarkern selbst können darüber hinaus auf die komplexen Strukturen von Ermüdungsmechanismen zurückgeführt werden, die sich auf verschiedenen Ebenen unterschiedlich stark manifestieren (muskulär, metabolisch, nerval, mental, etc.). Ferner wiesen Andersson et al. (2008) darauf hin, dass sich das zeitliche Verhaltensmuster zwischen den Ermüdungsmarkern kaum gleicht. Sie demonstrierten, dass noch 74 Stunden nach einem Fußballmatch die Sprungleistung reduziert sowie die CK-Aktivität und der empfundene Muskelschmerz erhöht waren, wohingegen sich die Sprintfähigkeit innerhalb der ersten fünf Stunden nach dem Match wieder erholte. Dies könnte ebenso die niedrigen Zusammenhänge zwischen der spezifischen Leistungsfähigkeit und den Surrogatmarkern aufklären. Der Zusammenhang eines Parameters mit der sportartspezifischen Leistungsfähigkeit und dementsprechend seine Tauglichkeit zum Monitoring von Ermüdung und Erholtheit sollte in der Sportpraxis daher möglichst auf individueller Ebene für jeden einzelnen Athleten im Längsschnitt identifiziert werden.

Anhand der Ergebnisse aus dem zweiten Untersuchungsmodul konnten keine signifikanten Unterschieden im Ermüdungs- und Erholungsverhalten zwischen den männlichen und weiblichen Sportlern festgestellt werden. Dennoch zeigte sich, dass die CK-Konzentration im Blut 24 Stunden nach dem hochintensiven Mikrozyklus bei den männlichen Athleten 64,8% über dem der weiblichen Teilnehmer lag. Dies kann in erster Linie auf den höheren CK-Gehalt der männlichen Muskelzellen zurückgeführt werden (Meyer & Meister, 2011; Mougios, 2007; Nédélec et al., 2012). Aufgrund der größeren Muskelmasse bzw. Muskelkraft und der damit einhergehenden höheren realisierbaren absoluten Trainingsintensität bzw. höheren mechanischen Belastung sowie der größeren Anteile an FT-Fasern sind männliche Athleten aber auch grundsätzlich anfälliger für muskulär bedingte Ermüdungserscheinungen (Leistungsverlust, Muskelzellschädigungen, Muskelkater, etc.) (Billaut & Bishop, 2009; Hunter, 2014). So zeigte Häkkinen (1993), dass die Leistungsfähigkeit sowie das neuromuskuläre Aktivierungsverhalten der männlichen Sportler im Vergleich zu den weiblichen Athleten nach einem hochintensiven Krafttraining stärker eingeschränkt war. Zudem brauchten die Männer länger, um sich von der belastungsbedingten Ermüdung zu erholen. In der Sportpraxis sollte daher der Einfluss des Geschlechts auf die im Rahmen

von HIT induzierten Ermüdungs- und Erholungscharakteristika bei der Belastungs- und Erholungssteuerung mit einkalkuliert werden. So tolerieren weibliche Athleten möglicherweise höhere HIT-Umfänge und erholen sich auch wieder schneller nach einer solchen Belastung als männliche Sportler.

Eine kontinuierliche Leistungsentwicklung sowie eine ideale Wettkampfvorbereitung setzt ein individuell optimales Verhältnis aus Belastung und Erholung voraus. Ein potentiell sensitives Diagnostikinventar für ein hierfür notwendiges Belastungs- und Ermüdungsmonitoring sind unter Berücksichtigung der Befunde aus dem zweiten Untersuchungsmodul in Tab. 4 dargestellt. Die Auflistung gilt für die durch HIT induzierten Funktionseinschränkungen. Verschiedene Belastungssituationen gehen mit unterschiedlich ausgeprägten Ermüdungsphänomenen einher. Daher sollte die Sensitivität praxistauglicher Surrogatmarker auch für andere Formen der körperlichen Arbeit (z.B. Krafttraining, hochvolumiges Ausdauertraining, etc.) im Detail evaluiert werden. Aufgrund individueller Einflussgrößen (u.a. Geschlecht) und um alle relevante Ebenen der Ermüdung abzubilden, empfiehlt sich zudem der Einsatz einer Kombination aus den genannten Parameter, deren Tauglichkeit jedoch auf individueller Basis überprüft werden muss.

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

	Kurzbeschreibung	Apparaturen	Parameter	Literatur
Leistungsdiagnostik				
CMJ	· Maximaler vertikaler Sprung nach einer vorausgehenden Kniegelenksbeugung von etwa 90°	· Kontaktplatte oder Kraftmessplatte · Computer mit entsprechender Software	· Maximale Sprunghöhe [cm] · Mittlere Sprunghöhe bei mehreren Sprüngen [cm]	· Tanner & Gore (2013)
MRJ	· Reaktive vertikale Mehrfachsprünge mit möglichst hohen Sprunghöhen bei gleichzeitig niedrigen Bodenkontaktzeiten	· Kontaktplatte oder Kraftmessplatte · Computer mit entsprechender Software	· Maximale und mittlere Sprunghöhe [cm] · Effizienzkoeffizient des Absprungs (Sprunghöhe [m] geteilt durch Bodenkontaktzeit [ms])	· Girard, Lattier, Micallef, & Millet (2006) · Voss, Witt, & Werthner (2007)
Neuromuskuläre Funktionsdiagnostik				
TMG	· Messung elektrisch stimulierter Muskelkontraktionseigenschaften	· Elektrischer Stimulator · Wegsensor · Computer mit entsprechender Software	· Kontraktionszeit [ms] · Muskelverformung [mm]	· García-Manso et al. (2012) · Rey, Lago-Peñas, & Lago-Ballesteros (2012)
Labordiagnostik				
CK	· Venöse oder kapillare Blutentnahme, Zentrifugation des entnommenen Blutes und Analyse der CK-Konzentration im Serum	· Blutröhrchen · Kanüle und Stauschlauch oder Lanzette und Kapillare · Zentrifuge	· Serumkonzentration von CK [U/l]	· Meeusen et al. (2013) · Meyer & Meister (2011)
Subjektive Empfindungsdiagnostik				
DOMS	· Quantifizierung des subjektiven Muskelschmerzempfindens mit visuell-analoger Skala	· Visuell-analoge Skala mit zehn cm langer Linie, deren Endpunkte mit „Keine Schmerzen“ und „Extrem Schmerzen“ beschriftet sind	· Subjektiv empfundener Muskelschmerz als Abstand von der vom Athleten gesetzten Markierung auf der Skala und dem linken Linienbeginn der Skala [mm]	· Cleather & Guthrie (2007)

CMJ = Countermovement Jump; MRJ = Multiple Rebound Jumps; TMG = Tensiomyographie; CK = Creatinkinase; DOMS = Delayed Onset Muscle Soreness

5.5 Evidenz der Wirksamkeit von aktiver Erholung

Im dritten Untersuchungsmodul wurde der Einfluss einer aktiven Erholung auf die durch einen viertägigen HIT-Mikrozyklus induzierten Ermüdungseffekte bei acht international gerankten Tennisspielern überprüft. Hierfür wurde das im zweiten Modul entwickelte Diagnostikinventar verwendet. Effekte von aktiver Erholung auf das Regenerationsverhalten wurden bislang zumeist nach einer einmaligen Belastung sowie im Rahmen einer einmaligen Applikation dieser Erholungsstrategie evaluiert. Demnach war der vorliegende Forschungsansatz einer der Ersten, der die Wirksamkeit von einer repetitiven Anwendung von aktiver Erholung während eines Trainingsblocks thematisierte. Der hochintensive Mikrozyklus führte innerhalb vier Tagen zu signifikanten Veränderungen bei den Ermüdungsmarkern. Die Leistungsfähigkeit war 24 Stunden nach der Trainingsphase reduziert sowie die CK-Aktivität im Serum und das Muskelschmerz- bzw. Ermüdungsempfinden erhöht. Vergleichbar zum zweiten Untersuchungsmodul resultierte das HIT-Programm auch im dritten Modul folglich in einem mittelfristigen Ermüdungszustand. Die aktive Erholung hatte dabei im Vergleich zur passiven Erholung keinerlei Einfluss auf das Belastungs-, Ermüdungs- und Erholungsverhalten der Athleten. Das Integrieren aktiver Erholungsstrategien im Rahmen von HIT ist demnach für den Regenerationsprozess weder förderlich noch schädlich.

Untersuchungen zur aktiven Erholung überprüften bisher vorrangig den Einfluss einer moderaten aeroben Aktivität großer Muskelgruppen auf die Eliminationsrate der durch intensive Belastungen verursachten akkumulierten Laktat- bzw. Milchsäurekonzentration (Baldari, Videira, Madeira, Sergio, & Guidetti, 2005; Del Coso, Hamouti, Aguado-Jimenez, & Mora-Rodriguez, 2010; Devlin et al., 2014; Kappenstein, Engel, Fernández-Fernández, & Ferrauti, 2015; Menzies et al., 2010; Monedero & Donne, 2000; Taoutaou et al., 1996). Hinsichtlich einer beschleunigten Laktatelimination bzw. pH-Wert-Einstellung durch dynamische Muskelaktivitäten herrscht Konsens. Auch die im dritten Modul durchgeführte aktive Erholung resultierte im Anschluss an jede HIT-Einheit im Vergleich zur passiven Erholung in einer signifikant schnelleren Regulation des Säure-Basen-Status. Ein Zusammenhang zwischen einer belastungsinduzierten Laktatanhäufung bzw. Azidose und muskulär oder zentralnervös bedingten Ermüdungsmechanismen konnte jedoch bis dato nicht eindeutig geklärt werden (Cairns, 2006; Westerblad, Allen, Lannergren, & Länergren, 2002). Auch hat Laktat, wie einstweilen geglaubt, nichts mit der Entstehung von Muskelkater zu tun (Böning & Beneke, 2008). Zudem liegt die Halbwertszeit des Blutlaktats bei passiver Erholung und in Abhängigkeit der Konzentrationsmenge zwischen 10 und 25 min (Rost, Appell, Graf, & Hartmann, 2001). Der Ruhewert wird in der Folge nach etwa 90 bis 100 min erreicht (Rost et al., 2001). Dies ist gewöhnlich kürzer als der zeitliche Abstand zwischen aufeinanderfolgenden Trainings- oder Wettkampfbelastungen in vielen Sportarten (Barnett, 2006). Insofern ist eine beschleunigte Laktatelimination für die meisten Disziplinen kein valider Indikator für die Qualität aktiver Erholungsmaßnahmen.

Aktuell wird auch über eine adaptationsmindernde Wirkung von aktiver Erholung infolge einer beschleunigten Laktatelimination diskutiert. So ist Laktat auf Zellebene ein wichtiges Signalmolekül (Godfrey, Whyte, Buckley, & Quinlivan, 2008). Dabei bewirkt eine Erhöhung von Laktat, wie sie insbesondere durch ein HIT induziert wird, die Bildung von Radikalen, welche ein transkriptionales Netzwerk aktivieren, das wiederum adaptive Zellreaktionen signalisiert (Brooks, Brooks & Brooks, 2008; Wahl, Zinner, Achtzehn, Bloch, & Mester, 2010). So werden beispielsweise angiogene Prozesse durch Laktat angeregt (Hunt, Aslam, Hussain, & Beckert, 2008). Ähnlich wie eine Hypoxie reguliert Laktat den HIF-1 (Hypoxie-induzierter Faktor) und erhöht die VEGF-Konzentration (vaskulärer endothelialer Wachstumsfaktor) in kultivierten Endothelzellen sowie die VEGF-Sekretion von Makrophagen (Constant et al., 2000; Fukumura et al., 2001; Goerges & Nugent, 2003; Wahl, Hägele, Zinner, Bloch, & Mester, 2010a). Eine erhöhte Laktatkonzentration ist somit ein für die Angiogenese wichtiger metabolischer Stimuli, der die Migration von Endothelzellen auslöst und die Freisetzung sogenannter Matrixmetalloproteinasen induziert, selbst unter pH-neutralen und ausreichend oxygenierten Bedingungen (Hunt et al., 2007; Wahl et al., 2010).

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-Mikrozyklus 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, Andersen, Spencer, & Hallén, 2015; Stacey, Gibala, Martin Ginis, & Timmons, 2010; Suzuki et al., 2004; Zainuddin, Sacco, Newton, & Nosaka, 2006). Folglich sollten im Rah-

men eines HIT alternative erholungsfördernde Maßnahmen zur Steigerung der Regenerationsgeschwindigkeit eingesetzt werden. Da jedoch in der vorliegenden Untersuchung auch keine negativen Effekte bzgl. mittelfristiger Ermüdungserscheinungen von aktiver Erholung im Vergleich zur passiven Erholung beobachtet wurden, können individuelle Präferenzen sowie Erfahrungen und Überzeugungen einen Einsatz aktiver Regenerationsstrategien im Rahmen von HIT rechtfertigen.

Bei der Entscheidung für oder gegen den Einsatz von aktiver Erholung sollte der Einfluss auf die Glykogenresyntheserate berücksichtigt werden. So berichteten mehrere Untersuchungen von einer gehemmten Wiedereinlagerung von Muskelglykogen durch aktive Regenerationsstrategien (Bonen, Ness, Belcastro, & Kirby, 1985; Choi, Cole, Goodpaster, Fink, & Costill, 1994; Fairchild et al., 2003; Fournier, Fairchild, Ferreira, & Bräu, 2004). Eine mögliche Erklärung hierfür ist die Oxidierung von Laktat im Verlauf anhaltender körperlicher Aktivität. Während passiver Erholung wird das Laktat hingegen nicht als Kraftstoff benötigt und kann in der Folge an der Glykogenresynthese mitwirken (Choi et al., 1994). Diese Hypothese wird durch Studien gestützt, die für eine Gluconeogenese aus Laktat notwendige Enzyme in der Muskulatur nachweisen konnten (Crabtree, Higgins, & Newsholme, 1972; McLane & Holloszy, 1979). Folglich ist Laktat möglicherweise wesentlich als Regulator an der Glykogenresynthese im Anschluss an intensive Belastungen beteiligt (Astrand, Hultman, Juhlin-Dannfelt, & Reynolds, 1986; Hermansen & Vaage, 1977). Ferner führt die anhaltende körperliche Arbeit im Rahmen aktiver Erholungsmaßnahmen zu einem fortwährenden Abbau eines Teils des übrig gebliebenen Muskelglykogens (Choi et al., 1994). Logischerweise ist der gemessene Muskelglykogengehalt im Anschluss an eine aktive Erholung im Vergleich zur passiven Erholung reduziert. Diesbezüglich besteht jedoch noch Klärungsbedarf, da ebenso eine Reihe von Studien keine Unterschiede in der Glykogenresyntheserate zwischen aktiver und passiver Erholung feststellen konnten (Bangsbo, Graham, Johansen, & Saltin, 1994; McAinch et al., 2004; Peters Futre, Noakes, Raine, & Terblanche, 1987).

In Einzelfallanalysen zeigte sich, dass einige Tennisspieler während des viertägigen Mikrozyklus von der aktiven Erholung profitierten. Beispielsweise reduzierte sich ihre Sprungleistung lediglich im Verlauf der durch passive Erholung komplettierten Trainingsphase. Die Ergebnisse des zweiten Untersuchungsmoduls fordern eine individuelle Überprüfung potentieller Ermüdungsmarker. Gleiches gilt womöglich für die Beurteilung der Wirksamkeit von Erholungsmaßnahmen. So verlangt eine valide Belastungs- und Erholungssteuerung im HIT den Einblick in die individuellen Anforderungen und Rahmenbedingungen der Sportart, der jeweiligen Teildisziplin oder Spielposition sowie der individuellen Belastungstoleranz und Beanspruchung des Athleten, seiner individuellen Response und seiner persönlichen Erfahrungen und Gewohnheiten hinsichtlich des Einsatzes von Regenerationsverfahren.

6 SCHLUSSFOLGERUNG UND PRAXISEMPFEHLUNGEN

Akute Belastungs- und mittelfristige Ermüdungsreaktionen variieren zwischen unterschiedlichen praxisüblichen HIT-Protokollen teils erheblich. HIT-Varianten mit längeren Intervallen führen zu den höchsten metabolischen und kardialen Belastungsreaktionen, während Wiederholungssprinttraining den höchsten Regenerationsbedarf verursacht. Insgesamt zeigte sich, dass praxisübliche HIT-Protokolle extrem belastend sind und bei einer Durchführung von mehreren Einheiten in kurzer Zeit in mittelfristig anhaltenden Ermüdungssymptomen resultieren. Für eine adäquate Belastungs- und Erholungssteuerung im HIT sollte jedoch berücksichtig werden, dass die akuten Belastungsreaktionen und mittelfristigen Ermüdungsmechanismen in Abhängigkeit des gewählten Protokolls variieren.

Wie bereits von Wahl et al. (2010a) erwähnt, macht der extreme Charakter von HIT den Einsatz für einige Adressatengruppen (z.B. Patienten oder Adipöse) unwahrscheinlich. Doch auch die in den letzten Jahren entstandene Euphorie hinsichtlich der Verwendung von HIT im Spitzensport muss basierend auf den vorliegenden Befunden gedämpft werden. Zwar wird angenommen, dass hochtrainierte Athleten bei höheren Intensitäten trainieren müssen als wenig Trainierte, um beispielsweise die maximale Sauerstoffaufnahme zu erhöhen (Wahl, Hägele, Zinner, Bloch, & Mester, 2010b). Hochintensive Trainingsbelastungen gehen jedoch mit erheblichem Regenerationsbedarf einher. Daher ist fraglich, zu welchem Zeitpunkt im Trainings- und Wettkampfprozess einzelne Einheiten oder ganze Blöcke hoher bis höchster Intensitäten adäquat eingebaut werden können.

Die Verwendung von HIT ist vor allem in den Sportspielen aufgrund der langen Wettkampfperiode bei vergleichsweise kurzer Übergangs- und Vorbereitungsperiode problematisch. Ein saisonbegleitender Einsatz von HIT ist hier schwierig, da der Zeitraum zwischen den Wettkampfbelastungen für eine ausreichende Regeneration nach HIT häufig nicht ausreicht. Insbesondere ein Wiederholungssprinttraining scheint daher während der Saison angesichts der beträchtlichen Muskelzellschädigungen sowie insbesondere aufgrund des erhöhten Verletzungsrisikos infolge der extremen muskelmechanischen Belastung nahezu ausgeschlossen. Doch auch im Verlauf einer Vorbereitungsperiode (z.B. während eines sogenannten intensiven Schockmikrozyklus im Rahmen einer Blockperiodisierung) sollte ein Wiederholungssprinttraining nur in begrenztem Maße eingesetzt werden (maximal ein- bis zweimal pro Woche). Für die Planung eines hochintensiven Schockmikrozyklus empfiehlt sich eine Variation aus mehreren HIT-Protokollen, um sowohl metabolische und kardiale als auch neuromuskuläre bzw. koordinative (z.B. Richtungswechsel) Belastungsstimuli hervorzurufen.

Praktikable und potentiell sensitive Parameter zur Diagnostik von Belastungs- bzw. Ermüdungseffekten im HIT sind die Bestimmung der Sprungleistung, Muskelkontraktilität und CK-Aktivität sowie des subjektiv empfundenen Muskelschmerzes. Ein Einsatz dieser Surrogatmarker in der (leistungs-)sportlichen Praxis erfordert jedoch aufgrund ihrer unzureichenden diagnostischen Effektivität die Überprüfung ihrer Eignung auf individueller Basis. Außerdem sollte eine Kombination aus mehreren Parametern verwendet werden, um möglichst alle Ermüdungsphänomene erfassen zu können.

Die Diagnostik der Sprungleistung bietet die Möglichkeit einer praktikablen Erfassung von neuromuskulären Funktionseinschränkungen. Hierfür empfehlen sich beispielsweise festinstallierte Kontaktmatten in den jeweiligen Sportstätten bzw. Leistungszentren. Kontaktmessplatten ermöglichen über die Registrierung von Flug- und/oder Bodenkontaktzeiten eine simple und engmaschige Bestimmung von Sprungdaten (z.B. Sprunghöhe beim Countermovement Jump oder Effizienzkoeffizient des Absprungs beim Multiple Rebound Jumps Test). So können mühelos individuelle Leistungsprofile erstellt werden. Zudem sind Messsysteme, die über Flug- und/oder Bodenkontaktzeiten die Sprungleistung erfassen, meist ohne großen Aufwand transportierbar, sodass die Ermittlung von Sprungdaten auch dezentral erfolgen kann.

Die fragebogen- oder skalenbasierte Evaluation des subjektiv empfundenen Beanspruchungs- bzw. Ermüdungsstatus ist ebenfalls sehr praktikabel. Vor allem dann, wenn die Daten webbasiert mittels Internetanwendungen oder plattformübergreifenden Applikationen erhoben und somit zeitnah weiterverarbeitet werden können. Beispielsweise kann die Erfragung des subjektiven Muskelschmerzempfindens am Smartphone der Tablet mittels digitalisierter visuell-analoge Skala erfolgen. Die erfassten Daten würden dann unter Einwilligung des Athleten automatisch über das Mobilfunknetz an den Trainer oder entsprechende Personen aus dem Betreuerteam übermittelt und über individuell kalibrierte und lernfähige Algorithmen in Kürze ausgewertet werden.

Zur Bestimmung der muskelkontraktilen Eigenschaften kann die Tensiomyographie verwendet werden. Die TMG wird bereits in unterschiedlichen leistungssportlichen Settings erfolgreich eingesetzt (u.a. FC Barcelona, Real Madrid C.F., UD Almeria, United States Olympic Committee, UK Sports Institute). Für die Anwendung der TMG ist allerdings gut geschultes Personal notwendig. Nur so können unter Einhaltung strenger Qualitätskriterien brauchbare Ergebnisse für die Messung des Regenerationsbedarfs gewonnen werden. Zudem ist die Durchführung TMG-basierter Muskelkontraktilitätstests sowie die Aggregierung der anfallenden Daten im Vergleich zur Sprungkraftdiagnostik oder zur subjektiven Empfindungsdiagnostik zeitaufwendiger. Dies geht unter anderem zu Lasten der Athletencompliance und Praktikabilität. In Abhängigkeit der organisatorischen und infrastrukturellen Bedingungen steht daher der Aufwand einer TMG-basierten Muskelfunktionsdiagnostik nicht immer in einem gesunden Verhältnis zum Nutzen.

Eine laufbasierte, niedrigintensive aerobe Aktivität erbrachte im vorliegenden Untersuchungskontext im Vergleich zur passiven Erholung keinen messbaren Vorteil. Da sich die aktive Erholung aber auch nicht negativ auf die Regenerationsprozesse nach HIT auswirkte, könnten individuelle Präferenzen sowie Erfahrungen und Überzeugungen den Einsatz einer solchen Regenerationsmaßnahme rechtfertigen. Diesbezüglich sollte auch ein potentieller Placeboeffekt bei der Anwendung aktiver Erholungsmaßnahmen im sportpraktischen Umfeld berücksichtigt werden. Durch eine entsprechend herbeigeführte Erwartungshaltung oder Konditionierung sind positive Veränderungen des subjektiven Befindens oder selbst der objektiv quantifizierbaren Ermüdungs- bzw. Erholungsprozesse nach einer aktiven Erholung im Einzelfall möglich.

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.

Eine aktive Erholung sollte zudem nicht unmittelbar im Anschluss an eine intensive Belastung absolviert werden, solange eine beschleunigte Laktatelimination nach dem Training oder Wettkampf von keiner sportpraktischen Bedeutung ist. So kann eine potentiell adaptationsmindernde Wirkung infolge einer beschleunigten Metabolisierung von Laktat vermieden werden. Bezüglich des Einflusses von Regenerationsinterventionen auf adaptive Vorgänge besteht jedoch noch Klärungsbedarf. Diesbezüglich folgen weitere Untersuchungen im Rahmen des Forschungsprojekts, in dessen Kontext die in dieser Arbeit dokumentierten Studien realisiert wurden.

7 ZUSAMMENFASSUNG

High-Intensity Ausdauertraining (HIT) ist eine in zahlreichen Sportarten etablierte und vielfach angewandte Trainingsmethode zur Verbesserung allgemeiner und sportartspezifischer Ausdauerleistungskomponenten. Die Leistungsentwicklung steht dabei in ständiger Wechselwirkung mit den durch vor allem hochintensive Belastungen ausgelösten Ermüdungs- und Erholungsprozessen. So resultiert HIT meist in einer reversiblen (im Sinne der Superkompensation beabsichtigten) Abnahme der Leistungsfähigkeit, die in Abhängigkeit von Belastungsdichte, Belastungsumfang und Belastungsintensität erst nach einigen Minuten, Stunden oder Tagen abklingt. Erfolgt keine adäquate Steuerung von Belastung und Erholung, nimmt das Risiko für chronische Überlastungsreaktionen zu. Dies betrifft insbesondere den Spitzensport, da Trainingsumfang, Trainingsintensität und Trainingsdichte sowie Wettkampfhäufigkeit und Leistungsdichte in vielen Disziplinen in den letzten Jahren deutlich angestiegen sind. Akute Belastungsreaktionen und mittelfristige Ermüdungsreaktionen im Rahmen unterschiedlicher HIT-Protokolle sowie Anwendungsmöglichkeiten praxisrelevanter Parameter zur Diagnostik von Ermüdung und Erholtheit und Wirkungsmechanismen verschiedener Erholungsmaßnahmen für spezifische Belastungssituationen sind bislang jedoch nicht lückenlos evaluiert worden. Daher war das Ziel dieser Dissertation, evidenzbasierte Praxisleitlinien für eine Belastungs- und Erholungssteuerung im HIT zu erarbeiten.

Das Arbeitsprogramm bestand aus drei Studien, die inhaltlich und chronologisch aufeinander aufbauten. Jede Untersuchung beinhaltete dabei einen in sich geschlossenen Forschungsansatz mit entsprechenden Zielstellungen. Die methodische Konzeption der Studien zwei und drei erfolgte dabei unter Berücksichtigung der Befunde aus den jeweils vorausgegangenen Untersuchungen.

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_{IFT}). 16 Athleten aus den Sportspielen (Mittelwert ± SD; Alter, 24,6 ± 2,7 Jahre; $\dot{V}O_2max$, 58,3 ± 5,9 ml/min/kg) absolvierten im Anschluss an eine Voruntersuchung zur Ermittlung von V_{IFT} innerhalb von fünf Wochen jeweils einmal wöchentlich in randomisierter Reihenfolge eines von fünf HIT-Programmen (P_{240}: 4 × 4 min bei 80% von V_{IFT}; P_{120}: 7 × 2 min bei 85% von V_{IFT}; P_{30}: 2 × 10 × 30 bei 90% von V_{IFT}; P_{15}: 3 × 9 × 15 s bei 95% von V_{IFT}; P_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 Gesamttrainingseinheit, 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 perzeptive 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-Absenkung verursachte. Mittelfristige Ermüdungseffekte ergaben sich insbesondere im Anschluss an das Wiederholungssprinttraining, mit signifikant höherem CK-Wert im Blut, Muskelschmerzempfinden 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-reaktives 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 V_{IFT}, 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, Muskelschmerzempfinden sowie subjektiv wahrgenommener Erholung- 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.

Akute Belastungs- und mittelfristige Ermüdungsreaktionen variieren zwischen unterschiedlichen praxisüblichen HIT-Protokolle teils erheblich. HIT-Varianten mit längeren Intervallen resultieren in den höchsten metabolischen und kardialen Belastungsreaktionen, während Wiederholungssprinttraining den höchsten Regenerationsbedarf verursacht. Praktikable und potentiell sensitive Parameter zur Diagnostik von Belastungs- bzw. Ermüdungseffekten im HIT sind die Bestimmung der Sprungleistung (CMJ), Sprungeffizienz (MRJ), Muskelkontraktilität und CK-Aktivität sowie des subjektiv empfundenen Muskelschmerzes. Ein Einsatz dieser Surrogatmarker in der (leistungs-) sportlichen Praxis erfordert jedoch aufgrund ihrer unzureichenden diagnostischen Effektivität die Überprüfung ihrer Eignung auf individueller Basis. AE war nicht in der Lage, die Regenerationsprozesse nach HIT zu beschleunigen. Gegenüber der PE besaß die AE keine Vorteile aber auch keine Nachteile. Da sich die AE im Vergleich zur PE also auch nicht negativ auf die Erholung ausgewirkt hat, könnten individuelle Präferenzen sowie Erfahrungen und Überzeugungen einen Einsatz von AE rechtfertigen.

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 (V_{IFT}). For this purpose, 16 well-trained intermittent sport players (mean ± SD; age, 24.6 ± 2.7 years; $\dot{V}O_2max$, 58.3 ± 5.9 ml·min·kg^{-1}) participated in five different running-based HIT-programs separated by six days in between (P_{240}: 4 × 4 min at 80% V_{IFT}; P_{120}: 7 × 2 min at 85% V_{IFT}; P_{30}: 2 × 10 × 30 s at 90% V_{IFT}; P_{15}: 3 × 9 × 15 s at 95%V_{IFT}; P_5: 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 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 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; $\dot{V}O_2max$, 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 mal 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.

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ABBILDUNGSVERZEICHNIS

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Publikation 1

Publikation 2

Publikation 3

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Theoretischer Hintergrund

Publikation 1

Publikation 2

Publikation 3

Gesamtdiskussion mit praktischen Empfehlungen

ABKÜRZUNGSVERZEICHNIS

$30\text{-}15_{IFT}$	=	30-15 Intermittent Fitness Test
ACT/AE	=	Active Recovery / Aktive Erholung
ANOVA	=	Varianzanalyse
AEB	=	Akutmaß zur Erfassung von Erholung und Beanspruchung
AVR	=	Anaerobic Velocity Reserve
BCAAs	=	Branched-Chain Amino Acids / verzweigtkettige Aminosäuren
BF	=	Biceps Femoris
Ca^{2+}-Ionen	=	Calcium-Ionen
CI	=	Confidence Interval / Konfidenzintervall
CK	=	Creatinkinase
CMJ	=	Countermovement Jump
COD	=	Change of Direction / Richtungswechsel
CRP	=	C-reaktives Protein
CV	=	Coefficient of Variation / Variationskoeffizient
Dm	=	Maximal Radial Muscle Belly Displacement / Maximale Muskelverformung
DOMS	=	Delayed Onset Muscle Soreness
EB	=	Emotional Balance / Emotionale Ausgeglichenheit
EBF-Sport	=	Erholungs-Belastungs-Fragebogen für Sportler
ES	=	Effect Size
ECG	=	Electrocardiography / Elektrokardiogramm
FSS	=	Freie Fettsäuren
FT-Fasern	=	Fast-Twitch-Fasern
HI(I)T	=	High-Intensity Interval Training
Hf/HR	=	Herzfrequenz
Hfmax/HRmax	=	Maximale Herzfrequenz
HRV	=	Herzfrequenzvariabilität
ICC	=	Intraclass Correlation Coefficient / Intra-Klassen-Korrelationskoeffizient
IGF	=	Insulin-like Growth Factor
IL	=	Interleukin
KEB/SRSS	=	Kurzskala zur Erfassung von Erholung und Beanspruchung
KWI	=	Kaltwasserimmersion
La	=	Blutlaktat
LA	=	Lack of Activation / Aktivierungsmangel
LPT_1	=	First Lactate Turn Point / Aerobe Laktatschwelle
LPT_2	=	Second Lactate Turn Point / Anaerobe Laktatschwelle

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 Sucessive 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@$\dot{V}O_2$max = An der maximalen Sauerstoffaufnahme verbrachte Belastungszeit
Tc = Contraction Time / Kontraktionszeit
TE = Typical Error / Standardfehler
TMG = Tensiomyographie
TNF α = 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
$\dot{V}O_2$ = Sauerstoffaufnahme
$\dot{V}O_2$max = Maximale Sauerstoffaufnahme

$v\dot{V}O_2max$	=	Laufgeschwindigkeit bei Erreichen der maximalen Sauerstoffaufnahme
W/R	=	Work/Rest Ratios / Belastungs-Erholungs-Verhältnis

ANHANG

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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; $\dot{V}O_{2max}$, 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

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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),

Table 1. Baseline physical characteristics of the athletes.

	Age (yrs)	Height (cm)	Body mass (kg)	Body fat (%)	$\dot{V}O_{2max}$ (mL·min·kg^{-1})
Overall (n = 22)	23.0±2.7	176.6±7.6	69.5±7.3	17.9±5.8	57.6±8.6
Male (n = 11)	22.9±1.9	181.6±5.3	73.8±6.4	14.6±3.7	62.9±8.3
Female (n = 11)	23.0±3.4	171.6±6.0	65.2±5.5	21.1±5.9	52.2±4.8

Parameters are shown as mean ± SD.

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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 (30-15$_{IFT}$) 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 $\dot{V}O_{2max}$. After familiarization with performance tests to minimize any learning effect, participants completed the 30-15$_{IFT}$ 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 (post$_1$), and following a 72 h recovery period (post$_2$), in which no training was allowed (Fig 1).

On all testing days (pre, post$_1$, post$_2$), 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, $post_1$, and $post_2$. 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 $\dot{V}O_{2max}$, 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 $km \cdot h^{-1}$, increasing 2 $km \cdot h^{-1}$ every 3 min with a constant incline of 0.5% until voluntary exhaustion. $\dot{V}O_2$ 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 $\dot{V}O_{2max}$.

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 $km \cdot h^{-1}$ for the first 30 s run and was increased by 0.5 $km \cdot h^{-1}$ 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_{IFT}) 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 non-motorized treadmill (NMT) (Force 3.0, Woodway GmbH, Weil am Rhein, Germany) pre,

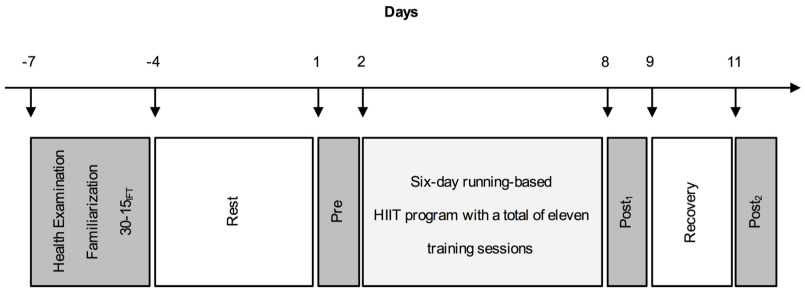

Fig 1. Study design. $30\text{-}15_{IFT}$ = 30–15 Intermittent Fitness Test, HIIT = high-intensity interval training.

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$post_1$, and $post_2$. 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 ($m \cdot s^{-1}$), n = 17, ICC = 0.92, TE = 0.10, CV = 1.5%).

Jump and linear sprint tests. On each testing day (pre, $post_1$, and $post_2$), countermovement jumps (CMJ) and multiple rebound jump tests (MRJ) were performed on a contact platform (Haynl-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, 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, $post_1$, and $post_2$. 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, 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, $post_1$, and $post_2$; 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-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, $post_1$, and $post_2$), 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% HR_{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 2. Six-day high-intensity interval training program.

	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6
	Straight-line runs	Straight-line runs	Straight-line runs		Straight-line runs	Straight-line runs
	4 x 4 min	7 x 2 min	4 x 4 min		4 x 4 min	7 x 2 min
a.m.	(r = 3 min)	(r = 2 min)	(r = 3 min)	Rest	(r = 3 min)	(r = 2 min)
	80% V_{IFT}	85% V_{IFT}	80% V_{IFT}		80% V_{IFT}	85% V_{IFT}
	TL: 231 ± 59	TL: 236 ± 48	TL: 210 ± 63		TL: 217 ± 71	TL: 256 ± 56
	Straight-line sprints	40m-shuttle runs	Straight-line sprints	40m-shuttle runs	Straight-line sprints	40m-shuttle runs
	4 x 6 x 5 s	2 x 12 x 30 s	4 x 6 x 5 s	2 x 12 x 30 s	4 x 6 x 5 s	2 x 12 x 30 s
p.m.	(r = 25 s; R = 5 min)	(r = 30 s; R = 3 min)	(r = 25 s; R = 5 min)	(r = 30 s; R = 3 min)	(r = 25 s; R = 5 min)	(r = 30 s; R = 3 min)
	all out	90% V_{IFT}	all out	90% V_{IFT}	all out	90% V_{IFT}
	TL: 207 ± 64	TL: 270 ± 67	TL: 225 ± 67	TL: 257 ± 59	TL: 232 ± 68	TL: 290 ± 56

V_{IFT}: 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_{IFT}, r = 30 s, R = 3 min] means that the subject had to run two series of 12 intervals at 90% V_{IFT} 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.

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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, $post_1$, and $post_2$) 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 $m \cdot s^{-1}$) than at baseline (pre: 5.02 ± 0.52 $m \cdot s^{-1}$) or after recovery ($post_2$: 4.97 ± 0.56 $m \cdot s^{-1}$). The respective changes were -0.18 ± 0.13 $m \cdot s^{-1}$ ($p = 0.001$; effect size = -1.51) from pre to $post_1$ and 0.12 ± 0.26 $m \cdot s^{-1}$ ($p = 0.003$; 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 $post_1$ compared to baseline values. However, these increases were not reversible between $post_1$ and $post_2$ (Table 3). Dm of the RF and BF, as well as CRP, and urea were not different at $post_1$ and $post_2$ 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.

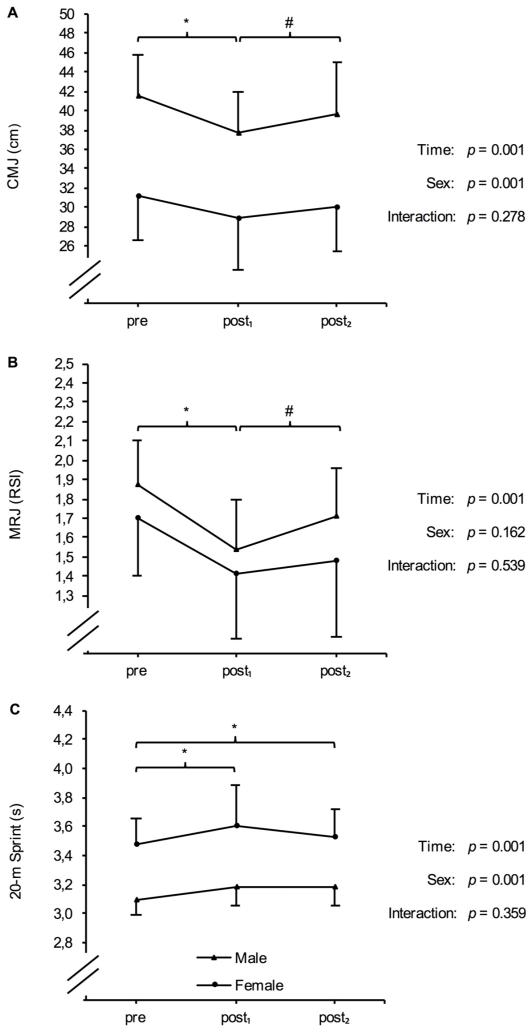

Fig 2. Mean (± SD) countermovement jump (CMJ) height (A), multiple rebound jumps (MRJ) performance (B) and 20-m sprint time (C) in males and females at baseline (pre), after a six-day high-intensity interval training program ($post_1$), and following 72 h of recovery ($post_2$). RSI = reactive strength index. *Significant difference compared to pre ($p < 0.05$). #Significant difference compared to $post_1$ ($p < 0.05$).

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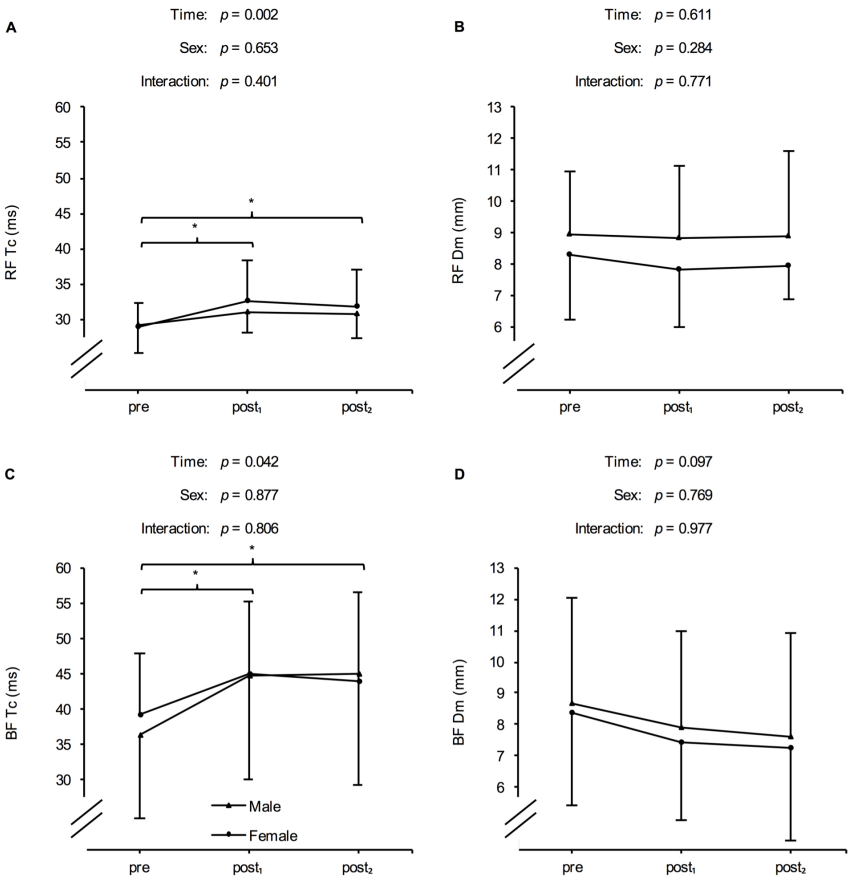

Fig 3. Mean (± SD) of the contraction time (Tc) and maximal radial muscle displacement (Dm) of the rectus femoris (RF) and biceps femoris (BF) in males and females at baseline (pre), after a six-day high-intensity interval training program ($post_1$), and following 72 h of recovery ($post_2$).
*Significant difference compared to pre ($p < 0.05$). #Significant difference compared to $post_1$ ($p < 0.05$).

doi:10.1371/journal.pone.0139801.g003

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

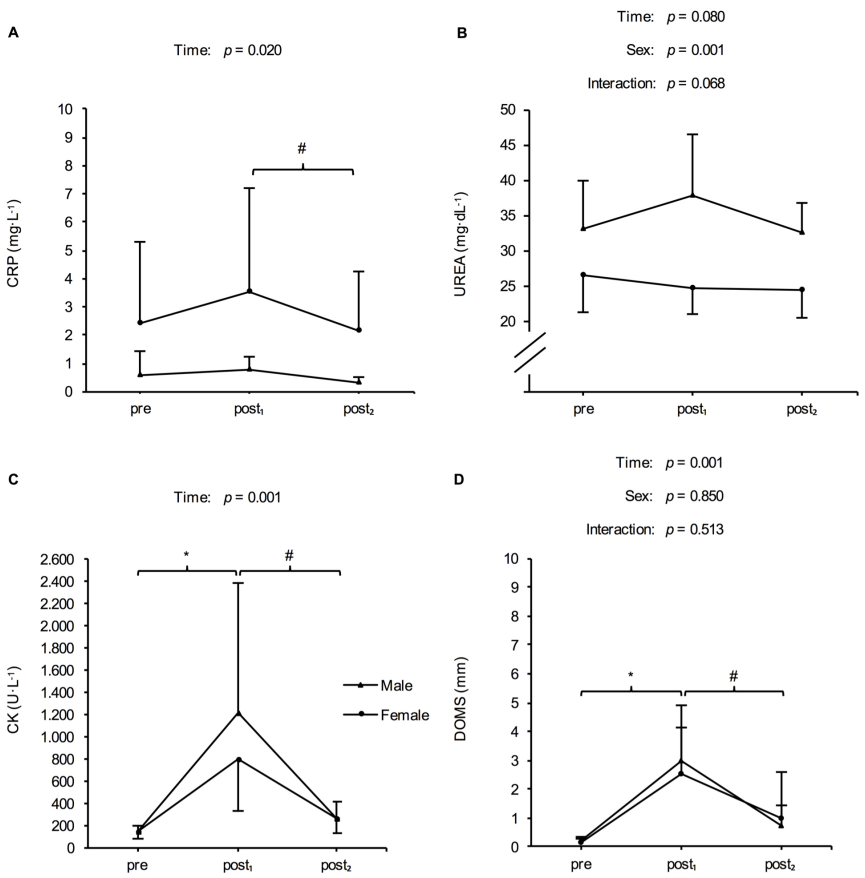

Fig 4. Mean (± SD) of the serum concentration of c-reactive protein (CRP), urea, and creatinkinase (CK) as well as of the rating of delayed onset muscle soreness (DOMS) in males and females at baseline (pre), after a six-day high-intensity interval training program ($post_1$), and following 72 h of recovery ($post_2$). *Significant difference compared to pre ($p < 0.05$). #Significant difference compared to $post_1$ ($p < 0.05$).

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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

Table 3. Markers of fatigue and recovery at baseline (pre), after a six-day high-intensity interval training program ($post_1$), and following 72 h of recovery ($post_2$) as well as percentage changes of performance and muscle contractile markers between testing days.

	pre		$post_1$	$post_2$	Time	pre-$post_1$		$post_1$-$post_2$		pre-$post_2$	
					p	%Δ ± CI	*d*	%Δ ± CI	*d*	%Δ ± CI	*d*
Performance markers											
CMJ (cm)	36.4±6.8	(33.4–39.4)	33.3±6.6*	(30.4–36.2)	34.8±6.9#	(31.8–37.9)	< 0.001	-8.4±2.9	-1.35	4.1±2.9	-0.68	-4.3±2.2	-0.79
MRJ (RSI)	1.79±0.28	(1.66–1.91)	1.48±0.30*	(1.34–1.61)	1.60±0.35#	(1.44–1.75)	< 0.001	-17.4±4.5	-1.60	6.5±4.5	-0.63	-10.9±5.3	-0.91
20-m Sprint (s)	3.28±0.24	(3.18–3.39)	3.40±0.30*	(3.26–3.53)	3.35±0.24*	(3.25–3.46)	< 0.001	3.4±1.8	-0.81	-1.2±1.6	-0.37	2.2±3.2	-0.65
Muscle contractile markers											
RF Tc (ms)	29.0±3.8	(27.3–30.7)	31.7±4.8*	(29.6–33.8)	31.2±4.6*	(29.2–33.2)	< 0.002	9.9±5.9	-0.72	-1.7±4.1	-0.17	8.2±6.0	-0.58
F Dm (mm)	8.6±2.1	(7.7–9.6)	8.3±2.2	(7.3–9.3)	8.4±2.1	(7.5–9.4)	< 0.611	-1.7±10.3	-0.17	2.1±6.2	-0.10	0.4±10.4	-0.11
BF Tc (ms)	37.7±10.7	(33.0–42.5)	44.9±12.9*	(39.2–50.6)	44.5±14.6*	(38.0–51.0)	< 0.042	28.7±24.9	-0.46	-8.1±21.5	-0.03	20.5±18.9	-0.59
BF Dm (mm)	8.5±3.3	(7.1–10.0)	7.7±2.9	(6.4–8.9)	7.4±3.2	(6.0–8.9)	< 0.097	-6.8±12.2	-0.39	-3.3±14.1	-0.10	-10.1±15.0	-0.46
Biochemical markers											
CK ($U{\cdot}L^{-1}$)	147±51	(125–170)	1010±887*	(617–1403)	269±134*#	(210–328)	< 0.001		-0.99		-0.95		-0.87
CRP ($mg{\cdot}L^{-1}$)	1.52±2.46	(0.33–2.70)	2.23±3.08	(0.74–3.71)	1.27±1.84#	(0.38–2.16)	< 0.020		-0.33		-0.66		-0.20
UREA ($mg{\cdot}dL^{-1}$)	29.6±7.2	(26.1–33.0)	30.9±9.4	(26.4–35.5)	28.2±5.9	(25.4–31.1)	< 0.080		-0.25		-0.46		-0.26
Subjective markers											
DOMS (mm)	0.2±0.1	(0.1–0.3)	2.7±1.8*	(2.0–3.5)	0.9±1.2#	(0.3–1.4)	< 0.001		-1.50		-1.44		-0.57

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

CI: 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 creatinkinase; CRP: C-reactive protein; DOMS: delayed onset muscle soreness.

*Significant difference compared to pre.

#Significant difference compared to $post_1$.

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

	Diagnostic effectiveness (%)		Misclassification rate (%)		Youden`s Index	
	Δpre-post$_1$	Δpost$_1$-post$_2$	Δpre-post$_1$	Δpost$_1$-post$_2$	Δpre-post$_1$	Δpost$_1$-post$_2$
Performance markers						
CMJ (cm)	63.6	60.0	36.4	40.0	0.01	0.20
MRJ (RSI)	68.2	60.0	31.8	40.0	0.08	0.20
20-m Sprint (s)	77.3	33.3	22.7	66.7	0.51	0.34
Muscle contractile markers						
RF Tc (ms)	68.2	40.0	31.8	60.0	0.38	0.21
RF Dm (mm)	50.0	66.7	50.0	33.3	0.04	0.30
BF Tc (ms)	54.5	40.0	45.5	60.0	0.03	0.23
BF Dm (mm)	50.0	60.0	50.0	40.0	0.11	0.20
Biochemical markers						
CK ($U{\cdot}L^{-1}$)	50.0	53.3	50.0	46.7	0.11	0.13
CRP ($mg{\cdot}L^{-1}$)	31.8	46.7	68.2	53.3	0.07	0.08
UREA ($mg{\cdot}dL^{-1}$)	30.0	46.7	70.0	53.3	0.10	0.00
Subjective markers						
DOMS (mm)	45.5	60.0	54.5	40.0	0.05	0.14

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.

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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 $> 1000\ U{\cdot}L^{-1}$ 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 $post_1$ 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 $post_1$ 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 $post_1$ and $post_2$ 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 $post_1$ and $post_2$. 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 $\dot{V}O_{2max}$ 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., 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 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.

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