Institut für Sportwissenschaft, Universität Rostock

Adaptabilität des motorischen Systems – Akute und chronische Anpassungen der neuromuskulären Funktion

Kumulative Dissertation zur Erlangung des akademischen Grades Doctor rerum humanarum (Dr. rer. hum.) der Medizinischen Fakultät der Universität Rostock

vorgelegt von
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Datum der Einreichung: 30.10.2013

Datum der Verteidigung: 27.05.2014

urn:nbn:de:gbv:28-diss2014-0162-6

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“[…] to move things is all that mankind can do, and that for such the sole executant is muscle, whether in whispering a syllable or in felling a forest.”

- Sir Charles Sherrington -

ABSTRACT

Körperliche Aktivität steigert die Lebensqualität, sie wirkt im Hinblick auf diverse Leiden präventiv und fördert bei vielen Pathologien Therapie als auch Rehabilitation. Bedingt durch die hohe Adaptabilität des motorischen Systems provoziert physische Aktivität spezifische Anpassungen, die in Abhängigkeit von der Art, Intensität und Dauer der jeweiligen Bewegungsintervention differieren. Zudem finden die Adaptationen auf verschiedenen Ebenen innerhalb des neuromuskulären Systems statt.

Generell können die Anpassungen im motorischen System in zwei Subkategorien differenziert werden. Dazu gehören (I) die akuten Anpassungen, die die Auswirkungen von einmaligen Bewegungsinterventionen auf das neuromuskuläre System umfassen. Des Weiteren können sich (II) chronische Anpassungen im neuromuskulären System einstellen, die z. B. durch die Applikation repetitiver Bewegungsreize über einen längeren Zeitraum oder das Ausbleiben von physischer Aktivität induziert werden.

Die vorliegende kumulative Dissertationsschrift befasst sich mit Teilaspekten der Adaptabilität des motorischen Systems. Die zugrundeliegende Leitfrage war dabei, welche Modulationen spezifische Bewegungsinterventionen und Alterungsprozesse im motorischen System provozieren. Innerhalb von vier Studien wurden akute und chronische Anpassungen der neuromuskulären Funktion analysiert. Im Hinblick auf die akuten Anpassungen wurden die Effekte von Ermüdung sowie kontraktionsinduzierten Muskelverletzungen und -schmerz auf die neuromuskuläre Funktion untersucht. Bei den chronischen Adaptationen stand der Einfluss von Training und Alterungsprozessen auf die neuromuskuläre Funktion im Fokus.

Die Experimente wurden an den Oberschenkelmuskeln durchgeführt. Aufgrund ihrer Bedeutung für die Kniestabilität sowie für alltägliche und sportliche Aktivitäten, wie z. B. das Gehen, Laufen und Springen, ist es von hoher Relevanz akute und chronische Adaptationen der neuromuskulären Funktion der Oberschenkelmuskeln zu untersuchen.

INHALTSVERZEICHNIS

ABKÜRZUNGSVERZEICHNIS

°	Grad
%	Prozent
°/s	Winkelgeschwindigkeit
α	alpha
β	beta
γ	gamma
ACL	anterior cruciate ligament
BF	M. biceps femoris
Ca^{2+}	Calcium-Ionen
d	Tag
DVZ	Dehnungs-Verkürzungs-Zyklus
EMD	electromechanical delay
EMG	Elektromyographie
h	Stunde
H_{max}	maximaler H-Reflex
H_{max}/M_{max}-Ratio	Quotient aus maximalem H-Reflex und maximaler M-Welle
H/Q-Ratio	Hamstring/Quadriceps-Ratio
H-Reflex	Hoffmann-Reflex
IGF-1	insulin-like growth factor 1
IL-6	Interleukin-6
iMVT	isometrisches maximales willkürliches Drehmoment
M.	Musculus
mm	Millimeter
M_{max}	maximale M-Welle
MRTD	maximal rate of torque development der willkürlich produzierten Drehmoment-Zeit-Kurve
$MRTD_{TT}$	maximal rate of torque development produziert durch den M. quadriceps femoris in Ruhebedingung
$MRTR_{TT}$	maximal rate of torque relaxation produziert durch den M. quadriceps femoris in Ruhebedingung
ms	Millisekunde
mV	Millivolt
MVC	maximum voluntary contraction
N.	Nervus
Nm	Newtonmeter
Nm/s	Newtonmeter pro Sekunde
PT	peak torque
RMS	root mean square
RMS-EMG	root mean square des EMG-Signals

$RMS\text{-}EMG_{iMVT}/M_{max}$	root mean square des EMG-Signals normalisiert zur maximalen M-Welle während des Drehmomentmaximums
$RMS\text{-}EMG/M_{max}$	root mean square des EMG-Signals normalisiert zur maximalen M-Welle
$RMS\text{-}EMG_{RTD}/M_{max}$	root mean square des EMG-Signals normalisiert zur maximalen M-Welle während des Drehmomentanstieges
RTD	rate of torque development
RTR	rate of torque relaxation
s	Sekunde
ST	M. semitendinosus/semimembranosus
T_b	Drehmomentniveau vor der Applikation der elektrischen Stimuli
TCT	twitch contraction time
THRT	twitch half relaxation time
TNF-α	tumor necrosis factor-α
TNF-β	tumor necrosis factor-β
V	Volt
VA	willkürliche oder volitive Aktivierung
VM	M. vastus medialis

ABBILDUNGSVERZEICHNIS

1. EINLEITUNG

Körperliche Aktivität steigert die Lebensqualität, sie wirkt im Hinblick auf diverse Leiden präventiv und fördert bei vielen Pathologien Therapie als auch Rehabilitation [182]. Bedingt durch die hohe Adaptabilität des motorischen Systems provoziert physische Aktivität spezifische Anpassungen, die in Abhängigkeit von der Art, Intensität und Dauer der jeweiligen Bewegungsintervention differieren. Zudem finden die Adaptationen auf verschiedenen Ebenen innerhalb des neuromuskulären Systems statt [66].

Generell können die Anpassungen im motorischen System in zwei Subkategorien differenziert werden. Dazu gehören (I) die akuten Anpassungen, die die Auswirkungen von einmaligen Bewegungsinterventionen auf das neuromuskuläre System umfassen. Zu dieser Subkategorie zählen u. a. die Effekte von Erwärmung, Dehnung, Ermüdung, kontraktionsinduzierten Muskelverletzungen und -schmerz sowie Potenzierungsmechanismen auf die neuromuskuläre Funktion. Des Weiteren können sich (II) chronische Anpassungen im neuromuskulären System einstellen, die z. B. durch die Applikation repetitiver Bewegungsreize über einen längeren Zeitraum oder das Ausbleiben von physischer Aktivität induziert werden. Diese beinhalten u. a. den Einfluss von Training, Regeneration nach Verletzungen, Inaktivität und Alterungsprozessen auf die neuromuskuläre Funktion [66].

Die vorliegende kumulative Dissertationsschrift befasst sich mit Teilaspekten der Adaptabilität des motorischen Systems. Die zugrundeliegende Leitfrage ist dabei, welche Modulationen spezifische Bewegungsinterventionen und Alterungsprozesse im motorischen System provozieren. Innerhalb von vier Studien wurden akute und chronische Anpassungen der neuromuskulären Funktion analysiert. Dabei standen die Anpassungen der neuromuskulären Funktion der Oberschenkelmuskeln in Abhängigkeit von der jeweiligen Fragestellung im Fokus. Aufgrund ihrer Bedeutung für die Kniestabilität [13, 130, 164] sowie für alltägliche und sportliche Aktivitäten, wie z. B. das Gehen, Laufen und Springen [126, 156, 237], ist es von hoher Relevanz akute und chronische Adaptationen der neuromuskulären Funktion der Oberschenkelmuskeln zu untersuchen.

Folgende eigene Publikationen beschäftigen sich mit akuten Anpassungen der neuromuskulären Funktion infolge physischer Aktivität.

EXPERIMENT I: Behrens, M., Mau-Moeller, A., Wassermann, F., Bruhn, S. (2013). Effect of fatigue on hamstring reflex responses and posterior-anterior tibial translation in men and women. *Plos One, 8* (2), e56988. [IF_{2011}: 4.092]. *Finanzierung: Gesellschaft für Orthopädisch-Traumatologische Sportmedizin (GOTS) / Bundesinstitut für Sportwissenschaft (BISp)*

EXPERIMENT II: Behrens, M., Mau-Moeller, A., Bruhn, S. (2012). Effect of exercise-induced muscle damage on neuromuscular function of the quadriceps muscle. *International Journal of Sports Medicine, 33* (8), 600-606. [IF_{2011}: 2.433] . *Finanzierung: Promotionsstipendium nach dem LGFG M-V*

In den Experimenten I und II wurden akute Anpassungen der neuromuskulären Funktion infolge von Ermüdungsinterventionen analysiert. Experiment I beschäftigte sich mit den Auswirkungen von Ermüdung auf die Kniestabilität und Reflexantworten der ischiocruralen Muskeln bei Männern und Frauen, während Experiment II die Effekte von kontraktionsinduzierten Muskelverletzungen auf die neuromuskuläre Funktion des M. quadriceps femoris eruierte. In beiden Experimenten wurden Ermüdungsinterventionen durchgeführt, wobei sich diese hinsichtlich der Belastungsgestaltung unterschieden und somit differente Beanspruchungsreaktionen hervorriefen. In Abbildung 1 ist die hypothetische Modulation der Leistungsfähigkeit des neuromuskulären Systems durch unterschiedliche Belastungsformen modellhaft dargestellt. Experiment I bezog sich auf die Leistungsfähigkeit infolge einer normalen Belastung, während Experiment II die Auswirkungen einer Überbelastung auf die Leistungsfähigkeit analysierte. Beide Belastungsformen führen zum Abfall der Leistungsfähigkeit, deren Ausmaß jedoch bei Überbelastung größer ist und eine längere Regenerationszeit benötigt.

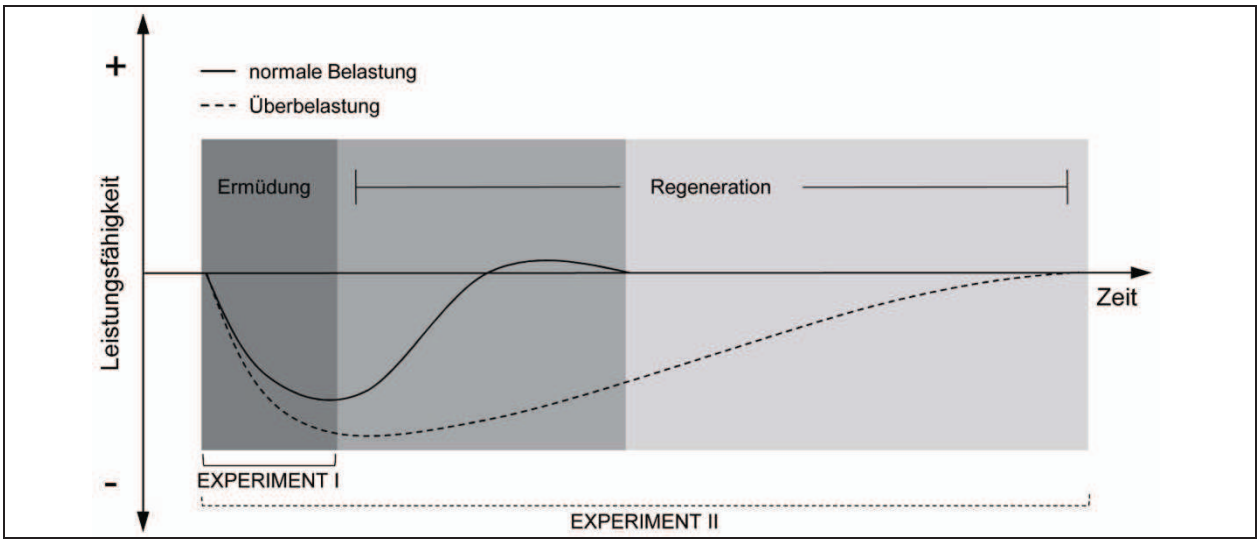

Abb. 1 Hypothetische Modulation der Leistungsfähigkeit des neuromuskulären Systems durch unterschiedliche Belastungsformen.

Chronische Anpassungen der neuromuskulären Funktion durch Training und im Verlauf des Alterungsprozesses werden in folgenden eigenen Publikationen beschrieben.

EXPERIMENT III: Behrens, M., Mau-Moeller, A., Bruhn, S. (2013). Effect of plyometric training on neural and mechanical properties of the knee extensor muscles. *International Journal of Sports Medicine* (in press). [IF_{2011}: 2.433]. *Finanzierung: Promotionsstipendium nach dem LGFG M-V*

EXPERIMENT IV: Mau-Moeller, A.*, **Behrens, M.***, Lindner, T., Bader, R., Bruhn, S. (2013). Age-related changes in neuromuscular function of the quadriceps muscle in physically active adults. *Journal of Electromyography and Kinesiology, 23* (3), 640-648. (* authors contributed equally to this work) [IF_{2011}: 1.969]. *Finanzierung: Promotionsstipendium nach dem LGFG M-V*

In den Experimenten III und IV wurden chronische Anpassungen der neuromuskulären Funktion des M. quadriceps femoris infolge regelmäßiger physischer Aktivität, d. h. Training, und durch den Alterungsprozess analysiert. Abbildung 2 zeigt modellhaft die hypothetische Modulation der Leistungsfähigkeit des neuromuskulären Systems durch Training und im Verlauf des Alterns. Während Training zu einer Steigerung der Leistungsfähigkeit führt, bewirken Alterungsprozesse und das Ausbleiben adäquater Bewegungsreize einen Abfall der Leistungsfähigkeit des neuromuskulären Systems.

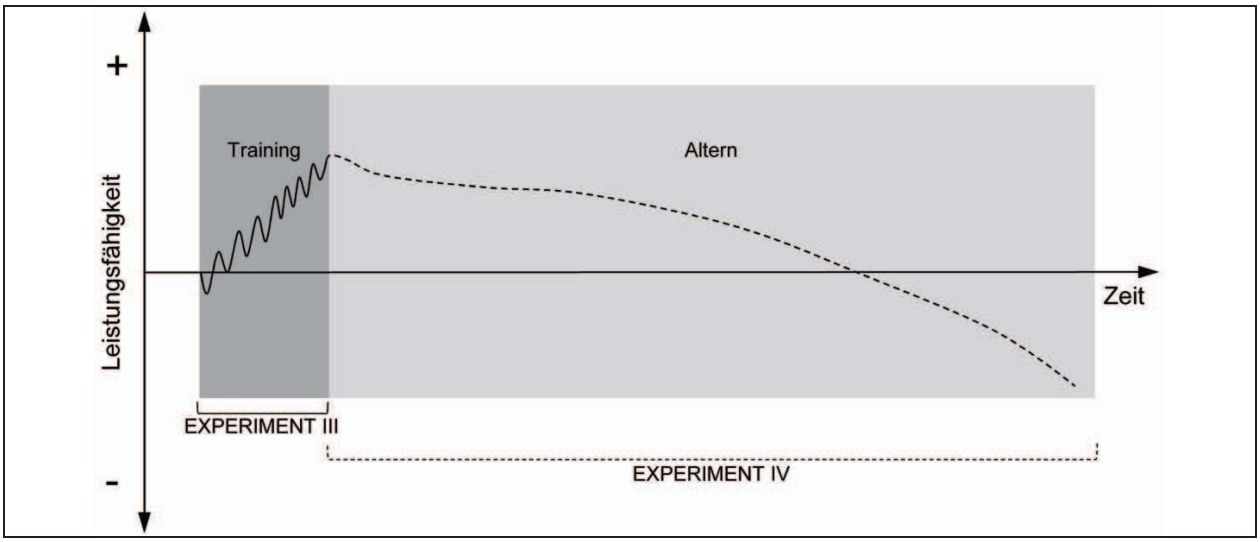

Abb. 2 Hypothetische Modulation der Leistungsfähigkeit des neuromuskulären Systems durch Training und im Verlauf des Alterns.

2. AKUTE ANPASSUNGEN DER NEUROMUSKULÄREN FUNKTION

2.1 EINFLUSS VON AKTIVITÄTSINDUZIERTER ERMÜDUNG AUF DIE NEURO-MUSKULÄRE FUNKTION

2.1.1 Darstellung des Forschungsdefizits

Anhaltende physische Aktivität führt zu Ermüdung, die als Reduktion der Fähigkeit des Muskels Kraft bzw. Leistung zu generieren, unabhängig davon, ob die Aktivität aufrecht erhalten werden kann oder nicht, definiert wird [24, 75, 144, 159]. Diese Definition impliziert, dass Ermüdung schon relativ zeitnah nach dem Beginn einer anhaltenden Aktivität einsetzt, obwohl das Individuum die Tätigkeit weiter ausführen kann. Die Ermüdung kann sich in unterschiedlichen Subsystemen manifestieren und involviert in Abhängigkeit von der Bewegungsaufgabe metabolische, kardiovaskuläre, biomechanische, psychologische und neuromuskuläre Faktoren [7, 10, 62, 82, 206].

Die neuromuskulären Faktoren der Ermüdung sind auf unterschiedlichen Ebenen zu lokalisieren. Zur besseren Übersicht können die Faktoren in zwei zentrale Subkategorien differenziert werden. Dazu zählen erstens die zentralen Elemente der Ermüdung, welche die Veränderung der Aktivität des cerebralen Cortex, der Basalganglien und des Cerebellums inkludieren. Darüber hinaus wird die Modulation der Impulse ausgehend vom motorischen Cortex, der Leitung dieser Impulse mittels deszendierender motorischer Bahnen und der Erregbarkeit der α-Motoneuronen dieser Ebene zugeschrieben. Zur zweiten Subkategorie zählen die peripheren Elemente, zu denen die Reizleitung in Richtung der Axonendigungen, die neuromuskuläre Übertragung, die Aktionspotentialausbreitung entlang des Plasmalemms der Muskelfaser, die Reizfortpflanzung in die transversalen Tubuli, die Ca^{2+}-Freisetzung aus dem sarkoplasmatischen Retikulum und die Bildung der Querbrücken gehören [141].

Die Untersuchung und Analyse von aktivitätsinduzierter muskulärer Ermüdung ist im Hinblick auf Verletzungen der unteren Extremitäten relevant. Es konnte nachgewiesen werden, dass die Inzidenz von Verletzungen, die nicht durch ein Foul verursacht wurden, in den letzten Minuten von Sportspielen erhöht ist [94, 177]. Diese Befunde werden durch die Feststellung flankiert, dass bestimmte Verletzungen in der späten Phase des Trainings oder Wettkampfes auftreten [57]. Die Ergebnisse weisen darauf hin, dass das Vorhandensein von aktivitätsinduzierter Ermüdung die Entstehung von Verletzungen begünstigen kann. Darüber hinaus wird im Zusammenhang mit non-kontakt Verletzungen der unteren Extremitäten während physischer Aktivität häufig auch Ermüdung als eine mögliche Ursache genannt [6, 16, 37, 47, 78, 83, 142, 145, 146, 160, 161, 185]. Dehnungsinduzierte Muskelverletzungen, Distorsionen, Luxationen sowie Ligamentrupturen im Bereich des Sprung- und Kniegelenks gehören zu den häufigsten Verletzungen im Sport [5, 6, 41, 84, 108, 196, 198].

Es konnte nachgewiesen werden, dass aktivitätsinduzierte Ermüdung nicht nur eine reduzierte Muskelkraft, eine verringerte Reflexamplitude und längere Reflexlaufzeiten implizieren kann [59, 155, 176], sondern ebenfalls eine Minderung der propriozeptiven Funktion möglich ist [204]. Dabei wird die Propriozeption vermutlich durch die Erhöhung der Schwelle für Muskelspindelentladungen und die Modulation der α-γ-Koaktivierung beeinflusst [19, 131, 227]. Die Modulation des afferenten Inputs in Richtung der α-Motoneuronen kann zu einer

inadäquaten Funktion der gelenkumspannenden Muskulatur führen. Dadurch werden die protektive reflektorische Aktivierung der Muskulatur und damit auch die aktive Gelenkstabilität reduziert. Dies führt zu einer nachteiligen Beeinflussung der neuromuskulären Kontrolle des Gelenks, die additional durch eine veränderte volitive Aktivierung verschlechtert wird. Das daraus resultierende Aktivierungsdefizit mindert die Fähigkeit der Muskulatur schnell auf Perturbationen reagieren zu können, die die anatomischen Limitierungen überschreiten und zu Verletzungen führen [186]. Es wird deutlich, dass ein ermüdungsbedingtes reflektorisches und volitives Aktivierungsdefizit die protektiven Ressourcen für das betreffende Gelenk minimiert und somit ein erhöhtes Verletzungsrisiko bestehen kann.

Studien weisen darauf hin, dass aktivitätsinduzierte Ermüdung als Risikofaktor für non-kontakt Verletzungen des vorderen Kreuzbandes (ACL-Verletzungen) angesehen werden kann [45, 67]. Die Experimente, die ACL-Verletzungsmechanismen im Zusammenhang mit Ermüdung analysiert haben, reflektierten zumeist auf kinetische und kinematische Daten [30, 45, 46, 67, 79, 142, 143, 165, 217, 218, 220].

Es existieren lediglich wenige Studien, die die Reflexantworten der knieumspannenden Muskulatur in den Fokus stellten [145, 232]. Wojtys et al. [232] haben die anteriore Tibiatranslation und die elektromyographischen Antworten der relaxierten knieumspannenden Muskulatur vor und nach einem Ermüdungsprotokoll, das mithilfe eines isokinetischen Dynamometers durchgeführt wurde, gemessen. Sie stellten eine erhöhte anteriore Tibiatranslation sowie verzögerte Muskelreflexe fest und schlussfolgerten, dass aktivitätsinduzierte Ermüdung eine Rolle bei der Pathomechanik von Knieverletzungen spielen kann. In einer Studie von Melnyk und Gollhofer [145] konnte gezeigt werden, dass die anteriore Tibiaverschiebung nach einem isokinetischen Ermüdungsprotokoll der ischiocruralen Muskulatur erhöht war. Sie führten die veränderte Kniestabilität auf die reduzierte Reflexaktivität des M. biceps femoris und des M. semitendinosus/semimembranosus zurück. In den beiden genannten Studien wurde jedoch keine Geschlechterdifferenzierung vorgenommen. Demzufolge wurde die hypothetische geschlechtsspezifische Modulation der anterioren Tibiatranslation und Reflexantworten im Zusammenhang mit aktivitätsinduzierter Ermüdung noch nicht betrachtet. Das ist vor dem Hintergrund einer beträchtlich höheren Inzidenz von ACL-Rupturen bei Frauen relevant, deren Ursachen noch nicht allumfassend aufgedeckt wurden [97]. Als primäre Einflussfaktoren werden Unterschiede in der passiven und aktiven Kniegelenkstabilität diskutiert. Die passive Kniegelenkstabilität hängt stark von der Laxizität der Bänder und der Geometrie der artikulären Oberflächen ab, während sich die aktive Stabilität u. a. stark auf Muskelaktivierungsmuster und -reaktionszeiten sowie die Muskelsteifigkeit stützt [99].

Die im Rahmen der Dissertation durchgeführte Studie sollte die Auswirkungen einer spezifischen Bewegungsintervention, welche Ermüdung induzierte, auf die Kniegelenkstabilität bei Männern sowie Frauen analysieren.

EXPERIMENT I: Behrens, M., Mau-Moeller, A., Wassermann, F., Bruhn, S. (2013). Effect of fatigue on hamstring reflex responses and posterior-anterior tibial translation in men and women. *Plos One, 8* (2), e56988.

2.1.2 Fragestellung

Innerhalb dieses Experiments wurde der akute Funktionsverlust der ischiocruralen Muskulatur aufgrund von ermüdender physischer Aktivität analysiert. Demzufolge sollten die Auswirkungen eines spezifischen Ermüdungsprotokolls auf die anteriore Tibiatranslation und Reflexantworten des M. biceps femoris und des M. semitendinosus/semimembranosus bei Männern und Frauen eruiert werden. Es wurde angenommen, dass die aktivitätsinduzierte Ermüdung die Reflexantworten der Hamstrings reduziert und die anteriore Tibiatranslation vergrößert. Zudem wurde geprüft, ob es einen Zusammenhang zwischen verschiedenen Hamstring/Quadriceps-Drehmoment-Ratios und der anterioren Tibiatranslation gibt.

2.1.3 Methoden

Im Folgenden wird der methodische Rahmen für das durchgeführte Experiment dargestellt. Dabei wird der methodische Ansatz global skizziert. Eine detaillierte Beschreibung der Personenstichprobe, experimentellen Prozedur, Datenaufnahme und -analyse sowie statistischen Analyse für das Experiment ist in der Publikation zu finden.

Für die Messung des akuten Funktionsverlustes der ischiocruralen Muskulatur aufgrund von ermüdender physischer Aktivität wurden die Probanden vor und nach einer Ermüdungsintervention, die aus repetitiven Sprüngen bis zur Erschöpfung bestand, untersucht. Die Messungen beinhalteten die Erhebung der anterioren Tibiatranslation mittels eines Kniearthrometers (Abbildung 3) und der Reflexantworten des M. biceps femoris und M. semitendinosus/semimembranosus mittels EMG. In Abbildung 4 sind die EMG- und Tibiatranslationssignale eines Probanden exemplarisch dargestellt.

Für die Messung der anterioren Tibiatranslation wurden die Probanden in 30° Kniebeugung (0° = volle Extension) untersucht. Die Untersuchungssituation sah die Einleitung einer standardisierten Kraft am Unterschenkel vor, die eine anteriore Tibiaverschiebung provozierte. Dafür wurde den Probanden eine Schlinge am Unterschenkel fixiert, die im Zusammenhang mit der relevanten Mechanik eine kontrollierte vordere Schublade auslöste. Mit der Versuchsanordnung konnte über Linearpotentiometer die Verschiebung der Tibia gegenüber dem Femur gemessen werden [32-34, 86].

Für die Messung der Muskelaktivität kamen Oberflächenelektroden zum Einsatz. Die Elektroden wurden in einer bipolaren Konfiguration auf dem M. biceps femoris und dem M. semitendinosus/semimembranosus appliziert. Die Referenzelektrode wurde auf der ipsilateralen Patella platziert.

Für die Analyse wurden die Daten der 15 Messversuche gemittelt. Die Muskelaktivität wurde nach Bruhn et al. [34] analysiert, d. h. das Tibiatranslationssignal zeigte den Beginn der Perturbation an und es wurden drei darauf folgende Zeitintervalle betrachtet (20-40, 40-60 und 60-95 ms). Zur Bestimmung des Ausmaßes der Reflexantworten wurde der root mean square (RMS) des EMG-Signals berechnet. Die Veränderung der Hintergrundaktivität wurde durch die Berechnung des RMS-EMG über 50 ms vor Beginn des Tibiatranslationssignals und die Subtraktion von den Reflexantworten berücksichtigt. Die maximale anteriore Tibiatranslation wurde anhand des Tibiatranslationssignals bestimmt.

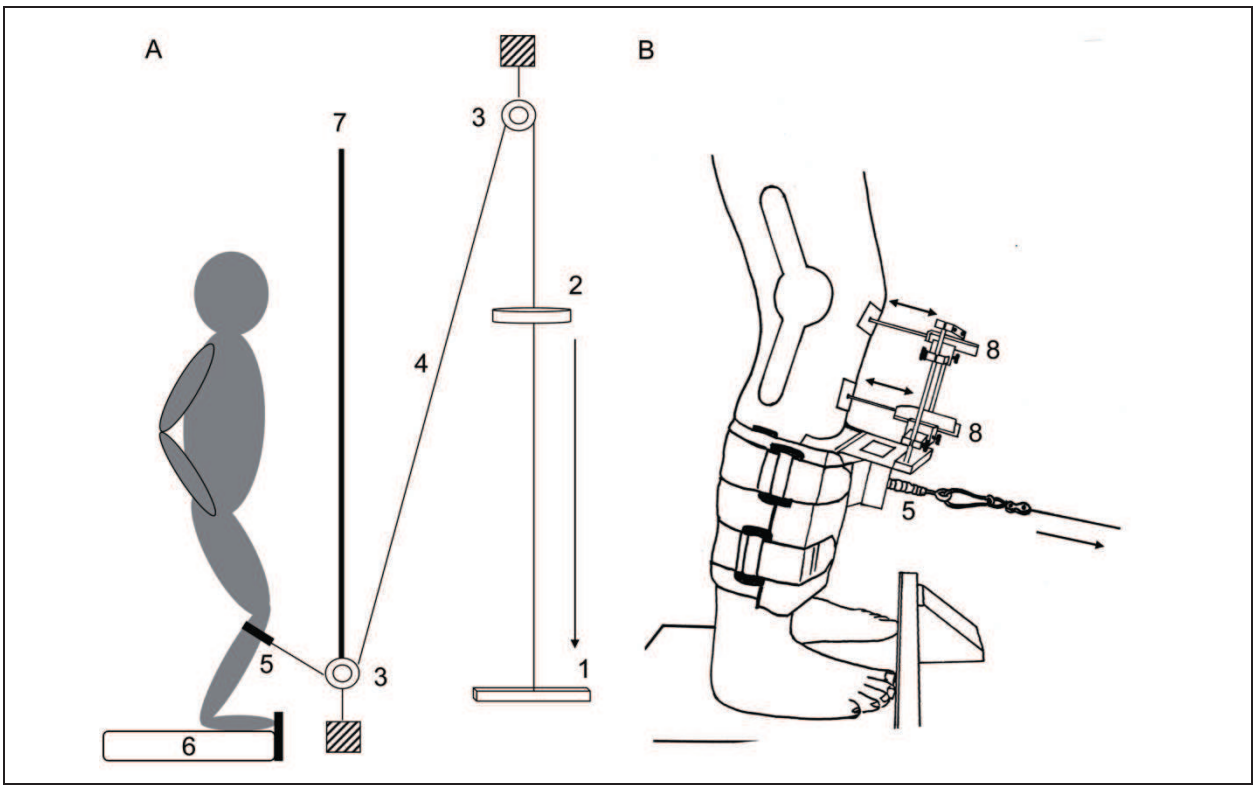

Abb. 3 Darstellung des Versuchsaufbaus zur Messung der funktionellen Kniestabilität.
A Experimentelles Setup, **B** Kniearthrometer | 1: Stopper, 2: Gewicht, 3: Seilzug, 4: Stahlseil, 5: Kraftaufnehmer, 6: Kraftmessplatte, 7: Sichtschutz, 8: Linearpotentiometer. Die Pfeile zeigen die Richtung der wirkenden Kraft an. Die anteriore Tibiatranslation wurde von den beiden Linearpotentiometern erfasst, die auf der Patella und Tuberositas tibiae platziert waren. Die an der Wade applizierte Kraft wurde mittels eines Kraftsensors (5) aufgezeichnet.

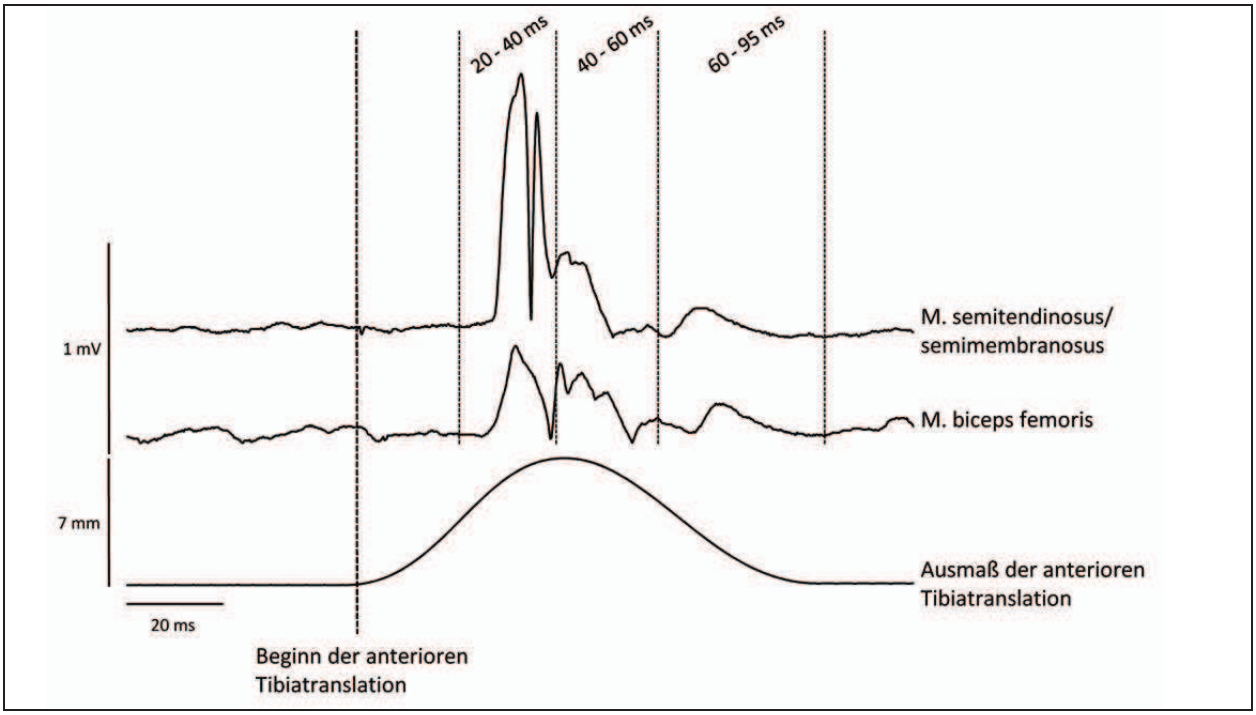

Abb. 4 EMG- und Tibiatranslationssignale eines Probanden.
In dieser Abbildung wurde das EMG-Signal gleichgerichtet, um die unterschiedlichen Anteile des Dehnungsreflexes zu visualisieren. Die dicke gestrichelte Linie markiert den Beginn der anterioren Tibiatranslation. Das EMG-Signal wurde in drei Zeitfenstern betrachtet (20-40, 40-60 und 60-95 ms).

2.1.4 Ergebnisse und Diskussion

Innerhalb dieses Kapitels werden die wichtigsten Ergebnisse des Experiments vorgestellt und anschließend kurz diskutiert. Die komplette Darstellung der Resultate der Studie ist in der angehängten Publikation zu finden.

Die durchgeführte Studie sollte den akuten Funktionsverlust der ischiocruralen Muskulatur aufgrund von ermüdender physischer Aktivität auf die anteriore Tibiatranslation und die Reflexantworten des M. biceps femoris und des M. semitendinosus/semimembranosus bei Männern und Frauen eruieren. Es wurde festgestellt, dass die anteriore Tibiatranslation bei den Frauen signifikant erhöht war. Die Veränderung in der Mechanik ging mit einer signifikanten Reduktion der Reflexantwort des M. biceps femoris in den Zeitfenstern 20-40 und 40-60 ms sowie des M. semitendinosus/semimembranosus im Zeitfenster 20-40 ms einher. Bei den Männern konnten keine statistisch signifikanten Veränderungen von Pre zu Post nachgewiesen werden (Abbildung 5).

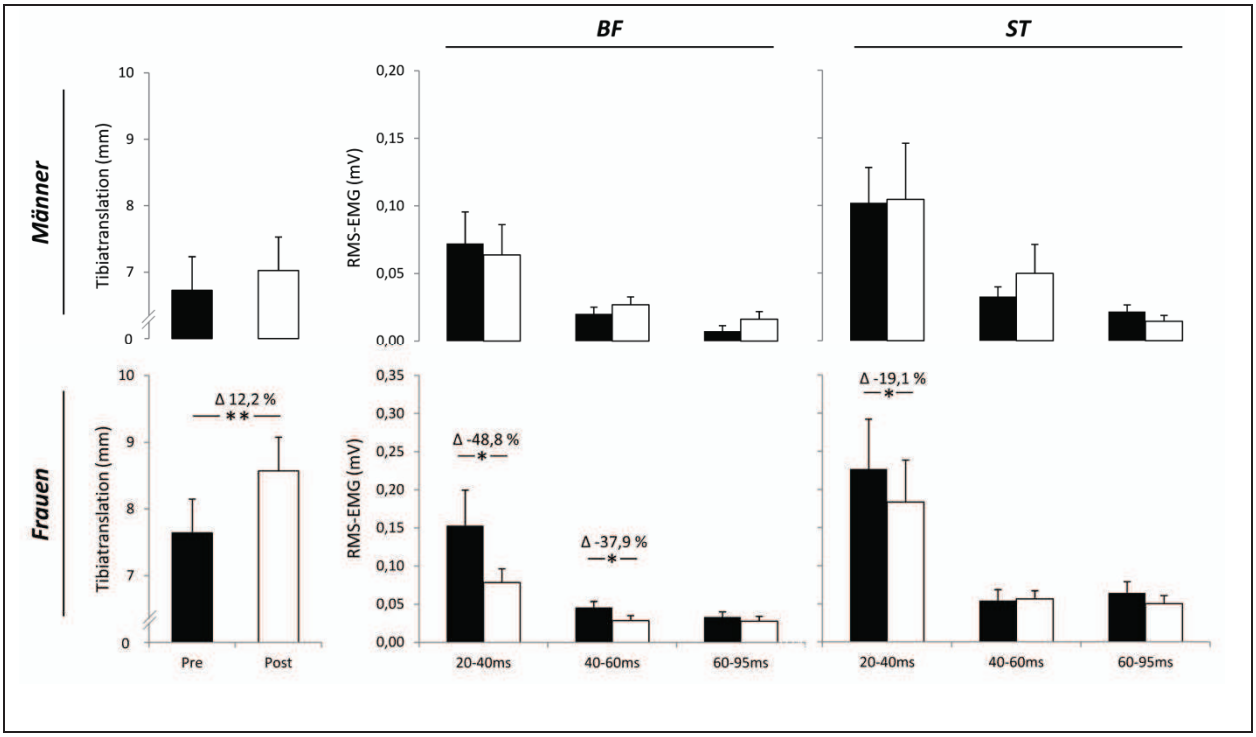

Abb. 5 Einfluss von Ermüdung auf die anteriore Tibiatranslation (*links*) und die Reflexantworten des M. biceps femoris (BF) und des M. semitendinosus/semimembranosus (ST) (*Mitte und rechts*).
Schwarze Balken: Pre, weiße Balken: Post. Die Daten sind als Mittelwerte mit Standardfehler dargestellt.
* zeigt eine signifikante Veränderung zum Pre-Wert an (* $P \leq 0,05$; ** $P \leq 0,01$).

Es wird angenommen, dass eine erhöhte Kniegelenklaxizität zur Pathomechanik von ACL-Rupturen beitragen kann [178]. Zudem konnte gezeigt werden, dass sportliche Aktivitäten, wie z. B. das Laufen [105, 115, 158] oder ein Volleyballtraining [125], eine akute Zunahme der Gelenklaxizität bewirken können. Innerhalb der genannten Experimente wurde die Kniegelenklaxizität jedoch in einer Untersuchungssituation analysiert, in der die Muskeln inaktiv waren und somit ihrer gelenkstabilisierenden Funktion nicht nachkommen konnten. Im Verlauf der hier vorliegenden Studie wurde die anteriore Tibiatranslation während einer Gewichtsbelastung gemessen. In dieser Untersuchungssituation wirkten Muskel- und axiale

Kräfte. Die durch Muskeln und das Körpergewicht generierten Kräfte können zu einer Reduktion der Translation und Rotation der Tibia beitragen [135, 224]. Dabei wird vor allem der ischiocruralen Muskulatur eine große Bedeutung zugeschrieben. Sie fungiert als Antagonist zur anterioren Tibiatranslation und schützt das ACL bei Bewegungen der Tibia relativ zum Femur [26, 106, 152]. Im Hinblick darauf wird der Muskelaktivierung durch Dehnungsreflexe eine potentielle Bedeutung für die Kniegelenkstabilität eingeräumt [73, 74]. Friemert et al. [74] fanden heraus, dass vor allem die Höhe der Muskelaktivität der „short latency response“ der Reflexantwort mit dem Ausmaß der anterioren Tibiatranslation korrespondiert. Dieser Phase der Reflexantwort trug innerhalb der durchgeführten Studie das Analysezeitfenster von 20-40 ms Rechnung. Das vorliegende Experiment zeigte diesen Zusammenhang bei den Frauen auf, während bei den Männern keine signifikante Veränderung durch die Ermüdungsintervention induziert wurde. Demnach war eine Reduktion der Reflexaktivität des M. biceps femoris in den Analysezeiträumen 20-40 und 40-60 ms sowie des M. semitendinosus/semimembranosus im Zeitfenster 20-40 ms bei den Frauen zu verzeichnen. Diese führte zu einer korrespondierenden signifikanten Zunahme der anterioren Tibiatranslation. Die Ergebnisse können mit einer unterschiedlichen Beanspruchung des neuromuskulären Systems bei Frauen und Männern begründet werden. Dabei scheint die neuromuskuläre Kontrolle in Abhängigkeit von der Bewegungsaufgabe moduliert zu werden. Rozzi et al. [187] haben herausgefunden, dass Frauen im Vergleich zu Männern eine erhöhte Aktivierung der ischiocruralen Muskulatur bei der Landung von einem Sprung aufweisen. Darüber hinaus konnten die Autoren eine erhöhte Kniegelenklaxizität und eine reduzierte propriozeptive Funktion bei Frauen feststellen. Sie kamen zu dem Schluss, dass die erhöhte Muskelaktivierung vermutlich ein Versuch des neuromuskulären Systems ist, diese Defizite zu kompensieren. Wenn dieser kompensatorische Mechanismus durch Ermüdung gestört wird, besteht eine erhöhte Verletzungsgefahr [187]. Demnach ist es vorstellbar, dass das verwendete Ermüdungsprotokoll eine stärkere Ermüdung in der ischiocruralen Muskulatur der Frauen bewirkte, die zu den reduzierten Reflexantworten beitrug. Dabei können folgende durch Ermüdung ausgelösten Prozesse eine Rolle spielen: (I) Veränderungen der intrafusalen Eigenschaften [29, 239], (II) Modulation der präsynaptischen Inhibition der Ia Afferenzen [58, 59, 184], (III) Modifikation der intrinsischen Eigenschaften von Motoneuronen [113, 176].

2.2 EINFLUSS VON KONTRAKTIONSINDUZIERTEN MUSKELVERLETZUNGEN AUF DIE NEUROMUSKULÄRE FUNKTION

2.2.1 Darstellung des Forschungsdefizits

Die kontraktionsinduzierte Muskelverletzung ist ein Phänomen, welches infolge ungewohnter physischer und ermüdender Aktivität auftritt. Je nach Ausmaß der Muskelverletzung beinhalten die mit ihr einhergehenden Symptome (I) die partielle Ruptur intrazellulärer Muskelstrukturen, des Sarkolemms und der extrazellulären Matrix [71, 72, 210], (II) die Beeinträchtigung der neuromuskulären Funktion [140, 172], (III) die Entstehung von Schwellungen und den temporären Verlust des vollen Bewegungsausmaßes sowie (IV) das Auftreten von Muskelschmerzen [51]. Die Muskelschmerzen, die mit Verzögerung auftreten und ihren Höhepunkt nach zwei bis drei Tagen erreichen [51], werden in der internationalen Literatur als „Delayed Onset Muscle Soreness“ und im Volksmund als „Muskelkater“ bezeichnet.

Es konnte gezeigt werden, dass exzentrische Muskelaktionen einen stärkeren Muskelschaden induzieren als konzentrische oder isometrische [38]. Exzentrische Muskelaktionen, gefolgt von konzentrischen, sind Bestandteil des Dehnungs-Verkürzungs-Zyklus (DVZ) und kommen bei alltäglichen und sportlichen Bewegungen, wie dem Gehen, Laufen und Springen, vor. Die mit exzentrischen Muskelaktionen einhergehenden Kräfte können zum Auftreten von kontraktionsinduzierten Muskelverletzungen führen. Dies ist bei langandauernden und/oder intensiven alltäglichen und sportlichen Aktivitäten, wie z. B. dem Laufen, repetitiven Springen und Krafttraining, der Fall. Demnach kommt die kontraktionsinduzierte Muskelverletzung häufig bei Freizeit- und Leistungssportlern sowie in der Rehabilitation nach Verletzungen und operativen Eingriffen vor. Dabei tritt sie vor allem bei Überbelastung und Überbeanspruchung auf [65].

Für den Menschen ist dabei der mit der Muskelverletzung einhergehende Funktionsverlust der Muskeln von Bedeutung, weil dieser zu Leistungseinbußen führt. Darüber hinaus kann eine inadäquate Muskelfunktion zu einem erhöhten Verletzungsrisiko beitragen. So kann eine verminderte neuromuskuläre Funktion der gelenkumspannenden Muskulatur z. B. die funktionelle Gelenkstabilität reduzieren [186] und somit das Verletzungsrisiko erhöhen. Andererseits ist bekannt, dass die kontraktionsinduzierten Muskelschäden reversibel sind, soweit die Schädigung ein gewisses Ausmaß nicht überschreitet und den betroffenen Strukturen Gelegenheit zur Regeneration gegeben wird. Die Auswirkungen von kontraktionsinduzierten Muskelverletzungen wurden in diversen Studien analysiert [17, 35, 50, 95, 172, 173, 175, 195, 205, 223, 225, 234]. Der Fokus der Studien lag dabei zum einen auf den Veränderungen von Prozessen auf Muskelebene bedingt durch den strukturellen Muskelschaden, d. h. Modulation der elektromechanischen Kopplung [101], Redistribution der Sarkomerlängen [153], selektive Zerstörung von Muskelfasern [72] und Beeinträchtigung der muskulären Glykogenresynthese [12]. Zum anderen wurde die Muskelfunktion in Bezug auf die Gelenkwinkel-Drehmoment-Relation [205, 234], Drehmoment-Winkelgeschwindigkeit-Relation [72], muskuläre Leistung [201], Sprungperformance [15], Sprintperformance [199], Ausdauerperformance [81] und neuromuskuläre Funktion [56] untersucht. Die Ergebnisse der

zitierten Studien zeigen, dass kontraktionsinduzierte Muskelverletzungen eine Abnahme der Muskelkraft und damit auch der Performance nach sich ziehen.
Es gibt jedoch nur wenige Studien, die zwischen dem Beitrag der Beeinträchtigungen auf Muskelebene und der Modulationen im Zentralnervensystem zur reduzierten Muskelkraft differenzieren konnten [171, 172, 175]. Dabei ist im Hinblick auf die reduzierte Muskelkraft die neuromuskuläre Funktion von Bedeutung. Diese beinhaltet erstens die periphere Ebene, d. h. die muskuläre Funktion, und zweitens die zentrale Ebene, d. h. die neuronale Funktion. Der Beitrag der beiden Ebenen zu der reduzierten Muskelkraft kann unter Zuhilfenahme von geeigneten Methoden, wie z. B. Stimulationsprotokollen, aufgeklärt werden.
Die Beeinträchtigungen auf der peripheren Ebene infolge von kontraktionsinduzierten Muskelverletzungen beinhalten u. a. die Störung der elektromechanischen Kopplung [101] und die Ruptur von Strukturen auf Sarkomerebene [174]. Die Modulation auf der zentralen Ebene beinhaltet u. a. die inadäquate willkürliche Aktivierung von Muskeln [171, 175]. Es wird angenommen, dass die reduzierte neuronale Ansteuerung ein Schutzmechanismus ist, der das tendomuskuläre System vor weiterem Schaden bewahrt [175]. Der mit der Muskelverletzung einhergehende Schmerz aktiviert vermutlich Gruppe III und IV Afferenzen [162], die das Potential besitzen Modulationen auf kortikaler [139] und spinaler Ebene [14] zu induzieren. Diese wiederum sollen zur reduzierten willkürlichen Aktivierung nach kontraktionsinduziertem Muskelschaden beitragen [175].
Es existieren nur wenige Studien, in denen der Beitrag der willkürlichen Aktivierung zur reduzierten Kraft mit adäquaten Methoden untersucht wurde [140, 171, 172, 175]. Die zitierten Studien konzentrierten sich auf die Armflexoren [171, 172], Plantarflexoren [175] und Knieextensoren [140]. In den genannten Untersuchungen konnte eine Beeinträchtigung der kontraktilen Eigenschaften der jeweiligen Muskulatur nachgewiesen werden. Im Hinblick auf die willkürliche Aktivierung wurden gegensätzliche Ergebnisse gefunden. Racinais et al. [175] konnten eine reduzierte willkürliche Aktivierung der Plantarflexoren bis 48 h nach der Intervention, die eine Muskelverletzung induzierte, nachweisen, während die Ergebnisse der anderen Studien keine Beeinträchtigungen für die Armflexoren und Knieextensoren zeigten [140, 172].
Neben der willkürlichen Aktivierung von Muskeln sind Modulationen innerhalb des Reflexbogens von Interesse, d. h. Änderungen in der Exzitabilität der α-Motoneuronen und/oder präsynaptischen Inhibition [238]. Für die Evaluation dieser Modulationen bietet sich die H-Reflexmethode an, die Anfang des 20. Jahrhunderts durch den deutschen Physiologen Paul Hoffmann etabliert wurde [100]. Avela et al. [14] konnten einen reduzierten H-Reflex nach kontraktionsinduziertem Muskelschaden nachweisen, während Racinais et al. [175] keine Veränderung des H-Reflexes dokumentierten.
Aufgrund der widersprüchlichen Ergebnisse und Unklarheiten sollte die folgende eigene Studie, im Hinblick auf den M. quadriceps femoris, zur Erweiterung des Wissens beitragen.

EXPERIMENT II: Behrens, M., Mau-Moeller, A., Bruhn, S. (2012). Effect of exercise-induced muscle damage on neuromuscular function of the quadriceps muscle. *International Journal of Sports Medicine, 33* (8), 600-606.

2.2.2 Fragestellung

Innerhalb dieser Studie wurde die Regeneration der neuromuskulären Funktion des M. quadriceps femoris nach einer Ermüdungsintervention, die zur Überbelastung und somit zu mikrostrukturellen Muskelverletzungen führte, untersucht. Es wurden die Auswirkungen von kontraktionsinduzierten Muskelverletzungen auf das isometrische maximale willkürliche Drehmoment (iMVT), die willkürliche Aktivierung, die kontraktilen Eigenschaften und den Schmerz des M. quadriceps femoris in einem Zeitraum von sieben Tagen analysiert. Zudem wurde der H-Reflex des M. vastus medialis abgeschätzt. Es wurde angenommen, dass das iMVT infolge der kontraktionsinduzierten Muskelverletzungen sinkt und die willkürliche Aktivierung, die kontraktilen Eigenschaften sowie der H-Reflex moduliert werden.

2.2.3 Methoden

Im Folgenden wird der methodische Rahmen für das durchgeführte Experiment dargestellt. Dabei wird der methodische Ansatz global skizziert. Eine detaillierte Beschreibung der Personenstichprobe, experimentellen Prozedur, Datenaufnahme und -analyse sowie statistischen Analyse für das Experiment ist in der Publikation zu finden.

Für die Untersuchung der Modulationen innerhalb des neuromuskulären Systems, ausgelöst durch kontraktionsinduzierte Muskelverletzungen, wurden Kraftmessungen und neurophysiologische Techniken kombiniert.

Zu den Messzeitpunkten wurden die Probanden auf einem Dynamometer fixiert, um die Drehmomente zu messen, die durch den M. quadriceps femoris produziert wurden.

Für die Messung der Muskelaktivität kamen Oberflächenelektroden zum Einsatz. Die Elektroden wurden in einer bipolaren Konfiguration auf dem M. rectus femoris, dem M. vastus medialis und dem M. vastus lateralis appliziert. Die Referenzelektrode wurde auf der ipsilateralen Patella platziert.

Die Analyse der Modulationen auf den unterschiedlichen Ebenen des neuromuskulären Systems wurde mithilfe der transkutanen elektrischen Stimulation des N. femoralis realisiert. Durch die Applikation von elektrischen Stimuli in Ruhebedingung und während der MVCs konnten unterschiedliche Ebenen des neuromuskulären Systems und deren Modulation näher untersucht werden. Dazu gehörte die Abschätzung der kontraktilen Eigenschaften des M. quadriceps femoris in Ruhebedingung, der Erregbarkeit der spinalen α-Motoneuronen des M. vastus medialis mittels der Exzitation der Ia Afferenzen und der willkürlichen Aktivierung des M. quadriceps femoris während MVC. In Abbildung 6 sind die Ebenen innerhalb des neuromuskulären Systems, auf denen akute und chronische Adaptationen induziert werden können und die genutzten Methoden zur Untersuchung dieser dargestellt.

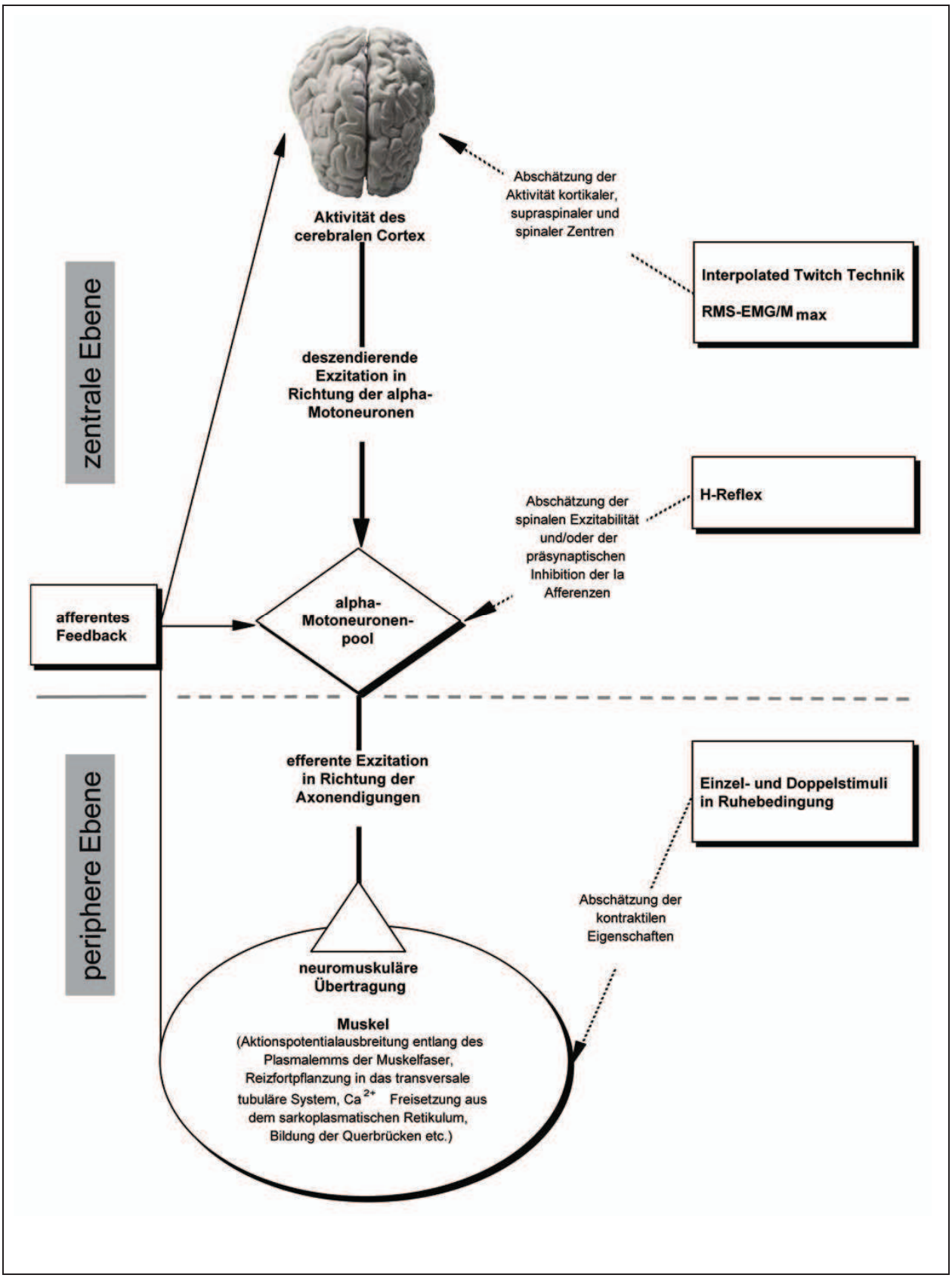

Abb. 6 Ebenen innerhalb des neuromuskulären Systems (*links*), auf denen akute und chronische Adaptationen induziert werden können und die genutzten Methoden zur Untersuchung dieser (*rechts*).

Abschätzung der kontraktilen Eigenschaften

Die Abschätzung der kontraktilen Eigenschaften des M. quadriceps femoris beinhaltete die Aufzeichnung der maximalen Muskelantworten im EMG (maximale M-Welle [M_{max}]) und der mechanischen Antworten der relaxierten Muskulatur auf supramaximale elektrische Einzel- und Doppelstimuli, die am N. femoralis appliziert wurden [87, 124, 222]. Die Stimulationsintensität betrug dabei 140 % der Intensität, mit der M_{max} im EMG und die korrespondierende maximale mechanische Antwort provoziert wurde. M_{max} repräsentiert das volle Ausmaß der Muskelaktivierung, induziert durch die direkte Depolarisation aller motorischen Axone. Es wird davon ausgegangen, dass diese maximale muskuläre Antwort mit der Entladung des gesamten Motoneuronenpools gleichgesetzt werden kann [163, 167]. Demnach stellt die supramaximale Stimulation sicher, dass alle Muskelfasern innerviert werden. Diese Stimulationsmethode erlaubt eine Abschätzung der kontraktilen Eigenschaften der betreffenden Muskulatur [80, 87, 124, 128]. Der involvierte Prozess umfasst die elektromechanische Kopplung inklusive der intrazellulären Ca^{2+}-Kinetik. Durch den Pre-Post-Vergleich der Amplitude von M_{max} und der korrespondierenden mechanischen Antwort kann auf differente Anpassungen des neuromuskulären Systems geschlossen werden. In Abbildung 7 sind die unterschiedlichen berechenbaren Parameter dargestellt.

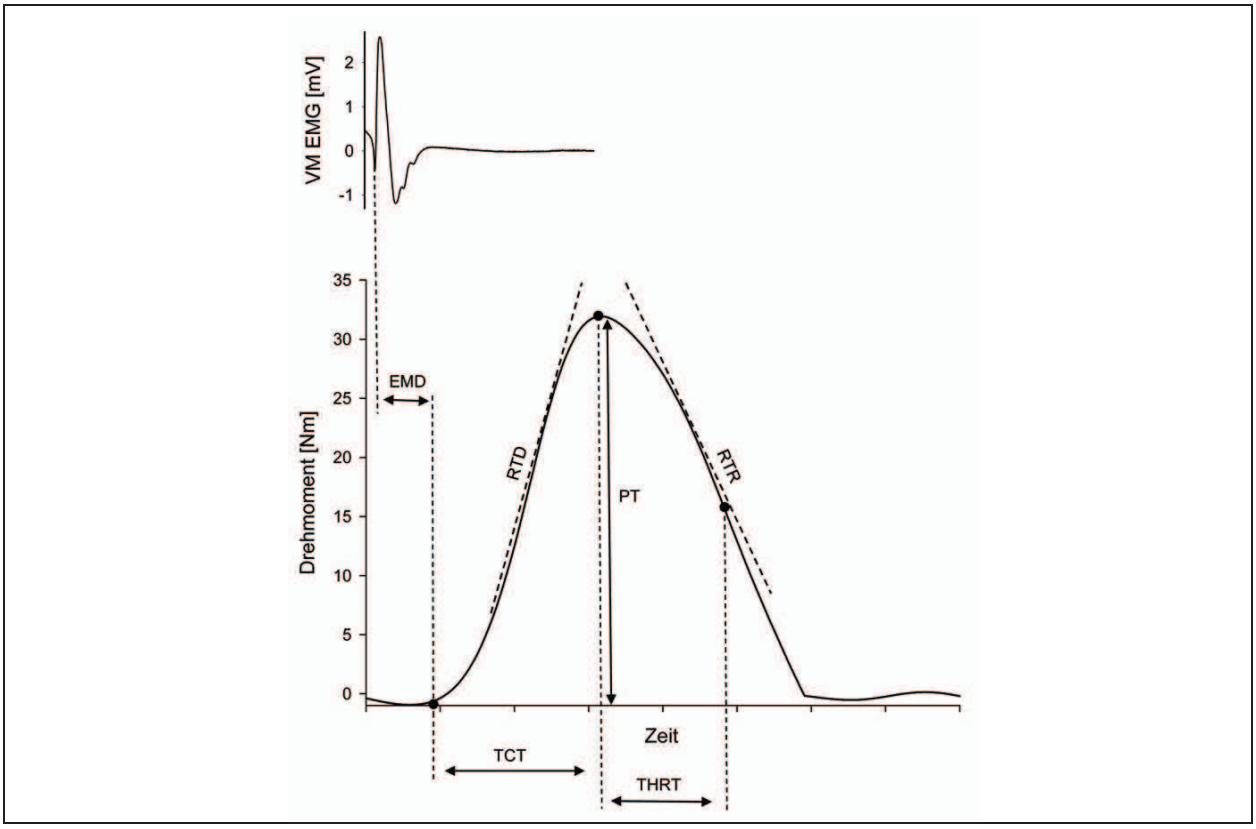

Abb. 7 Maximale M-Welle des M. vastus medialis (VM) (*oben*) und Drehmoment des M. quadriceps femoris (*unten*) eines Probanden induziert durch die supramaximale Stimulation des N. femoralis.
RTD: rate of torque development, RTR: rate of torque relaxation, PT: peak torque, EMD: electromechanical delay, TCT: twitch contraction time, THRT: twitch half relaxation time.

Abschätzung der Exzitabilität der spinalen α-Motoneuronen

Mit der H-Reflextechnik kann die Modulation der Exzitabilität der spinalen α-Motoneuronen und/oder präsynaptischen Inhibition der Ia Afferenzen infolge von kurz- oder langfristigen Interventionen abgeschätzt werden [2, 167, 197, 238]. Die Auslösung des H-Reflexes basiert auf der kurzen elektrischen Stimulation eines gemischten peripheren Nervens, der sowohl afferente als auch efferente Axone beinhaltet [166, 238]. Aufgrund der elektrischen Reizung mit einer geringen Stromstärke werden die Ia-Spindelafferenzen depolarisiert und das Aktionspotential breitet sich entsprechend dem Alles-oder-Nichts-Gesetz in Richtung der homonymen α-Motoneuronen aus. Im Rückenmark erfolgt eine Umschaltung der Erregung auf die α-Motoneuronen, deren Depolarisation Aktionspotentiale generiert, die sich über den efferenten Schenkel des Reflexbogens bis zum Muskel ausbreiten. Die durch die Elektrostimulation induzierte Reflexantwort des Muskels wird mittels Oberflächenelektroden elektromyografisch erfasst. Die Reflexantwort variiert in Abhängigkeit von der applizierten Stromstärke. Demnach erscheint bei geringen Stromstärken zunächst der H-Reflex. Mit zunehmender Stimulationsintensität erhöht sich der H-Reflex, bis er sein Maximum erreicht, um sich anschließend wieder zu reduzieren. Eine Erhöhung der Reizstärke impliziert außerdem die Generierung eines weiteren Potentials, das als M-Welle bezeichnet wird und durch die Stimulation der motorischen Axone entsteht. Diese direkte Muskelantwort wird durch eine efferente Reizleitung in Richtung des Muskels ausgelöst und erreicht bei einer bestimmten Stimulationsintensität ihr Maximum, welches als M_{max} bezeichnet wird [163, 167, 238]. Die maximale Amplitude des H-Reflexes im Verhältnis zur maximalen M-Welle (H_{max}/M_{max}-Ratio) ermöglicht die Abschätzung der spinalen Exzitabilität und/oder präsynaptischen Inhibition der Ia Afferenzen für einen gegebenen Zustand [238]. Veränderungen des Verhältnisses beider Amplituden durch Interventionen dokumentieren Adaptationen auf der Ebene des spinalen sensomotorischen Systems. Im Hinblick auf die durchgeführten Experimente wurde die elektrische Stimulation am N. femoralis appliziert und die elektromyographische Erfassung der evozierten Potentiale erfolgte am M. vastus medialis. Abbildung 8 zeigt die evozierten Potentiale (H_{max} und M_{max}) eines Probanden.

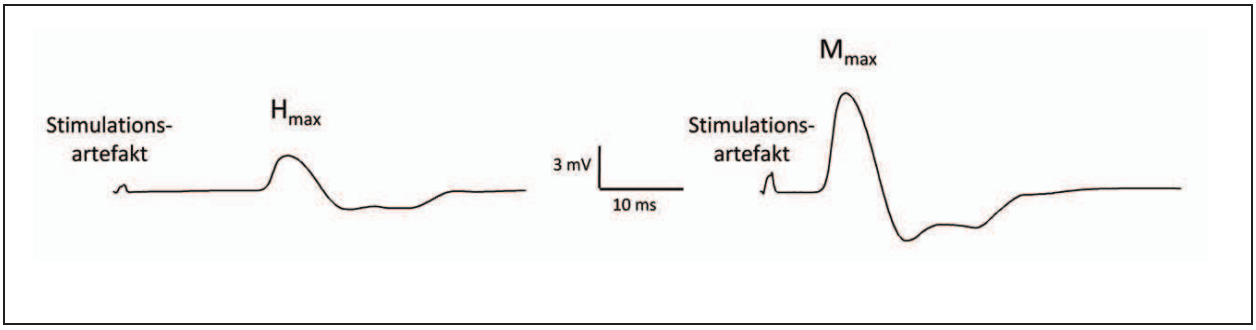

Abb. 8 Maximaler H-Reflex (H_{max}) und maximale M-Welle (M_{max}) des M. vastus medialis einer Versuchsperson.

Abschätzung der willkürlichen Aktivierung

Die Abschätzung der willkürlichen Aktivierung des M. quadriceps femoris erfolgte zum einen über die Berechnung des normalisierten RMS-EMG (RMS-EMG/M_{max}) und zum anderen mittels der Interpolated Twitch Technik.

Der Vorteil der normalisierten EMG-Daten gegenüber den „einfachen" EMG-Daten liegt in der Reduktion von potentiellen Fehlerquellen. Dazu gehören eine veränderte Elektrodenposition bei Wiederholungsmessungen sowie die Inter-Session-Variabilität der Hautimpedanz, des subkutanen Fettgewebes und der Faszien [219]. Der RMS-EMG/M_{max} wurde herangezogen, um die Aktivierung des M. quadriceps femoris während der initialen Phase des Drehmomentanstieges (RMS-EMG_{RTD}/M_{max}) und des Drehmomentmaximums (RMS-EMG_{iMVT}/M_{max}) zu analysieren.

Als zweites Verfahren wurde die Interpolated Twitch Technik verwendet. Mit dieser Methode ist es möglich, die volitive Aktivierung eines Muskels zu messen [8, 77, 122, 149]. Anders gesagt, kann somit das Ausmaß des Aktivierungsdefizits für den relevanten Muskel abgeschätzt werden. Dieses wird über die supramaximale Stimulation eines peripheren Nervens während einer statischen MVC und der Messung des korrespondierenden mechanischen Outputs eruiert. Es wird angenommen, dass die supramaximale Aktivierung der motorischen Axone alle Muskelfasern rekrutiert [163], demnach auch solche, die bei der willkürlichen Anstrengung nicht in den Kontraktionsprozess einbezogen werden können. Wenn die elektrische Stimulation keinen additionalen mechanischen Output produziert, ist der Muskel durch den deszendierenden Input von kortikalen, supraspinalen und spinalen Zentren voll aktiviert. Wird das generierte Drehmoment durch die Stimuli jedoch erhöht, weist das auf eine submaximale volitive Aktivierung hin [117].

Die Quantifizierung der volitiven Aktivierung (VA) erfolgt über die Formel:

$$\% \text{ VA} = (1 - (\text{superimposed twitch} * (T_b * \text{iMVT}^{-1}) * \text{control twitch}^{-1})) * 100$$ [140, 212]

T_b bezeichnet das Drehmomentniveau vor der Applikation der elektrischen Stimuli, welches aufgrund der Fluktuation der Drehmoment-Zeit-Kurve unter dem iMVT liegen kann. Die beschriebene Formel wirkt dieser Fehlerquelle entgegen. In Abbildung 9 kann die Methode anhand von Rohdaten eines Probanden nachvollzogen werden. Die Versuchsperson hatte die Aufgabe eine isometrische MVC durchzuführen. Die elektrische Stimulation des N. femoralis erfolgte 2 s nach dem Beginn der Kontraktion und generierte den „superimposed twitch". Das Ende der Kontraktion löste eine weitere 2 s später erfolgende elektrische Stimulation aus, die den „control twitch" produzierte. Mithilfe der in der Formel aufgeführten Parameter kann auf die willkürliche Aktivierung in Prozent geschlossen werden. Die Abbildung zeigt zudem weitere Parameter, die anhand der Rohdaten berechnet werden können und Aufschluss über die neuromuskuläre Funktion geben.

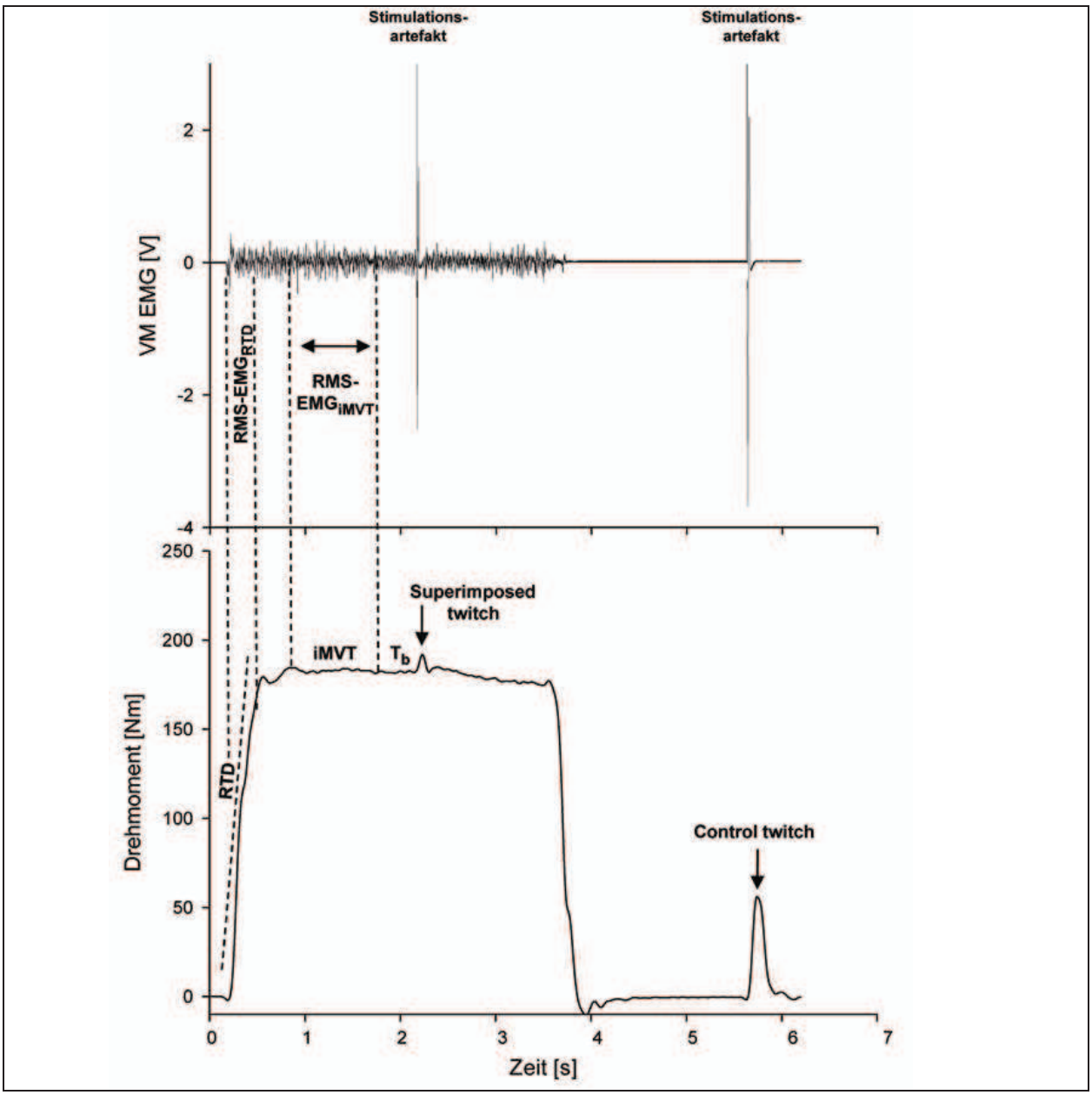

Abb. 9 EMG des M. vastus medialis (VM) (*oben*) und das isometrische maximale willkürliche Drehmoment (iMVT) des M. quadriceps femoris (*unten*) eines Probanden.
Die Stimulationsartefakte im EMG markieren die Zeitpunkte der Stimulation, auf die der M. quadriceps femoris mit einem Drehmomentanstieg reagierte. RTD: rate of torque development, T_b: Drehmomentniveau vor der Applikation der elektrischen Stimuli, RMS-EMG$_{RTD}$: Aktivierung des M. quadriceps femoris während der initialen Phase des Drehmomentanstieges, RMS-EMG$_{iMVT}$: Aktivierung des M. quadriceps femoris während des Drehmomentmaximums.

2.2.4 Ergebnisse und Diskussion

Die durchgeführte Studie untersuchte die Regeneration der neuromuskulären Funktion des M. quadriceps femoris nach einer Ermüdungsintervention, welche zur Überbelastung und somit zu mikrostrukturellen Muskelverletzungen führte. Dafür wurden die Auswirkungen von kontraktionsinduzierten Muskelverletzungen auf das iMVT, die willkürliche Aktivierung, die kontraktilen Eigenschaften und den Schmerz des M. quadriceps femoris in einem Zeitraum von sieben Tagen (Post, 24 h, 48 h, 72 h, 7 d) analysiert. Zudem wurde die Exzitabilität des α-Motoneuronenpools des M. vastus medialis über Ia Afferenzen zu den Messzeitpunkten abgeschätzt. Die kontraktionsinduzierten Muskelverletzungen wurden mithilfe eines Dynamometers durch vier Sätze mit jeweils 25 maximalen willkürlichen konzentrisch-exzentrischen Kontraktionen bei einer Geschwindigkeit von 60°/s induziert.

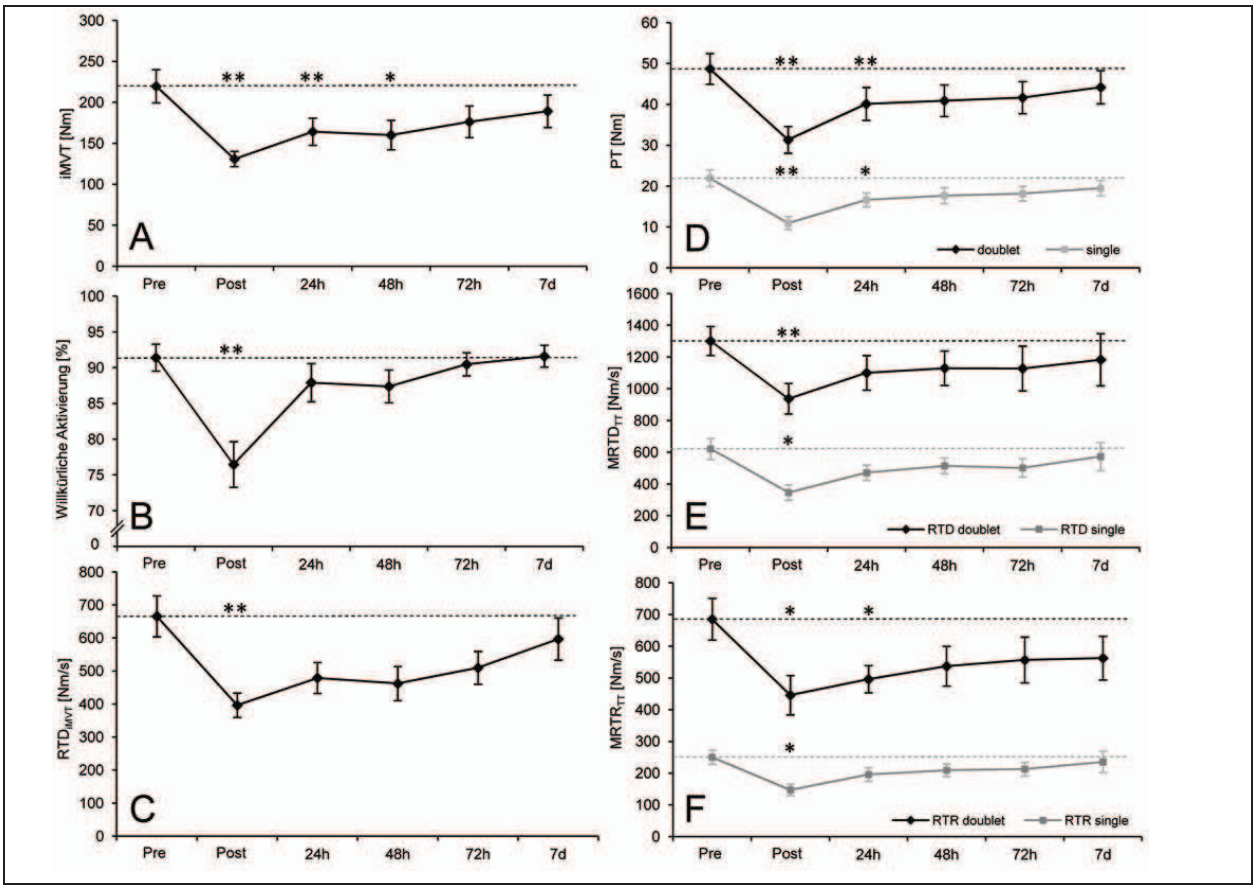

Abb. 10 Einfluss von kontraktionsinduzierten Muskelverletzungen auf das willkürliche Drehmoment, die Muskelaktivierung und die kontraktilen Eigenschaften des M. quadriceps femoris.
A Isometrisches maximales willkürliches Drehmoment (iMVT), **B** willkürliche Aktivierung, **C** rate of torque development der willkürlich produzierten Drehmoment-Zeit-Kurve im Zeitfenster 0-200 ms (RTD_{iMVT}), **D** peak torque produziert durch den M. quadriceps femoris in Ruhebedingung (PT), induziert durch die elektrische Stimulation des N. femoralis mit supramaximalen Doppel- (doublet) und Einzelstimuli (single), **E** maximal rate of torque development produziert durch den M. quadriceps femoris in Ruhebedingung ($MRTD_{TT}$), induziert durch die elektrische Stimulation des N. femoralis mit supramaximalen Doppel- (doublet) und Einzelstimuli (single), **F** maximal rate of torque relaxation produziert durch den M. quadriceps femoris in Ruhebedingung ($MRTR_{TT}$), induziert durch die elektrische Stimulation des N. femoralis mit supramaximalen Doppel- (doublet) und Einzelstimuli (single). Die Daten sind als Mittelwerte mit Standardfehler dargestellt. * zeigt eine signifikante Veränderung zum Pre-Wert an (* $P \leq 0{,}05$; ** $P \leq 0{,}01$).

Unmittelbar nach der Intervention (Post) war das iMVT signifikant reduziert. Dafür kann eine signifikante Abnahme der willkürlichen Aktivierung und der kontraktilen Eigenschaften des M. quadriceps femoris verantwortlich gemacht werden. Die Verminderung der Muskelaktivierung und -funktion zum Zeitpunkt Post weist auf interventionsbedingte zentrale [75] und periphere Modulationen hin [148]. Dabei ist die deutliche Abnahme der Parameter vermutlich überwiegend auf die akuten Effekte der Ermüdung zurückzuführen [150, 202]. Die Folgemessungen ergaben eine signifikante Reduktion des iMVT bis 48 h nach der Intervention, die primär auf die Verschlechterung der kontraktilen Funktion zurückzuführen war. Die willkürliche Aktivierung, abgeschätzt mittels Interpolated Twitch Technik, zeigte zwar keine signifikante Änderung, war aber nach 24 h um 3,8 % und nach 48 h um 4,4 % vermindert. Abbildung 10 zeigt die Zusammenfassung der wichtigsten Ergebnisse. Demnach scheinen akute Adaptationen in der Peripherie eine größere Rolle für die Abnahme des Drehmoments nach kontraktionsinduzierten Muskelverletzungen zu spielen, wobei die Modulation der Muskelaktivierung nicht ausgeschlossen werden kann. Die Messung des Muskelschmerzes ergab eine signifikante Zunahme zu den Messzeitpunkten 24 h, 48 h und 72 h. Die neuronalen Parameter wiesen jedoch keine Korrespondenz mit der Schmerzentwicklung auf.

Die Reduktion des iMVT durch kontraktionsinduzierte Muskelverletzungen konnte in unterschiedlichen Studien nachgewiesen werden [140, 172, 175]. Im Hinblick auf den Beitrag der Muskelinaktivierung zur Abnahme des iMVT konnten Racinais et al. [175] eine reduzierte willkürliche Aktivierung der Plantarflexoren bis 48 h nach der Intervention, die eine Muskelverletzung induziert hat, nachweisen. Im Widerspruch dazu stehen die Ergebnisse von Martin et al. [140] und Prasartwuth et al. [172], die keine Veränderung dieses Parameters für die Knieextensoren und Armflexoren finden konnten. Durch den kombinierten Ansatz der vorliegenden Studie, der die Abschätzung der willkürlichen Aktivierung über die Interpolated Twitch Technik und den normalisierten RMS-EMG umfasste, konnte keine Modulation der Muskelaktivierung festgestellt werden. Zudem zeigte die Exzitabilität der α-Motoneuronen des M. vastus medialis, abgeschätzt mittels der H-Reflextechnik, ebenfalls keine Veränderung. Demnach scheint der mit kontraktionsinduzierten Muskelverletzungen einhergehende Muskelschmerz keine Modulation dieser neuronalen Parameter zu provozieren.

Die Verschlechterung der kontraktilen Funktion nach kontraktionsinduzierten Muskelverletzungen wurde schon zuvor beobachtet [172]. Die Abnahme der Muskelfunktion wird dabei primär durch die Beeinträchtigung der elektromechanischen Kopplung verursacht. Dabei spielen vermutlich die partielle Ruptur transversaler Tubuli [213], die Abnahme der intrazellulären Ca^{2+}-Konzentration und eine reduzierte Ca^{2+}-Sensitivität eine Rolle [20, 21]. Darüber hinaus wird eine Rechtsverschiebung der Längen-Spannungs-Relation durch den strukturellen Muskelschaden diskutiert [109, 171], die durch eine ungleichmäßige Dehnung und Ruptur von Sarkomeren zustande kommt [38]. Demzufolge muss der Muskel länger sein, damit die Myofilamente optimal interagieren können.

3. CHRONISCHE ANPASSUNGEN DER NEUROMUSKULÄREN FUNKTION

3.1 EINFLUSS EINES PLYOMETRISCHEN TRAININGS AUF DIE NEUROMUSKULÄRE FUNKTION

3.1.1 Darstellung des Forschungsdefizits

Regelmäßige physische Aktivität, z. B. Training, führt zur Steigerung der Leistungsfähigkeit des neuromuskulären Systems. Im Hinblick auf eine Erhöhung der Muskelkraft bietet sich Krafttraining an, welches neuronale und muskuläre Adaptationen provoziert. Dabei wird der Kraftzuwachs in der initialen Phase des Trainings primär neuronalen Anpassungen zugeschrieben, denen muskuläre Adaptationen folgen [66].

Zu den neuronalen Adaptationen infolge von Krafttraining gehört eine zunehmende Verbesserung der intermuskulären Koordination, vor allem bei Kraftleistungen, die das Zusammenspiel mehrerer Muskeln erfordern [188]. Des Weiteren kann es zur Erhöhung der Muskelaktivierung des Agonisten kommen, die mit einer erhöhten Rekrutierung und Feuerfrequenz von motorischen Einheiten begründet wird [60, 192]. Ein Anstieg der agonistischen Muskelaktivität infolge eines Krafttrainings wurde in diversen Studien belegt, die zur Detektion der trainingsbedingten Veränderungen die Elektromyographie (EMG) [88-90, 118, 157] oder Stimulationstechniken [2, 55, 61, 69, 179] verwendeten. Es gibt Hinweise darauf, dass die durch Krafttraining induzierten Anpassungen primär auf spinaler Ebene lokalisiert sind und kortikale Adaptationen eine untergeordnete Rolle spielen [44, 60, 104]. Zudem kann Krafttraining die Koaktivierung des Antagonisten reduzieren [43], was wiederum zur Abnahme der zu überwindenden Kraft für den Agonisten führt. Außerdem kann eine verminderte Koaktivierung zur Erhöhung der agonistischen Muskelaktivität führen, indem die reziproke Inhibition des Agonisten abnimmt [70].

Die primäre Anpassung auf Muskelebene infolge von Krafttraining ist die Zunahme des Muskelquerschnitts, die als Muskelhypertrophie bezeichnet wird [70]. Nach der Initiierung des ersten Trainingsstimulus werden diverse zelluläre und hormonelle Signalwege aktiviert, die zur Hypertrophie der beanspruchten Muskulatur führen [22, 28, 52]. Die beiden dabei involvierten Hauptprozesse sind (I) die Erhöhung der Muskelproteinsynthese [123] und (II) die Proliferation von Satellitenzellen im Muskel [110]. Trotz des zeitnahen Einsetzens der genannten Prozesse ist eine messbare Zunahme des Muskeldickenwachstums bei zuvor untrainierten Personen erst nach ca. vier bis acht Wochen nachweisbar [4, 11, 76, 98]. Eine neuere Untersuchung zeigte jedoch, dass eine signifikante Muskelquerschnittzunahme bereits nach drei Wochen eintreten kann [200]. Die beiden Prozesse, die das Muskelwachstum induzieren, setzen zwar zeitnah nach der Applikation des ersten Trainingsstimulus ein, tragen aber in unterschiedlichem Ausmaß zur Hypertrophie der beanspruchten Muskulatur bei. Demnach ist die Erhöhung der Muskelproteinsynthese der primäre Faktor für das Muskeldickenwachstum und die Aktivierung von Satellitenzellen steht diesem nach [11]. Dabei sind die Typ II Muskelfasern stärker von der Hypertrophie betroffen als die Typ I Muskelfasern [40, 209, 215].

Neben dem Dickenwachstum des Muskelgewebes durch Krafttraining kommt es zu weiteren Adaptationen. Diese beinhalten Anpassungen innerhalb der Muskelfasertypen, die durch eine Abnahme der Typ IIX und eine Zunahme der Typ IIA Muskelfasern gekennzeichnet sind [40, 90,

92]. Darüber hinaus kann es zur Veränderung der Sehne und des Bindegewebes [70, 119] sowie der Muskelarchitektur kommen [1, 112].
Krafttraining kann mittels unterschiedlicher Trainingsregimes durchgeführt werden, die wiederum spezifische Anpassungen provozieren. Dazu gehören u. a. das Training an Geräten, Training mit freien Gewichten, Elektromyostimulationstraining und plyometrisches Training.
Der Begriff plyometrisches Training bezieht sich auf sportliche Übungen, die den DVZ involvieren [137]. Der DVZ ist durch eine exzentrische Muskelaktion, auf die eine konzentrische folgt, gekennzeichnet und kommt bei alltäglichen und sportlichen Bewegungen, wie dem Gehen, Laufen und Springen, vor. Plyometrisches Training kommt bei gesunden Menschen zur Anwendung, es wird aber auch für spezielle Patientenpopulationen, z. B. Menschen mit Osteoporose, empfohlen [137].
Die Auswirkungen eines plyometrischen Trainings auf den menschlichen Organismus wurden in diversen Studien thematisiert [31, 85, 107, 111, 121, 127, 132, 133, 136, 189, 207, 231]. Der Fokus lag dabei zum einen auf der Wirkung dieser Trainingsform auf die sportliche Leistung, z. B. die Sprung-, Sprint- und Laufperformance [31, 121, 194, 207]. Zum anderen wurden neuromuskuläre Adaptationen [85, 127, 132, 133, 189, 214] und muskuloskeletale Adaptationen [9, 111, 121, 231, 233] untersucht. Obwohl diverse Studien eine Kraftsteigerung nach plyometrischem Training dokumentieren konnten [91, 189, 230], existieren nur wenige Studien, die die zugrundeliegenden neuromuskulären Adaptationen analysiert haben. Kyrolainen et al. [127] untersuchten neuronale Anpassungen mittels EMG und muskuläre Adaptationen mithilfe von Muskelbiopsien. Nach dem Training stellten die Autoren eine erhöhte maximale Kraft der Plantarflexoren und einen Anstieg der korrespondierenden EMG-Aktivität fest, jedoch keine Änderungen auf muskulärer Ebene. Kubo et al. [121] konnten ebenfalls eine Steigerung der Kraft der Plantarflexoren nach plyometrischem Training nachweisen. Mithilfe der Interpolated Twitch Technik konnten die Autoren zeigen, dass eine erhöhte willkürliche Aktivierung zur Kraftsteigerung beigetragen hat. Im Hinblick auf die Knieextensoren konnten Kyrolainen et al. [127] keine Steigerung der maximalen Kraft, aber der Kraftanstiegssteilheit, nach einem plyometrischen Training feststellen. Malisoux et al. [133] untersuchten muskuläre Adaptationen des M. vastus medialis infolge eines plyometrischen Trainings mittels Muskelbiopsien und stellten eine Erhöhung des Querschnitts für Typ-I, -IIA und -IIA/IIX Muskelfasern fest. Die Autoren konnten außerdem eine Steigerung der Kraft und Sprunghöhe dokumentieren.
Aufgrund der übersichtlichen Anzahl an Studien, die sich mit neuronalen sowie muskulären Adaptationen und ihrem Beitrag zu einer gesteigerten maximalen Kraft nach plyometrischem Training beschäftigten, kommen Markovic und Mikulic [137] in ihrem Review-Artikel über plyometrisches Training auf S. 869 zu dem Fazit: „[…] our current knowledge about plyometric training-induced changes in neural function is limited“. Hier wird der Forschungsbedarf deutlich.
Es existieren nur wenige Studien, die die neuromuskuläre Funktion des M. quadriceps femoris vor und nach plyometrischem Training mit geeigneten Methoden analysiert haben. Die folgende durchgeführte Studie sollte dazu beitragen das Wissen in dieser Hinsicht zu erweitern.

EXPERIMENT III: Behrens, M., Mau-Moeller, A., Bruhn, S. (2013). Effect of plyometric training on neural and mechanical properties of the knee extensor muscles. *International Journal of Sports Medicine* (in press).

3.1.2 Fragestellung

Innerhalb dieses Experiments wurde die Modulation der Leistungsfähigkeit des neuromuskulären Systems durch Training untersucht. Es wurden die Auswirkungen eines achtwöchigen plyometrischen Trainings auf das iMVT, die willkürliche Aktivierung, die Anstiegssteilheit der Drehmoment-Zeit-Kurve (rate of torque development [RTD]), die kontraktilen Eigenschaften des M. quadriceps femoris, den H-Reflex des M. vastus medialis und die Sprungperformance analysiert. Es wurde davon ausgegangen, dass die Trainingsintervention zur Erhöhung der genannten Parameter führt.

3.1.3 Methoden

Für die Untersuchung der Modulationen innerhalb des neuromuskulären Systems, ausgelöst durch ein plyometrisches Training, wurde auf das gleiche Methodenrepertoire zurückgegriffen, das in Kapitel 2.2.3 beschrieben ist. Dieses beinhaltete jeweils Kraftmessungen und neurophysiologische Techniken. Eine detaillierte Beschreibung der Personenstichprobe, experimentellen Prozedur, Datenaufnahme und -analyse sowie statistischen Analyse für das Experiment ist in der Publikation zu finden.

Zu den Messzeitpunkten wurden die Probanden auf einem Dynamometer fixiert, um die Drehmomente zu messen, die durch den M. quadriceps femoris produziert wurden.

Für die Messung der Muskelaktivität kamen Oberflächenelektroden zum Einsatz. Die Elektroden wurden in einer bipolaren Konfiguration auf dem M. rectus femoris, dem M. vastus medialis und dem M. vastus lateralis appliziert. Die Referenzelektrode wurde auf der ipsilateralen Patella platziert.

Die Analyse der Modulationen auf den unterschiedlichen Ebenen des neuromuskulären Systems wurde mithilfe der transkutanen elektrischen Stimulation des N. femoralis realisiert. Durch die Applikation von elektrischen Stimuli in Ruhebedingung und während der MVCs konnten unterschiedliche Ebenen des neuromuskulären Systems und deren Modulation näher untersucht werden. Dazu gehörte die Abschätzung der kontraktilen Eigenschaften des M. quadriceps femoris in Ruhebedingung, der Erregbarkeit der spinalen α-Motoneuronen des M. vastus medialis mittels der Exzitation der Ia Afferenzen und der willkürlichen Aktivierung des M. quadriceps femoris während MVC.

3.1.4 Ergebnisse und Diskussion

In diesem Kapitel werden die wichtigsten Ergebnisse des Experiments vorgestellt und anschließend kurz diskutiert. Die komplette Darstellung der Resultate der Studie ist in der angehängten Publikation zu finden.

Innerhalb dieses Experiments wurde die Modulation der Leistungsfähigkeit des neuromuskulären Systems durch Training untersucht. Die durchgeführte Studie analysierte die Auswirkungen eines achtwöchigen plyometrischen Trainings auf das iMVT, die willkürliche Aktivierung, die RTD und die kontraktilen Eigenschaften des M. quadriceps femoris sowie die Sprungperformance. Zudem wurde die Exzitabilität des α-Motoneuronenpools des M. vastus medialis über Ia Afferenzen zu den Messzeitpunkten abgeschätzt. Die Messung der neuromuskulären Funktion erfolgte in zwei Kniewinkeln, d. h. 80° und 45° Knieflexion (0° = volle

Extension), um potentielle winkelspezifische Trainingsanpassungen aufzudecken. Das Training erfolgte zwei Mal pro Woche und beinhaltete Countermovement Jumps, Squat Jumps und Drop Jumps.

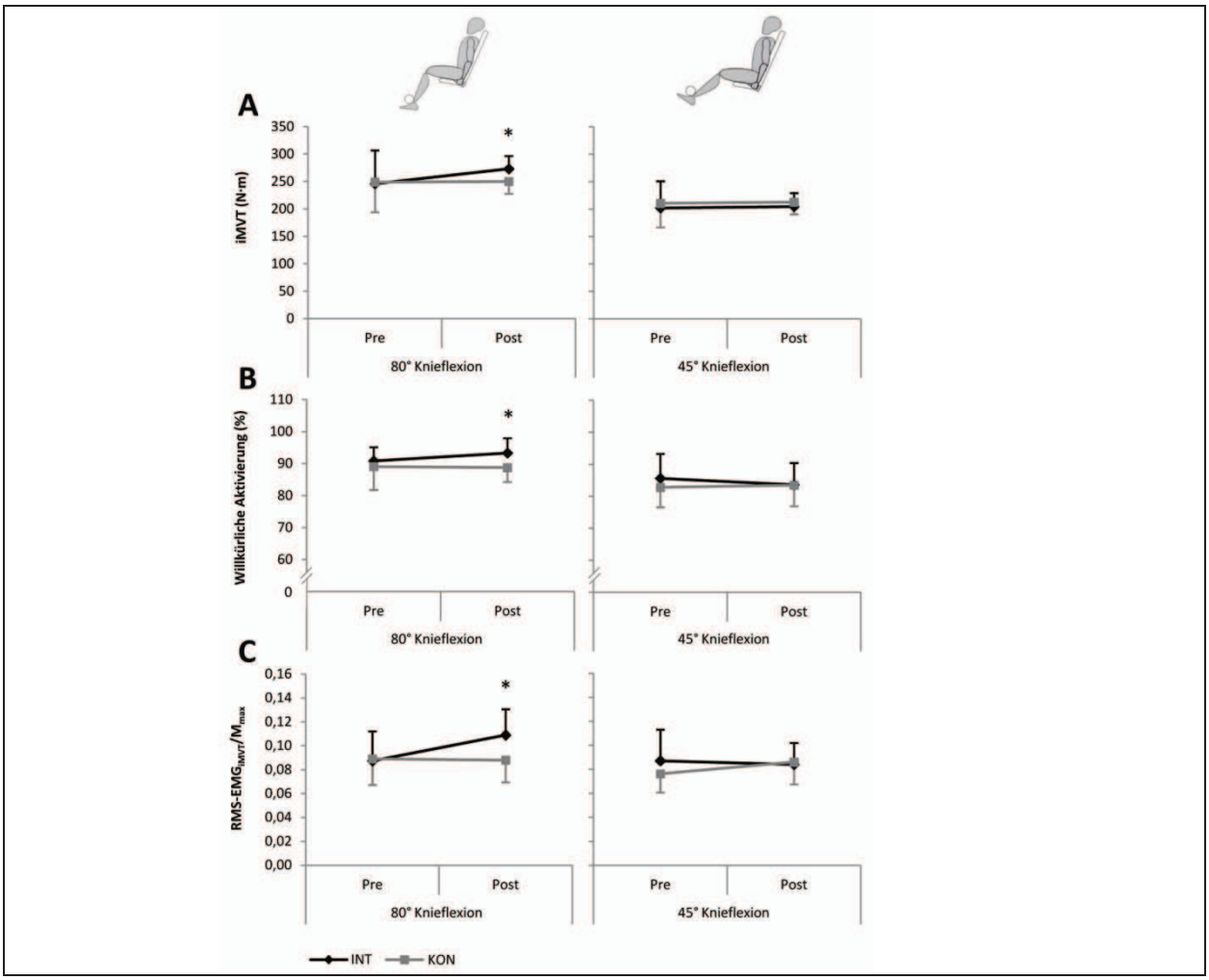

Abb. 11 Einfluss eines plyometrischen Trainings auf das willkürliche Drehmoment und die Aktivierung des M. quadriceps femoris.
A Isometrisches maximales willkürliches Drehmoment (iMVT), **B** willkürliche Aktivierung, **C** normalisierte Muskelaktivität während des isometrischen maximalen willkürlichen Drehmoments (RMS-EMG$_{iMVT}$/M$_{max}$) bei 80° und 45° Knieflexion vor und nach einem plyometrischen Training. INT: Interventionsgruppe, KON: Kontrollgruppe. Die Daten sind als Mittelwerte mit Standardabweichung dargestellt. * zeigt einen signifikanten Unterschied zwischen den Gruppen an (* P ≤ 0,05).

Die Ergebnisse zeigen eine Erhöhung der mechanischen und neuronalen Parameter bei MVC für die Knieflexion von 80° aber nicht für die Knieflexion von 45° (Abbildung 11). Diese Resultate weisen auf eine winkelspezifische Trainingsadaptation der neuromuskulären Funktion des M. quadriceps femoris hin.

Nach dem Training war das iMVT der Interventionsgruppe signifikant höher als das der Kontrollgruppe. Dieser Befund deckt sich mit den Ergebnissen anderer Studien, die ebenfalls eine Erhöhung der maximalen willkürlichen Kraft nach einem plyometrischen Training nachweisen konnten [91, 127, 189, 207]. Die Steigerung des iMVT in der aktuellen Studie kann auf einen erhöhten efferenten Output der α-Motoneuronen zurückgeführt werden. Diese Annahme wird durch die Ergebnisse beider Messverfahren zur Abschätzung der willkürlichen

Aktivierung gestützt. Die kontraktilen Eigenschaften zeigten keinen trainingsbedingten Gruppenunterschied. Auf Grundlage dieser Resultate kann davon ausgegangen werden, dass die applizierte Trainingsmaßnahme primär neuronale Anpassungen provoziert hat. Zu ähnlichen Ergebnissen kamen Kyrolainen et al. [127], die eine Erhöhung der Muskelaktivierung der Plantarflexoren infolge eines plyometrischen Trainings nachweisen konnten, während keine muskulären Veränderungen detektiert wurden. Darüber hinaus existieren Studien, die neuronale und hypertrophische Veränderungen nach einem Sprungtraining zeigen konnten [121, 133]. Generell wird allerdings angenommen, dass primär neuronale Anpassungen für den Anstieg der willkürlichen maximalen Kraft in den ersten Wochen eines Krafttrainings verantwortlich sind [60, 91, 154]. Aufgrund der Winkelspezifik der Anpassungen in der vorliegenden Studie kann primär von neuronalen Adaptationen ausgegangen werden [116]. Die Ergebnisse einer Studie von Thepaut-Mathieu et al. [216] weisen darauf hin, dass neuronale Anpassungen lediglich die Gelenkwinkel und damit Muskellängen betreffen, die im Training Verwendung fanden. Damit geht einher, dass eine Kraftsteigerung ebenfalls nur in den spezifischen Gelenkwinkeln zu beobachten ist. Dagegen scheinen sich hypertrophische Adaptationen auf die maximale willkürliche Kraft in allen Gelenkwinkeln auszuwirken [116, 191].
Voigt et al. [226] fanden bei ihren Probanden erhöhte Dehnungsreflexe und H-Reflexe infolge eines vierwöchigen plyometrischen Trainings. Die Autoren gingen davon aus, dass eine erhöhte Muskelspindelsensitivität sowie eine reduzierte präsynaptische Inhibition der Ia Afferenzen zu den Ergebnissen beitrugen. In der aktuellen Studie bestand das Training größtenteils aus Sprungübungen, die zwischen ~80° und 0° Knieflexion (0° = volle Extension) durchgeführt wurden. Dabei entsprachen 80° Knieflexion dem Umkehrpunkt beim Countermovement Jump und der Startposition beim Squat Jump. Demnach waren die Gewichtsbelastung und damit der Anspruch an die Kraftleistung im besagten Kniewinkel am höchsten. Zudem sind die Anteile des M. quadriceps femoris und damit auch die Muskelspindeln bei 80° Knieflexion länger. Das kann zu einer gesteigerten Erregung des M. quadriceps femoris Motoneuronenpools über Ia Afferenzen führen, die bei einer Knieflexion von 45° nicht erfolgt [27, 122]. Demnach ist es möglich, dass das plyometrische Training die Spindelsensitivität ausschließlich bei 80° Knieflexion erhöht hat und damit zur Steigerung des Ia-afferenten Inputs beitrug. Dieser könnte wiederum zur Steigerung des gesamten exzitatorischen Outputs im Sinne einer gesteigerten willkürlichen Aktivierung während MVC beigetragen haben.

3.2 EINFLUSS DES ALTERNS AUF DIE NEUROMUSKULÄRE FUNKTION

3.2.1 Darstellung des Forschungsdefizits

Im Verlauf des Alterungsprozesses kommt es zu einer Reduktion der Leistungsfähigkeit des neuromuskulären Systems. Die Abnahme der neuromuskulären Funktion des gesunden alternden Menschen ist durch strukturelle und funktionelle Veränderungen bedingt.

Die strukturellen Veränderungen beinhalten u. a. eine Abnahme des Volumens von Neuronen im Lobus frontalis [93, 190] und in der Substantia alba des Cerebrums [138]. Darüber hinaus spielen Modulationen im peripheren Nervensystem eine Rolle. Dazu gehören die Reduktion der Anzahl und des Durchmessers myelinisierter Axone [151] und die Abnahme der axonalen Leitungsgeschwindigkeit [147] aufgrund verminderter Myelinisierung und veränderter Internodiumlängen [3]. Es kommt des Weiteren zu einem zunehmenden Verlust von α-Motoneuronen, induziert durch Apoptose, die Reduktion der Aktivität von insulinähnlichen Wachstumsfaktoren (insulin-like growth factor 1, IGF-1), die erhöhte Konzentration spezifischer Zytokine (tumor necrosis factor (TNF)-α, TNF-β, Interleukin (IL)-6) und oxidativen Stress [3]. Außerdem kann eine altersbedingte Abnahme der Muskelfaseranzahl und Atrophie von Muskelfasern konstatiert werden. Dabei sind die Typ II Muskelfasern stärker betroffen als die Typ I Muskelfasern [68, 117, 129]. Als primäre Ursache dafür wird eine reduzierte myofibrilläre Proteinsynthese im Alter diskutiert [18]. Zudem weisen ältere Individuen eine sogenannte anabole Resistenz auf [180], die durch eine reduzierte Fähigkeit zur Erhöhung der Muskelproteinsynthese charakterisiert ist. Demnach reagieren jüngere und ältere Menschen unterschiedlich auf anabole Stimuli, wie z. B. Nahrungsaufnahme und physische Aktivität [53, 180, 229]. Des Weiteren wurde die Reduktion der kontraktilen Kinetik [54] sowie die Modifikation der Muskelarchitektur [157] beim älteren Menschen nachgewiesen. Im Hinblick auf die Veränderung der Muskelarchitektur sind primär zwei Faktoren relevant: (I) die Reduktion der Muskelfaszikellänge und (II) die Abnahme des Fiederungswinkels der Muskulatur [120, 157] bedingt durch die Abnahme der Sarkomere in Serie. Dadurch werden die Längen-Spannungs-Relation, Kraft-Geschwindigkeits-Relation und Leistungs-Geschwindigkeits-Relation des relevanten Muskels negativ beeinflusst [157].

Die altersbedingten funktionellen Veränderungen inkludieren u. a. die Modulation der Aktivität des zerebralen Cortexes während differenter Aufgaben, die mittels funktioneller Magnetresonanztomographie [96, 181] und transkranieller Magnetstimulation [193] nachgewiesen wurden. Zudem konnte bei älteren Menschen eine erhöhte Koaktivierung antagonistischer Muskeln festgestellt werden [23, 36], die zur Reduktion der Kraftproduktion der agonistischen Muskeln beiträgt. Die Ursache liegt in der Erhöhung der zu überwindenden Kraft für den Agonisten. Außerdem kann eine erhöhte Koaktivierung zur Abnahme der agonistischen Muskelaktivität führen, indem die reziproke Inhibition des Agonisten zunimmt. Des Weiteren deuten H-Reflex Studien darauf hin, dass die spinalen α-Motoneuronen im Altersgang eine reduzierte Erregbarkeit aufweisen [64, 221] und es zu einer Modifikation von Hemmungsmechanismen, wie z. B. der präsynaptischen Inhibition von Ia Afferenzen [63] und der reziproken Inhibition [114], bei spezifischen Bewegungsaufgaben kommt.

Die genannten und weitere Faktoren sind mitverantwortlich für die Abnahme der Muskelkraft mit zunehmendem Alter, die in der internationalen Literatur als „Dynapenia“ charakterisiert wird [49, 134]. Die verminderte mechanische Muskelperformance, die eine reduzierte willkürliche maximale und explosive Kraft sowie Leistung im höheren Alter beinhaltet, impliziert eine Verschlechterung der funktionellen Kapazität während der Ausübung von Alltagstätigkeiten. Zu diesen Tätigkeiten gehören z. B. das Gehen, Treppensteigen und Aufstehen von einem Stuhl [3]. Demnach ist die altersbedingte Abnahme der maximalen und explosiven Kraftproduktion [203] für die Sturzinzidenz und Mobilität im Alltag von enormer Relevanz. Es konnte gezeigt werden, dass die Kraft der unteren Extremitäten mit der Häufigkeit von Sturzereignissen und der Fähigkeit auf Perturbationen der posturalen Kontrolle adäquat reagieren zu können im Zusammenhang steht [168, 169]. Darüber hinaus wurden signifikante Korrelationen zwischen der Explosivkraft eines Individuums und der Ganggeschwindigkeit sowie der Zeit zum Absolvieren einer definierten Anzahl von Treppenstufen gefunden [25]. Izquierdo et al. [102] wiesen nach, dass die Fähigkeit der Beinextensoren zur schnellen Kraftproduktion mit der posturalen Kontrolle zusammen hängt.

Aufgrund der Relevanz der maximalen und explosiven Kraft für die Mobilität im Alter ist die Aufdeckung der Ursachen für die Abnahme dieser Kraftfähigkeiten von Bedeutung. Obwohl die altersbedingte Abnahme der Maximal- und Explosivkraft des M. quadriceps femoris mehrfach nachgewiesen wurde [102, 103, 170], sind die zugrundeliegenden neuromuskulären Mechanismen ungenügend untersucht. Darüber hinaus sind die Ergebnisse der existierenden Studien zu dieser Thematik partiell widersprüchlich. So konnten Roos et al. [183] und Wilder et al. [228] keine altersbedingte Veränderung der willkürlichen Aktivierung des M. quadriceps femoris während isometrischer maximaler willkürlicher Kontraktionen (maximum voluntary contraction [MVC]) feststellen, während Stevens et al. [211] und Stackhouse et al. [208] ein altersbedingtes Aktivierungsdefizit der Knieextensoren während isometrischer MVC bei gesunden Männern und Frauen aufdecken konnten.

Die unterschiedlichen Ergebnisse sind zum Teil auf methodische Defizite zurückzuführen. Die folgende durchgeführte Studie sollte in dieser Hinsicht zur Erweiterung des Wissens beitragen.

EXPERIMENT IV: Mau-Moeller, A.*, **Behrens, M.***, Lindner, T., Bader, R., Bruhn, S. (2013). Age-related changes in neuromuscular function of the quadriceps muscle in physically active adults. *Journal of Electromyography and Kinesiology, 23* (3), 640-648. (* authors contributed equally to this work)

3.2.2 Fragestellung

Diese Studie untersuchte den Abfall der Leistungsfähigkeit des neuromuskulären Systems infolge des Alterungsprozesses. Dazu wurde der Beitrag altersbedingter neuronaler und muskulärer Veränderungen zur Reduktion des iMVT und der RTD analysiert. Zudem wurde der H-Reflex des M. vastus medialis zwischen älteren und jüngeren Probanden verglichen. Es wurde angenommen, dass die Reduktion neuronaler sowie muskulärer Parameter für die Abnahme der maximalen willkürlichen Kraft im Altersgang verantwortlich ist.

3.2.3 Methoden

Für die Untersuchung der Modulationen innerhalb des neuromuskulären Systems, ausgelöst durch den Alterungsprozess, wurde auf das gleiche Methodenrepertoire zurückgegriffen, das in Kapitel 2.2.3 beschrieben ist. Dieses beinhaltete jeweils Kraftmessungen und neurophysiologische Techniken. Eine detaillierte Beschreibung der Personenstichprobe, experimentellen Prozedur, Datenaufnahme und -analyse sowie statistischen Analyse für das Experiment ist in der Publikation zu finden.

Zu den Messzeitpunkten wurden die Probanden auf einem Dynamometer fixiert, um die Drehmomente zu messen, die durch den M. quadriceps femoris produziert wurden.

Für die Messung der Muskelaktivität kamen Oberflächenelektroden zum Einsatz. Die Elektroden wurden in einer bipolaren Konfiguration auf dem M. rectus femoris, dem M. vastus medialis und dem M. vastus lateralis appliziert. Die Referenzelektrode wurde auf der ipsilateralen Patella platziert.

Die Analyse der Modulationen auf den unterschiedlichen Ebenen des neuromuskulären Systems wurde mithilfe der transkutanen elektrischen Stimulation des N. femoralis realisiert. Durch die Applikation von elektrischen Stimuli in Ruhebedingung und während der MVCs konnten unterschiedliche Ebenen des neuromuskulären Systems und deren Modulation näher untersucht werden. Dazu gehörte die Abschätzung der kontraktilen Eigenschaften des M. quadriceps femoris in Ruhebedingung, der Erregbarkeit der spinalen α-Motoneuronen des M. vastus medialis mittels der Exzitation der Ia Afferenzen und der willkürlichen Aktivierung des M. quadriceps femoris während MVC.

3.2.4 Ergebnisse und Diskussion

In diesem Kapitel werden die wichtigsten Ergebnisse des Experiments vorgestellt und anschließend kurz diskutiert. Die komplette Darstellung der Resultate der Studie ist in der angehängten Publikation zu finden.

Das durchgeführte Experiment untersuchte den Abfall der Leistungsfähigkeit des neuromuskulären Systems infolge des Alterungsprozesses. Innerhalb einer Querschnittstudie wurde der Beitrag altersbedingter neuronaler und muskulärer Veränderungen zur Reduktion des iMVT und der RTD analysiert. Zudem wurde die Exzitabilität des α-Motoneuronenpools des M. vastus medialis über Ia Afferenzen zwischen älteren und jüngeren Probanden verglichen.

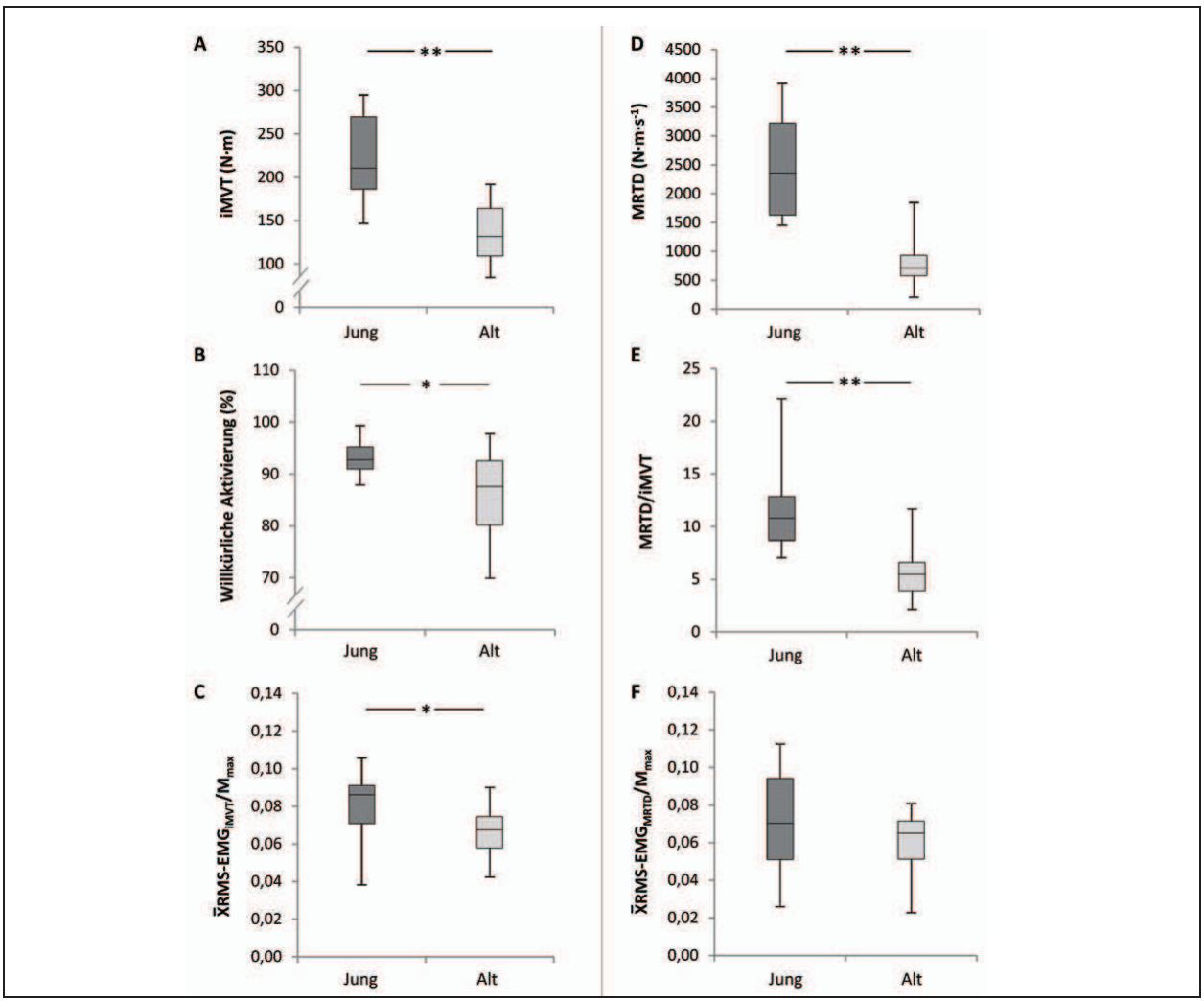

Abb. 12 Einfluss des Alterns auf das maximale und explosive willkürliche Drehmoment und die Muskelaktivierung des M. quadriceps femoris.
A Isometrisches maximales willkürliches Drehmoment (iMVT), **B** willkürliche Aktivierung, **C** normalisierte Muskelaktivität während des isometrischen maximalen willkürlichen Drehmoments ($\bar{x}$RMS-EMG$_{iMVT}$/M$_{max}$), **D** maximal rate of torque development der willkürlich produzierten Drehmoment-Zeit-Kurve (MRTD), **E** maximal rate of torque development der willkürlich produzierten Drehmoment-Zeit-Kurve normalisiert zum isometrischen maximalen willkürlichen Drehmoment (MRTD/iMVT), **F** normalisierte Muskelaktivität während der maximal rate of torque development der willkürlich produzierten Drehmoment-Zeit-Kurve ($\bar{x}$RMS-EMG$_{MRTD}$/M$_{max}$). * zeigt einen signifikanten Unterschied zwischen den Gruppen an (* P ≤ 0,05; ** P ≤ 0,01).

Das iMVT der älteren Probanden war im Vergleich zum iMVT der jüngeren Probanden signifikant reduziert. Dem Unterschied in der Mechanik lag eine differente willkürliche Aktivierung zugrunde. Demnach war die Muskelaktivierung der älteren Männer und Frauen signifikant geringer als die der jüngeren. Dieser Befund konnte durch die Interpolated Twitch Technik sowie den normalisierten RMS-EMG abgesichert werden (Abbildung 12). Darüber hinaus wiesen die älteren Probanden eine signifikant geringere maximale RTD auf. Weitere Unterschiede konnten im Hinblick auf die kontraktilen Eigenschaften des M. quadriceps femoris und die fettfreie Masse des untersuchten Beines gefunden werden.
Die Reduktion der maximalen willkürlichen Kraft der Knieextensoren im Altersgang ist gut belegt [48, 134] und die Ergebnisse der vorliegenden Studie bestätigen diesen Sachverhalt. Die Resultate des Experiments weisen auf eine reduzierte willkürliche Aktivierung des M.

quadriceps femoris bei den älteren Probanden hin. Demnach scheint ein reduzierter efferenter Output der α-Motoneuronen mitverantwortlich für die Abnahme des iMVT mit zunehmendem Alter zu sein. Es existieren Studien, die ein Aktivierungsdefizit bei älteren Menschen nachweisen konnten [208, 211], während andere Experimente keine altersbedingte Abnahme der willkürlichen Aktivierung fanden [39, 183, 228]. Die widersprüchlichen Ergebnisse sind vermutlich auf unterschiedliche elektrische Stimulationsmethoden und heterogene Probandenpopulationen zurückzuführen. Ein Aktivierungsdefizit wurde zumeist dann festgestellt, wenn die „Central Activation Ratio“ zur Abschätzung der willkürlichen Aktivierung verwendet wurde [208, 211]. Wenn die Interpolated Twitch Technik zur Analyse der Muskelaktivierung herangezogen wurde, konnte größtenteils kein Unterschied in der Aktivierung der Knieextensoren bei jüngeren und älteren Erwachsenen festgestellt werden [39, 183, 228]. In der vorliegenden Studie wurden zwei Ansätze zur Messung der willkürlichen Aktivierung des M. quadriceps femoris verwendet. Zum einen erfolgte die Abschätzung der Muskelaktivierung über die Berechnung des normalisierten RMS-EMG und zum anderen mittels der Interpolated Twitch Technik. Zudem wurde für die Berechnung der willkürlichen Aktivierung, die mittels der Interpolated Twitch Technik erhoben wurde, eine korrigierte Formel verwendet. Diese Formel berücksichtigt das Drehmomentniveau vor der Applikation der elektrischen Stimuli, welches aufgrund der Fluktuation der Drehmoment-Zeit-Kurve unter dem iMVT liegen kann.

In der vorliegenden Studie wurden eine schlechtere Kontraktilität der Knieextensoren sowie eine reduzierte Muskelmasse des untersuchten Beines bei den älteren Probanden gefunden. Diese Befunde entsprechen den Ergebnissen anderer Studien, die ebenfalls eine altersbedingte Veränderung der kontraktilen Eigenschaften nachweisen konnten [42, 183, 228]. Im Hinblick auf die Muskelmasse korrespondieren die Ergebnisse des vorliegenden Experiments ebenfalls mit der Datenlage anderer Untersuchungen [235, 236]. Als Ursache kann die altersbedingte Abnahme der Muskelfaseranzahl und Atrophie von Muskelfasern ausgemacht werden. Diese Prozesse wirken sich stärker auf die Typ II Muskelfasern als auf die Typ I Muskelfasern aus [68, 117, 129]. Als primäre Ursache dafür wird eine reduzierte myofibrilläre Proteinsynthese im Alter diskutiert [18]. Darüber hinaus wurde eine anabole Resistenz älterer Personen nachgewiesen [180], die durch eine reduzierte Fähigkeit zur Erhöhung der Muskelproteinsynthese charakterisiert ist. Demzufolge reagieren jüngere und ältere Menschen unterschiedlich auf anabole Stimuli, wie z. B. Nahrungsaufnahme und physische Aktivität [53, 180, 229].

4. ZUSAMMENFASSUNG

Die Ergebnisse der durchgeführten Studien haben zur Erweiterung des Wissens über akute und chronische Anpassungen der neuromuskulären Funktion beigetragen. Es konnte gezeigt werden, dass physische Aktivität sowie der Alterungsprozess, der durch einen generellen Leistungsverlust aber auch die Zunahme von physischer Inaktivität gekennzeichnet ist, die neuromuskuläre Funktion der Oberschenkelmuskeln modulieren kann.

In den Experimenten I und II wurden spezifische akute Anpassungen untersucht und die Auswirkungen von unterschiedlich belastenden Ermüdungsinterventionen auf die neuromuskuläre Funktion der Oberschenkelmuskeln analysiert. Es wurde festgestellt, dass die Leistungsfähigkeit des neuromuskulären Systems ermüdungsbedingt abnimmt und sich spezifische akute Adaptationen einstellen. So zeigte Experiment I „Effect of fatigue on hamstring reflex responses and posterior-anterior tibial translation in men and women", dass die spezifische Ermüdungsintervention unterschiedliche Auswirkungen auf die anteriore Tibiatranslation und die Reflexantworten des M. biceps femoris und des M. semitendinosus/semimembranosus der Männer und Frauen hatte. Auf Grundlage der Ergebnisse ist es vorstellbar, dass eine reduzierte Reflexaktivierung der ischiocruralen Muskulatur mit einer korrespondierenden Erhöhung der anterioren Tibiatranslation zur erhöhten Inzidenz für ACL-Rupturen bei Frauen beitragen könnte. In Experiment II „Effect of exercise-induced muscle damage on neuromuscular function of the quadriceps muscle" wurde eine überbelastende Ermüdungsintervention durchgeführt, die zur längerfristigen Modulation der neuromuskulären Funktion führte. Die Studie zeigte, dass die Abnahme der willkürlichen Aktivierung und der kontraktilen Funktion des M. quadriceps femoris zum Kraftverlust unmittelbar nach der Intervention, die eine Muskelverletzung induziert hat, beitrug. Im darauf folgenden Zeitraum schienen die beeinträchtigten kontraktilen Eigenschaften der Knieextensoren hauptverantwortlich für die Reduktion des iMVT zu sein. Die Studie zeigte, dass es keine Korrespondenz zwischen den neurophysiologischen Parametern und dem Schmerzverlauf gibt.

In den Experimenten III und IV wurden spezifische chronische Anpassungen der neuromuskulären Funktion untersucht. Experiment III beschäftigte sich mit der Modulation der neuromuskulären Funktion des M. quadriceps femoris durch gezielte repetitive physische Aktivität, während Experiment IV den Einfluss des Alterns und zunehmender physischer Inaktivität auf die neuromuskuläre Funktion des M. quadriceps femoris untersuchte. In der Publikation „Effect of plyometric training on neural and mechanical properties of the knee extensor muscles", die aus Experiment III hervorging, wurde dokumentiert, dass das plyometrische Training primär neuronale Anpassungen provozierte, die wiederum zur Steigerung des iMVT des M. quadriceps femoris beitrugen. Darüber hinaus konnten winkelspezifische neuronale Adaptationen nachgewiesen werden, die bis dato lediglich nach isometrischen Trainingsregimes beobachtet wurden [116, 216]. Das Experiment IV „Age-related changes in neuromuscular function of the quadriceps muscle in physically active adults" brachte neue Erkenntnisse im Hinblick auf die Kontribution des Zentralnervensystems zur reduzierten Kraft der Knieextensoren im Alter. Die Abschätzung der willkürlichen Aktivierung des M.

quadriceps femoris mithilfe von zwei differenten Verfahren zeigte eine altersbedingte Reduktion. Demnach tragen scheinbar Modulationen auf neuronaler und muskulärer Ebene zur Abnahme der maximalen Kraft im Altersgang bei.

5. LITERATUR

1. Aagaard, P., Andersen, J.L., Dyhre-Poulsen, P., Leffers, A.M., Wagner, A., Magnusson, S.P., Halkjaer-Kristensen, J. & Simonsen, E.B. (2001). A mechanism for increased contractile strength of human pennate muscle in response to strength training: changes in muscle architecture. *J Physiol, 534*(Pt. 2), 613-623.
2. Aagaard, P., Simonsen, E.B., Andersen, J.L., Magnusson, P. & Dyhre-Poulsen, P. (2002). Neural adaptation to resistance training: changes in evoked V-wave and H-reflex responses. *J Appl Physiol, 92*(6), 2309-2318.
3. Aagaard, P., Suetta, C., Caserotti, P., Magnusson, S.P. & Kjaer, M. (2010). Role of the nervous system in sarcopenia and muscle atrophy with aging: strength training as a countermeasure. *Scand J Med Sci Sports, 20*(1), 49-64.
4. Abe, T., DeHoyos, D.V., Pollock, M.L. & Garzarella, L. (2000). Time course for strength and muscle thickness changes following upper and lower body resistance training in men and women. *Eur J Appl Physiol, 81*(3), 174-180.
5. Agre, J.C. (1985). Hamstring injuries. Proposed aetiological factors, prevention, and treatment. *Sports Med, 2*(1), 21-33.
6. Alentorn-Geli, E., Myer, G.D., Silvers, H.J., Samitier, G., Romero, D., Lazaro-Haro, C. & Cugat, R. (2009). Prevention of non-contact anterior cruciate ligament injuries in soccer players. Part 1: Mechanisms of injury and underlying risk factors. *Knee Surg Sports Traumatol Arthrosc*
7. Allen, D.G., Lamb, G.D. & Westerblad, H. (2008). Skeletal muscle fatigue: cellular mechanisms. *Physiol Rev, 88*(1), 287-332.
8. Allen, G.M., Gandevia, S.C. & McKenzie, D.K. (1995). Reliability of measurements of muscle strength and voluntary activation using twitch interpolation. *Muscle Nerve, 18*(6), 593-600.
9. Allison, S.J., Folland, J.P., Rennie, W.J., Summers, G.D. & Brooke-Wavell, K. (2013). High impact exercise increased femoral neck bone mineral density in older men: a randomised unilateral intervention. *Bone, 53*(2), 321-328.
10. Ament, W. & Verkerke, G.J. (2009). Exercise and fatigue. *Sports Med, 39*(5), 389-422.
11. Andersen, J.L. & Aagaard, P. (2010). Effects of strength training on muscle fiber types and size; consequences for athletes training for high-intensity sport. *Scand J Med Sci Sports, 20 Suppl 2*, 32-38.
12. Asp, S., Daugaard, J.R., Kristiansen, S., Kiens, B. & Richter, E.A. (1998). Exercise metabolism in human skeletal muscle exposed to prior eccentric exercise. *J Physiol, 509 (Pt 1)*, 305-313.
13. Aune, A.K., Cawley, P.W. & Ekeland, A. (1997). Quadriceps muscle contraction protects the anterior cruciate ligament during anterior tibial translation. *Am J Sports Med, 25*(2), 187-190.
14. Avela, J., Finni, J. & Komi, P.V. (2006). Excitability of the soleus reflex arc during intensive stretch-shortening cycle exercise in two power-trained athlete groups. *Eur J Appl Physiol, 97*(4), 486-493.

15. Avela, J., Kyrolainen, H., Komi, P.V. & Rama, D. (1999). Reduced reflex sensitivity persists several days after long-lasting stretch-shortening cycle exercise. *J Appl Physiol, 86*(4), 1292-1300.
16. Baker, J., Frankel, V.H. & Burnstein, A. (1972). Fatigue fractures: Biomechanical concideration. *J Bone Joint Surg Am, 54*(6), 1345-1346.
17. Bakhtiary, A.H., Safavi-Farokhi, Z. & Aminian-Far, A. (2007). Influence of vibration on delayed onset of muscle soreness following eccentric exercise. *Br J Sports Med, 41*(3), 145-148.
18. Balagopal, P., Rooyackers, O.E., Adey, D.B., Ades, P.A. & Nair, K.S. (1997). Effects of aging on in vivo synthesis of skeletal muscle myosin heavy-chain and sarcoplasmic protein in humans. *Am J Physiol, 273*(4 Pt 1), E790-800.
19. Balestra, C., Duchateau, J. & Hainaut, K. (1992). Effects of fatigue on the stretch reflex in a human muscle. *Electroencephalogr Clin Neurophysiol, 85*(1), 46-52.
20. Balnave, C.D. & Allen, D.G. (1995). Intracellular calcium and force in single mouse muscle fibres following repeated contractions with stretch. *J Physiol, 488*, 25-36.
21. Balnave, C.D., Davey, D.F. & Allen, D.G. (1997). Distribution of sarcomere length and intracellular calcium in mouse skeletal muscle following stretch-induced injury. *J Physiol, 502*, 649-659.
22. Bamman, M.M., Petrella, J.K., Kim, J.S., Mayhew, D.L. & Cross, J.M. (2007). Cluster analysis tests the importance of myogenic gene expression during myofiber hypertrophy in humans. *J Appl Physiol, 102*(6), 2232-2239.
23. Barry, B.K. & Carson, R.G. (2004). The consequences of resistance training for movement control in older adults. *J Gerontol A Biol Sci Med Sci, 59*(7), 730-754.
24. Barry, B.K. & Enoka, R.M. (2007). The neurobiology of muscle fatigue: 15 years later. *Integr Comp Biol, 47*(4), 465-473.
25. Bean, J.F., Kiely, D.K., Herman, S., Leveille, S.G., Mizer, K., Frontera, W.R. & Fielding, R.A. (2002). The relationship between leg power and physical performance in mobility-limited older people. *J Am Geriatr Soc, 50*(3), 461-467.
26. Beard, D.J., Kyberd, P.J., Fergusson, C.M. & Dodd, C.A. (1993). Proprioception after rupture of the anterior cruciate ligament. An objective indication of the need for surgery? *J Bone Joint Surg Br, 75*(2), 311-315.
27. Becker, R. & Awiszus, F. (2001). Physiological alterations of maximal voluntary quadriceps activation by changes of knee joint angle. *Muscle Nerve, 24*(5), 667-672.
28. Bickel, C.S., Slade, J., Mahoney, E., Haddad, F., Dudley, G.A. & Adams, G.R. (2005). Time course of molecular responses of human skeletal muscle to acute bouts of resistance exercise. *J Appl Physiol, 98*(2), 482-488.
29. Bongiovanni, L.G. & Hagbarth, K.E. (1990). Tonic vibration reflexes elicited during fatigue from maximal voluntary contractions in man. *J Physiol, 423*, 1-14.
30. Borotikar, B.S., Newcomer, R., Koppes, R. & McLean, S.G. (2008). Combined effects of fatigue and decision making on female lower limb landing postures: central and peripheral contributions to ACL injury risk. *Clin Biomech (Bristol, Avon), 23*(1), 81-92.

31. Brown, M.E., Mayhew, J.L. & Boleach, L.W. (1986). Effect of plyometric training on vertical jump performance in high school basketball players. *J Sports Med Phys Fitness, 26*(1), 1-4.
32. Bruhn, S., *Functional knee stability*. 1999, University of Stuttgart: Stuttgart. p. 149 p.
33. Bruhn, S. & Gollhofer, A. (2002). Evaluation of mechanical and neurophysiological effects of wearing bandages for the knee joint in functional testing situations. *Sportverletz Sportschaden, 16*(1), 15-21.
34. Bruhn, S., Leukel, C. & Gollhofer, A. (2011). Differential effects of stimulus characteristics during knee joint perturbation on hamstring and quadriceps reflex responses. *Hum Mov Sci, 30*(6), 1079-1091.
35. Bulbulian, R. & Bowles, D.K. (1992). Effect of downhill running on motoneuron pool excitability. *J Appl Physiol, 73*(3), 968-973.
36. Burnett, R.A., Laidlaw, D.H. & Enoka, R.M. (2000). Coactivation of the antagonist muscle does not covary with steadiness in old adults. *J Appl Physiol, 89*(1), 61-71.
37. Burr, D.B. (1997). Bone, exercise, and stress fractures. *Exerc Sport Sci Rev, 25*, 171-194.
38. Byrne, C., Twist, C. & Eston, R. (2004). Neuromuscular function after exercise-induced muscle damage: theoretical and applied implications. *Sports Med, 34*(1), 49-69.
39. Callahan, D.M., Foulis, S.A. & Kent-Braun, J.A. (2009). Age-related fatigue resistance in the knee extensor muscles is specific to contraction mode. *Muscle Nerve, 39*(5), 692-702.
40. Campos, G.E., Luecke, T.J., Wendeln, H.K., Toma, K., Hagerman, F.C., Murray, T.F., Ragg, K.E., Ratamess, N.A., Kraemer, W.J. & Staron, R.S. (2002). Muscular adaptations in response to three different resistance-training regimens: specificity of repetition maximum training zones. *Eur J Appl Physiol, 88*(1-2), 50-60.
41. Canale, S.T., Cantler, E.D., Jr., Sisk, T.D. & Freeman, B.L., 3rd. (1981). A chronicle of injuries of an American intercollegiate football team. *Am J Sports Med, 9*(6), 384-389.
42. Cannon, J., Kay, D., Tarpenning, K.M. & Marino, F.E. (2006). Normalized lengthening peak torque is associated with temporal twitch characteristics in elderly women but not young women. *Acta Physiol (Oxf), 188*(1), 53-62.
43. Carolan, B. & Cafarelli, E. (1992). Adaptations in coactivation after isometric resistance training. *J Appl Physiol, 73*(3), 911-917.
44. Carroll, T.J., Riek, S. & Carson, R.G. (2002). The sites of neural adaptation induced by resistance training in humans. *J Physiol, 544*(Pt 2), 641-652.
45. Chappell, J.D., Herman, D.C., Knight, B.S., Kirkendall, D.T., Garrett, W.E. & Yu, B. (2005). Effect of fatigue on knee kinetics and kinematics in stop-jump tasks. *Am J Sports Med, 33*(7), 1022-1029.
46. Chappell, J.D., Yu, B., Kirkendall, D.T. & Garrett, W.E. (2002). A comparison of knee kinetics between male and female recreational athletes in stop-jump tasks. *Am J Sports Med, 30*(2), 261-267.

47. Christina, K.A., White, S.C. & Gilchrist, L.A. (2001). Effect of localized muscle fatigue on vertical ground reaction forces and ankle joint motion during running. *Hum Mov Sci, 20*(3), 257-276.
48. Clark, B.C. & Manini, T.M. (2012). What is dynapenia? *Nutrition, 28*(5), 495-503.
49. Clark, B.C. & Taylor, J.L. (2011). Age-related changes in motor cortical properties and voluntary activation of skeletal muscle. *Curr Aging Sci, 4*(3), 192-199.
50. Clarkson, P.M. & Hubal, M.J. (2002). Exercise-induced muscle damage in humans. *Am J Phys Med Rehabil, 81*(11 Suppl), S52-69.
51. Clarkson, P.M., Nosaka, K. & Braun, B. (1992). Muscle function after exercise-induced muscle damage and rapid adaptation. *Med Sci Sports Exerc, 24*(5), 512-520.
52. Coffey, V.G. & Hawley, J.A. (2007). The molecular bases of training adaptation. *Sports Med, 37*(9), 737-763.
53. Cuthbertson, D., Smith, K., Babraj, J., Leese, G., Waddell, T., Atherton, P., Wackerhage, H., Taylor, P.M. & Rennie, M.J. (2005). Anabolic signaling deficits underlie amino acid resistance of wasting, aging muscle. *Faseb J, 19*(3), 422-424.
54. D'Antona, G., Pellegrino, M.A., Adami, R., Rossi, R., Carlizzi, C.N., Canepari, M., Saltin, B. & Bottinelli, R. (2003). The effect of ageing and immobilization on structure and function of human skeletal muscle fibres. *J Physiol, 552*(Pt 2), 499-511.
55. Del Balso, C. & Cafarelli, E. (2007). Adaptations in the activation of human skeletal muscle induced by short-term isometric resistance training. *J Appl Physiol, 103*(1), 402-411.
56. Deschenes, M.R., Brewer, R.E., Bush, J.A., McCoy, R.W., Volek, J.S. & Kraemer, W.J. (2000). Neuromuscular disturbance outlasts other symptoms of exercise-induced muscle damage. *J Neurol Sci, 174*(2), 92-99.
57. Dornan, P. (1971). A report on 140 hamstring injuries. *Austral J Sports Med, 4*, 30-36.
58. Duchateau, J., Balestra, C., Carpentier, A. & Hainaut, K. (2002). Reflex regulation during sustained and intermittent submaximal contractions in humans. *J Physiol, 541*(Pt 3), 959-967.
59. Duchateau, J. & Hainaut, K. (1993). Behaviour of short and long latency reflexes in fatigued human muscles. *J Physiol, 471*, 787-799.
60. Duchateau, J., Semmler, J.G. & Enoka, R.M. (2006). Training adaptations in the behavior of human motor units. *J Appl Physiol, 101*(6), 1766-1775.
61. Duclay, J., Martin, A., Robbe, A. & Pousson, M. (2008). Spinal reflex plasticity during maximal dynamic contractions after eccentric training. *Med Sci Sports Exerc, 40*(4), 722-734.
62. Dugan, S.A. & Frontera, W.R. (2000). Muscle fatigue and muscle injury. *Phys Med Rehabil Clin N Am, 11*(2), 385-403.
63. Earles, D., Vardaxis, V. & Koceja, D. (2001). Regulation of motor output between young and elderly subjects. *Clin Neurophysiol, 112*(7), 1273-1279.
64. Earles, D.R., Koceja, D.M. & Shively, C.W. (2000). Environmental changes in soleus H-reflex excitability in young and elderly subjects. *Int J Neurosci, 105*(1-4), 1-13.

65. Eichner, E.R. (1995). Overtraining: consequences and prevention. *J Sports Sci, 13 Spec No*, S41-48.
66. Enoka, R.M. (2008). *Neuromechanics of Human Movement.* Champaign: Human Kinetics.
67. Fagenbaum, R. & Darling, W.G. (2003). Jump landing strategies in male and female college athletes and the implications of such strategies for anterior cruciate ligament injury. *Am J Sports Med, 31*(2), 233-240.
68. Faulkner, J.A., Larkin, L.M., Claflin, D.R. & Brooks, S.V. (2007). Age-related changes in the structure and function of skeletal muscles. *Clin Exp Pharmacol Physiol, 34*(11), 1091-1096.
69. Fimland, M.S., Helgerud, J., Gruber, M., Leivseth, G. & Hoff, J. (2009). Functional maximal strength training induces neural transfer to single-joint tasks. *Eur J Appl Physiol, 107*(1), 21-29.
70. Folland, J.P. & Williams, A.G. (2007). The adaptations to strength training : morphological and neurological contributions to increased strength. *Sports Med, 37*(2), 145-168.
71. Friden, J. & Lieber, R.L. (2001). Eccentric exercise-induced injuries to contractile and cytoskeletal muscle fibre components. *Acta Physiol Scand, 171*(3), 321-326.
72. Friden, J., Sjostrom, M. & Ekblom, B. (1983). Myofibrillar damage following intense eccentric exercise in man. *Int J Sports Med, 4*(3), 170-176.
73. Friemert, B., Bumann-Melnyk, M., Faist, M., Schwarz, W., Gerngross, H. & Claes, L. (2005). Differentiation of hamstring short latency versus medium latency responses after tibia translation. *Exp Brain Res, 160*(1), 1-9.
74. Friemert, B., Franke, S., Gollhofer, A., Claes, L. & Faist, M. (2010). Group I afferent pathway contributes to functional knee stability. *J Neurophysiol, 103*(2), 616-622.
75. Gandevia, S.C. (2001). Spinal and supraspinal factors in human muscle fatigue. *Physiol Rev, 81*(4), 1725-1789.
76. Garfinkel, S. & Cafarelli, E. (1992). Relative changes in maximal force, EMG, and muscle cross-sectional area after isometric training. *Med Sci Sports Exerc, 24*(11), 1220-1227.
77. Garrandes, F., Colson, S.S., Pensini, M., Seynnes, O. & Legros, P. (2007). Neuromuscular fatigue profile in endurance-trained and power-trained athletes. *Med Sci Sports Exerc, 39*(1), 149-158.
78. Gefen, A. (2002). Biomechanical analysis of fatigue-related foot injury mechanisms in athletes and recruits during intensive marching. *Med Biol Eng Comput, 40*(3), 302-310.
79. Gehring, D., Melnyk, M. & Gollhofer, A. (2009). Gender and fatigue have influence on knee joint control strategies during landing. *Clin Biomech (Bristol, Avon), 24*(1), 82-87.
80. Girard, O., Millet, G.P., Micallef, J.P. & Racinais, S. (2011). Alteration in neuromuscular function after a 5 km running time trial. *Eur J Appl Physiol, 112*(6), 2323-2330.
81. Gleeson, M., Blannin, A.K., Zhu, B., Brooks, S. & Cave, R. (1995). Cardiorespiratory, hormonal and haematological responses to submaximal cycling performed 2 days after eccentric or concentric exercise bouts. *J Sports Sci, 13*(6), 471-479.

82. Green, H.J. (1997). Mechanisms of muscle fatigue in intense exercise. *J Sports Sci, 15*(3), 247-256.
83. Gregory, M.G., Nicole, D.J., Kristin, A.D., Sarah, E.M. & Thomas, W.K. (2007). Effect of fatigue on neuromuscular function at the ankle. *J Sport Rehabil, 16*, 295-306.
84. Gross, M.T. (1987). Effects of recurrent lateral ankle sprains on active and passive judgements of joint position. *Phys Ther, 67*(10), 1505-1509.
85. Grosset, J.F., Piscione, J., Lambertz, D. & Perot, C. (2009). Paired changes in electromechanical delay and musculo-tendinous stiffness after endurance or plyometric training. *Eur J Appl Physiol, 105*(1), 131-139.
86. Gruber, M., Bruhn, S. & Gollhofer, A. (2006). Specific adaptations of neuromuscular control and knee joint stiffness following sensorimotor training. *Int J Sports Med, 27*(8), 636-641.
87. Gruber, M., Gruber, S.B., Taube, W., Schubert, M., Beck, S.C. & Gollhofer, A. (2007). Differential effects of ballistic versus sensorimotor training on rate of force development and neural activation in humans. *J Strength Cond Res, 21*(1), 274-282.
88. Hakkinen, K., Alen, M., Kraemer, W.J., Gorostiaga, E., Izquierdo, M., Rusko, H., Mikkola, J., Hakkinen, A., Valkeinen, H., Kaarakainen, E., Romu, S., Erola, V., Ahtiainen, J. & Paavolainen, L. (2003). Neuromuscular adaptations during concurrent strength and endurance training versus strength training. *Eur J Appl Physiol, 89*(1), 42-52.
89. Hakkinen, K., Kallinen, M., Linnamo, V., Pastinen, U.M., Newton, R.U. & Kraemer, W.J. (1996). Neuromuscular adaptations during bilateral versus unilateral strength training in middle-aged and elderly men and women. *Acta Physiol Scand, 158*(1), 77-88.
90. Hakkinen, K., Newton, R.U., Gordon, S.E., McCormick, M., Volek, J.S., Nindl, B.C., Gotshalk, L.A., Campbell, W.W., Evans, W.J., Hakkinen, A., Humphries, B.J. & Kraemer, W.J. (1998). Changes in muscle morphology, electromyographic activity, and force production characteristics during progressive strength training in young and older men. *J Gerontol A Biol Sci Med Sci, 53*(6), B415-423.
91. Häkkinen, K., Pakarinen, A., Kyrolainen, H., Cheng, S., Kim, D.H. & Komi, P.V. (1990). Neuromuscular adaptations and serum hormones in females during prolonged power training. *Int J Sports Med, 11*(2), 91-98.
92. Hather, B.M., Tesch, P.A., Buchanan, P. & Dudley, G.A. (1991). Influence of eccentric actions on skeletal muscle adaptations to resistance training. *Acta Physiol Scand, 143*(2), 177-185.
93. Haug, H. & Eggers, R. (1991). Morphometry of the human cortex cerebri and corpus striatum during aging. *Neurobiol Aging, 12*(4), 336-338; discussion 352-335.
94. Hawkins, R.D. & Fuller, C.W. (1998). An examination of the frequency and severity of injuries and incidents at three levels of professional football. *Br J Sports Med, 32*(4), 326-332.
95. Hedayatpour, N., Falla, D., Arendt-Nielsen, L. & Farina, D. (2010). Effect of delayed-onset muscle soreness on muscle recovery after a fatiguing isometric contraction. *Scand J Med Sci Sports, 20*(1), 145-153.

96. Heuninckx, S., Wenderoth, N., Debaere, F., Peeters, R. & Swinnen, S.P. (2005). Neural basis of aging: the penetration of cognition into action control. *J Neurosci, 25*(29), 6787-6796.
97. Hewett, T.E., Myer, G.D. & Ford, K.R. (2006). Anterior cruciate ligament injuries in female athletes: Part 1, mechanisms and risk factors. *Am J Sports Med, 34*(2), 299-311.
98. Housh, D.J., Housh, T.J., Johnson, G.O. & Chu, W.K. (1992). Hypertrophic response to unilateral concentric isokinetic resistance training. *J Appl Physiol, 73*(1), 65-70.
99. Hughes, G. & Watkins, J. (2006). A risk-factor model for anterior cruciate ligament injury. *Sports Med, 36*(5), 411-428.
100. Hultborn, H. & Nielsen, J.B. (1995). H-reflexes and F-responses are not equally sensitive to changes in motoneuronal excitability. *Muscle Nerve, 18*(12), 1471-1474.
101. Ingalls, C.P., Warren, G.L., Williams, J.H., Ward, C.W. & Armstrong, R.B. (1998). E-C coupling failure in mouse EDL muscle after in vivo eccentric contractions. *J Appl Physiol, 85*(1), 58-67.
102. Izquierdo, M., Aguado, X., Gonzalez, R., Lopez, J.L. & Hakkinen, K. (1999). Maximal and explosive force production capacity and balance performance in men of different ages. *Eur J Appl Physiol Occup Physiol, 79*(3), 260-267.
103. Izquierdo, M., Hakkinen, K., Anton, A., Garrues, M., Ibanez, J., Ruesta, M. & Gorostiaga, E.M. (2001). Maximal strength and power, endurance performance, and serum hormones in middle-aged and elderly men. *Med Sci Sports Exerc, 33*(9), 1577-1587.
104. Jensen, J.L., Marstrand, P.C. & Nielsen, J.B. (2005). Motor skill training and strength training are associated with different plastic changes in the central nervous system. *J Appl Physiol, 99*(4), 1558-1568.
105. Johannsen, H.V., Lind, T., Jakobsen, B.W. & Kroner, K. (1989). Exercise-induced knee joint laxity in distance runners. *Br J Sports Med, 23*(3), 165-168.
106. Johansson, H., Sjolander, P. & Sojka, P. (1990). Activity in receptor afferents from the anterior cruciate ligament evokes reflex effects on fusimotor neurones. *Neurosci Res, 8*(1), 54-59.
107. Johnson, B.A., Salzberg, C.L. & Stevenson, D.A. (2011). A systematic review: plyometric training programs for young children. *J Strength Cond Res, 25*(9), 2623-2633.
108. Johnson, M.B. & Johnson, C.L. (1993). Electromyographic response of peroneal muscles in surgical and nonsurgical injured ankles during sudden inversion. *J Orthop Sports Phys Ther, 18*(3), 497-501.
109. Jones, C., Allen, T., Talbot, J., Morgan, D.L. & Proske, U. (1997). Changes in the mechanical properties of human and amphibian muscle after eccentric exercise. *Eur J Appl Physiol, 76*(1), 21-31.
110. Kadi, F., Charifi, N., Denis, C., Lexell, J., Andersen, J.L., Schjerling, P., Olsen, S. & Kjaer, M. (2005). The behaviour of satellite cells in response to exercise: what have we learned from human studies? *Pflugers Arch, 451*(2), 319-327.
111. Kato, T., Terashima, T., Yamashita, T., Hatanaka, Y., Honda, A. & Umemura, Y. (2006). Effect of low-repetition jump training on bone mineral density in young women. *J Appl Physiol, 100*(3), 839-843.

112. Kawakami, Y., Abe, T., Kuno, S.Y. & Fukunaga, T. (1995). Training-induced changes in muscle architecture and specific tension. *Eur J Appl Physiol Occup Physiol, 72*(1-2), 37-43.
113. Kernell, D. & Monster, A.W. (1982). Motoneurone properties and motor fatigue. An intracellular study of gastrocnemius motoneurones of the cat. *Exp Brain Res, 46*(2), 197-204.
114. Kido, A., Tanaka, N. & Stein, R.B. (2004). Spinal excitation and inhibition decrease as humans age. *Can J Physiol Pharmacol, 82*(4), 238-248.
115. Kirkley, A., Mohtadi, N. & Ogilvie, R. (2001). The effect of exercise on anterior-posterior translation of the normal knee and knees with deficient or reconstructed anterior cruciate ligaments. *Am J Sports Med, 29*(3), 311-314.
116. Kitai, T.A. & Sale, D.G. (1989). Specificity of joint angle in isometric training. *Eur J Appl Physiol Occup Physiol, 58*(7), 744-748.
117. Klass, M., Baudry, S. & Duchateau, J. (2007). Voluntary activation during maximal contraction with advancing age: a brief review. *Eur J Appl Physiol, 100*(5), 543-551.
118. Komi, P.V., Viitasalo, J.T., Rauramaa, R. & Vihko, V. (1978). Effect of isometric strength training of mechanical, electrical, and metabolic aspects of muscle function. *Eur J Appl Physiol Occup Physiol, 40*(1), 45-55.
119. Kongsgaard, M., Aagaard, P., Kjaer, M. & Magnusson, S.P. (2005). Structural Achilles tendon properties in athletes subjected to different exercise modes and in Achilles tendon rupture patients. *J Appl Physiol, 99*(5), 1965-1971.
120. Kubo, K., Kanehisa, H., Azuma, K., Ishizu, M., Kuno, S.Y., Okada, M. & Fukunaga, T. (2003). Muscle architectural characteristics in young and elderly men and women. *Int J Sports Med, 24*(2), 125-130.
121. Kubo, K., Morimoto, M., Komuro, T., Yata, H., Tsunoda, N., Kanehisa, H. & Fukunaga, T. (2007). Effects of plyometric and weight training on muscle-tendon complex and jump performance. *Med Sci Sports Exerc, 39*(10), 1801-1810.
122. Kubo, K., Tsunoda, N., Kanehisa, H. & Fukunaga, T. (2004). Activation of agonist and antagonist muscles at different joint angles during maximal isometric efforts. *Eur J Appl Physiol, 91*(2-3), 349-352.
123. Kumar, V., Atherton, P., Smith, K. & Rennie, M.J. (2009). Human muscle protein synthesis and breakdown during and after exercise. *J Appl Physiol, 106*(6), 2026-2039.
124. Kuu, S., Ereline, J., Gapeyeva, H., Kolts, I. & Pääsuke, M. (2005). Age-related changes in contractile properties of plantar flexor muscles in physically active women. *Kinesiology, 37*(2), 133-140.
125. Kvist, J., Cunningham, D. & Tigerstrand-Wejlemark, H. (2006). Gender differences in post-exercise sagittal knee translation: a comparison between elite volleyball players and swimmers. *Knee, 13*(2), 132-136.
126. Kyrolainen, H., Avela, J. & Komi, P.V. (2005). Changes in muscle activity with increasing running speed. *J Sports Sci, 23*(10), 1101-1109.

127. Kyrolainen, H., Avela, J., McBride, J.M., Koskinen, S., Andersen, J.L., Sipila, S., Takala, T.E. & Komi, P.V. (2005). Effects of power training on muscle structure and neuromuscular performance. *Scand J Med Sci Sports, 15*(1), 58-64.
128. Lepers, R., Maffiuletti, N.A., Rochette, L., Brugniaux, J. & Millet, G.Y. (2002). Neuromuscular fatigue during a long-duration cycling exercise. *J Appl Physiol, 92*(4), 1487-1493.
129. Lexell, J. (1993). Ageing and human muscle: observations from Sweden. *Can J Appl Physiol, 18*(1), 2-18.
130. Lloyd, D.G. & Buchanan, T.S. (2001). Strategies of muscular support of varus and valgus isometric loads at the human knee. *J Biomech, 34*(10), 1257-1267.
131. Macefield, G., Gandevia, S.C. & Burke, D. (1990). Perceptual responses to microstimulation of single afferents innervating joints, muscles and skin of the human hand. *J Physiol, 429*, 113-129.
132. Malisoux, L., Francaux, M., Nielens, H., Renard, P., Lebacq, J. & Theisen, D. (2006). Calcium sensitivity of human single muscle fibers following plyometric training. *Med Sci Sports Exerc, 38*(11), 1901-1908.
133. Malisoux, L., Francaux, M., Nielens, H. & Theisen, D. (2006). Stretch-shortening cycle exercises: an effective training paradigm to enhance power output of human single muscle fibers. *J Appl Physiol, 100*(3), 771-779.
134. Manini, T.M. & Clark, B.C. (2012). Dynapenia and aging: an update. *J Gerontol A Biol Sci Med Sci, 67*(1), 28-40.
135. Markolf, K.L., Gorek, J.F., Kabo, J.M. & Shapiro, M.S. (1990). Direct measurement of resultant forces in the anterior cruciate ligament. An in vitro study performed with a new experimental technique. *J Bone Joint Surg Am, 72*(4), 557-567.
136. Markovic, G. (2007). Does plyometric training improve vertical jump height? A meta-analytical review. *Br J Sports Med, 41*(6), 349-355.
137. Markovic, G. & Mikulic, P. (2010). Neuro-musculoskeletal and performance adaptations to lower-extremity plyometric training. *Sports Med, 40*(10), 859-895.
138. Marner, L., Nyengaard, J.R., Tang, Y. & Pakkenberg, B. (2003). Marked loss of myelinated nerve fibers in the human brain with age. *J Comp Neurol, 462*(2), 144-152.
139. Martin, P.G., Weerakkody, N., Gandevia, S.C. & Taylor, J.L. (2008). Group III and IV muscle afferents differentially affect the motor cortex and motoneurones in humans. *J Physiol, 586*(5), 1277-1289.
140. Martin, V., Millet, G.Y., Lattier, G. & Perrod, L. (2004). Effects of recovery modes after knee extensor muscles eccentric contractions. *Med Sci Sports Exerc, 36*(11), 1907-1915.
141. McComas, A.J. (1996). *Skeletal muscle - Form and function.* Champaign: Human Kinetics.
142. McLean, S.G., Fellin, R.E., Suedekum, N., Calabrese, G., Passerallo, A. & Joy, S. (2007). Impact of fatigue on gender-based high-risk landing strategies. *Med Sci Sports Exerc, 39*(3), 502-514.

143. McLean, S.G. & Samorezov, J.E. (2009). Fatigue-induced ACL injury risk stems from a degradation in central control. *Med Sci Sports Exerc, 41*(8), 1661-1672.
144. Meeusen, R., Watson, P., Hasegawa, H., Roelands, B. & Piacentini, M.F. (2006). Central fatigue: the serotonin hypothesis and beyond. *Sports Med, 36*(10), 881-909.
145. Melnyk, M. & Gollhofer, A. (2007). Submaximal fatigue of the hamstrings impairs specific reflex components and knee stability. *Knee Surg Sports Traumatol Arthrosc, 15*(5), 525-532.
146. Melnyk, M., Schloz, C., Schmitt, S. & Gollhofer, A. (2009). Neuromuscular ankle joint stabilisation after 4-weeks WBV training. *Int J Sports Med, 30*(6), 461-466.
147. Metter, E.J., Conwit, R., Metter, B., Pacheco, T. & Tobin, J. (1998). The relationship of peripheral motor nerve conduction velocity to age-associated loss of grip strength. *Aging (Milano), 10*(6), 471-478.
148. Millet, G.Y. & Lepers, R. (2004). Alterations of neuromuscular function after prolonged running, cycling and skiing exercises. *Sports Med, 34*(2), 105-116.
149. Millet, G.Y., Lepers, R., Maffiuletti, N.A., Babault, N., Martin, V. & Lattier, G. (2002). Alterations of neuromuscular function after an ultramarathon. *J Appl Physiol, 92*(2), 486-492.
150. Millet, G.Y., Martin, V., Lattier, G. & Ballay, Y. (2003). Mechanisms contributing to knee extensor strength loss after prolonged running exercise. *J Appl Physiol, 94*(1), 193-198.
151. Mittal, K.R. & Logmani, F.H. (1987). Age-related reduction in 8th cervical ventral nerve root myelinated fiber diameters and numbers in man. *J Gerontol, 42*(1), 8-10.
152. More, R.C., Karras, B.T., Neiman, R., Fritschy, D., Woo, S.L. & Daniel, D.M. (1993). Hamstrings--an anterior cruciate ligament protagonist. An in vitro study. *Am J Sports Med, 21*(2), 231-237.
153. Morgan, D.L. & Allen, D.G. (1999). Early events in stretch-induced muscle damage. *J Appl Physiol, 87*(6), 2007-2015.
154. Moritani, T. & deVries, H.A. (1979). Neural factors versus hypertrophy in the time course of muscle strength gain. *Am J Phys Med, 58*(3), 115-130.
155. Motl, R.W. & Dishman, R.K. (2003). Acute leg-cycling exercise attenuates the H-reflex recorded in soleus but not flexor carpi radialis. *Muscle Nerve, 28*(5), 609-614.
156. Murdock, G.H. & Hubley-Kozey, C.L. (2012). Effect of a high intensity quadriceps fatigue protocol on knee joint mechanics and muscle activation during gait in young adults. *Eur J Appl Physiol, 112*(2), 439-449.
157. Narici, M.V., Maganaris, C.N., Reeves, N.D. & Capodaglio, P. (2003). Effect of aging on human muscle architecture. *J Appl Physiol, 95*(6), 2229-2234.
158. Nawata, K., Teshima, R., Morio, Y., Hagino, H., Enokida, M. & Yamamoto, K. (1999). Anterior-posterior knee laxity increased by exercise. Quantitative evaluation of physiologic changes. *Acta Orthop Scand, 70*(3), 261-264.
159. Nybo, L. & Secher, N.H. (2004). Cerebral perturbations provoked by prolonged exercise. *Prog Neurobiol, 72*(4), 223-261.

160. Nyland, J.A., Caborn, D.N., Shapiro, R. & Johnson, D.L. (1999). Crossover Cutting During Hamstring Fatigue Produces Transverse Plane Knee Control Deficits. *J Athl Train, 34*(2), 137-143.
161. Nyland, J.A., Shapiro, R., Stine, R.L., Horn, T.S. & Ireland, M.L. (1994). Relationship of fatigued run and rapid stop to ground reaction forces, lower extremity kinematics, and muscle activation. *J Orthop Sports Phys Ther, 20*(3), 132-137.
162. O'Connor, P.J. & Cook, D.B. (1999). Exercise and pain: the neurobiology, measurement, and laboratory study of pain in relation to exercise in humans. *Exerc Sport Sci Rev, 27*, 119-166.
163. Palmieri, R.M., Ingersoll, C.D. & Hoffman, M.A. (2004). The Hoffmann Reflex: Methodologic Considerations and Applications for Use in Sports Medicine and Athletic Training Research. *J Athl Train, 39*(3), 268-277.
164. Palmieri-Smith, R.M., McLean, S.G., Ashton-Miller, J.A. & Wojtys, E.M. (2009). Association of quadriceps and hamstrings cocontraction patterns with knee joint loading. *J Athl Train, 44*(3), 256-263.
165. Pappas, E., Hagins, M., Sheikhzadeh, A., Nordin, M. & Rose, D. (2009). Peak biomechanical variables during bilateral drop landings: comparisons between sex (female/male) and fatigue (pre-fatigue/post-fatigue). *N Am J Sports Phys Ther, 4*(2), 83-91.
166. Pierrot-Deseilligny, E. & Burke, D. (2005). *The Circuitry of the Human Spinal Cord.* Cambridge: Cambridge University Press.
167. Pierrot-Deseilligny, E. & Mazevet, D. (2000). The monosynaptic reflex: a tool to investigate motor control in humans. Interest and limits. *Neurophysiol Clin, 30*(2), 67-80.
168. Pijnappels, M., Reeves, N.D., Maganaris, C.N. & van Dieen, J.H. (2008). Tripping without falling; lower limb strength, a limitation for balance recovery and a target for training in the elderly. *J Electromyogr Kinesiol, 18*(2), 188-196.
169. Pijnappels, M., van der Burg, P.J., Reeves, N.D. & van Dieen, J.H. (2008). Identification of elderly fallers by muscle strength measures. *Eur J Appl Physiol, 102*(5), 585-592.
170. Porter, M.M., Myint, A., Kramer, J.F. & Vandervoort, A.A. (1995). Concentric and eccentric knee extension strength in older and younger men and women. *Can J Appl Physiol, 20*(4), 429-439.
171. Prasartwuth, O., Allen, T.J., Butler, J.E., Gandevia, S.C. & Taylor, J.L. (2006). Length-dependent changes in voluntary activation, maximum voluntary torque and twitch responses after eccentric damage in humans. *J Physiol, 571*(Pt 1), 243-252.
172. Prasartwuth, O., Taylor, J.L. & Gandevia, S.C. (2005). Maximal force, voluntary activation and muscle soreness after eccentric damage to human elbow flexor muscles. *J Physiol, 567*(Pt 1), 337-348.
173. Proske, U. & Allen, T.J. (2005). Damage to skeletal muscle from eccentric exercise. *Exerc Sport Sci Rev, 33*(2), 98-104.
174. Proske, U. & Morgan, D.L. (2001). Muscle damage from eccentric exercise: mechanism, mechanical signs, adaptation and clinical applications. *J Physiol, 537*(Pt 2), 333-345.

175. Racinais, S., Bringard, A., Puchaux, K., Noakes, T.D. & Perrey, S. (2008). Modulation in voluntary neural drive in relation to muscle soreness. *Eur J Appl Physiol, 102*(4), 439-446.
176. Racinais, S., Girard, O., Micallef, J.P. & Perrey, S. (2007). Failed excitability of spinal motoneurons induced by prolonged running exercise. *J Neurophysiol, 97*(1), 596-603.
177. Rahnama, N., Reilly, T. & Lees, A. (2002). Injury risk associated with playing actions during competitive soccer. *Br J Sports Med, 36*(5), 354-359.
178. Ramesh, R., Von Arx, O., Azzopardi, T. & Schranz, P.J. (2005). The risk of anterior cruciate ligament rupture with generalised joint laxity. *J Bone Joint Surg Br, 87*(6), 800-803.
179. Reeves, N.D., Narici, M.V. & Maganaris, C.N. (2004). Effect of resistance training on skeletal muscle-specific force in elderly humans. *J Appl Physiol, 96*(3), 885-892.
180. Rennie, M.J., Selby, A., Atherton, P., Smith, K., Kumar, V., Glover, E.L. & Philips, S.M. (2010). Facts, noise and wishful thinking: muscle protein turnover in aging and human disuse atrophy. *Scand J Med Sci Sports, 20*(1), 5-9.
181. Riecker, A., Groschel, K., Ackermann, H., Steinbrink, C., Witte, O. & Kastrup, A. (2006). Functional significance of age-related differences in motor activation patterns. *Neuroimage, 32*(3), 1345-1354.
182. Robert-Koch-Institut, *Gesundheit in Deutschland. Gesundheitsberichterstattung des Bundes*. 2006, Robert Koch-Institut: Berlin.
183. Roos, M.R., Rice, C.L., Connelly, D.M. & Vandervoort, A.A. (1999). Quadriceps muscle strength, contractile properties, and motor unit firing rates in young and old men. *Muscle Nerve, 22*(8), 1094-1103.
184. Rotto, D.M. & Kaufman, M.P. (1988). Effect of metabolic products of muscular contraction on discharge of group III and IV afferents. *J Appl Physiol, 64*(6), 2306-2313.
185. Rowe, A., Wright, S., Nyland, J., Caborn, D.N. & Kling, R. (1999). Effects of a 2-hour cheerleading practice on dynamic postural stability, knee laxity, and hamstring extensibility. *J Orthop Sports Phys Ther, 29*(8), 455-462.
186. Rozzi, S., Yuktanandana, P., Pincivero, D. & Lephart, S.M. (2000). Role of fatigue on proprioception and neuromuscular control. In F.H. Fu (Ed.), *Proprioception and neuromuscular control in joint stability* (pp. 375-383). Champaign: Human Kinetics.
187. Rozzi, S.L., Lephart, S.M., Gear, W.S. & Fu, F.H. (1999). Knee joint laxity and neuromuscular characteristics of male and female soccer and basketball players. *Am J Sports Med, 27*(3), 312-319.
188. Rutherford, O.M. & Jones, D.A. (1986). The role of learning and coordination in strength training. *Eur J Appl Physiol Occup Physiol, 55*(1), 100-105.
189. Saez-Saez de Villarreal, E., Requena, B. & Newton, R.U. (2010). Does plyometric training improve strength performance? A meta-analysis. *J Sci Med Sport, 13*(5), 513-522.
190. Salat, D.H., Buckner, R.L., Snyder, A.Z., Greve, D.N., Desikan, R.S., Busa, E., Morris, J.C., Dale, A.M. & Fischl, B. (2004). Thinning of the cerebral cortex in aging. *Cereb Cortex, 14*(7), 721-730.

191. Sale, D. & MacDougall, D. (1981). Specificity in strength training: a review for the coach and athlete. *Can J Appl Sport Sci, 6*(2), 87-92.
192. Sale, D.G. (2003). Neural Adaptation to Strength Training. In P. V. Komi (Ed.), *Strength and Power in Sport* (pp. 281-314). Oxford: Blackwell Science.
193. Sale, M.V. & Semmler, J.G. (2005). Age-related differences in corticospinal control during functional isometric contractions in left and right hands. *J Appl Physiol, 99*(4), 1483-1493.
194. Salonikidis, K. & Zafeiridis, A. (2008). The effects of plyometric, tennis-drills, and combined training on reaction, lateral and linear speed, power, and strength in novice tennis players. *J Strength Cond Res, 22*(1), 182-191.
195. Saxton, J.M. & Donnelly, A.E. (1996). Length-specific impairment of skeletal muscle contractile function after eccentric muscle actions in man. *Clin Sci (Lond), 90*(2), 119-125.
196. Schache, A.G., Wrigley, T.V., Baker, R. & Pandy, M.G. (2009). Biomechanical response to hamstring muscle strain injury. *Gait Posture, 29*(2), 332-338.
197. Schieppati, M. (1987). The Hoffmann reflex: a means of assessing spinal reflex excitability and its descending control in man. *Prog Neurobiol, 28*(4), 345-376.
198. Schneider, S., Seither, B., Tönges, S. & Schmitt, H. (2006). Sports injuries: population based representative data on incidence, diagnosis, sequelae, and high risk groups. *Br J Sports Med, 40*, 334-339.
199. Semark, A., Noakes, T.D., St Clair Gibson, A. & Lambert, M.I. (1999). The effect of a prophylactic dose of flurbiprofen on muscle soreness and sprinting performance in trained subjects. *J Sports Sci, 17*(3), 197-203.
200. Seynnes, O.R., de Boer, M. & Narici, M.V. (2007). Early skeletal muscle hypertrophy and architectural changes in response to high-intensity resistance training. *J Appl Physiol, 102*(1), 368-373.
201. Sherman, W.M., Armstrong, L.E., Murray, T.M., Hagerman, F.C., Costill, D.L., Staron, R.C. & Ivy, J.L. (1984). Effect of a 42.2-km footrace and subsequent rest or exercise on muscular strength and work capacity. *J Appl Physiol, 57*(6), 1668-1673.
202. Sidhu, S.K., Bentley, D.J. & Carroll, T.J. (2009). Locomotor exercise induces long-lasting impairments in the capacity of the human motor cortex to voluntarily activate knee extensor muscles. *J Appl Physiol, 106*(2), 556-565.
203. Skelton, D.A., Greig, C.A., Davies, J.M. & Young, A. (1994). Strength, power and related functional ability of healthy people aged 65-89 years. *Age Ageing, 23*(5), 371-377.
204. Skinner, H.B., Wyatt, M.P., Hodgdon, J.A., Conard, D.W. & Barrack, R.L. (1986). Effect of fatigue on joint position sense of the knee. *J Orthop Res, 4*(1), 112-118.
205. Skurvydas, A., Brazaitis, M. & Kamandulis, S. (2011). Muscle-damaging exercise affects isokinetic torque more at short muscle length. *J Strength Cond Res, 25*(5), 1400-1406.
206. Song, M., Segala, D.B., Dingwell, J.B. & Chelidze, D. (2009). Slow-time changes in human EMG muscle fatigue states are fully represented in movement kinematics. *J Biomech Eng, 131*(2), 021004.

207. Spurrs, R.W., Murphy, A.J. & Watsford, M.L. (2003). The effect of plyometric training on distance running performance. *Eur J Appl Physiol, 89*(1), 1-7.
208. Stackhouse, S.K., Stevens, J.E., Lee, S.C., Pearce, K.M., Snyder-Mackler, L. & Binder-Macleod, S.A. (2001). Maximum voluntary activation in nonfatigued and fatigued muscle of young and elderly individuals. *Phys Ther, 81*(5), 1102-1109.
209. Staron, R.S., Malicky, E.S., Leonardi, M.J., Falkel, J.E., Hagerman, F.C. & Dudley, G.A. (1990). Muscle hypertrophy and fast fiber type conversions in heavy resistance-trained women. *Eur J Appl Physiol Occup Physiol, 60*(1), 71-79.
210. Stauber, W.T., Clarkson, P.M., Fritz, V.K. & Evans, W.J. (1990). Extracellular matrix disruption and pain after eccentric muscle action. *J Appl Physiol, 69*(3), 868-874.
211. Stevens, J.E., Stackhouse, S.K., Binder-Macleod, S.A. & Snyder-Mackler, L. (2003). Are voluntary muscle activation deficits in older adults meaningful? *Muscle Nerve, 27*(1), 99-101.
212. Strojnik, V. & Komi, P.V. (1998). Neuromuscular fatigue after maximal stretch-shortening cycle exercise. *J Appl Physiol, 84*(1), 344-350.
213. Takekura, H., Fujinami, N., Nishizawa, T., Ogasawara, H. & Kasuga, N. (2001). Eccentric exercise-induced morphological changes in the membrane systems involved in excitation-contraction coupling in rat skeletal muscle. *J Physiol, 533*(Pt 2), 571-583.
214. Taube, W., Leukel, C., Lauber, B. & Gollhofer, A. (2011). The drop height determines neuromuscular adaptations and changes in jump performance in stretch-shortening cycle training. *Scand J Med Sci Sports*
215. Tesch, P.A. (1988). Skeletal muscle adaptations consequent to long-term heavy resistance exercise. *Med Sci Sports Exerc, 20*(5 Suppl), S132-134.
216. Thepaut-Mathieu, C., Van Hoecke, J. & Maton, B. (1988). Myoelectrical and mechanical changes linked to length specificity during isometric training. *J Appl Physiol, 64*(4), 1500-1505.
217. Thomas, A.C., McLean, S.G. & Palmieri-Smith, R.M. (2010). Quadriceps and hamstrings fatigue alters hip and knee mechanics. *J Appl Biomech, 26*(2), 159-170.
218. Thomas, A.C., Palmieri-Smith, R.M. & McLean, S.G. (2011). Isolated hip and ankle fatigue are unlikely risk factors for anterior cruciate ligament injury. *Scand J Med Sci Sports, 21*(3), 359-368.
219. Tillin, N.A., Pain, M.T. & Folland, J.P. (2011). Short-term unilateral resistance training affects the agonist-antagonist but not the force-agonist activation relationship. *Muscle Nerve, 43*(3), 375-384.
220. Tsai, L.C., Sigward, S.M., Pollard, C.D., Fletcher, M.J. & Powers, C.M. (2009). Effects of Fatigue and Recovery on Knee Mechanics during Side-Step Cutting. *Med Sci Sports Exerc, 41*(10), 1952-1957.
221. Tsuruike, M., Koceja, D.M., Yabe, K. & Shima, N. (2003). Age comparison of H-reflex modulation with the Jendrassik maneuver and postural complexity. *Clin Neurophysiol, 114*(5), 945-953.

222. Van Cutsem, M., Duchateau, J. & Hainaut, K. (1998). Changes in single motor unit behaviour contribute to the increase in contraction speed after dynamic training in humans. *J Physiol, 513 (Pt 1)*, 295-305.
223. Vickers, A.J. (2001). Time course of muscle soreness following different types of exercise. *BMC Musculoskelet Disord, 2*, 5.
224. Victor, J., Labey, L., Wong, P., Innocenti, B. & Bellemans, J. (2010). The influence of muscle load on tibiofemoral knee kinematics. *J Orthop Res, 28*(4), 419-428.
225. Vila-Cha, C., Hassanlouei, H., Farina, D. & Falla, D. (2011). Eccentric exercise and delayed onset muscle soreness of the quadriceps induce adjustments in agonist-antagonist activity, which are dependent on the motor task. *Exp Brain Res*, DOI 10.1007/s00221-00011-02942-00222.
226. Voigt, M., Chelli, F. & Frigo, C. (1998). Changes in the excitability of soleus muscle short latency stretch reflexes during human hopping after 4 weeks of hopping training. *Eur J Appl Physiol Occup Physiol, 78*(6), 522-532.
227. Watson, J.D., Colebatch, J.G. & McCloskey, D.I. (1984). Effects of externally imposed elastic loads on the ability to estimate position and force. *Behav Brain Res, 13*(3), 267-271.
228. Wilder, M.R. & Cannon, J. (2009). Effect of age on muscle activation and twitch properties during static and dynamic actions. *Muscle Nerve, 39*(5), 683-691.
229. Wilkes, E.A., Selby, A.L., Atherton, P.J., Patel, R., Rankin, D., Smith, K. & Rennie, M.J. (2009). Blunting of insulin inhibition of proteolysis in legs of older subjects may contribute to age-related sarcopenia. *Am J Clin Nutr, 90*(5), 1343-1350.
230. Wilson, G.J., Newton, R.U., Murphy, A.J. & Humphries, B.J. (1993). The optimal training load for the development of dynamic athletic performance. *Med Sci Sports Exerc, 25*(11), 1279-1286.
231. Witzke, K.A. & Snow, C.M. (2000). Effects of plyometric jump training on bone mass in adolescent girls. *Med Sci Sports Exerc, 32*(6), 1051-1057.
232. Wojtys, E.M., Wylie, B.B. & Huston, L.J. (1996). The effects of muscle fatigue on neuromuscular function and anterior tibial translation in healthy knees. *Am J Sports Med, 24*(5), 615-621.
233. Wu, Y.K., Lien, Y.H., Lin, K.H., Shih, T.T., Wang, T.G. & Wang, H.K. (2010). Relationships between three potentiation effects of plyometric training and performance. *Scand J Med Sci Sports, 20*(1), e80-86.
234. Yeung, S.S. & Yeung, E.W. (2008). Shift of peak torque angle after eccentric exercise. *Int J Sports Med, 29*(3), 251-256.
235. Young, A., Stokes, M. & Crowe, M. (1984). Size and strength of the quadriceps muscles of old and young women. *Eur J Clin Invest, 14*(4), 282-287.
236. Young, A., Stokes, M. & Crowe, M. (1985). The size and strength of the quadriceps muscles of old and young men. *Clin Physiol, 5*(2), 145-154.
237. Zajac, F.E. (1993). Muscle coordination of movement: a perspective. *J Biomech, 26 Suppl 1*, 109-124.

238. Zehr, P.E. (2002). Considerations for use of the Hoffmann reflex in exercise studies. *Eur J Appl Physiol, 86*(6), 455-468.
239. Zhang, L.Q. & Rymer, W.Z. (2001). Reflex and intrinsic changes induced by fatigue of human elbow extensor muscles. *J Neurophysiol, 86*(3), 1086-1094.

6. SELBSTSTÄNDIGKEITSERKLÄRUNG

Ich versichere hiermit, dass ich die eingereichte Dissertation selbständig und ohne fremde Hilfe verfasst, andere als die von mir angegebenen Quellen und Hilfsmittel nicht benutzt und die den benutzten Quellen wörtlich oder inhaltlich entnommenen Stellen als solche kenntlich gemacht habe.

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8. EIGENE PUBLIKATIONEN

I. Behrens, M., Mau-Moeller, A., Wassermann, F., Bruhn, S. (2013). Effect of fatigue on hamstring reflex responses and posterior-anterior tibial translation in men and women. *Plos One, 8* (2), e56988. [IF_{2011}: 4.092].

II. Behrens, M., Mau-Moeller, A., Bruhn, S. (2012). Effect of exercise-induced muscle damage on neuromuscular function of the quadriceps muscle. *International Journal of Sports Medicine, 33* (8), 600-606. [IF_{2011}: 2.433].

III. Behrens, M., Mau-Moeller, A., Bruhn, S. (2013). Effect of plyometric training on neural and mechanical properties of the knee extensor muscles. *International Journal of Sports Medicine* (in press). [IF_{2011}: 2.433].

IV. Mau-Moeller, A.*, **Behrens, M.***, Lindner, T., Bader, R., Bruhn, S. (2013). Age-related changes in neuromuscular function of the quadriceps muscle in physically active adults. *Journal of Electromyography and Kinesiology, 23* (3), 640-648. (* authors contributed equally to this work) [IF_{2011}: 1.969].

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PLOS ONE

Effect of Fatigue on Hamstring Reflex Responses and Posterior-Anterior Tibial Translation in Men and Women

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Abstract

Anterior cruciate ligament (ACL) rupture ranks among the most common injuries in sports. The incidence of ACL injuries is considerably higher in females than in males and the underlying mechanisms are still under debate. Furthermore, it has been suggested that muscle fatigue can be a risk factor for ACL injuries. We investigated gender differences in hamstring reflex responses and posterior-anterior tibial translation (TT) before and after fatiguing exercise. We assessed the isolated movement of the tibia relative to the femur in the sagittal plane as a consequence of mechanically induced TT in standing subjects. The muscle activity of the hamstrings was evaluated. Furthermore, isometric maximum voluntary torque (iMVT) and rate of torque development (RTD) of the hamstrings (H) and quadriceps (Q) were measured and the MVT H/Q as well as the RTD H/Q ratios were calculated. After fatigue, reflex onset latencies were enhanced in women. A reduction of reflex responses associated with an increased TT was observed in females. Men showed no differences in these parameters. Correlation analysis revealed no significant associations between parameters for TT and MVT H/Q as well as RTD H/Q. The results of the present study revealed that the fatigue protocol used in this study altered the latency and magnitude of reflex responses of the hamstrings as well as TT in women. These changes were not found in men. Based on our results, it is conceivable that the fatigue-induced decrease in neuromuscular function with a corresponding increase in TT probably contributes to the higher incidence of ACL injuries in women.

Citation: Behrens M, Mau-Moeller A, Wassermann F, Bruhn S (2013) Effect of Fatigue on Hamstring Reflex Responses and Posterior-Anterior Tibial Translation in Men and Women. PLoS ONE 8(2): e56988. doi:10.1371/journal.pone.0056988

Editor: Junming Yue, The University of Tennessee Health Science Center, United States of America

Received October 2, 2012; **Accepted** January 16, 2013; **Published** February 27, 2013

Funding: This study was supported by the German-Austrian-Swiss Society for Orthopaedic Traumatologic Sports Medicine (GOTS) and the Bundesinstitut fuer Sportwissenschaft (BISp). The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Competing Interests: The authors have declared that no competing interests exist.

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Introduction

Anterior cruciate ligament (ACL) rupture ranks among the most common injuries in sports [1] and is associated with long recovery times and high socio-economic costs. The incidence of ACL injuries is considerably higher in females than in males and the underlying mechanisms are still under debate [2]. It has been argued that differences in the passive and active stability of the tibiofemoral joint could be responsible for the higher injury rate. The passive stability of the knee joint depends largely on the laxity of the ligaments and the geometry of the articular surfaces. Active stability relies on the patellar tendon-tibia shaft angle, muscle activity pattern, muscle reaction time, time to peak torque and muscle stiffness [3].

Furthermore, it has been suggested that muscle fatigue can be a risk factor for ACL injuries [2,3]. Several studies have shown that muscle fatigue is associated with decreased joint proprioception and postural stability as well as increased joint laxity [4–6]. Fatigue has also been shown to alter the control of lower extremity mechanics during landing, side-step cutting and running [7–10]. Epidemiological data suggest that injury rates tend to be higher at the end of matches [11,12], suggesting fatigue could be related to injury. Therefore, fatigue may play an important role in the pathomechanics of knee joint injuries [13].

Several studies have focused on hamstring reflex responses and their role in resisting posterior-anterior tibial translation (TT), which served as a criterion for functional knee stability [14,15]. A study by Friemert et al. [14] has revealed that these reflex responses originate from primary and secondary spindle afferents in the hamstring muscles. Some studies have investigated the effect of fatigue on reflex responses of the hamstring muscles and TT [13,16]. These studies have revealed that fatigue can modulate the timing and magnitude of reflex activity as well as increase TT. However, the effect of fatigue on gender-specific hamstring reflex responses and TT has not been sufficiently investigated.

Furthermore, it has been suggested that a low hamstrings/quadriceps (H/Q) strength ratio can be an indicator for knee joint injury risk [17–19]. According to a study by Krosshaug et al. [20], ACL injuries occur between 17 and 50 ms after initial ground contact. Therefore, Zebis et al. [19] introduced a H/Q ratio that takes the ability of the subject to rapidly develop force within 50 ms into account. The authors proposed that this rate of torque development (RTD) H/Q ratio could be used in addition to the traditional H/Q ratio, derived from the peak force values during maximum voluntary contraction (MVC), to describe the potential for knee joint stabilization.

The purpose of the present study was to analyze gender differences in hamstring reflex responses and TT before and after a fatigue protocol. We assessed the isolated movement of the tibia relative to the femur in the sagittal plane as a consequence of mechanically induced TT in standing subjects. The muscle activity of the lateral and medial hamstrings was evaluated. Furthermore, isometric maximum voluntary torque (iMVT) and RTD of the

hamstrings and quadriceps were measured and MVT H/Q as well as RTD H/Q ratios were calculated.

It was hypothesized that, due to fatigue, reflex components of the muscles are impaired and TT is altered. In addition, we presumed that there is an association between TT and the MVT H/Q ratio as well as the RTD H/Q ratio. We assumed that the main outcome variables would show gender-specific differences.

Methods

Subjects and study design

Fifty healthy subjects (25 males: 25.4±2.7 years, 78.8±7.8 kg, 180.7±5.3 cm/25 females: 23.3±2.2 years, 64.9±8.9 kg, 168.3±3.6 cm) with no history of neurological disorders or injuries participated. Before testing, subjects were instructed to refrain from consuming alcohol and caffeine in the 24 h preceding the experiment and not to perform any strenuous exercise in the 48 h prior to the measurements. All persons signed informed consent. The study was conducted according to the declaration of Helsinki and was approved by the ethics committee of the University of Rostock (A 2011 129). During the experiment, participants were examined with regard to reflex responses and TT before and after a fatiguing jumping task. The measurements were performed using a knee arthrometer [16,21–23] (Fig. 1). In addition, before the execution of the fatigue protocol, the subjects performed isometric MVCs for the hamstrings and the quadriceps using a dynamometer. The experiment required approximately 2 h per person.

Measurement of posterior-anterior tibial translation

Participants were examined in bipedal stance with the knees in 30° flexion (0° = full extension). In order to standardize the stance position between the trials, subjects stood on a force plate (sampling frequency: 40 Hz, GKS 1000®, IMM Holding GmbH, Germany). Subjects were thereby provided with online feedback about their center of pressure. Furthermore, to avoid the influence of acoustic signals on the subjects they wore ear protection (Bilsom Thunder T3). The subjects had no information on the point in time of the perturbation. A standardized force was applied to the proximal shank of the dominant leg using a pulley system in order to induce TT. A device was attached to the tibia to secure two linear potentiometers (measuring accuracy: <0.01 mm, linearity: ±0.7%, Type CLR13–50; Megatron, Germany) that were placed on the patella and the tibial tuberosity (Fig. 1). The knee arthrometer enabled us to measure the translational movement of the tibia relative to the femur in the sagittal plane. The interface pressure between the knee arthrometer and the subjects' tibia was controlled by an air pressure recorder (Kikuhime, TT MediTrade, Denmark). The interface pressure was kept constant before and after the fatiguing exercise. The locations of the subjects' feet on the force plate, the stabilizing device and the linear potentiometers were marked in order to ensure the same positions before and after the fatigue protocol. The applied force that induced TT was

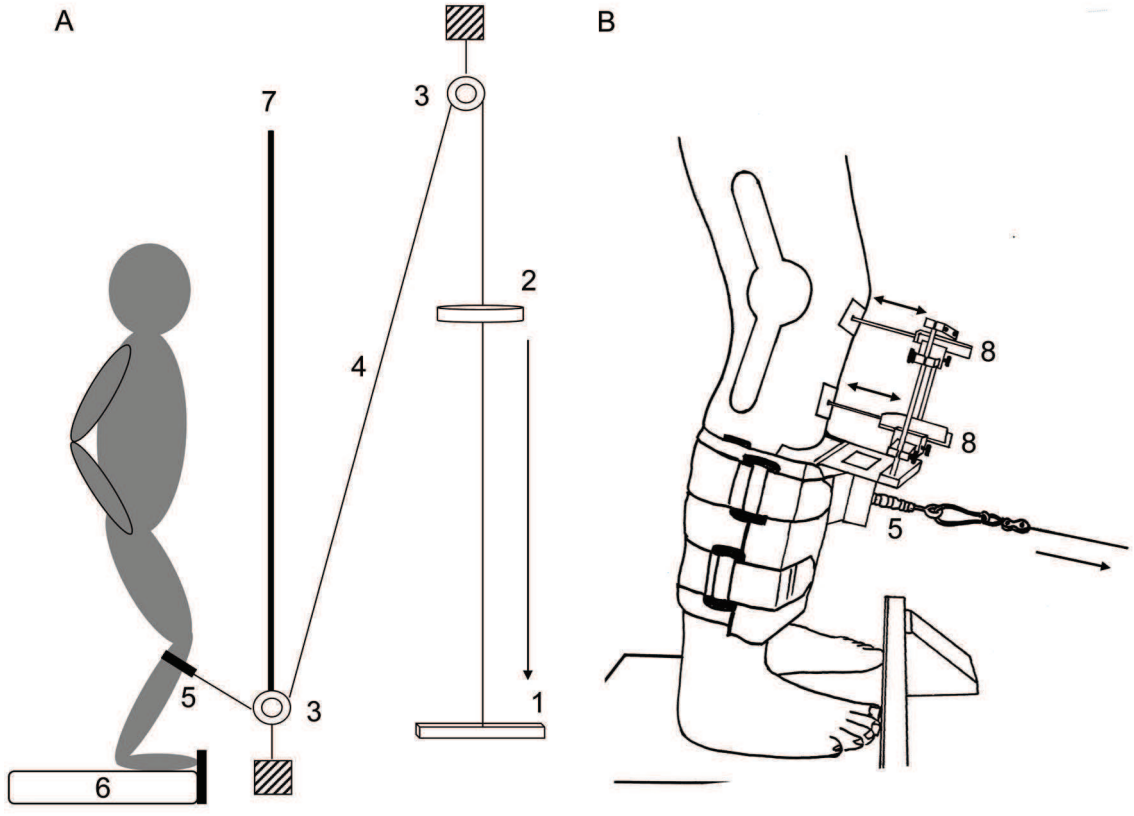

Figure 1. Schematic drawing of the experimental setup. A: Experimental setup, B: Measurement system | 1: stopper, 2: falling weight, 3: pulley, 4: steel rope, 5: force transducer, 6: force plate, 7: visual cover, 8: linear potentiometer. Arrows indicate the direction of the force. Posterior-anterior tibial translation was assessed by two linear potentiometers (8) placed on the patella and the tibial tuberosity. A force transducer (5) was used to measure the force transmitted to the shank.
doi:10.1371/journal.pone.0056988.g001

controlled using a force transducer (measuring range: 0–5000 N, sensitivity: −3.42 to 3.36 $pC \cdot N^{-1}$, linearity: ±0.2–0.3%; Kistler, Switzerland). The force sensor was placed between the stabilizing device and the pulley system. TT was elicited 15 times in order to familiarize the subject with the measurement. Thereafter, further 15 perturbations were applied before and immediately after the fatigue protocol. The inter stimulus interval was varied between ~8 s and ~14 s to avoid anticipation.

EMG and torque recordings

Surface EMG was recorded using bipolar Ambu® Blue Sensor N electrodes. The electrodes were attached to the shaved, abraded and cleaned skin over the biceps femoris (BF) and semitendinosus/semimembranosus (ST) of the dominant leg (resistance between electrodes <5 kΩ). The electrodes were applied with a center-to-center distance of 2 cm over the muscle bellies and in line with the presumed direction of the underlying muscle fibers. The reference electrode was attached to the patella. Signals were amplified (2500x), band-pass filtered (10–1300 Hz) and digitized (sampling frequency: 5 kHz) through an analog-to-digital converter (DAQ Card™-6024E, National Instruments, USA).

Torque was measured using a CYBEX NORM dynamometer (Computer Sports Medicine®, Inc., Stoughton, MA). A hip joint angle and knee joint angle of 30°, respectively (0° = full extension), was chosen in order to mimic the posture and therefore the length of the muscles during the measurement of TT. The axis of the dynamometer was aligned with the anatomical knee flexion-extension axis and the lever arm was attached to the anterior aspect of the shank 2–3 cm above the lateral malleolus. Straps across the waist and chest prevented excessive movements. The iMVT was tested by asking the subjects to exert isometric knee extensions and flexions against the lever arm of the dynamometer for 3 s. For each trial, subjects were thoroughly instructed to act as forcefully and as fast as possible. They were motivated by strong verbal encouragement and online visual feedback of the instantaneous dynamometer torque provided on a digital oscilloscope (HM1508, HAMEG Instruments, Germany). Care was taken that the iMVT trials were performed without an apparent countermovement or pre-tension (change in baseline torque <0.5 Nm during 200 ms prior to contraction onset). A rest period of 2 min was allowed between trials. The maximal attempts were recorded until the coefficient of variance of five consecutive trials was below 5% [24]. The EMG, linear potentiometer, force and torque signals were stored on a hard drive for later analysis with custom built LABVIEW® based software (Imago, Pfisoft, Germany).

Fatigue protocol

Fatigue was induced by repetitive jumping performed between ~90° and 0° knee flexion (0° = full extension). The fatigue protocol consisted of consecutive maximal countermovement jumps [25], each one separated by 4 s according to the sound of a digital metronome. The jumps were performed until the subjects reached a fatigued state defined as the inability to reach 50% of their maximal jump height for 3 consecutive jumps or until the subjects reached an intolerable state of dyspnea or exhaustion. Rate of perceived exertion (RPE) was assessed using the Borg 6–20 scale.

Data analysis

In order to analyze the data, the EMG signals of each subject were averaged. The EMG onset latencies were defined as the time between onset of TT and onset of significant muscular activity, e.g. the beginning of EMG deflection (average EMG baseline value measured over 100 ms ± 3 standard deviations). Muscle activity was analyzed according to Bruhn et al. [23], that is, the TT signal indicated the onset of perturbation and muscle activity was calculated over different time intervals relative to the onset of TT, i.e. 20–40, 40–60 and 60–95 ms (Fig. 2), using the root mean square of the EMG signal (RMS-EMG). In order to assess background activity before and after fatigue, RMS-EMG was calculated over 50 ms prior to the onset of TT. Consequently, background activity was subtracted from the reflex responses. Maximum TT was determined based on the TT curves.

The torque signals were corrected for the effect of gravity and the three best maximum voluntary contractions were retained for analysis. The iMVT was defined as the highest peak torque value. Explosive voluntary muscle strength was determined by analyzing the average RTD over time intervals of 0–50, 0–100 and 0–200 ms relative to the onset of contraction. The identification of torque onset was made manually according to the method of Tillin et al. [26]. It has been suggested that this is the best method for detecting signal onsets [27]. MVT H/Q as well as RTD H/Q ($RTD_{0-50,\ 0-100,\ 0-200}$ H/Q) ratios were calculated as follows [19]: MVT H/Q = Hamstrings MVT/Quadriceps MVT and RTD_x H/Q = Hamstrings RTD_x/Quadriceps RTD_x, where x denotes the analyzed time interval (0–50, 0–100, 0–200 ms).

Statistical analysis

Data were checked for normal distribution using the Kolmogorov-Smirnov test. Differences between the values before and after the fatigue protocol were tested for significance by repeated measures ANOVA. Differences between the groups were tested for significance by the unpaired Student's t test. Correlations between parameters were calculated using Pearson's correlation coefficient. In each case the level of significance was established at $p \leq 0.05$. SPSS 20.0 (SPSS Inc., Chicago, IL, USA) was used for statistical analysis. Effect size (ES) was calculated with the statistical software package G*Power (Version 3.1.5) [28]. The ES characterizes the effectiveness of an intervention. Furthermore, it is used to determine whether a statistically significant difference is a difference of practical importance. ES-values = 0.10 indicate small, ES = 0.25 medium and ES = 0.40 large effects [29]. Data are presented as group mean values ± standard error of the mean in the figures and as group mean values ± standard deviation in the table.

Results

During the fatiguing exercise, men performed 159.8±90.9 and women 150.6±62.4 jumps. RPE using the Borg 6–20 scale was 15.2±2.0 for males and 14.8±1.6 for females. The force applied to the proximal shank of the dominant leg remained constant during the pre- and post-test (Table 1). Reflex onset latencies of BF and ST were significantly delayed in women at post-test (Table 1). BF muscle activity decreased after fatigue in women in the time intervals 20–40 ms ($F = 4.99$, $P = 0.035$, $\eta^2 = 0.166$, ES = 0.45) and 40–60 ms ($F = 7.22$, $P = 0.013$, $\eta^2 = 0.224$, ES = 0.54). The reflex response of BF between 60–95 ms ($F = 0.455$, $P = 0.506$, $\eta^2 = 0.018$, ES = 0.14) was unchanged in women after the fatigue protocol. The reflex activity of ST was significantly reduced after fatigue between 20–40 ms in women ($F = 5.38$, $P = 0.029$, $\eta^2 = 0.183$, ES = 0.47), but not between 40–60 ms ($F = 0.051$, $P = 0.823$, $\eta^2 = 0.002$, ES = 0.045) and 60–95 ms ($F = 3.001$, $P = 0.096$, $\eta^2 = 0.111$, ES = 0.35). TT increased significantly after the fatigue protocol in females ($F = 11.86$, $P = 0.002$, $\eta^2 = 0.322$, ES = 0.69) (Fig. 3).

No statistical differences in the reflex responses of BF between 20–40 ms ($F = 0.601$, $P = 0.448$, $\eta^2 = 0.032$, ES = 0.18), 40–60 ms

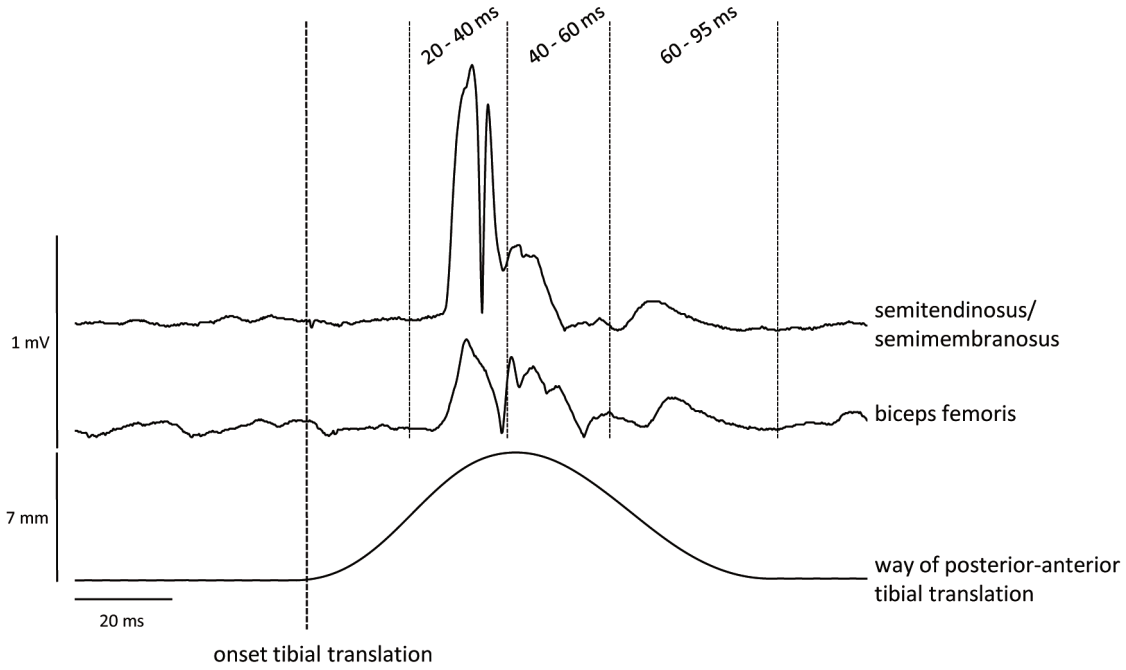

Figure 2. EMG and tibial translation data from one representative subject. EMG activity of biceps femoris and semitendinosus/semimembranosus as well as posterior-anterior tibial translation of one subject. In the figure, EMG data is rectified in order to visualize the different parts of the hamstring stretch reflex. The vertical bold line indicates the onset of posterior-anterior tibial translation. Three different time intervals were analyzed (20–40, 40–60 and 60–95 ms).
doi:10.1371/journal.pone.0056988.g002

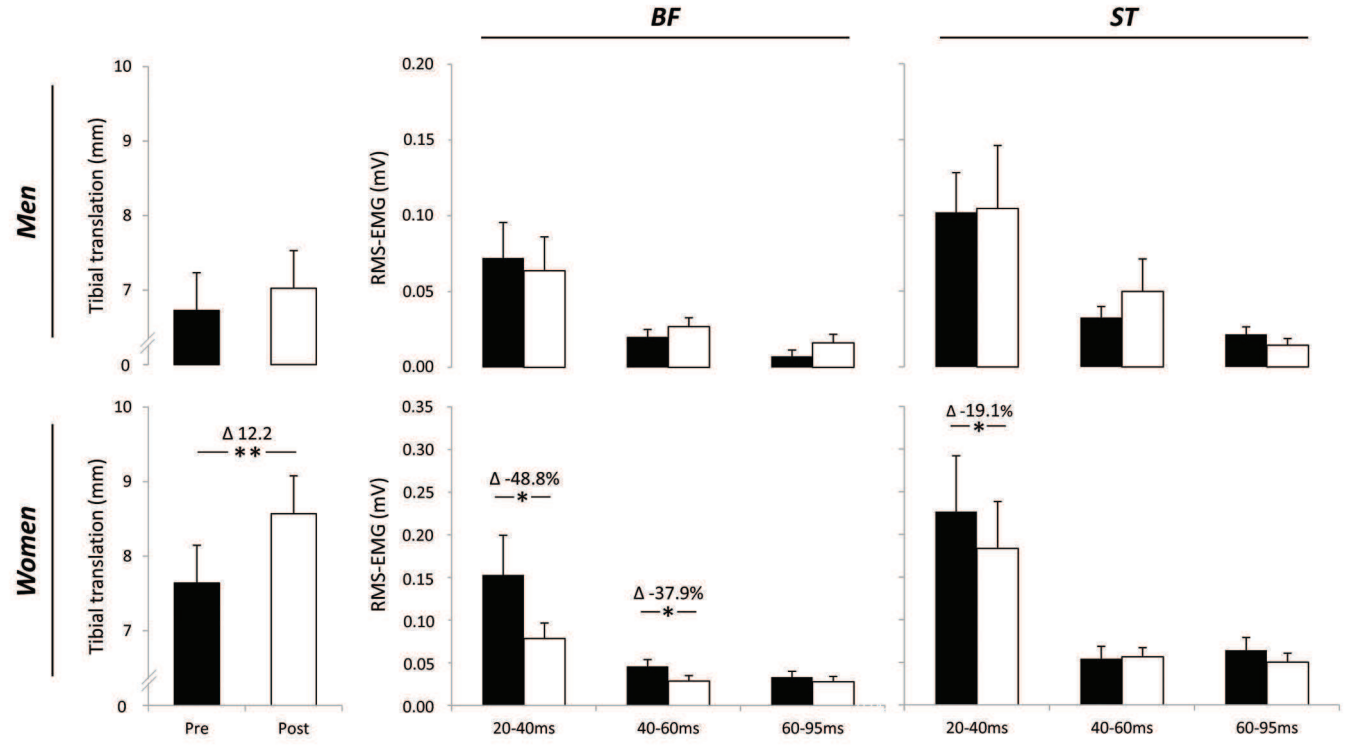

Figure 3. Effect of fatigue on tibial translation (left) and reflex responses (middle and right). Filled bars: Pre, open bars: Post, BF: biceps femoris, ST: semitendinosus/semimembranosus. Data are displayed as means ± standard error of the mean. * denotes a significant difference compared to the pre-measurement, *$P \leq 0.05$; **$P \leq 0.01$.
doi:10.1371/journal.pone.0056988.g003

Table 1. Force applied to the proximal shank of the dominant leg and EMG onset latencies before and after the fatigue protocol for men and women. In addition, weight-normalized isometric maximum voluntary torque (iMVT), weight-normalized rate of torque development ($RTD_{0–50,\ 0–100,\ 0–200}$) of the hamstrings (H) and quadriceps (Q), MVT H/Q ratio and RTD H/Q ratios ($RTD_{0–50,\ 0–100,\ 0–200}$ H/Q) for men and women.

Parameter	Men			Women		
	Pre	*Post*	*P*	*Pre*	*Post*	*P*
Force (N)	249.73±11.48	243.74±22.32	NS (0.220)	241.48±13.53	240.15±12.29	NS (0.468)
EMG onset latencies (ms)						
BF	23.60±2.57	24.59±2.17	NS (0.080) ES = 0.47	22.10±2.23	23.05±2.56 *	0.022 (ES = 0.57)
ST	22.92±2.59	24.92±2.27	NS (0.058) ES = 0.61	22.37±2.30	23.42±2.54 *	0.017 (ES = 0.62)
	H	*Q*		*H*	*Q*	
iMVT ($N{\cdot}m{\cdot}kg^{-1}$)	1.21±0.20 ††	2.07±0.35 ††		0.87±0.16 ††	1.79±0.33 ††	
RTD ($N{\cdot}m{\cdot}s^{-1}{\cdot}kg^{-1}$)						
0–50 ms	2.76±2.77	10.80±6.61		1.72±1.01	7.37±4.38	
0–100 ms	5.72±2.06 ††	10.93±4.18 †		3.75±1.42 ††	8.29±3.18 †	
0–200 ms	4.70±0.94 ††	7.90±2.00		3.32±0.77 ††	6.83±1.73	
	H/Q			*H/Q*		
MVT H/Q ratio	0.59±0.08 ††			0.50±0.10 ††		
RTD H/Q ratio						
0–50 ms	0.50±0.53			0.46±0.71		
0–100 ms	0.63±0.43			0.51±0.25		
0–200 ms	0.62±0.16 †			0.50±0.12 †		

BF: biceps femoris, ST: semitendinosus/semimembranosus. * denotes a significant difference compared to the pre-measurement, *$P \leq 0.05$. † indicates a significant difference between men and women, †$P \leq 0.05$; ††$P \leq 0.01$. ES = effect size. Values are means ± standard deviation.

doi:10.1371/journal.pone.0056988.t001

($F = 4.118$, $P = 0.057$, $\eta^2 = 0.186$, ES = 0.48) and 60–95 ms ($F = 1.429$, $P = 0.247$, $\eta^2 = 0.074$, ES = 0.28) were observed in men. The reflex activity of ST was not altered in the time intervals 20–40 ms ($F = 0.008$, $P = 0.930$, $\eta^2 = 0.000$, ES = 0.00), 40–60 ms ($F = 0.806$, $P = 0.382$, $\eta^2 = 0.045$, ES = 0.22) and 60–95 ms ($F = 1.744$, $P = 0.204$, $\eta^2 = 0.093$, ES = 0.32). TT did not change following the fatigue protocol in males ($F = 0.932$, $P = 0.346$, $\eta^2 = 0.047$, ES = 0.22).

The weight-normalized iMVT of the hamstring and the quadriceps muscle was significantly higher in men than in women ($P \leq 0.001$). Furthermore, weight-normalized $RTD_{0–100}$ and $RTD_{0–200}$ of the hamstrings ($P \leq 0.01$) but only $RTD_{0–100}$ of the quadriceps ($P \leq 0.05$) were significantly higher in males than in females. The MVT H/Q ratio and the $RTD_{0–200}$ H/Q ratio were significantly different between men and women ($P = 0.003$ and $P = 0.013$, respectively) (Table 1). Correlation analysis for all participants revealed no significant associations between the parameters for TT and the MVT H/Q ratio as well as RTD H/Q ratio (Table 2).

Discussion

The purpose of this study was to elucidate the effect of fatigue, induced by repetitive jumping, on reflex activity of the hamstrings and TT in men and women. In addition, we presumed that there is an association between the extent of TT and the MVT H/Q ratio as well as the RTD H/Q ratio.

Reflex onset latencies were enhanced in women after the fatiguing task. The results revealed a fatigue-induced reduction of reflex responses in women associated with an increased TT. Men showed no significant differences in the parameters after the fatigue protocol. Correlation analysis revealed no significant associations between the parameters for TT and the MVT H/Q ratio as well as the RTD H/Q ratio.

Tibial translation

It has been assumed that increased joint laxity may contribute to increased ACL injury risk [30]. Several studies have found significant increases in anterior knee laxity, for example, after running [31–33] or a regular workout in volleyball [34]. However, only Kvist et al. [34] have compared males and females after fatiguing exercise and found an increase in TT for men. Nevertheless, these studies measured anterior knee laxity while subjects were relaxed. In contrast, in the current study TT was measured in a functional weight-bearing situation. In a situation such as this, axial loading and forces due to muscle contraction could reduce rotation and translation compared to the passive condition [35,36]. The present study found an increase in TT in women but not in men after the fatiguing exercise. The ES of 0.69 indicates that the fatigue protocol induced a large effect regarding the parameter TT in females. Studies using a similar methodology have shown that an isokinetic fatigue protocol performed with a dynamometer can increase TT [13,16]. However, only the study by Wojtys et al. [13] has focused on gender-specific differences but it used a very small sample size (six men and four women). The authors reported no gender difference in any parameter.

In general, knee ligamentous structures probably undergo some increase in laxity during exercise, thereby placing athletes at risk for ligamentous injury [6]. It is assumed that this is due to the fact that joint structures, particularly the ligaments, exhibit viscoelastic properties [37]. Therefore, cyclic stress of the ligamentous structures leads to time-dependent and stress-dependent modifications and therefore increased ligamentous laxity [6,37]. However, the muscles that cross the knee joint play a large role in

Table 2. Correlations between posterior-anterior tibial translation and strength parameters for all subjects.

Parameter	MVT H/Q ratio	RTD_{0-50} H/Q ratio	RTD_{0-100} H/Q ratio	RTD_{0-200} H/Q ratio
Tibial translation Pre	−0.07	0.10	0.25	0.09
Tibial translation Post	−0.24	−0.09	0.11	−0.03
Tibial translation diff.	−0.28	−0.30	−0.21	−0.19

Tibial translation diff. stands for the difference between the tibial translation before and after fatigue.
doi:10.1371/journal.pone.0056988.t002

maintaining physiological kinematics of the knee. Muscle activity is able to induce large changes in strains as well as forces experienced by the ACL [38].

Reflex responses

The fast activation of muscles by means of reflexes may play a substantial role in the stabilization of the knee joint [14]. It has been suggested that the direct reflex arc between the ACL and the hamstrings makes only a minor contribution to the biphasic reflex response in the hamstring muscles [39]. Therefore, it has been suspected that the reflex response is mainly generated by hamstring stretch reflexes [15]. In the current study, delayed reflex onset latencies for BF and ST were found in women after the fatigue protocol. The ES for the parameters (BF = 0.57 and ST = 0.62) indicate that the fatiguing exercise had a large effect on the latencies of both muscles. The onset latencies for BF and ST in men were not statistically different after fatigue. However, ES of 0.47 and 0.61, respectively, indicate that the fatigue protocol provoked a large effect for men as well. Similar results were reported by Melnyk and Gollhofer [16] who found an increased latency of reflex responses after submaximal fatigue. The authors have argued that the slowing of reflex responses probably does not play a substantial role in functional knee stability. Nevertheless, the authors have not focused on gender-specific differences.

In the current study, the reflex response of the females was significantly reduced in BF for the time intervals 20-40 and 40–60 ms (ES = 0.45 and ES = 0.54, respectively). The same was true for ST from 20–40 ms (ES = 0.47). The results of the present study correspond with the results of Melnyk and Gollhofer [16] who found significantly decreased iEMG values for the short latency response and medium latency response of the hamstring stretch reflex after an isokinetic concentric-eccentric fatigue protocol. As before, gender-specific differences were not investigated. Moore et al. [40] have investigated vastus lateralis reflex activity induced by a standardized tendon tap with a spring-loaded reflex hammer before and after fatiguing isokinetic contractions. The authors reported a significant increase in reflex amplitude in men and a tendency to a reduction in women. They concluded that males and females might respond differently to fatigue. A reason for this could be that men and women activate their muscles differently according to the requirements of the movement task. For example, Rozzi et al. [41] have observed that women show greater muscle activity of the lateral hamstring muscle when landing from a jump, and possess increased knee joint laxity as well as longer time to detect knee joint motion compared with men. The authors concluded that the greater EMG peak amplitude and area in women might be an attempt of the nervous system to compensate for the greater joint laxity and proprioceptive deficit. Furthermore, the authors argued that an interruption of this compensatory mechanism, for example due to fatigue, might increase joint laxity that may cause ligament injury. The greater muscle activity of the hamstrings when landing from a jump in females may be an explanation for the differing results in the present study between men and women regarding reflex responses and TT. It is conceivable that the fatigue protocol used in this study, which consisted of repetitive jumps performed until exhaustion, induced more fatigue in the hamstring muscles of women and therefore impaired hamstring reflex responses.

The decrease in muscle activity in distinct time intervals in women could be explained by different physiological processes: (i) fatigue-induced changes in intrafusal properties, (ii) presynaptic inhibition (PSI) of Ia afferents and (iii) changes in intrinsic properties of motoneurons. Fatigue-induced changes in intrafusal properties are assumed to occur during sustained MVCs and probably reduce intrafusal contraction force and thereby the fusimotor-driven afferent discharge [42]. Furthermore, it has been suggested that submaximal isometric fatiguing exercise is also able to change intrafusal properties and, in turn, reflex responses [43]. Another explanation might be the decline in transmission from Ia afferents to motoneurons due to PSI mediated by group III and IV afferents [44]. These afferents are sensitive to several parameters associated with either metabolic fatigue or muscle damage [45]. It has been found that these afferents have a powerful input to inhibitory interneurons which induce PSI of Ia afferent terminals [46]. In addition, the possibility of changes in intrinsic properties of motoneurons should be taken into account [47]. It has been found that motoneurons can experience an intrinsic adaptation in firing frequency to a constant excitatory drive [48]. The decrease in jump height below 50% during the fatigue protocol and the RPE indicated that the jumping exercise was exhaustive. Therefore, it can be assumed that the exercise caused a stressful metabolic loading and thereby stimulated group III and IV afferents that probably induced PSI of Ia afferents. Furthermore, it is conceivable that the reflex inhibition of the motoneuron pool was accompanied by changes in the intrinsic properties of motoneurons.

The results of the present study revealed that the fatigue protocol used in this study altered the latency as well as magnitude of reflex responses of the hamstring muscles and TT in women. These changes were not found in men. The authors of various studies have suggested that the hamstring muscles play an important role in maintaining knee stability and that they protect the ACL during movements of the tibia relative to the femur [49–51]. Therefore, decreased reflex responses of the hamstring muscles and in turn an increased TT might contribute to the pathomechanics of knee joint injuries. It has been shown that female athletes have an increased risk for ACL injuries [2]. Based on our results it is conceivable that the fatigue-induced decrease in neuromuscular function with a corresponding increase in TT probably contributes to the higher incidence of ACL injuries in women.

Limits of the study

The measurements used in this study were performed in a functional weight-bearing situation. Only a few studies have investigated the effect of fatigue on tibial translation and muscular responses with a similar methodology [13,16]. Therefore, comparisons with studies that used another methodology, e.g. experiments that measured anterior knee laxity while subjects were relaxed [31–33], should be viewed with caution. Furthermore, a study by Bruhn et al. [23] has revealed that hamstring reflex responses are modulated according to the stimulus characteristics, i.e. stretch velocity vs. stimulus amplitude. It is possible that men and women respond differentially with regard to stimulus characteristics. In this respect, it is noteworthy that body weight may have influenced the response to the constant stimulus and in turn the observed differences in the neuromuscular response and TT. Moreover, we assume that the fatigue protocol used in this study caused a stressful metabolic loading and thereby modulated the reflex responses. Unfortunately, we have not measured metabolic data that could support this assumption.

Acknowledgments

The authors would like to thank Detlef Werner for technical support, Rike Pahnke for drawing of the measurement system and Michael Wolter as well as Daniel Lexow for their help during data acquisition.

Author Contributions

Conceived and designed the experiments: MB AM SB. Performed the experiments: MB FW AM. Analyzed the data: MB AM FW. Contributed reagents/materials/analysis tools: MB AM FW SB. Wrote the paper: MB AM FW SB.

References

1. Hootman JM, Dick R, Agel J (2007) Epidemiology of collegiate injuries for 15 sports: summary and recommendations for injury prevention initiatives. J Athl Train 42: 311–319.
2. Hewett TE, Myer GD, Ford KR (2006) Anterior cruciate ligament injuries in female athletes: Part 1, mechanisms and risk factors. Am J Sports Med 34: 299–311.
3. Hughes G, Watkins J (2006) A risk-factor model for anterior cruciate ligament injury. Sports Med 36: 411–428.
4. Miura K, Ishibashi Y, Tsuda E, Okamura Y, Otsuka H, et al. (2004) The effect of local and general fatigue on knee proprioception. Arthroscopy 20: 414–418.
5. Gribble PA, Hertel J (2004) Effect of lower-extremity muscle fatigue on postural control. Arch Phys Med Rehabil 85: 589–592.
6. Rozzi SL, Lephart SM, Fu FH (1999) Effects of Muscular Fatigue on Knee Joint Laxity and Neuromuscular Characteristics of Male and Female Athletes. J Athl Train 34: 106–114.
7. McLean SG, Fellin RE, Suedekum N, Calabrese G, Passerallo A, et al. (2007) Impact of fatigue on gender-based high-risk landing strategies. Med Sci Sports Exerc 39: 502–514.
8. Tsai LC, Sigward SM, Pollard CD, Fletcher MJ, Powers CM (2009) Effects of Fatigue and Recovery on Knee Mechanics during Side-Step Cutting. Med Sci Sports Exerc 41: 1952–1957.
9. Derrick TR, Dereu D, McLean SP (2002) Impacts and kinematic adjustments during an exhaustive run. Med Sci Sports Exerc 34: 998–1002.
10. Gehring D, Melnyk M, Gollhofer A (2009) Gender and fatigue have influence on knee joint control strategies during landing. Clin Biomech (Bristol, Avon) 24: 82–87.
11. Price RJ, Hawkins RD, Hulse MA, Hodson A (2004) The Football Association medical research programme: an audit of injuries in academy youth football. Br J Sports Med 38: 466–471.
12. Hawkins RD, Hulse MA, Wilkinson C, Hodson A, Gibson M (2001) The association football medical research programme: an audit of injuries in professional football. Br J Sports Med 35: 43–47.
13. Wojtys EM, Wylie BB, Huston LJ (1996) The effects of muscle fatigue on neuromuscular function and anterior tibial translation in healthy knees. Am J Sports Med 24: 615–621.
14. Friemert B, Franke S, Gollhofer A, Claes L, Faist M (2010) Group I afferent pathway contributes to functional knee stability. J Neurophysiol 103: 616–622.
15. Friemert B, Bumann-Melnyk M, Faist M, Schwarz W, Gerngross H, et al. (2005) Differentiation of hamstring short latency versus medium latency responses after tibia translation. Exp Brain Res 160: 1–9.
16. Melnyk M, Gollhofer A (2007) Submaximal fatigue of the hamstrings impairs specific reflex components and knee stability. Knee Surg Sports Traumatol Arthrosc 15: 525–532.
17. Aagaard P, Simonsen EB, Magnusson SP, Larsson B, Dyhre-Poulsen P (1998) A new concept for isokinetic hamstring: quadriceps muscle strength ratio. Am J Sports Med 26: 231–237.
18. Aagaard P, Simonsen EB, Beyer N, Larsson B, Magnusson P, et al. (1997) Isokinetic muscle strength and capacity for muscular knee joint stabilization in elite sailors. Int J Sports Med 18: 521–525.
19. Zebis MK, Andersen LL, Ellingsgaard H, Aagaard P (2011) Rapid hamstring/quadriceps force capacity in male vs. female elite soccer players. J Strength Cond Res 25: 1989–1993.
20. Krosshaug T, Nakamae A, Boden BP, Engebretsen L, Smith G, et al. (2007) Mechanisms of anterior cruciate ligament injury in basketball: video analysis of 39 cases. Am J Sports Med 35: 359–367.
21. Bruhn S (1999) Improved measurement of knee-joint stability. The Michael-Jager Prize for a Stuttgart research group. Orthopade 28: 819.
22. Gruber M, Bruhn S, Gollhofer A (2006) Specific adaptations of neuromuscular control and knee joint stiffness following sensorimotor training. Int J Sports Med 27: 636–641.
23. Bruhn S, Leukel C, Gollhofer A (2011) Differential effects of stimulus characteristics during knee joint perturbation on hamstring and quadriceps reflex responses. Hum Mov Sci 30: 1079–1091.
24. Behrens M, Mau-Moeller A, Bruhn S (2012) Effect of Exercise-induced Muscle Damage on Neuromuscular Function of the Quadriceps Muscle. Int J Sports Med 33: 600–606.
25. Masci I, Vannozzi G, Gizzi L, Bellotti P, Felici F (2010) Neuromechanical evidence of improved neuromuscular control around knee joint in volleyball players. Eur J Appl Physiol 108: 443–450.
26. Tillin NA, Jimenez-Reyes P, Pain MT, Folland JP (2010) Neuromuscular performance of explosive power athletes versus untrained individuals. Med Sci Sports Exerc 42: 781–790.
27. Tillin NA, Pain MT, Folland JP (2011) Short-term unilateral resistance training affects the agonist-antagonist but not the force-agonist activation relationship. Muscle Nerve 43: 375–384.
28. Faul F, Erdfelder E, Lang AG, Buchner A (2007) G*Power 3: a flexible statistical power analysis program for the social, behavioral, and biomedical sciences. Behav Res Methods 39: 175–191.
29. Cohen J (1988) Statistical Power for the Behavioral Sciences. NJ: Erlbaum. 590 p.
30. Ramesh R, Von Arx O, Azzopardi T, Schranz PJ (2005) The risk of anterior cruciate ligament rupture with generalised joint laxity. J Bone Joint Surg Br 87: 800–803.
31. Nawata K, Teshima R, Morio Y, Hagino H, Enokida M, et al. (1999) Anterior-posterior knee laxity increased by exercise. Quantitative evaluation of physiologic changes. Acta Orthop Scand 70: 261–264.
32. Johannsen HV, Lind T, Jakobsen BW, Kroner K (1989) Exercise-induced knee joint laxity in distance runners. Br J Sports Med 23: 165–168.
33. Kirkley A, Mohtadi N, Ogilvie R (2001) The effect of exercise on anterior-posterior translation of the normal knee and knees with deficient or reconstructed anterior cruciate ligaments. Am J Sports Med 29: 311–314.
34. Kvist J, Cunningham D, Tigerstrand-Wejlemark H (2006) Gender differences in post-exercise sagittal knee translation: a comparison between elite volleyball players and swimmers. Knee 13: 132–136.
35. Markolf KL, Gorek JF, Kabo JM, Shapiro MS (1990) Direct measurement of resultant forces in the anterior cruciate ligament. An in vitro study performed with a new experimental technique. J Bone Joint Surg Am 72: 557–567.
36. Victor J, Labey L, Wong P, Innocenti B, Bellemans J (2010) The influence of muscle load on tibiofemoral knee kinematics. J Orthop Res 28: 419–428.
37. Weisman G, Pope MH, Johnson RJ (1980) Cyclic loading in knee ligament injuries. Am J Sports Med 8: 24–30.
38. Takeda Y, Xerogeanes JW, Livesay GA, Fu FH, Woo SL (1994) Biomechanical function of the human anterior cruciate ligament. Arthroscopy 10: 140–147.
39. Friemert B, Faist M, Spengler C, Gerngross H, Claes L, et al. (2005) Intraoperative direct mechanical stimulation of the anterior cruciate ligament elicits short- and medium-latency hamstring reflexes. J Neurophysiol 94: 3996–4001.
40. Moore BD, Drouin J, Gansneder BM, Shultz SJ (2002) The differential effects of fatigue on reflex response timing and amplitude in males and females. J Electromyogr Kinesiol 12: 351–360.
41. Rozzi SL, Lephart SM, Gear WS, Fu FH (1999) Knee joint laxity and neuromuscular characteristics of male and female soccer and basketball players. Am J Sports Med 27: 312–319.
42. Bongiovanni LG, Hagbarth KE (1990) Tonic vibration reflexes elicited during fatigue from maximal voluntary contractions in man. J Physiol 423: 1–14.

43. Zhang LQ, Rymer WZ (2001) Reflex and intrinsic changes induced by fatigue of human elbow extensor muscles. J Neurophysiol 86: 1086–1094.
44. Duchateau J, Balestra C, Carpentier A, Hainaut K (2002) Reflex regulation during sustained and intermittent submaximal contractions in humans. J Physiol 541: 959–967.
45. Rotto DM, Kaufman MP (1988) Effect of metabolic products of muscular contraction on discharge of group III and IV afferents. J Appl Physiol 64: 2306–2313.
46. Duchateau J, Hainaut K (1993) Behaviour of short and long latency reflexes in fatigued human muscles. J Physiol 471: 787–799.
47. Racinais S, Girard O, Micallef JP, Perrey S (2007) Failed excitability of spinal motoneurons induced by prolonged running exercise. J Neurophysiol 97: 596–603.
48. Kernell D, Monster AW (1982) Motoneurone properties and motor fatigue. An intracellular study of gastrocnemius motoneurones of the cat. Exp Brain Res 46: 197–204.
49. Beard DJ, Kyberd PJ, Fergusson CM, Dodd CA (1993) Proprioception after rupture of the anterior cruciate ligament. An objective indication of the need for surgery? J Bone Joint Surg Br 75: 311–315.
50. Johansson H, Sjolander P, Sojka P (1990) Activity in receptor afferents from the anterior cruciate ligament evokes reflex effects on fusimotor neurones. Neurosci Res 8: 54–59.
51. More RC, Karras BT, Neiman R, Fritschy D, Woo SL, et al. (1993) Hamstrings--an anterior cruciate ligament protagonist. An in vitro study. Am J Sports Med 21: 231–237.

Effect of Exercise-induced Muscle Damage on Neuromuscular Function of the Quadriceps Muscle

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Key words
- muscle damage
- spinal excitability
- twitch contraction
- interpolated twitch technique

Abstract

Exercise-induced muscle injury is commonly accompanied by a reduction of muscular strength. It has been suggested that this reduction in voluntary force is attributable to "peripheral" and "central" mechanisms within the neuromuscular system. The quadriceps muscle of 15 subjects was damaged with four bouts of 25 maximal voluntary concentric-eccentric contractions at a speed of 60 °/s. In a time period of 7 days, we investigated the contribution of agonist muscle activation and contractile properties (CP) to changes in isometric maximum voluntary torque (iMVT). In order to provide a comprehensive assessment, the neural drive to muscles was estimated with the interpolated twitch technique and root mean square of the EMG signal. CP were evaluated by analysing the twitch torque signal induced by single and doublet stimulation. Furthermore, we measured changes in alpha motoneuron excitability of vastus medialis at the spinal level due to muscle soreness using the H reflex technique. The iMVT was impaired at post, 24 h and 48 h, while rate of torque development and voluntary activation (VA) were only decreased immediately after the intervention. CP were impaired immediately after exercise and at 24 h. Maximal H reflex (H_{max}), maximal M wave (M_{max}) and the H_{max}/M_{max}-ratio were not affected. Sensation of muscle soreness assessment revealed impairments at 24 h, 48 h and 72 h. Data suggest that reduced VA and altered CP contribute to the force loss immediately after concentric-eccentric exercise. Thereafter, the impairment of CP seems to be mainly responsible for the reduced iMVT. In addition, there is no evidence for an association between muscle soreness and VA as well as between muscle soreness and spinal excitability.

accepted after revision
January 26, 2012

Bibliography
DOI http://dx.doi.org/10.1055/s-0032-1304642
Published online: 2012
Int J Sports Med

ISSN 0172-4622

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Introduction

It has been shown that unaccustomed eccentric exercise, which involves the active lengthening of muscles, is frequently followed by muscle damage [7]. It has been suggested that the reduced voluntary force due to muscle damage is attributable to "peripheral" and "central" mechanisms within the neuromuscular system [28]. "Peripheral" mechanisms include disturbance to excitation-contraction coupling [42] and disruption at the level of sarcomeres [30]. The "central" mechanism is an inadequate voluntary activation of muscles [29,31]. It has been assumed that the reduced neural drive to the muscles is an attempt of the neuromuscular system to prevent further injury of the muscle-tendon unit [31]. The muscle pain associated with muscle damage after eccentric exercise is believed to reflect activity in group III and IV muscle afferents [27] which has the ability to induce modulations at the spinal level [3] and/or in the motor cortex [21]. It has been suggested that these modulations impair voluntary activation of muscles [29]. Several studies have investigated neuromuscular function of the elbow flexors [28,29] and the plantar flexors in relation to muscle damage [31]. They have shown that contractile properties of muscles were impaired. However, these studies have found conflicting results concerning voluntary activation. While Prasartwuth et al. [29] have observed no change in voluntary activation of the elbow flexors. Racinais et al. [31] have shown a decrease in voluntary activation of the plantar flexors up to 48 h after exercise. Another study of Prasartwuth et al. [28] suggests that the relation of voluntary activation and exercise-induced muscle damage depends on the length of muscles. The authors have found a significant decrease in voluntary activation of the elbow

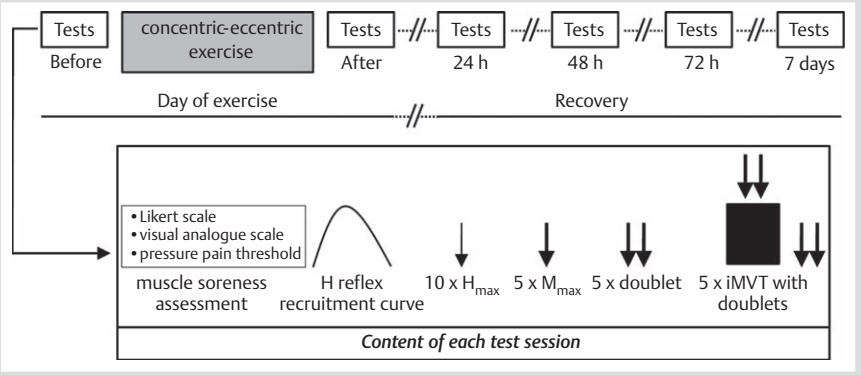

Fig. 1 Experimental design. The thin arrow indicates stimulation at H_{max} intensity, the thick arrow indicates stimulation at supramaximal intensity, double thick arrow indicates doublet at supramaximal intensity.

flexors only at short muscle lengths. The knee extensors have already been investigated with regard to muscle damage but these studies were primarily interested in voluntary force production [35,43] or the effect of different recovery modes [4,22]. Consequently, there is some lack of clarity about the underlying neuromuscular mechanisms with regard to exercise-induced muscle damage of the quadriceps muscle. In this context, it is of interest whether voluntary activation and/or spinal excitability would be linked to muscle damage. The neuromuscular function of the quadriceps in relation to muscle soreness is of particular interest because this muscle is crucial for many daily and sports activities, e.g. walking, running and jumping. Therefore, in the present study we damaged the quadriceps muscle with concentric-eccentric exercise and induced muscle soreness. In the following time period of seven days we investigated the contribution of agonist muscle activation and contractile properties to changes in isometric maximum voluntary torque (iMVT). In order to provide a comprehensive assessment, the neural drive to muscles was estimated with the interpolated twitch technique and root mean square of the EMG signal (RMS-EMG) during iMVT normalized to the maximal M wave (M_{max}). Contractile properties were evaluated by analysing the twitch torque signal induced by electrical stimulation. Furthermore, we measured changes in alpha motoneuron excitability at the spinal level due to muscle soreness using the H reflex technique. We hypothesized that exercise-induced muscle damage would reduce voluntary force and voluntary activation. Furthermore, we assumed that exercise-induced muscle damage would impair contractile properties and modify spinal excitability.

Materials and Methods

Subjects

Fifteen subjects (8 males, 7 females, 27.2 ± 3.8 yrs, 68.5 ± 10.6 kg, 174.2 ± 7.4 cm) with no history of neurological disorders or injuries participated. Before testing, subjects were instructed to refrain from consuming alcohol and caffeine in the 24 h preceding the experiment and not to perform any strenuous exercise in the 48 h previous to the experiment. All persons signed informed consent prior to investigation. The study was conducted according to the declaration of Helsinki and was approved by the university ethics committee. The study meets the ethical standards of the journal [18].

Experimental procedure

The subjects participated in 5 sessions at the laboratory (**Fig. 1**). No warm-up was realized before each experiment in order to avoid H reflex and M wave potentiation [13]. The concentric-eccentric exercise and the measurements were done with the quadriceps muscle of the right leg using a CYBEX NORM dynamometer (Computer Sports Medicine®, Inc., Stoughton, MA). Throughout the testing sessions the subjects were comfortably seated in a standardized position on the dynamometer. Before testing the subjects sat passively on the dynamometer for ~15 min in order to minimize potentiation effects from walking to the laboratory. The first experimental session consisted of neuromuscular tests followed by concentric-eccentric exercise followed by further neuromuscular testing immediately after the exercise. On the second, third, fourth and seventh day, subjects returned to the laboratory and were investigated again. The content of each session is displayed in **Fig. 1**. The exercise consisted of four bouts of 25 maximal voluntary concentric-eccentric contractions at a speed of 60°/s, between 90° and 170° of knee joint angle [19]. A rest period of 3 min was allowed between the bouts.

Electrical stimulation

Transcutaneous electrical femoral nerve stimulation was used to produce the stimulus-response curve. A hand-held stimulation probe was used to locate the optimum site of stimulation. Consequently, the femoral nerve was stimulated using a cathode ball electrode which was fixed to the subject's femoral triangle, 3–5 cm below the inguinal ligament. The anode was a self-adhesive electrode (35 × 45 mm, Spes Medica, Italy) placed over the greater trochanter. The percutaneous electrical stimuli were single (1 ms duration, 400 V) and paired rectangular pulses (1 ms duration, 10 ms apart, 400 V) delivered by a constant-current stimulator (Digitimer® DS7A, Hertfordshire, UK). Constant inter stimulus intervals (ISI) were provided by a Digitimer® train/delay generator (DG2A, Hertfordshire, UK). The testing procedure included random stimulation (ISI 10 s, i.e., 0.1 Hz) with different current intensities, resulting in a recruitment curve, until identification of peak-to-peak maximal H reflex (H_{max}) and M_{max} of vastus medialis [13,15]. The 5–10 current intensities around H_{max} were then repeated with two stimuli given at each current. Afterwards H_{max} and M_{max} were elicited and recorded 10 and 5 times, respectively. An example of the evoked potentials of one representative subject is displayed in **Fig. 2**. A stimulation intensity of 40% greater than that needed for maximal twitch response and concomitant M_{max} was used for evaluating contractile properties and voluntary activation. Resting

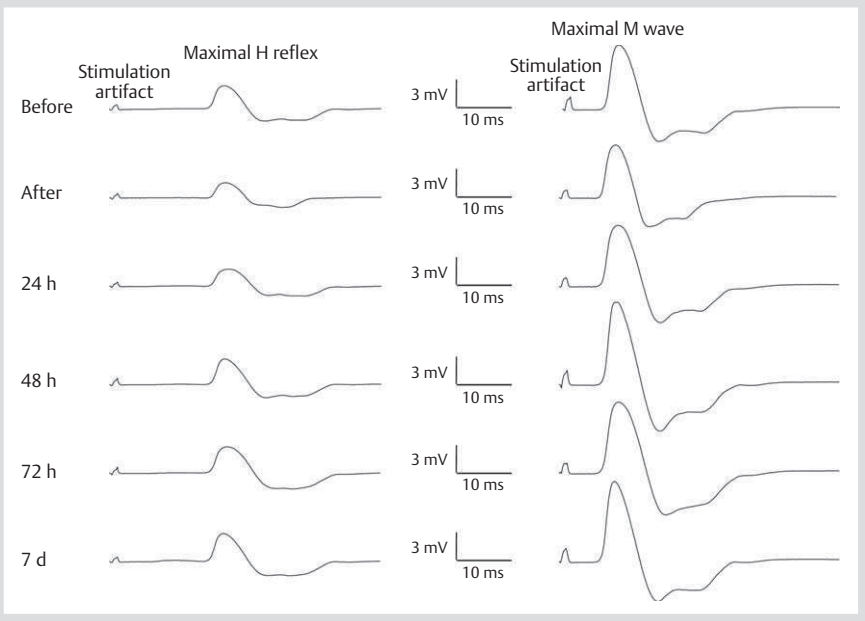

Fig. 2 Evoked potentials of one subject at the different points in time. Each drawing represents the average of ten maximal H reflexes and 5 maximal M waves, respectively.

twitch responses were evoked using single and doublet stimulation. Voluntary activation was assessed using the interpolated twitch technique [2], i.e., 2 s after torque onset, during the plateau phase, and 2 s after the end of the maximal voluntary contraction a transcutaneous supramaximal doublet was given to the femoral nerve.

EMG and torque recordings

To record bipolar surface EMG Ambu® Blue Sensor N electrodes (2 cm diameter) were used that were firmly attached to the shaved, abraded and cleaned skin over vastus medialis, vastus lateralis and rectus femoris muscle of the right leg. The resistance between electrodes was measured with a digital multimeter (MY-68, McVoice, Germany) and kept below 5 kΩ. The electrodes were applied with a center-to-center distance of 2 cm over the muscle bellies. The recording electrodes were in line with the presumed direction of the underlying muscle fibres. The reference electrode was attached to the patella of the ipsilateral leg. Signals were amplified (2 500×), band-pass filtered (10–450 Hz) and digitized with a sampling frequency of 5 kHz through an analogue-to-digital converter (DAQ Card™-6024E, National Instruments, USA). Both, the EMG and torque signals were sampled at 5 kHz and stored on a hard drive for later analysis with a custom built LABVIEW® based program (Imago, Pfisoft, Germany).

Torque signals were measured using a CYBEX NORM dynamometer (Computer Sports Medicine®, Inc., Stoughton, MA). The knee and hip joint angles of 80° (0°=full extension), respectively, were consistently maintained during the experiments. The axis of the dynamometer was aligned with the anatomical knee flexion-extension axis and the lever arm was attached to the anterior aspect of the shank 2–3 cm above the lateral malleolus. Straps across the waist and the chest prevented excessive movements. The iMVT was tested by asking the subjects to exert isometric knee extensions against the lever arm of the dynamometer for 3 s. For each trial, subjects were thoroughly instructed to act as forcefully and as fast as possible. They were motivated by strong verbal encouragement and online visual feedback of the instantaneous dynamometer torque provided on a digital oscilloscope (HM1508, HAMEG Instruments, Germany). Care was taken that the iMVT trials were performed without an apparent countermovement or pre-tension (change of baseline force <0.3 Nm during the 200 ms prior to contraction onset). A rest period of 2 min was allowed between the trials. The maximal attempts were recorded until the coefficient of variance of 5 subsequent trials was below 5%. The participants performed up to 10 maximal voluntary contractions.

Muscle soreness assessment

The sensation of muscle soreness was assessed using the Likert scale with 7 items (from 0: no pain to 6: severe pain), the visual analogue scale of 10 cm (0 cm: no pain, 10 cm: extreme pain) and the pressure pain threshold. Pressure pain threshold was measured over the vastus medialis, vastus lateralis and rectus femoris muscle using a stamp and an air pressure recorder (Kikuhime, TT MediTrade, Denmark). The stamp was placed on the relevant muscles in a vertical position and was pressed down. The air pressure recorder was located between the stamp and the muscles. The subjects were asked to announce any pain due to muscle soreness and then the indicating pressure was recorded as the pressure pain threshold.

Data analysis

H_{max} and M_{max} amplitudes were measured peak-to-peak. The H_{max}/M_{max}-ratio was calculated which can be considered as a global index of modulations at the spinal level due to alterations in alpha motoneuron excitability and/or presynaptic inhibition of primary muscle spindle afferents [44]. The resting twitch torques were analyzed regarding their (i) peak torque (PT), i.e. the highest value of twitch torque signal; (ii) maximal rate of torque development ($MRTD_{TT}$), i.e. the highest value of the first derivative of the twitch torque signal (time interval between data points=0.2 ms) and (iii) maximal rate of torque relaxation ($MRTR_{TT}$), i.e. the lowest value of the first derivative of the twitch torque signal (time interval between data points=0.2 ms). Voluntary activation was calculated with the formula $\%VA = (1 - \text{superimposed twitch} \times (T_b/\text{iMVT}) \times \text{control twitch}^{-1}) \times 100$ [22,36], where T_b is the torque level immediately before the superimposed twitch and iMVT the maximum voluntary torque. This formula counteracts the problem that, in some cases, the

instant the superimposed doublet is delivered does not represent the maximal torque level. In order to bypass this limitation, Strojnik and Komi [36] suggested a correction of the original equation that takes the torque level before the superimposed twitch and the maximal torque into account. In the corrected trials T_b was ≥95% of iMVT. The trials that did not meet this criterion were discarded from the analysis. The best maximal voluntary contractions were taken for further analyses, i.e. the attempt with the highest iMVT and the highest rate of torque development (RTD), respectively. The following parameters were calculated: iMVT, RTD (RTD_{iMVT}), i.e., the average of the differentiated torque signal over a time period of 0–200 ms and RMS of the EMG signal during a 200 ms period at iMVT (RMS-EMG_{iMVT}), i.e., 200 ms prior to the electrical stimuli if the maximal torque level was reached at the instant the superimposed stimuli were delivered and 100 ms on either side of iMVT if the maximal torque level was reached before the superimposed stimuli were administered. In order to normalize muscle activity, RMS-EMG_{iMVT} of vastus medialis, vastus lateralis and rectus femoris was divided by their respective M_{max} values (RMS-EMG_{iMVT}/M_{max}) [39]. Furthermore, the average sum of RMS-EMG_{iMVT}/M_{max} of vastus medialis, vastus lateralis and rectus femoris ($Ø\sum$RMS-EMG_{iMVT}/M_{max}) was calculated.

Statistical analysis

Data were checked for normal distribution using the Kolmogorov-Smirnov test. Differences between the values before and after the concentric-eccentric exercise were tested for significance by one-way repeated measures ANOVA. Further, the confidence interval was adjusted using the Bonferroni correction in order to counteract the problem of multiple comparisons. In each case the level of significance was established at $p \leq 0.05$. SPSS 19 (SPSS Inc., Chicago, IL, USA) was used for statistical analysis. Data are presented as group mean values ± standard error of the mean in the figures and as group mean values ± standard deviation in the table.

Results

Isometric maximum voluntary torque, rate of torque development, voluntary activation and contractile properties

The iMVT was significantly impaired at post (−40.4%, p=0.001), at 24 h (−25.2%, p=0.005) and at 48 h (−27.1%, p=0.016) (**Fig. 3a**), while voluntary activation and RTD_{iMVT} were only significantly decreased immediately after the intervention (−16.34%, p<0.001; −40.42%, p<0.001, respectively) (**Fig. 3b, c**). Peak torques of the resting twitch responses of the quadriceps, produced by single and doublet stimulation, were significantly reduced immediately after the maximal voluntary concentric-eccentric exercise (−50.1%, single: p=0.001; −35.7%, doublet: p<0.001) and at 24 h (−24.2%, single: p=0.046; −17.6%, doublet: p=0.007) (**Fig. 3d**). $MRTD_{TT}$ of the resting twitch torques, induced by single and doublet stimulation, revealed a significant impairment immediately after exercise (−44.3%, single: p=0.028; −28.0%, doublet: p=0.006) (**Fig. 3e**). $MRTR_{TT}$ of the resting twitch torque, induced by single stimulation, was only decreased immediately after the intervention (−41.2%, p=0.011), whereas $MRTR_{TT}$, induced by doublet stimulation, was significantly decreased after exercise (−34.9%, p=0.017) and at 24 h (−27.6%, doublet: p=0.016) (**Fig. 3f**).

Evoked potentials and muscle activity

The maximal voluntary concentric-eccentric exercise did not affect H_{max}, M_{max} and the H_{max}/M_{max}-ratio of vastus medialis. Furthermore, M_{max} of vastus lateralis and rectus femoris did not change significantly. Muscle activity expressed by the average sum of RMS-EMG_{iMVT}/M_{max} of vastus medialis, vastus lateralis and rectus femoris ($Ø\sum$RMS-EMG_{iMVT}/M_{max}) was significantly reduced only after exercise (−27.3%, p=0.011). However, the RMS-EMG_{iMVT}/M_{max}-ratio of the individual muscles was not significantly different after the exercise, at 24 h, 48 h, 72 h and 7 days but tended towards a reduction immediately after the concentric-eccentric contractions (vastus medialis: −24.7%, p=0.072; vastus lateralis: −32,9%, p=0.083; rectus femoris: −28.0%, p=0.075). The mean values ± standard deviations are displayed in **Table 1**.

Muscle soreness

Sensation of muscle soreness assessment using the Likert scale, visual analogue scale and pressure pain threshold revealed significant impairments at 24 h, 48 h and 72 h ($p \leq 0.002$, for all assessments) (**Fig. 4**; pressure pain threshold is not displayed). Sensation of muscle soreness peaked at 48 h and recovered thereafter.

Discussion

The maximal voluntary concentric-eccentric exercise induced a significant decrease in iMVT of the quadriceps muscle. The pronounced reduction of iMVT and RTD_{iMVT} immediately after the exercise appeared to be caused by an alteration in voluntary activation and muscle contractile properties. The decreased voluntary activation and resting twitch torque after the intervention indicate a central modulation [17] and the occurrence of peripheral alterations [24]. The iMVT showed a clear significant reduction until 48 h after exercise and a slow recovery towards control values. Similar reductions in MVT were found after eccentric exercise of the elbow flexors [28], the knee extensors [22] and the plantar flexors [31]. The decreased iMVT in this study was associated with decreased contractile properties of the quadriceps at 24 h. Voluntary activation was not significantly impaired at 24 h (−3.8%, p=0.132) and 48 h (−4.4%, p=0.184), but tended towards a reduction. At 48 h probably both, the non-significant reduction in voluntary activation and contractile properties (−19.3%, single: p=0.324; −16.0%, doublet: p=0.247), respectively, contributed to the persistently decreased iMVT (−27.1%, p=0.016). Sensation of muscle soreness occurred 24 h, 48 h and 72 h after concentric-eccentric exercise. However, changes in voluntary activation did not follow this time course.

It has been shown that fatiguing exercise has the ability to change voluntary activation [25, 34] which was the case in the present study immediately after the concentric-eccentric exercise. Muscle activity expressed by the $Ø\sum$RMS-EMG_{iMVT}/M_{max}-ratio, that takes the activity of all investigated muscles into account, was significantly reduced directly after exercise. This result is in accordance with the decrement of voluntary activation. The RMS-EMG_{iMVT}/M_{max}-ratio of vastus lateralis, vastus medialis and rectus femoris, respectively, was not significantly different in the time period of seven days but tended toward a reduction immediately after the concentric-eccentric exercise. Several studies have shown that eccentric exercise decreases surface EMG amplitude during isometric maximal voluntary

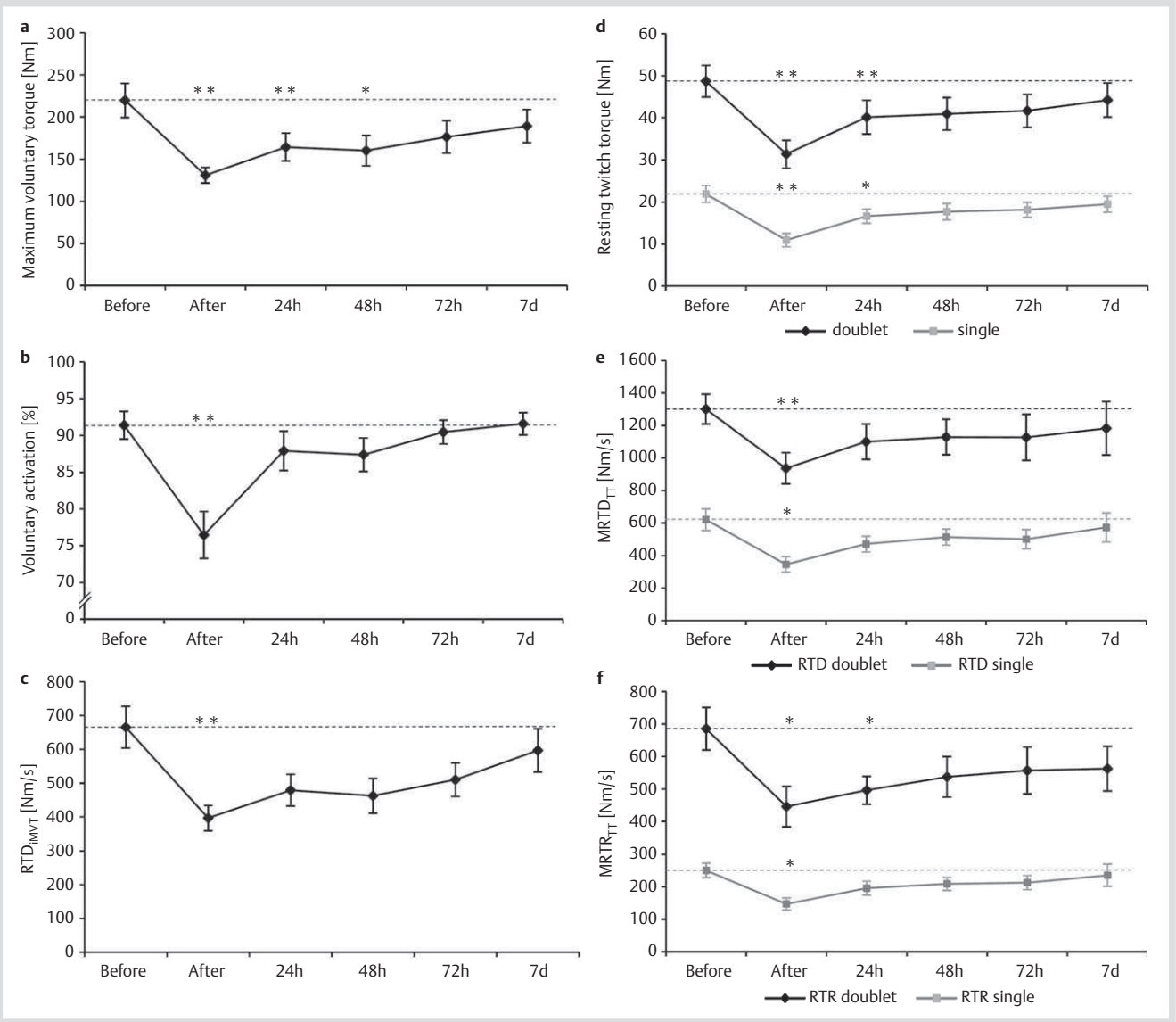

Fig. 3 **a** Isometric maximum voluntary torque (iMVT), **b** voluntary activation, **c** rate of torque development (RTD_{iMVT}), **d** resting twitch torque, **e** maximal rate of torque development of the resting twitch torque ($MRTD_{TT}$), **f** maximal rate of torque relaxation of the resting twitch torque ($MRTR_{TT}$). *denotes a significant difference to the pre-value (* ≤ 0.05; ** ≤ 0.01). Data are presented as means ± standard error of the mean.

Table 1 Maximal M wave (M_{max}), maximal H reflex (H_{max}), H_{max}/M_{max}-ratio and RMS-EMG_{iMVT}/M_{max} of vastus medialis (VM) as well as M_{max} and RMS-EMG_{iMVT}/M_{max} of vastus lateralis (VL) and rectus femoris (RF) at the different points in time. Furthermore, the average sum of RMS-EMG_{iMVT}/M_{max} of VM, VL and RF ($Ø\sum$RMS-EMG_{iMVT}/M_{max}) is displayed. Data are means (± SD). *denotes a significant difference to the pre-value. (RM ANOVA = one-way repeated measures ANOVA).

	Day of exercise		Recovery				RM ANOVA
	Before	After	24 h	48 h	72 h	7 days	
Vastus medialis							
M_{max}(mV)	9.28 (3.10)	8.74 (2.87)	9.04 (2.45)	8.86 (3.49)	8.23 (3.05)	8.94 (2.78)	NS
H_{max}(mV)	2.12 (1.75)	1.90 (1.77)	1.73 (1.70)	1.84 (1.94)	1.71 (1.63)	1.88 (1.69)	NS
H_{max}/M_{max}-ratio	0.24 (0.14)	0.21 (0.12)	0.19 (0.14)	0.19 (0.13)	0.21 (0.14)	0.21 (0.14)	NS
RMS-EMG_{iMVT}/M_{max}	0.054 (0.023)	0.041 (0.019)	0.049 (0.014)	0.044 (0.019)	0.048 (0.017)	0.045 (0.026)	NS
Vastus lateralis							
M_{max}(mV)	8.37 (3.84)	7.50 (3.39)	7.50 (3.20)	8.27 (3.75)	7.29 (3.37)	8.17 (2.96)	NS
RMS-EMG_{iMVT}/M_{max}	0.045 (0.022)	0.030 (0.019)	0.048 (0.021)	0.041 (0.016)	0.046 (0.027)	0.050 (0.027)	NS
Rectus femoris							
M_{max}(mV)	3.64 (2.03)	3.52 (2.11)	3.41 (2.07)	3.39 (2.25)	2.75 (1.84)	2.86 (1.63)	NS
RMS-EMG_{iMVT}/M_{max}	0.054 (0.022)	0.039 (0.024)	0.059 (0.023)	0.043 (0.017)	0.044 (0.018)	0.045 (0.019)	NS
VM, VL & RF							
$Ø\sum$RMS-EMG_{iMVT}/M_{max}	0.051 (0.018)	0.037 (0.015)*	0.052 (0.016)	0.043 (0.012)	0.046 (0.013)	0.046 (0.019)	p = 0.013

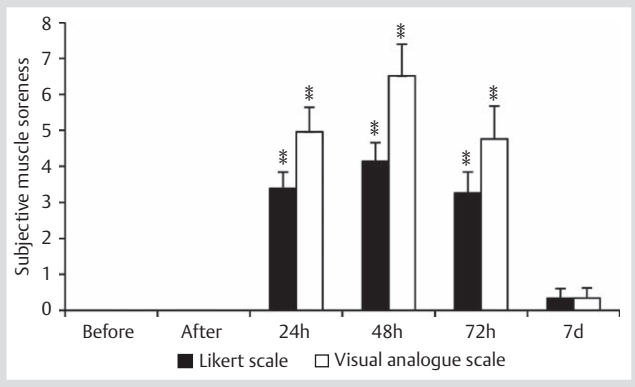

Fig. 4 Subjective muscle soreness measured in the experimental sessions (assessed using the Likert scale and visual analogue scale). *denotes a significant difference to the pre-value (** ≤ 0.01). Data are presented as means ± standard error of the mean.

contractions [23,41], while others did not [33,40]. Although surface EMG has been classically used to estimate the neural drive to muscles, it is only a crude indicator of the neural activation of muscles [11,12]. Furthermore, studies of Dideriksen et al. [8,9] have demonstrated that the estimation of neural drive using surface EMG amplitude should be viewed critically. The potential sites of muscle damage-induced "central modulation" were assumed within the motor cortex and at the supraspinal as well as the spinal level [16]. It has been argued, that muscle soreness has the potential to increase discharge of group III and IV afferents [27]. Due to the fact that group III and IV afferents have little or no background discharge their activation induces pronounced increases in their input to the central nervous system [16]. Therefore, it has been suggested that these afferents have the ability to induce modulations in the motor cortex [21] and/or at the spinal level [3]. In the present study the interpolated twitch technique was used for the assessment of voluntary activation [26]. This method measures the drive of the motoneurons to the muscles but does not assess the descending drive reaching the motoneurons [38]. Therefore, the used technique is not capable of distinguishing between alterations in the motor cortex and at the supraspinal level. In this study sensation of muscle soreness occurred 24 h, 48 h and 72 h after concentric-eccentric exercise. However, changes in voluntary activation did not follow this time course indicating that muscle pain does not necessarily change voluntary activation of the quadriceps significantly. This is in line with the results of Prasartwuth et al. [29] who failed to show an association between muscle soreness and voluntary drive of the elbow flexor muscles examined at 90° of elbow flexion. In contrast, Racinais et al. [31] have found a decreased voluntary activation of the plantar flexors in relation to muscle soreness induced by backward downhill walking. Another study of Prasartwuth et al. [28] suggests that the relation of voluntary activation and muscle soreness depends on the length of muscles. The authors have found a significant decrease in voluntary activation of the elbow flexors only at short muscle lengths, i.e., 60° and 70° of elbow flexion. In this context, it is noteworthy that there are some methodological issues in the use of the interpolated twitch technique [14]. The evaluation of voluntary activation with the standard formula %VA = (1 − superimposed twitch × control twitch^{-1}) × 100 [2] calculates the activation of muscles at the instant the superimposed stimulus is delivered. This is unlikely to be the moment of the maximal torque level because of the fluctuations of the torque signal. In order to bypass this limitation, we used a correction of the original equation that takes the torque level before the superimposed twitch and the maximal torque into account [36]. Furthermore, it is unlikely that the relationship between the superimposed twitch torque and the voluntary torque is linear. Therefore Folland and Williams [14] recommend to calculate voluntary activation on the basis of extrapolation of the twitch-voluntary force relationship, which was not done in the present study.

Besides the output of motoneurons, it is of interest to look at modulations in the reflex pathways in relation to muscle soreness. This study used the H reflex technique in order to detect modulations at the spinal level [32] due to muscle soreness. In this context, the alteration in the activity of group III and IV afferents in relation to muscle soreness is of interest. It has been suggested that increased discharge of these afferents can induce presynaptic inhibition of primary muscle spindle afferents [3,10] and therefore reduce H reflex amplitude. However, the results of the present study failed to show significant modulations in alpha motoneuron excitability and/or presynaptic inhibition of primary muscle spindle afferents of vastus medialis.

The concentric-eccentric exercise induced profound changes in contractile properties of the quadriceps muscle. These alterations were previously observed after muscle damage [29]. The M wave amplitudes of vastus medialis, vastus lateralis and rectus femoris did not change significantly after the exercise indicating that altered excitation of the sarcolemma seems unlikely to have caused the decrease in contractile properties. Therefore, it is suggested that the impairment of excitation-contraction coupling seems a rational explanation. In animal experiments, it has been shown that eccentric exercise, as downhill running in rats, is capable of rupturing the t-tubules and hence is able to impair the excitation-contraction coupling process [37]. Furthermore, eccentric damage of muscles decreases tetanic intracellular calcium concentration [5,6]. Due to this failure of tetanic calcium release the activation of contractile proteins is reduced [1,6]. In addition to this, reduced calcium sensitivity might contribute to the reduced force production [5]. A further consideration is that changes in contractile properties after the concentric-eccentric exercise may involve changes in the length-tension relation of the quadriceps. There is evidence that the peak of this relation shifts towards longer muscle lengths [20,28]. It has been suggested that non-uniform lengthening and disruption of sarcomeres are responsible for it [7]. Consequently, muscle length must be increased in order to produce the same myofilament overlap. Therefore, probably both, impairment of excitation-contraction coupling and the shift of the length-tension relation contributed to the changes in contractile properties.

In summary, data suggest that reduced voluntary activation and contractile properties contribute to the force loss immediately after concentric-eccentric exercise. In the time period thereafter, the impairment of contractile properties seems to be mainly responsible for the reduced iMVT. In addition, there is no evidence for an association between muscle soreness and voluntary activation as well as between muscle soreness and spinal excitability in the present study.

Acknowledgements

The authors would like to thank Detlef Werner for the technical support and Franziska Wassermann for her help during data acquisition. This study was supported by a grant of LGF M-V (Landesgraduiertenfoerderung Mecklenburg Vorpommern).

References

1 *Allen DG*. Skeletal muscle function: role of ionic changes in fatigue, damage and disease. Clin Exp Pharmacol Physiol 2004; 31: 485–493
2 *Allen GM*, *Gandevia SC*, *McKenzie DK*. Reliability of measurements of muscle strength and voluntary activation using twitch interpolation. Muscle Nerve 1995; 18: 593–600
3 *Avela J*, *Finni J*, *Komi PV*. Excitability of the soleus reflex arc during intensive stretch-shortening cycle exercise in two power-trained athlete groups. Eur J Appl Physiol 2006; 97: 486–493
4 *Bakhtiary AH*, *Safavi-Farokhi Z*, *Aminian-Far A*. Influence of vibration on delayed onset of muscle soreness following eccentric exercise. Br J Sports Med 2007; 41: 145–148
5 *Balnave CD*, *Allen DG*. Intracellular calcium and force in single mouse muscle fibres following repeated contractions with stretch. J Physiol 1995; 488: 25–36
6 *Balnave CD*, *Davey DF*, *Allen DG*. Distribution of sarcomere length and intracellular calcium in mouse skeletal muscle following stretch-induced injury. J Physiol 1997; 502: 649–659
7 *Byrne C*, *Twist C*, *Eston R*. Neuromuscular function after exercise-induced muscle damage: theoretical and applied implications. Sports Med 2004; 34: 49–69
8 *Dideriksen JL*, *Enoka RM*, *Farina D*. Neuromuscular adjustments that constrain submaximal EMG amplitude at task failure of sustained isometric contractions. J Appl Physiol 2011; 111: 485–494
9 *Dideriksen JL*, *Farina D*, *Enoka RM*. Influence of fatigue on the simulated relation between the amplitude of the surface electromyogram and muscle force. Philos Transact A Math Phys Eng Sci 2010; 368: 2765–2781
10 *Duchateau J*, *Balestra C*, *Carpentier A*, *Hainaut K*. Reflex regulation during sustained and intermittent submaximal contractions in humans. J Physiol 2002; 541: 959–967
11 *Farina D*, *Holobar A*, *Merletti R*, *Enoka RM*. Decoding the neural drive to muscles from the surface electromyogram. Clin Neurophysiol 2010; 121: 1616–1623
12 *Farina D*, *Merletti R*, *Enoka RM*. The extraction of neural strategies from the surface EMG. J Appl Physiol 2004; 96: 1486–1495
13 *Folland JP*, *Wakamatsu T*, *Fimland MS*. The influence of maximal isometric activity on twitch and H-reflex potentiation, and quadriceps femoris performance. Eur J Appl Physiol 2008; 104: 739–748
14 *Folland JP*, *Williams AG*. Methodological issues with the interpolated twitch technique. J Electromyogr Kinesiol 2007; 17: 317–327
15 *Fuchs DP*, *Sanghvi N*, *Wieser J*, *Schindler-Ivens S*. Pedaling alters the excitability and modulation of vastus medialis H-reflexes after stroke. Clin Neurophysiol 2011; 122: 2036–2043
16 *Gandevia SC*. Neural control in human muscle fatigue: changes in muscle afferents, motoneurones and motor cortical drive. Acta Physiol Scand 1998; 162: 275–283
17 *Gandevia SC*. Spinal and supraspinal factors in human muscle fatigue. Physiol Rev 2001; 81: 1725–1789
18 *Harriss DJ*, *Atkinson G*. Update – Ethical standards in sport and exercise science research. Int J Sports Med 2011; 32: 819–821
19 *Hedayatpour N*, *Falla D*, *Arendt-Nielsen L*, *Farina D*. Effect of delayed-onset muscle soreness on muscle recovery after a fatiguing isometric contraction. Scand J Med Sci Sports 2010; 20: 145–153
20 *Jones C*, *Allen T*, *Talbot J*, *Morgan DL*, *Proske U*. Changes in the mechanical properties of human and amphibian muscle after eccentric exercise. Eur J Appl Physiol 1997; 76: 21–31
21 *Martin PG*, *Weerakkody N*, *Gandevia SC*, *Taylor JL*. Group III and IV muscle afferents differentially affect the motor cortex and motoneurones in humans. J Physiol 2008; 586: 1277–1289
22 *Martin V*, *Millet GY*, *Lattier G*, *Perrod L*. Effects of recovery modes after knee extensor muscles eccentric contractions. Med Sci Sports Exerc 2004; 36: 1907–1915
23 *Michaut A*, *Pousson M*, *Babault N*, *Van Hoecke J*. Is eccentric exercise-induced torque decrease contraction type dependent? Med Sci Sports Exerc 2002; 34: 1003–1008
24 *Millet GY*, *Lepers R*. Alterations of neuromuscular function after prolonged running, cycling and skiing exercises. Sports Med 2004; 34: 105–116
25 *Millet GY*, *Martin V*, *Lattier G*, *Ballay Y*. Mechanisms contributing to knee extensor strength loss after prolonged running exercise. J Appl Physiol 2003; 94: 193–198
26 *Morton JP*, *Atkinson G*, *MacLaren DP*, *Cable NT*, *Gilbert G*, *Broome C*, *McArdle A*, *Drust B*. Reliability of maximal muscle force and voluntary activation as markers of exercise-induced muscle damage. Eur J Appl Physiol 2005; 94: 541–548
27 *O'Connor PJ*, *Cook DB*. Exercise and pain: the neurobiology, measurement, and laboratory study of pain in relation to exercise in humans. Exerc Sport Sci Rev 1999; 27: 119–166
28 *Prasartwuth O*, *Allen TJ*, *Butler JE*, *Gandevia SC*, *Taylor JL*. Length-dependent changes in voluntary activation, maximum voluntary torque and twitch responses after eccentric damage in humans. J Physiol 2006; 571: 243–252
29 *Prasartwuth O*, *Taylor JL*, *Gandevia SC*. Maximal force, voluntary activation and muscle soreness after eccentric damage to human elbow flexor muscles. J Physiol 2005; 567: 337–348
30 *Proske U*, *Morgan DL*. Muscle damage from eccentric exercise: mechanism, mechanical signs, adaptation and clinical applications. J Physiol 2001; 537: 333–345
31 *Racinais S*, *Bringard A*, *Puchaux K*, *Noakes TD*, *Perrey S*. Modulation in voluntary neural drive in relation to muscle soreness. Eur J Appl Physiol 2008; 102: 439–446
32 *Schieppati M*. The Hoffmann reflex: a means of assessing spinal reflex excitability and its descending control in man. Prog Neurobiol 1987; 28: 345–376
33 *Semmler JG*, *Tucker KJ*, *Allen TJ*, *Proske U*. Eccentric exercise increases EMG amplitude and force fluctuations during submaximal contractions of elbow flexor muscles. J Appl Physiol 2007; 103: 979–989
34 *Sidhu SK*, *Bentley DJ*, *Carroll TJ*. Locomotor exercise induces long-lasting impairments in the capacity of the human motor cortex to voluntarily activate knee extensor muscles. J Appl Physiol 2009; 106: 556–565
35 *Skurvydas A*, *Brazaitis M*, *Kamandulis S*. Muscle-damaging exercise affects isokinetic torque more at short muscle length. J Strength Cond Res 2011; 25: 1400–1406
36 *Strojnik V*, *Komi PV*. Neuromuscular fatigue after maximal stretch-shortening cycle exercise. J Appl Physiol 1998; 84: 344–350
37 *Takekura H*, *Fujinami N*, *Nishizawa T*, *Ogasawara H*, *Kasuga N*. Eccentric exercise-induced morphological changes in the membrane systems involved in excitation-contraction coupling in rat skeletal muscle. J Physiol 2001; 533: 571–583
38 *Taylor JL*. Point: the interpolated twitch does/does not provide a valid measure of the voluntary activation of muscle. J Appl Physiol 2009; 107: 354–355
39 *Tillin NA*, *Pain MT*, *Folland JP*. Short-term unilateral resistance training affects the agonist-antagonist but not the force-agonist activation relationship. Muscle Nerve 2011; 43: 375–384
40 *Turner TS*, *Tucker KJ*, *Rogasch NC*, *Semmler JG*. Impaired neuromuscular function during isometric, shortening, and lengthening contractions after exercise-induced damage to elbow flexor muscles. J Appl Physiol 2008; 105: 502–509
41 *Vila-Cha C*, *Hassanlouei H*, *Farina D*, *Falla D*. Eccentric exercise and delayed onset muscle soreness of the quadriceps induce adjustments in agonist-antagonist activity, which are dependent on the motor task. Exp Brain Res 2011, doi:10.1007/s00221-011-2942-2
42 *Warren GL*, *Ingalls CP*, *Lowe DA*, *Armstrong RB*. Excitation-contraction uncoupling: major role in contraction-induced muscle injury. Exerc Sport Sci Rev 2001; 29: 82–87
43 *Yeung SS*, *Yeung EW*. Shift of peak torque angle after eccentric exercise. Int J Sports Med 2008; 29: 251–256
44 *Zehr PE*. Considerations for use of the Hoffmann reflex in exercise studies. Eur J Appl Physiol 2002; 86: 455–468

Effect of Plyometric Training on Neural and Mechanical Properties of the Knee Extensor Muscles

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Key words
- maximal voluntary strength
- explosive voluntary strength
- voluntary activation
- angular specificity

Abstract

This study investigated neuromuscular adaptations of the knee extensors after 8 weeks of plyometric training. 23 subjects were randomly assigned to an intervention group and a control group. We measured isometric maximum voluntary torque (iMVT), rate of torque development (RTD) and impulse (IMP) over different time intervals. The neural drive to muscles was estimated with the interpolated twitch technique and normalized root mean square of the EMG signal. Contractile properties, H reflexes as well as jump height in squat jump (SJ) and countermovement jump (CMJ) were evaluated. Neuromuscular testing was performed at 2 knee angles, i.e., 80° and 45° (0° = full extension). The iMVT at 80° knee flexion was 23.1 N·m (95% CI: 0.1–46.1 N·m, $P=0.049$) higher at post-test for the intervention group compared with controls. The same was true for RTD and IMP in the time interval 0–50 ms [308.7 $N \cdot m \cdot s^{-1}$ (95% CI: 28.8–588.6 $N \cdot m \cdot s^{-1}$, $P=0.033$) and 0.32 N·m·s (95% CI: 0.05–0.60 N·m·s, $P=0.026$), respectively]. These changes were accompanied by enhanced neural drive to the quadriceps muscle. Jump height in SJ and CMJ was higher at post-test for the intervention group compared with controls. Parameters at 45° knee flexion, contractile properties and evoked potentials did not differ between groups. Although hypertrophic changes were not measured, data suggest that the training regime probably induced mainly neural adaptations that were specifically related to the knee angle. The strength gains at 80° knee flexion likely contributed to the enhanced jump height in SJ and CMJ.

accepted after revision
March 01, 2013

Bibliography
DOI http://dx.doi.org/10.1055/s-0033-1343401
Published online: 2013
Int J Sports Med

ISSN 0172-4622

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Introduction

Plyometric training is commonly used for physical conditioning of healthy people and patients, for example individuals suffering from osteoporosis [6,7,26,33]. It has been shown that plyometric training induces musculoskeletal adaptations [17,19,42,43] as well as neuromuscular adaptations [13,21–23,30,36] and can be used to improve athletic performance, for example jumping, sprinting and distance running performance [6,19,32,34].

Numerous studies have examined the effect of plyometric training on strength of the lower-extremity muscles and have shown increases in maximal voluntary force [8,21,34] and rate of force development [21,34] under isometric conditions. Although several studies have reported strength gains following plyometric training only a few studies focused on the possible neuromuscular mechanisms. Kubo et al. [19] have shown that isometric maximum voluntary torque (iMVT) of the plantar flexor muscles but not the rate of torque development (RTD) was increased after 12 weeks of plyometric training. The improved voluntary strength was associated with increased voluntary activation, assessed by the interpolated twitch technique, and muscle volume. Similar results could be observed by Kyrolainen et al. [21] who have found that 15 weeks of plyometric training improved isometric maximum voluntary force but not rate of force development of the plantar flexors. The changes in voluntary force were accompanied by increased muscle activation measured by surface electromyography (EMG) while the muscle fibre distribution and areas of the lateral gastrocnemius muscle were unchanged. Unlike their results for the plantar flexors, the authors have observed an increased rate of force development for the knee extensors, but have found no change in maximal voluntary strength and muscle activ-

ity during maximal voluntary contraction. In contrast, Malisoux et al. [23] have found that knee extensors maximal voluntary strength was enhanced following 8 weeks of maximal effort stretch-shortening cycle exercise training. The strength gain was accompanied by increases in single-fiber cross-sectional area and improvement in contractile properties of chemically skinned single muscle fibers.

In summary, data suggest that increases in maximal and explosive voluntary strength after plyometric training could be due to neural and muscular adaptations, but there is a lack of clarity about the underlying neuromuscular mechanisms with regard to the quadriceps muscle. Most studies that investigated neuromuscular adaptations after plyometric training by using EMG did not normalize muscle activity during maximum voluntary contraction and during force development to a maximal compound muscle action potential (M_{max}). Normalizing EMG amplitude to M_{max} may reduce errors due to electrode relocation and between-session variability in skin impedance, subcutaneous fat and fascia [39]. Furthermore, studies that have used the interpolated twitch technique to assess alterations in voluntary activation of muscles after plyometric training used the standard formula $\%VA = (1 - \text{superimposed twitch} \times \text{control twitch}^{-1}) \times 100$ [19] that calculates neural activation of muscles at the instant the superimposed stimulus is delivered. This is unlikely to be the moment of the maximal torque. In order to bypass this limitation, we used a correction of the original equation that takes the torque level before the superimposed twitch and the maximal torque into account [35]. Thus, the simultaneous application of 2 different methods, i.e., normalized EMG amplitude and corrected calculation of voluntary activation, allows greater insight into the modulations of neural drive to the quadriceps muscle after the training intervention.

It has been shown that voluntary activation of the quadriceps muscle differs depending on the muscle length and, therefore, the knee angle [3,20]. Accordingly, voluntary activation is higher in longer muscles than in shorter muscles, i.e., the activation level at the knee-flexed position is higher than that at the knee-extended position. Hence, we wanted to assess if plyometric training induces angle-specific adaptations and performed the neuromuscular tests at 2 knee angles, i.e., 80° and 45° knee flexion (0° = full extension).

Therefore, the present study was designed to elucidate neuromuscular adaptations of the quadriceps muscle after plyometric training. We investigated the contribution of quadriceps muscle activation and contractile properties to changes in iMVT following 8 weeks of plyometric training. In order to provide a comprehensive assessment, the neural drive to muscles was estimated with the interpolated twitch technique and root mean square of the EMG signal during iMVT ($RMS\text{-}EMG_{iMVT}$) normalized to M_{max}. Additionally, alterations of explosive voluntary strength and muscle activation in the early contraction phase after plyometric training were analyzed. Modifications at the muscle level were only assessed by analyzing the twitch torque signal induced by electrical stimulation, i.e., muscle volume was not measured. Changes in α-motoneuron excitability and/or presynaptic inhibition of primary muscle spindle afferents of vastus medialis were assessed using the H reflex technique. The neuromuscular tests were performed at 2 knee angles, i.e., 80° and 45° knee flexion (0° = full extension) in order to detect hypothetical angle-specific adaptations. In addition, jump height in countermovement jump (CMJ) and squat jump (SJ) was estimated.

We hypothesized that plyometric training would increase iMVT as well as RTD and that these changes would be associated with modulations in muscle activation at the maximum and onset of voluntary contraction. Furthermore, we assumed that plyometric training would modify contractile properties as well as α-motoneuron excitability via Ia afferents and increase jump height.

Materials and Methods

Subjects

In order to get a homogenous sample of subjects with adequate jump technique, 23 recreational volleyball players with no history of neurological disorders or injuries volunteered for the study. The sample size was similar to those in previous studies [21,34]. The study was a parallel-group randomized controlled trial. The subjects were randomly assigned to an intervention group and a control group using randomization by a computer-generated table of random numbers. The intervention group consisted of 13 subjects (8 males, 5 females, age: 24 ± 3 years, height: 183 ± 9 cm, body mass: 77 ± 10 kg), while 10 subjects were assigned to the control group. Owing to illness unrelated to the intervention, 3 participants dropped out and therefore 7 subjects (5 males, 2 females, age: 26 ± 5 years, height: 183 ± 8 cm, body mass: 77 ± 13 kg) served as controls. The subjects of the intervention and the control group were recreationally active (moderate exercise ≤3 times per week) and were recruited among the same recreational sports groups so that their normal weekly activity can be regarded as similar. The participants had never performed a systematic plyometric training program prior to this study. Before testing, subjects were instructed to refrain from consuming alcohol and caffeine in the 24 h preceding the experiments and not to perform any strenuous exercise in the 48 h previous to the measurements. All persons were informed of the procedures to be utilized and signed informed consent prior to investigation. The study was conducted according to the declaration of Helsinki and was approved by the university ethics committee. The study meets the ethical standards of the journal [16].

Training

The intervention group trained twice a week for 8 weeks. The training sessions were controlled by experienced supervisors and included CMJs, SJs and drop jumps from a height of 40 cm. The subjects performed 3 sets of each exercise. In the first 2 weeks the subjects performed 6 repetitions per set and thereafter 7 repetitions per set (rest interval between jumps = 4 s). The rest interval between the sets was 90 s. The jump exercises were performed with maximal effort in order to achieve explosive force production and maximal jump height. Participants' normal weekly activity did not change during the experimental period.

Experimental procedure

All subjects were tested before and after an 8 week period. Testing included different neuromuscular tests (**Fig. 1**). No warm-up was performed before the neuromuscular tests in order to avoid H reflex and M wave potentiation [11]. The measurements were made on the quadriceps muscle of the right leg. Throughout the testing sessions the subjects were comfortably seated in a standardized position on a CYBEX NORM dynamometer (Com-

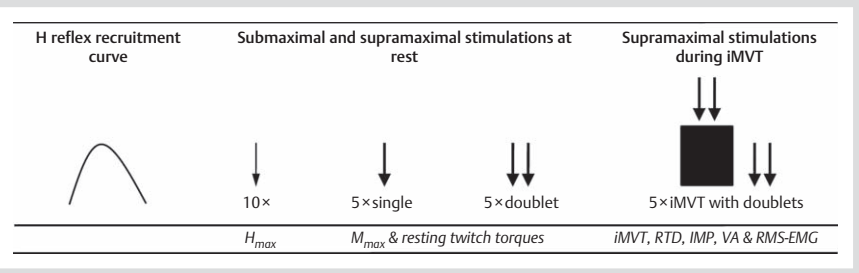

Fig. 1 An overview of the procedures carried out during neuromuscular testing. The neuromuscular tests were performed at 2 knee angles, i.e., 80° and 45° knee flexion (0° = full extension). Submaximal and supramaximal electrical stimulation at rest at both knee angles was done prior to the voluntary contractions. The *thin arrow* indicates stimulation at H_{max} intensity, the *thick arrow* indicates stimulation at supramaximal intensity, *double thick arrow* indicates doublet at supramaximal intensity. H_{max}: maximal H reflex, M_{max}: maximal M wave, iMVT: isometric maximum voluntary torque, RTD: rate of torque development, IMP: impulse, VA: voluntary activation, RMS-EMG: root mean square of the EMG signal.

puter Sports Medicine®, Inc., Stoughton, MA). Before neuromuscular testing, the subjects sat passively on the dynamometer for ~15 min in order to minimize potentiation effects from walking to the laboratory. The measurements were carried out at 2 knee angles, i.e., 80° and 45° (0° = full extension), in a random order. The neuromuscular tests were performed to assess voluntary activation and contractile properties of the quadriceps muscle. The excitability of the α-motoneuron pool of the vastus medialis muscle via Ia afferents was assessed using the H reflex technique [4, 11, 12]. Submaximal and supramaximal electrical stimulation at rest at both knee angles was carried out prior to the voluntary contractions. Jump height in CMJ and SJ was estimated using a force plate (Kistler® type 9290AD, Winterthur, Switzerland). Before the jump tests, all subjects underwent a standardized warm up on a cycle ergometer for 5 min at 60 W. The measurement of neuromuscular function of the quadriceps and jump performance took place on different days separated by at least 24 h.

Electrical stimulation

Transcutaneous electrical femoral nerve stimulation was used to produce the stimulus-response curve at the 2 knee angles, i.e., 80° and 45° (0° = full extension). Prior to attaching the stimulation electrodes, the skin was prepared by shaving and cleaning the relevant area. A hand-held stimulation probe was used to locate the optimum site of stimulation. Consequently, the femoral nerve was stimulated using a cathode ball electrode which was fixed to the subject's femoral triangle, 3–5 cm below the inguinal ligament. The anode was a self-adhesive electrode (35 × 45 mm, Spes Medica, Italy) placed over the greater trochanter. The percutaneous electrical stimuli were single (1 ms duration, 400 V maximal voltage) and paired rectangular pulses (1 ms duration, 10 ms apart, 400 V maximal voltage) delivered by a constant-current stimulator (Digitimer® DS7A, Hertfordshire, UK). Constant inter stimulus intervals (ISI) were provided by a Digitimer® train/delay generator (DG2A, Hertfordshire, UK). The testing procedure included random stimulation (ISI 7 s) with different current intensities, resulting in a recruitment curve, until identification of peak-to-peak maximal H reflex (H_{max}) and maximal M wave (M_{max}) of the vastus medialis muscle. The 5–10 current intensities around H_{max} were then repeated with 2 stimuli given at each current. Afterwards H_{max} and M_{max} were elicited and recorded 10 and 5 times, respectively [4, 5]. A stimulation intensity of 40% greater than that needed for maximal twitch response and concomitant M_{max} was used for evaluating contractile properties and voluntary activation. Resting twitch responses were evoked using single and doublet stimulation. Voluntary activation was assessed using the interpolated twitch technique [2], i.e., 2 s after torque onset, during the plateau phase, and 2 s after the end of the maximum voluntary contraction a transcutaneous supramaximal doublet was given to the femoral nerve.

EMG and torque recordings

Surface EMG was recorded using bipolar EMG Ambu® Blue Sensor N electrodes (2 cm diameter). The electrodes were firmly attached to the shaved, abraded and cleaned skin over vastus medialis, vastus lateralis and rectus femoris muscle of the right leg. The resistance between electrodes was measured with a digital multimeter (MY-68, McVoice, Germany) and was kept below 5 kΩ. The electrodes were applied with a center-to-center distance of 2 cm over the middle of the muscle bellies. The recording electrodes were in line with the presumed direction of the underlying muscle fibers. The reference electrode was attached to the patella of the ipsilateral leg. Signals were amplified (2 500×), band-pass filtered (10–450 Hz) and digitized with a sampling frequency of 5 kHz through an analog-to-digital converter (DAQ Card™-6024E, National Instruments, USA). Both the EMG and torque signals were sampled at 5 kHz and stored on a hard drive for later analysis with a custom-built LABVIEW® based program (Imago, Pfisoft, Germany).

Torque signals were measured using a CYBEX NORM dynamometer (Computer Sports Medicine®, Inc., Stoughton, MA). The individual positioning for each participant was similar before and after the training intervention. The hip joint angle of 80° was consistently maintained during the experiments, while the measurements were performed at 80° and 45° of knee flexion (0° = full extension), respectively. The axis of the dynamometer was aligned with the anatomical knee flexion-extension axis, and the lever arm was attached to the anterior aspect of the shank 2–3 cm above the lateral malleolus. The shin cushion was removed to avoid artifacts in the torque signal. A shin guard ensured that subjects could exert maximal forces without discomfort. Straps across the waist and the chest prevented excessive movements. The iMVT was tested by asking the subjects to exert isometric knee extensions against the lever arm of the dynamometer for 3 s. For each trial, subjects were thoroughly instructed to act as forcefully and as fast as possible. They were motivated by strong verbal encouragement and online visual feedback of the instantaneous dynamometer torque provided on a digital oscilloscope (HM1508, HAMEG Instruments, Germany). Care was taken to ensure that the iMVT trials were performed

without an apparent countermovement or pre-tension (change of baseline torque < 0.5 Nm during the 200 ms prior to contraction onset). A rest period of 2 min was allowed between the trials. The maximal attempts were recorded until the coefficient of variance of 5 subsequent trials was below 5 %.

Jump height

The participants performed maximal vertical CMJs and SJs on a one-dimensional force plate (Kistler® type 9290AD, Winterthur, Switzerland). The vertical ground reaction force was sampled at 500 Hz. The SJs were performed from ~80 ° knee flexion (0 ° = full extension) with hands akimbo and the trunk as straight as possible. During the CMJs, subjects stood in an upright position with hands on the hips and were instructed to begin the jump with a downward movement, which was immediately followed by an upward movement. As warm up, subjects completed up to 6 CMJs and SJs, respectively, with immediate feedback about their jump height. During the test, participants performed 4 maximal CMJs and SJs, respectively, with a resting period of at least 30 s between jumps. The subjects were instructed to perform the jumps with maximal effort in order to achieve explosive force production and maximal jump height. The best trial in terms of maximal jumping height was taken for further analysis.

Data analysis

The resting twitch torques were analyzed regarding their (i) peak torque (PT), the highest value of twitch torque signal, (ii) maximal rate of torque development ($MRTD_{TT}$) – the highest value of the first derivative of the twitch torque signal and (iii) maximal rate of torque relaxation ($MRTR_{TT}$) – the lowest value of the first derivative of the twitch torque signal. Voluntary activation was calculated with the formula %VA = (1 – superimposed twitch × (T_b/iMVT) × control twitch^{-1}) × 100 [27,35], where T_b is the torque level immediately before the superimposed twitch and iMVT the maximum voluntary torque. This formula counteracts the problem that, in some cases, the instant the superimposed doublet is delivered does not represent the maximal torque level. In the analyzed trials T_b was ≥ 95 % of iMVT, and the trials that did not meet this criterion were discarded from the analysis. The torque signals were corrected for the effect of gravity and the 3 best maximum voluntary contractions were retained for analysis. Explosive voluntary muscle strength was determined by analyzing the average RTD and the impulse (IMP) over time intervals of 0–50 ms, 50–100 ms, 100–150 ms and 150–200 ms relative to the onset of contraction. The muscle activation during the early phase of contraction was analyzed by calculating the RMS-EMG over time intervals of 0–50 ms, 50–100 ms, 100–150 ms and 150–200 ms relative to the onset of the EMG signals. RMS-EMG during iMVT ($RMS\text{-}EMG_{iMVT}$) was calculated over a 200 ms period at iMVT, i. e., 200 ms prior to the electrical stimuli if the maximal torque level was reached at the instant the superimposed stimuli were delivered and 100 ms on either side of iMVT if the maximal torque level was reached before the superimposed stimuli were administered. In order to normalize muscle activity, RMS-EMG of vastus medialis, vastus lateralis and rectus femoris was divided by their respective M_{max} values ($RMS\text{-}EMG/M_{max}$). Furthermore, $RMS\text{-}EMG/M_{max}$ was averaged across the 3 muscles to calculate quadriceps activation during the early phase of contraction and at iMVT ($RMS\text{-}EMG_{RTD}/M_{max}$ and $RMS\text{-}EMG_{iMVT}/M_{max}$, respectively). The identification of torque and EMG onsets was performed manually according to the method of Tillin et al. [38]. It has been suggested that this is the best method for detecting signal onsets [39]. H_{max} and M_{max} amplitudes were measured peak-to-peak. The H_{max}/M_{max}-ratio was calculated, which can be considered as a global index of modulations at the spinal level due to alterations in α-motoneuron excitability and/or presynaptic inhibition of primary muscle spindle afferents [45]. This data should be viewed with caution, because the H reflex of the vastus medialis muscle could be elicited in only 8 subjects of the intervention group and 3 subjects of the control group.

Statistical analysis

We had access to 23 subjects. Unfortunately, 3 subjects dropped out. However, none of the subjects withdrew from the study for a reason related to the study treatment. Therefore, a total of 20 participants completed the study. The data of these subjects were collected successfully. Data were checked for normal distribution using the Kolmogorov-Smirnov test. Differences between groups at pre-test were tested with independent t-tests. Furthermore, the statistical analysis consisted of ANCOVA with baseline measurements and gender entered as covariates [40]. The level of significance was established at $p \leq 0.05$. SPSS 20.0 (SPSS Inc., Chicago, IL, USA) was used for statistical analysis. Data are presented as group mean values ± standard deviation for the pre-test and adjusted means ± standard deviation for the post-test in the figures and in the table. In the "Results" section, data are presented as difference between means (95 % confidence interval).

Results

The subjects' characteristics and values of the analyzed parameters were not found to be different between the groups at pre-test (**Table 1** and **Fig. 2–4**). Jump height in SJ and CMJ was 4.5 cm (1.4–7.6 cm, $P = 0.007$) and 4.6 cm (0.4–8.8 cm, $P = 0.035$) higher at post-test, respectively, for the intervention group compared with controls (**Fig. 2**). With regard to the neuromuscular tests, all parameters at 45 ° knee flexion did not differ between groups after the training. Significant differences of several parameters at post-test were observed only at 80 ° knee flexion as detailed below. The iMVT was 23.1 N · m (0.1–46.1 N · m, $P = 0.049$) higher at post-test for the intervention group compared with the control group. Voluntary activation and $RMS\text{-}EMG_{iMVT}/M_{max}$ were significantly different between both groups after the training [4.6 % (0.1–9.1 %, $P = 0.049$) and 0.021 (0.001–0.042, $P = 0.039$), respectively] (**Fig. 3a–c**).

Furthermore, the slope of the moment-time curve in the time interval 0–50 ms was 308.7 N · m · s^{-1} (28.8–588.6 N · m · s^{-1}, $P = 0.033$) steeper for the intervention group compared with controls. Accordingly, there was a difference between groups at post-test regarding the parameter IMP in the time interval 0–50 ms [0.32 N · m · s (0.05–0.60 N · m · s, $P = 0.026$)] (**Fig. 4a, b**). In the initial phase of quadriceps contraction, i. e., 0–50 ms, the normalized muscle activity was 0.027 (−0.003–0.057, $P = 0.075$) higher at post-test for the intervention group compared with the control group, although this difference did not reach statistical significance. $RMS\text{-}EMG_{RTD}/M_{max}$ in the time interval 50–100 ms was significantly different between groups after the intervention [0.027 (0.000–0.054, $P = 0.048$)] (**Fig. 4c**).

No statistical differences between groups in the twitch mechanical parameters and evoked potentials at 80 ° and 45 ° knee flex-

Table 1 Twitch mechanical parameters and evoked potentials at 80° and 45° knee flexion (0° = full extension) before and after the training for the intervention (INT) and control (CON) group. Diff. (95% CI): difference between means (95% confidence interval), PTT: peak twitch torque, $MRTD_{TT}$: maximal rate of torque development, $MRTR_{TT}$: maximal rate of torque relaxation, H_{max}: maximal H reflex, M_{max}: maximal M wave, VM: vastus medialis. Data are means ± SD for the pre-test and adjusted means ± SD for the post-test.

Parameter	Pre				Post			
	INT	CON	Diff.	P	INT	CON	Diff. (95% CI)	P
PTT (N·m)								
Single at 80°	32.0 ± 18.4	31.2 ± 9.5	0.8	0.926	27.6 ± 8.1	25.3 ± 11.1	2.3 (−5.9 to 10.4)	0.563
Doublet at 80°	64.7 ± 23.2	73.2 ± 21.2	−8.5	0.455	60.6 ± 10.2	60.1 ± 14.0	0.5 (−9.7 to 10.8)	0.909
Single at 45°	24.5 ± 11.3	22.3 ± 6.2	2.2	0.643	20.7 ± 8.2	19.3 ± 8.3	1.4 (−6.9 to 9.7)	0.726
Doublet at 45°	58.3 ± 20.4	61.1 ± 15.6	−2.8	0.772	50.1 ± 12.9	52.8 ± 12.9	−2.7 (−15.5 to 10.1)	0.663
MRTDTT ($N \cdot m \cdot s^{-1}$)								
Single at 80°	926.6 ± 435.2	871.0 ± 249.6	55.6	0.856	778.5 ± 407.5	626.5 ± 556.7	152.0 (−255.3 to 559.2)	0.441
Doublet at 80°	1799.4 ± 922.9	2104.8 ± 706.6	−305.4	0.478	1539.2 ± 413.5	1524.6 ± 566.0	14.6 (−400.3 to 429.4)	0.942
Single at 45°	833.1 ± 522.5	691.0 ± 162.2	142.1	0.514	665.1 ± 284.8	603.6 ± 286.9	61.5 (−225.9 to 349.1)	0.656
Doublet at 45°	2021.5 ± 731.7	1941.3 ± 677.1	80.2	0.823	1704.0 ± 502.7	1813.5 ± 504.3	−109.5 (−612.5 to 393.6)	0.651
MRTRTT ($N \cdot m \cdot s^{-1}$)								
Single at 80°	397.6 ± 274.7	371.8 ± 110.9	25.8	0.823	342.5 ± 98.6	298.5 ± 134.7	44.0 (−54.6 to 142.6)	0.359
Doublet at 80°	809.5 ± 395.1	996.4 ± 329.2	−186.9	0.324	795.0 ± 180.5	736.5 ± 248.0	58.5 (−124.1 to 241.2)	0.507
Single at 45°	366.4 ± 205.4	309.0 ± 78.2	57.4	0.508	302.6 ± 128.5	275.4 ± 129.5	27.2 (−102.7 to 157.1)	0.663
Doublet at 45°	959.6 ± 453.0	978.0 ± 244.1	−18.4	0.926	768.9 ± 214.1	838.5 ± 214.5	−69.6 (−283.2 to 144.1)	0.500
H_{max} VM (mV)								
80°	1.87 ± 1.39	1.03 ± 0.58	0.84	0.382	2.44 ± 1.58	1.40 ± 2.37	1.04 (−1.44 to 3.51)	0.345
45°	1.93 ± 1.70	1.39 ± 0.75	0.54	0.636	2.51 ± 1.57	1.41 ± 1.90	1.10 (−0.91 to 3.10)	0.238
M_{max} VM (mV)								
80°	7.72 ± 3.15	8.82 ± 4.29	−1.10	0.543	9.65 ± 2.65	10.71 ± 2.67	−1.06 (−3.72 to 1.60)	0.412
45°	7.95 ± 3.12	8.87 ± 3.61	−0.92	0.579	9.25 ± 2.60	9.79 ± 2.61	−0.54 (−3.14 to 2.06)	0.665
H_{max}/M_{max}-ratio VM								
80°	0.23 ± 0.12	0.10 ± 0.03	0.13	0.127	0.28 ± 0.14	0.07 ± 0.21	0.21 (−0.02 to 0.43)	0.068
45°	0.24 ± 0.13	0.12 ± 0.04	0.12	0.164	0.28 ± 0.18	0.13 ± 0.23	0.15 (−0.10 to 0.40)	0.189

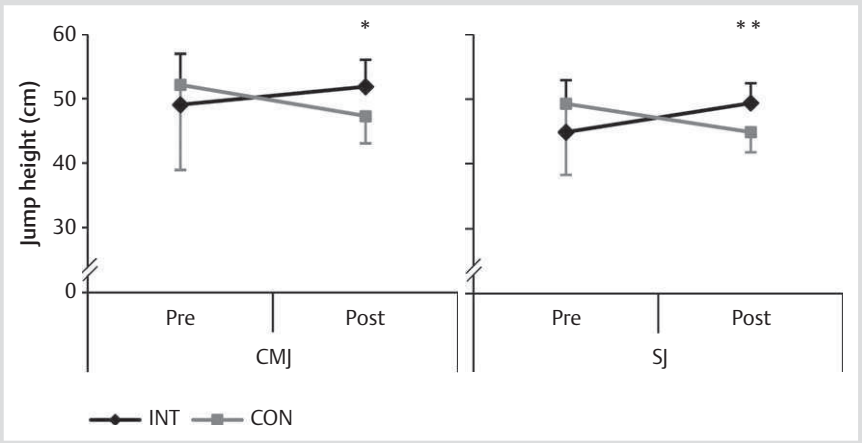

Fig. 2 Jump height in countermovement jump (CMJ) and squat jump (SJ) before and after the plyometric training for the intervention (INT) and control (CON) group. * denotes a significant difference between groups, $^{*}P \leq 0.05$; $^{**}P \leq 0.01$.

ion were observed after the training (▶ **Table 1**; M_{max} of vastus lateralis and rectus femoris muscle is not displayed).

Discussion

The present study was designed to elucidate the neuromuscular adaptations of the knee extensors after plyometric training as well as the effect of the training regime on jump height in CMJ and SJ. The results demonstrate a knee angle-specific adaptation of neuromuscular function, i.e., a modulation at 80° knee flexion but not at 45° knee flexion. It was found that iMVT as well as RTD and IMP in the time interval 0–50 ms were significantly different between groups after the intervention. The increased mechanical output of the intervention group was accompanied by an enhanced neural drive to the quadriceps muscle, as evidenced by increases in voluntary activation as well as normalized muscle activity during iMVT and the initial phase of contraction ($RMS\text{-}EMG_{iMVT}/M_{max}$ and $RMS\text{-}EMG_{RTD}/M_{max}$, respectively). The intervention did not affect H_{max}, M_{max} and the H_{max}/M_{max}-ratio of the vastus medialis muscle. Furthermore, adaptations at the muscle level seem unlikely because twitch mechanical parameters did not change. However, hypertrophic changes cannot be ruled out, because muscle volume was not measured. The strength gains at 80° knee flexion probably contributed to the enhanced jump height in SJ and CMJ.

The iMVT at 80° knee flexion was significantly higher for the intervention group compared with controls after the training. This is in line with results of other studies that reported increased maximal voluntary strength after plyometric training [15,21,30,34]. The strength enhancement was probably due to increases in efferent motoneuron output, as evidenced by

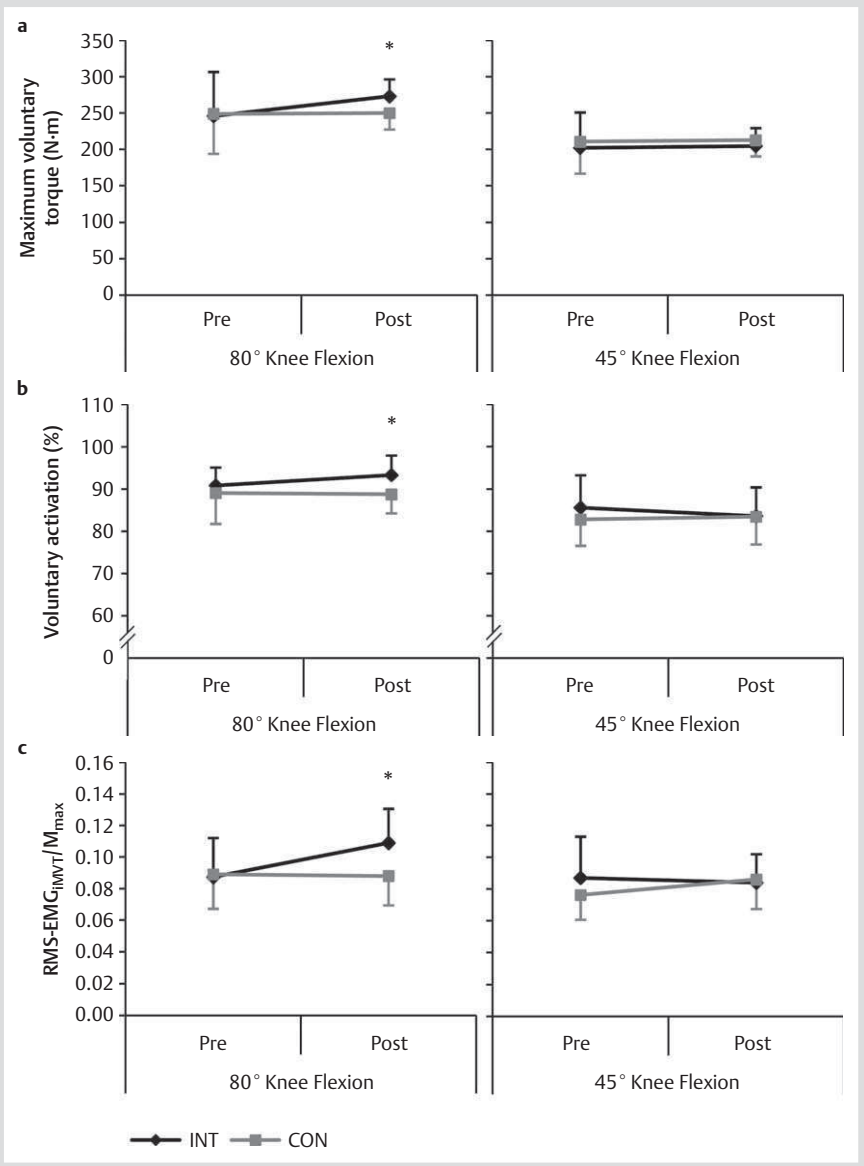

Fig. 3 Isometric maximum voluntary torque (iMVT, **a**), voluntary activation **b** and normalized muscle activity during iMVT (RMS-EMG$_{iMVT}$/M$_{max}$, **c**) at 80° and 45° knee flexion before and after the plyometric training. INT: intervention group, CON: control group. * denotes a significant difference between groups, *$P \leq 0.05$.

changes in both voluntary activation and normalized muscle activity (RMS-EMG$_{iMVT}$/M$_{max}$). These data and the results of the twitch mechanical parameters suggest that the training regime induced mainly neural adaptations although alterations in muscle volume were not tested in this study. Similar results were obtained by Kyrolainen et al. [21]. The authors have found that 15 weeks of plyometric training improved isometric maximum voluntary force of the plantar flexors accompanied by increased muscle activity and unchanged muscle fiber distribution as well as areas of the lateral gastrocnemius muscle. It has been argued that neural adaptations play a decisive role in the early phase of strength gain [10, 15, 28]. Studies have shown that increases in muscle strength in the first weeks of training can be attributed primarily to increased muscle activity and that muscle fiber hypertrophy plays a minor role [14, 21]. In contrast, studies have found that increased strength after plyometric training could be attributed to both an enhancement of voluntary activation and hypertrophic changes [19, 23]. In this study it was found that RTD and IMP in the time interval 0–50 ms at 80° knee flexion were increased after plyometric training. Similar results were observed by Kyrolainen et al. [21] who have found that 15 weeks of plyometric training improved maximal rate of force development of the knee extensors. Unfortunately, the authors did not analyze muscle activation during the early phase of contraction. In the present study, the neural drive to the quadriceps muscle was analyzed by calculating normalized muscle activity (RMS-EMG$_{RTD}$/M$_{max}$) over time intervals of 0–50 ms, 50–100 ms, 100–150 ms and 150–200 ms relative to the onset of the EMG signals. The results indicate that the improvements in RTD and IMP in the time interval 0–50 ms could be ascribed to increased muscle activation during torque development. This is in line with the findings of Häkkinen et al. [15] who have measured maximal voluntary strength in females after 16 weeks of power training that included different jumping exercises with a special sledge apparatus. The authors have reported a reduced time to produce 500 N force accompanied by increased muscle activity of vastus lateralis and vastus medialis. In addition, they could not find significant hypertrophic changes in the vastus lateralis muscle and they concluded that the adaptations were mainly of neural origin. The results of the present study support this assumption because twitch mechanical parameters were not different between groups after training. However, it should be noted that muscle volume was not measured directly. A study of Kubo et al. [19] has yielded different results. The authors have shown that

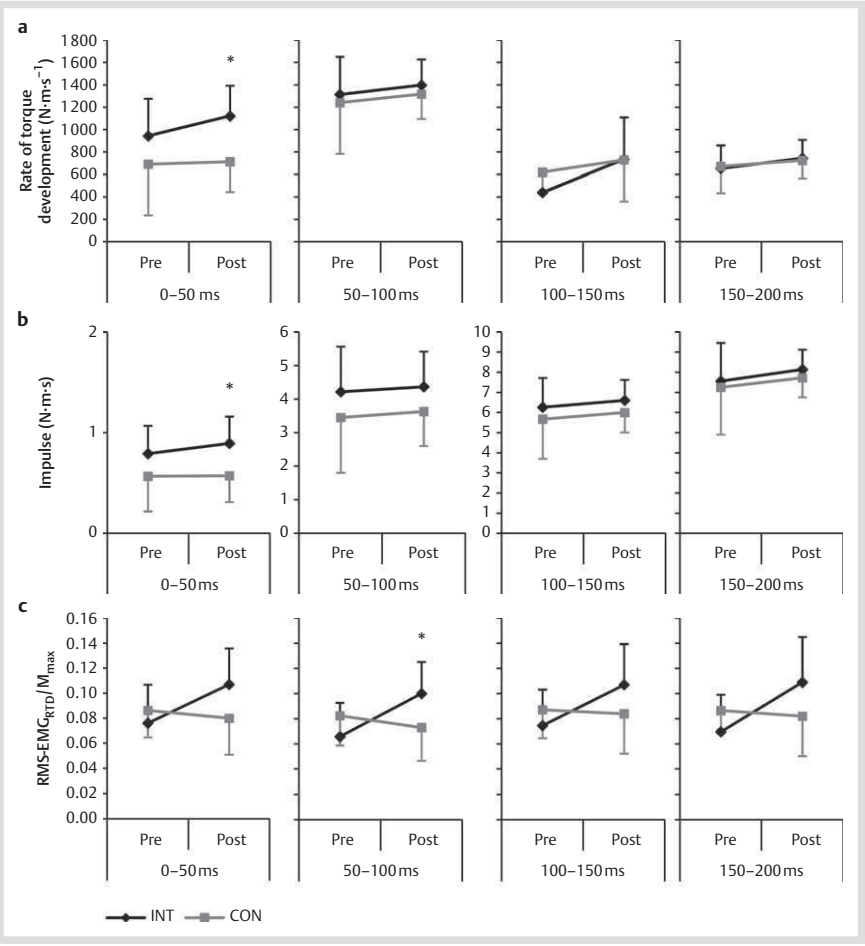

Fig. 4 Rate of torque development **a**, impulse **b** and normalized muscle activity during the initial phase of contraction (RMS-EMG$_{RTD}$/M$_{max}$, **c**) at 80° knee flexion before and after the plyometric training. The parameters were calculated in time intervals of 0–50 ms, 50–100 ms, 100–150 ms and 150–200 ms from the onset of the respective signals. INT: intervention group, CON: control group. * denotes a significant difference between groups, *$P \leq 0.05$.

RTD of the plantar flexors did not change after 12 weeks of plyometric training, whereas maximal voluntary strength, voluntary activation during iMVT and muscle volume were increased.

Interestingly, data suggest that the increased neural drive to the quadriceps muscle during iMVT and the initial phase of contraction was specifically related to the knee angle because adaptations were only observable at 80° knee flexion and not at 45° knee flexion. Studies have shown that voluntary activation of the quadriceps muscle differs depending on the muscle length and, therefore, the knee angle [3,20]. Accordingly, voluntary activation is higher in longer muscles than in shorter muscles, i.e., the activation level at the knee-flexed position is higher than that at the knee-extended position. It has been suggested that the physiological basis for this phenomenon must be considered in relation to the amount of afferent input to the quadriceps motoneuron pool [20]. In the flexed position, the muscle spindle length is increased, which leads to an enhanced steady-state excitation of quadriceps motoneurons via Ia afferents. The heightened Ia afferent input to the motoneuron pool in turn increases the total excitatory drive and, thus, voluntary activation. Furthermore, it has been shown that a change in the joint angle induces tension changes of knee joint ligaments [24], which have the ability to modulate the γ innervation of muscle spindles [29].

The results of the present study indicate an angular specificity of training. Several studies have shown that training effects could depend on angular position selected for training [18,37]. It has been suggested that this angular specificity could be attributed to neural adaptations rather than to hypertrophic changes [18]. This assumption is supported by the results of Thepaut-Mathieu et al. [37], who have found that the neural drive to muscles was greater at the joint angles trained. In contrast, it has been proposed that hypertrophic changes tend to enhance force at all joint angles [18,31]. A study of Voigt et al. [41] has revealed that the soleus tendon reflex at rest as well as the H reflex during the stance phase of a jumping task were enhanced after 4 weeks of hopping training. The authors hypothesized that the sensitivity of the muscle spindles as well as the extent of presynaptic inhibition of Ia afferents might have changed. In the present study, it is conceivable that the angle-specific adaptation is the result of the characteristics of the training regime, because the training consisted to a large extent of SJ and CMJ exercises that were performed between ~80° and 0° knee flexion (0° = full extension). As mentioned before, in a flexed position, i.e. ~80 knee flexion, the muscle spindle length is increased, leading to an enhanced excitation of quadriceps motoneurons via Ia afferents, which is not the same at 45° knee flexion [3,20]. It might be that the training modulated spindle sensitivity only at 80° knee flexion, which, in turn, led to an increment in Ia afferent input to the quadriceps motoneuron pool at this knee angle. This could have increased the total excitatory drive and, therefore, voluntary activation exclusively at 80° knee flexion. The H reflex of vastus medialis evoked at rest did not change indicating that α-motoneuron excitability and/or presynaptic inhibition of Ia afferents was not altered at rest. However, we should take into account that H reflexes evoked during voluntary contraction [1] and/or during performance [41] probably represent a more functional assessment of training-induced adaptations at the

spinal level. Thus, a reduction of inhibitory inputs to the quadriceps motoneuron pool could be an explanation as well.

Jump height in SJ and CMJ was significantly higher at post-test for the intervention group compared with controls. This is in line with the results of several studies that reported increased jump height after plyometric training [9,25]. The increased maximal voluntary strength and explosive voluntary strength at 80° knee flexion probably contributed to the enhanced jump height in SJ and CMJ. It has been shown that jump height is sensitive to muscle strength and speed, i.e., stronger and faster muscles are important factors to achieve greater jump heights. These factors enable the subject to exert more force on the ground [44].

In summary, the present study revealed that neural adaptations were mainly responsible for the enhancement of maximum voluntary strength and explosive voluntary strength at 80° of knee flexion, as evidenced by increases in voluntary activation as well as normalized muscle activity during iMVT and the initial phase of contraction. Adaptations at the muscle level seem unlikely because twitch mechanical parameters and evoked potentials induced by supramaximal electrical stimulation did not change. Interestingly, data suggest that the neural adaptations were specifically related to the knee angle. Until now, this angular specificity of adaptation was primarily shown for isometric training regimes [18,37]. The findings of the present study indicate that an angular specificity may also appear after plyometric training that contains primarily eccentric and concentric muscle actions.

Acknowledgements

The authors would like to thank Detlef Werner for the technical support and Benjamin Pohlenz for his help during data acquisition. This study was supported by a grant of LGF M-V (Landesgraduiertenfoerderung Mecklenburg Vorpommern).

References

1 *Aagaard P*, *Simonsen EB*, *Andersen JL*, *Magnusson P*, *Dyhre-Poulsen P*. Neural adaptation to resistance training: changes in evoked V-wave and H-reflex responses. J Appl Physiol 2002; 92: 2309–2318
2 *Allen GM*, *Gandevia SC*, *McKenzie DK*. Reliability of measurements of muscle strength and voluntary activation using twitch interpolation. Muscle Nerve 1995; 18: 593–600
3 *Becker R*, *Awiszus F*. Physiological alterations of maximal voluntary quadriceps activation by changes of knee joint angle. Muscle Nerve 2001; 24: 667–672
4 *Behrens M*, *Mau-Moeller A*, *Bruhn S*. Effect of exercise-induced muscle damage on neuromuscular function of the quadriceps muscle. Int J Sports Med 2012; 33: 600–606
5 *Behrens M*, *Mau-Moeller A*, *Zschorlich V*, *Bruhn S*. Repetitive peripheral magnetic stimulation (15 Hz RPMS) of the human soleus muscle did not affect spinal excitability. Journal of Sports Science and Medicine 2011; 10: 39–44
6 *Brown ME*, *Mayhew JL*, *Boleach LW*. Effect of plyometric training on vertical jump performance in high school basketball players. J Sports Med Phys Fitness 1986; 26: 1–4
7 *Chimera NJ*, *Swanik KA*, *Swanik CB*, *Straub SJ*. Effects of plyometric training on muscle-activation strategies and performance in female athletes. J Athl Train 2004; 39: 24–31
8 *Cornu C*, *Almeida Silveira MI*, *Goubel F*. Influence of plyometric training on the mechanical impedance of the human ankle joint. Eur J Appl Physiol 1997; 76: 282–288
9 *de Villarreal ES*, *Kellis E*, *Kraemer WJ*, *Izquierdo M*. Determining variables of plyometric training for improving vertical jump height performance: a meta-analysis. J Strength Cond Res 2009; 23: 495–506
10 *Duchateau J*, *Semmler JG*, *Enoka RM*. Training adaptations in the behavior of human motor units. J Appl Physiol 2006; 101: 1766–1775
11 *Folland JP*, *Wakamatsu T*, *Fimland MS*. The influence of maximal isometric activity on twitch and H-reflex potentiation, and quadriceps femoris performance. Eur J Appl Physiol 2008; 104: 739–748
12 *Fuchs DP*, *Sanghvi N*, *Wieser J*, *Schindler-Ivens S*. Pedaling alters the excitability and modulation of vastus medialis H-reflexes after stroke. Clin Neurophysiol 2011; 122: 2036–2043
13 *Grosset JF*, *Piscione J*, *Lambertz D*, *Perot C*. Paired changes in electromechanical delay and musculo-tendinous stiffness after endurance or plyometric training. Eur J Appl Physiol 2009; 105: 131–139
14 *Häkkinen K*, *Komi PV*, *Tesch PA*. Effect of combined concentric and eccentric strength training and detraining on force-time, muscle fiber and metabolic characteristics of leg extensor muscles. Scand J Sports Sci 1981; 3: 50–58
15 *Häkkinen K*, *Pakarinen A*, *Kyrolainen H*, *Cheng S*, *Kim DH*, *Komi PV*. Neuromuscular adaptations and serum hormones in females during prolonged power training. Int J Sports Med 1990; 11: 91–98
16 *Harriss DJ*, *Atkinson G*. Ethical standards in sport and exercise science research. Int J Sports Med 2011; 32: 819–821
17 *Kato T*, *Terashima T*, *Yamashita T*, *Hatanaka Y*, *Honda A*, *Umemura Y*. Effect of low-repetition jump training on bone mineral density in young women. J Appl Physiol 2006; 100: 839–843
18 *Kitai TA*, *Sale DG*. Specificity of joint angle in isometric training. Eur J Appl Physiol 1989; 58: 744–748
19 *Kubo K*, *Morimoto M*, *Komuro T*, *Yata H*, *Tsunoda N*, *Kanehisa H*, *Fukunaga T*. Effects of plyometric and weight training on muscle-tendon complex and jump performance. Med Sci Sports Exerc 2007; 39: 1801–1810
20 *Kubo K*, *Tsunoda N*, *Kanehisa H*, *Fukunaga T*. Activation of agonist and antagonist muscles at different joint angles during maximal isometric efforts. Eur J Appl Physiol 2004; 91: 349–352
21 *Kyrolainen H*, *Avela J*, *McBride JM*, *Koskinen S*, *Andersen JL*, *Sipila S*, *Takala TE*, *Komi PV*. Effects of power training on muscle structure and neuromuscular performance. Scand J Med Sci Sports 2005; 15: 58–64
22 *Malisoux L*, *Francaux M*, *Nielens H*, *Renard P*, *Lebacq J*, *Theisen D*. Calcium sensitivity of human single muscle fibers following plyometric training. Med Sci Sports Exerc 2006; 38: 1901–1908
23 *Malisoux L*, *Francaux M*, *Nielens H*, *Theisen D*. Stretch-shortening cycle exercises: an effective training paradigm to enhance power output of human single muscle fibers. J Appl Physiol 2006; 100: 771–779
24 *Markolf KL*, *Gorek JF*, *Kabo JM*, *Shapiro MS*. Direct measurement of resultant forces in the anterior cruciate ligament. An in vitro study performed with a new experimental technique. J Bone Joint Surg Am 1990; 72: 557–567
25 *Markovic G*. Does plyometric training improve vertical jump height? A meta-analytical review. Br J Sports Med 2007; 41: 349–355
26 *Markovic G*, *Mikulic P*. Neuro-musculoskeletal and performance adaptations to lower-extremity plyometric training. Sports Med 2010; 40: 859–895
27 *Martin V*, *Millet GY*, *Lattier G*, *Perrod L*. Effects of recovery modes after knee extensor muscles eccentric contractions. Med Sci Sports Exerc 2004; 36: 1907–1915
28 *Moritani T*, *deVries HA*. Neural factors versus hypertrophy in the time course of muscle strength gain. Am J Phys Med 1979; 58: 115–130
29 *Rice DA*, *McNair PJ*. Quadriceps arthrogenic muscle inhibition: neural mechanisms and treatment perspectives. Semin Arthritis Rheum 2010; 40: 250–266
30 *Saez-Saez de Villarreal E*, *Requena B*, *Newton RU*. Does plyometric training improve strength performance? A meta-analysis. J Sci Med Sport 2010; 13: 513–522
31 *Sale D*, *MacDougall D*. Specificity in strength training: a review for the coach and athlete. Can J Appl Sport Sci 1981; 6: 87–92
32 *Salonikidis K*, *Zafeiridis A*. The effects of plyometric, tennis-drills, and combined training on reaction, lateral and linear speed, power, and strength in novice tennis players. J Strength Cond Res 2008; 22: 182–191
33 *Saunders PU*, *Telford RD*, *Pyne DB*, *Peltola EM*, *Cunningham RB*, *Gore CJ*, *Hawley JA*. Short-term plyometric training improves running economy in highly trained middle and long distance runners. J Strength Cond Res 2006; 20: 947–954
34 *Spurrs RW*, *Murphy AJ*, *Watsford ML*. The effect of plyometric training on distance running performance. Eur J Appl Physiol 2003; 89: 1–7
35 *Strojnik V*, *Komi PV*. Neuromuscular fatigue after maximal stretch-shortening cycle exercise. J Appl Physiol 1998; 84: 344–350
36 *Taube W*, *Leukel C*, *Lauber B*, *Gollhofer A*. The drop height determines neuromuscular adaptations and changes in jump performance in stretch-shortening cycle training. Scand J Med Sci Sports 2011; 22: 671–683

37 *Thepaut-Mathieu C*, *Van Hoecke J*, *Maton B*. Myoelectrical and mechanical changes linked to length specificity during isometric training. J Appl Physiol 1988; 64: 1500–1505
38 *Tillin NA*, *Jimenez-Reyes P*, *Pain MT*, *Folland JP*. Neuromuscular performance of explosive power athletes versus untrained individuals. Med Sci Sports Exerc 2010; 42: 781–790
39 *Tillin NA*, *Pain MT*, *Folland JP*. Short-term unilateral resistance training affects the agonist-antagonist but not the force-agonist activation relationship. Muscle Nerve 2011; 43: 375–384
40 *Vickers AJ*, *Altman DG*. Statistics notes: Analysing controlled trials with baseline and follow up measurements. BMJ 2001; 323: 1123–1124
41 *Voigt M*, *Chelli F*, *Frigo C*. Changes in the excitability of soleus muscle short latency stretch reflexes during human hopping after 4 weeks of hopping training. Eur J Appl Physiol 1998; 78: 522–532
42 *Witzke KA*, *Snow CM*. Effects of plyometric jump training on bone mass in adolescent girls. Med Sci Sports Exerc 2000; 32: 1051–1057
43 *Wu YK*, *Lien YH*, *Lin KH*, *Shih TT*, *Wang TG*, *Wang HK*. Relationships between three potentiation effects of plyometric training and performance. Scand J Med Sci Sports 2010; 20: e80–e86
44 *Zajac FE*. Muscle coordination of movement: a perspective. J Biomech 1993; 26 (Suppl 1): 109–124
45 *Zehr PE*. Considerations for use of the Hoffmann reflex in exercise studies. Eur J Appl Physiol 2002; 86: 455–468

Journal of Electromyography and Kinesiology 23 (2013) 640–648

Contents lists available at SciVerse ScienceDirect

Journal of Electromyography and Kinesiology

journal homepage: www.elsevier.com/locate/jelekin

Age-related changes in neuromuscular function of the quadriceps muscle in physically active adults

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ARTICLE INFO

Article history:
Received 5 July 2012
Received in revised form 10 December 2012
Accepted 10 January 2013

Keywords:
Aging
Quadriceps muscle
Dynapenia
Voluntary activation
Interpolated twitch technique
Electromyography
Spinal excitability
H reflex
M wave
Contractile properties
Twitch contraction

ABSTRACT

Substantial evidence exists for the age-related decline in maximal strength and strength development. Despite the importance of knee extensor strength for physical function and mobility in the elderly, studies focusing on the underlying neuromuscular mechanisms of the quadriceps muscle weakness are limited.

The aim of this study was to investigate the contributions of age-related neural and muscular changes in the quadriceps muscle to decreases in isometric maximal voluntary torque (iMVT) and explosive voluntary strength. The interpolated twitch technique and normalized surface electromyography (EMG) signal during iMVT were analyzed to assess changes in neural drive to the muscles of 15 young and 15 elderly volunteers. The maximal rate of torque development as well as rate of torque development, impulse and neuromuscular activation in the early phase of contraction were determined. Spinal excitability was estimated using the *H* reflex technique. Changes at the muscle level were evaluated by analyzing the contractile properties and lean mass.

The age-related decrease in iMVT was accompanied by a decline in voluntary activation and normalized surface EMG amplitude. Mechanical parameters of explosive voluntary strength were reduced while the corresponding muscle activation remained primarily unchanged. The spinal excitability of the vastus medialis was not different while *M* wave latency was longer. Contractile properties and lean mass were reduced.

In conclusion, the age-related decline in iMVT of the quadriceps muscle might be due to a reduced neural drive and changes in skeletal muscle properties. The decrease in explosive voluntary strength seemed to be more affected by muscular than by neural changes.

1. Introduction

The age-related decrease in muscle strength (dynapenia) and strength development is well-documented and might be caused by neural modulations and alterations of skeletal muscle properties (Clark and Taylor, 2011; Manini and Clark, 2011). However, the results differ in part between the investigated muscle groups possibly due to differences in the function and physiological profile of the muscles (Clark and Taylor, 2011).

Numerous studies have described the modulations of voluntary strength and morphology of the quadriceps muscle with aging (Porter et al., 1995; Roos et al., 1999). Due to its functional importance for gait (Murdock and Hubley-Kozey, 2012) and maintaining posture (Barbeau et al., 2000), it is surprising that studies focusing on the underlying neuromuscular mechanisms of the knee extensor weakness are limited. Moreover, the reported results are partially conflicting, particularly with regard to changes in neural drive to the quadriceps muscle (Harridge et al., 1999; Roos et al., 1999; Stevens et al., 2003; Wilder and Cannon, 2009). The ability to voluntarily activate the knee extensors during isometric contractions has been estimated using either the interpolated twitch technique or the central activation ratio. The inconsistent findings may be primarily related to methodological limitations and differences of these two techniques (Klass et al., 2007). Thus, the simultaneous application of two different methods may provide greater insight into modulations of neural drive to the muscle. Only one study has considered a twofold approach to detecting age-related changes in efferent motoneuron output to the knee extensors (Cannon et al., 2007). Besides the interpolated twitch technique, Cannon et al. have used normalized muscle activity during isometric maximal strength to estimate the neural drive. Both parameters were not changed indicating no differences in efferent motoneuron output to the quadriceps muscle. However, the authors used the standard formula for estimating voluntary activation. It has been demonstrated that the interpolated twitch technique has its limitations when the standard formula is applied (Folland and Williams, 2007). The moment when the stimulus is delivered is not always the time point of maximal strength. Thus, the use of a corrected

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1050-6411/$ - see front matter
http://dx.doi.org/10.1016/j.jelekin.2013.01.009

formula which includes the variations of the torque level has been recommended (Strojnik and Komi, 1998). Only Suetta et al. (2009) applied this corrected formula and also demonstrated no age-related change in voluntary activation under isometric conditions. However, these authors did not use a second method to assess the neural drive to the muscle in order to confirm their results.

It is further noteworthy that age-related changes in neural drive to the quadriceps during strength development have been less frequently investigated. Dynapenia of the quadriceps is a determinant of fall risk (Lord et al., 1994) and mortality (Newman et al., 2006). Considering the fact that the capacity to activate the muscle quickly may be more essential for maintaining balance and preventing falls than the maximal strength (Schultz, 1995), neuromuscular modulations of the quadriceps muscle during strength development should be investigated more closely.

In summary, there is some lack of knowledge about the neuromuscular mechanisms behind the modulations of maximal strength and strength development of the quadriceps muscle with advancing age. The aim of the present study was to investigate the contributions of age-related neural and muscular changes to decreases in isometric maximal voluntary torque (iMVT) and explosive voluntary strength. Explosive voluntary strength was analyzed by calculating the maximal rate of torque development (MRTD) as well as the rate of torque development (RTD) and impulse (IMP) in the early phase of contraction in time intervals of 0–50 ms, 50–100 ms, 100–150 ms and 150–200 ms after torque onset. The neural drive to the muscle during iMVT was estimated using two approaches: the interpolated twitch technique (corrected formula) and the root mean square of the EMG signal (RMS–EMG) normalized to maximal *M* wave (M_{max}). The neural drive to the muscle during MRTD and RTD was assessed using the RMS–EMG normalized to the M_{max} and normalized to the RMS–EMG during iMVT. Age-related changes in α-motoneuron excitability via Ia afferents were determined with the *H* reflex technique. Modulations at the skeletal muscle level were evaluated by analyzing the twitch torque signal induced by supramaximal electrical stimulation and by determining the lean mass of the leg.

It was hypothesized that iMVT, MRTD, RTD and IMP were reduced with aging accompanied by changes in neural drive to the quadriceps muscle. It was further assumed that aging would affect spinal excitability, contractile properties and skeletal muscle mass.

2. Methods

2.1. Subjects

Thirty physically active and healthy subjects with no history of neurological and musculoskeletal disorders or injuries volunteered to participate in this study. Physically active and healthy subjects were chosen in order to take account of the problem of lack of motivation and disuse atrophy. The subjects were assigned to two groups: (1) young, (2) elderly, each with 15 participants who refrained from consuming caffeine or alcohol and performing strenuous leg exercise 24 h and 48 h preceding the experiment. Prior to participation written informed consent was obtained from all subjects. The study was conducted according to the declaration of Helsinki and approved by the local ethics committee (A 2009 52). Subject characteristics are displayed in Table 1.

Table 1
Subject characteristics and lean mass of the leg. *Denotes a significant difference between the groups (*<0.05, **<0.01).

	Young (n = 15)	Elderly (n = 15)	p
Men, n (%)	8 (53.3)	8 (53.3)	
Age, yrs, Mean (SD)	25.3 (3.6)	69.6 (3.1)	0.000**
Weight, kg, Mean (SD)	71.3 (11.6)	72.6 (9.9)	0.750
Height, m, Mean (SD)	1.75 (0.9)	1.69 (0.9)	0.058
Physical activity, h/week, Mean (SD)	4.6 (3.6)	3.7 (3.3)	0.134
Lean mass, kg, Mean (SD)	9.27 (1.65)	7.79 (1.72)	0.028*

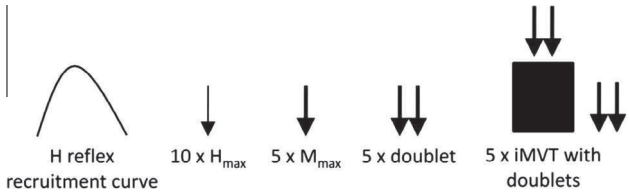

Fig. 1. Schematic illustration of neuromuscular tests. The *thin arrow* indicates stimulation at H_{max} intensity, the *thick arrow* indicates stimulation at supramaximal intensity, *double thick arrow* indicates doublet at supramaximal intensity.

2.2. Experimental procedure

The subjects participated in three experimental sessions. The first session included the measurement of body composition using dual-energy X-ray absorptiometry (DXA) followed by two sessions of neuromuscular tests: (1) familiarization, (2) experiment, separated by one week. Subjects were seated in a standardized position on a CYBEX NORM dynamometer. Prior to the experiment subjects sat passively for 15 min without any warm-up in order to avoid potentiation effects (Folland et al., 2008). The neuromuscular tests were carried out on the quadriceps muscle of the dominant leg. A schematic illustration of the neuromuscular tests is displayed in Fig. 1.

2.3. Torque and EMG recordings

Torque signals were measured using a CYBEX NORM dynamometer (Computer Sports Medicine®, Inc., Stoughton, MA). The tests were performed in a sitting position with a knee angle of 75° and a hip angle of 50° (0° = full extension). The iMVT was tested by asking the subjects to exert isometric knee extensions as forcefully and as fast as possible against the lever arm of the dynamometer for 3 s. The maximal attempts were recorded until the coefficient of variance of five subsequent trials was below 5%. A rest period of 2 min was allowed between the trials.

The surface EMG was recorded using bipolar EMG Ambu® Blue Sensor N electrodes (2 cm diameter). The electrodes were applied to the shaved, abraded and cleaned skin over the middle of the muscle bellies of the vastus medialis, vastus lateralis and rectus femoris. Signals were amplified (2500×), band-pass filtered (10–450 Hz) and digitized with a sampling frequency of 5 kHz through an analog-to-digital converter (DAQ Card™-6024E, National Instruments, USA). The EMG and torque signals were analyzed using a custom built LABVIEW® based program (Imago, Pfisoft, Germany). A detailed description of EMG and torque measurements has been given previously (Behrens et al., 2012).

2.4. Transcutaneuous electrical stimulation

Transcutaneuous electrical stimulation was used to assess voluntary activation, spinal excitability and contractile properties of the quadriceps muscle. The anode (self-adhesive electrode, 35 × 45 mm, Spes Medica, Italy) was placed over the greater trochanter. The cathode (ball electrode, 1 cm diameter) was fixed to the subject's femoral triangle, 3–5 cm below the inguinal ligament.

One millisecond rectangular pulses (400 V) were applied to the femoral nerve by a constant-current stimulator (Digitimer® DS7A, Hertfordshire, UK). The inter stimulus intervals (ISI 10 s, i.e. 0.1 Hz) were provided by a Digitimer® train/delay generator (DG2A, Hertfordshire, UK). Different current intensities were randomly delivered to the femoral nerve to evoke the maximal *H* reflex (H_{max}) of the vastus medialis and M_{max} of the vastus medialis, rectus femoris and vastus lateralis. The H_{max} and M_{max} were elicited and recorded 5 and 10 times, respectively. The evaluation of contractile properties and voluntary activation was realized at a stimulation intensity of 40% above the level that was needed for eliciting a maximal twitch response and concomitant M_{max}. Resting twitch responses were evoked using single and doublet (ISI 10 ms, i.e. 100 Hz) stimulation. Voluntary activation was assessed using the interpolated twitch technique (Allen et al., 1995). A transcutaneous supramaximal doublet was given 2 s after torque onset, during the plateau phase. The second doublet was applied 2 s after the contraction.

2.5. Lean body mass

Leg lean body mass was assessed using DXA. A Lunar Prodigy densitometer (General Electric (GE) Medical System Lunar, Madison, WI, USA) measured the attenuation of X-rays (76 kV, 0.15 mA, 0.4 μGy) while a total body scan was performed in the supine position with arms at the side. The scan time took 6–7 min, using the standard speed mode (irradiation time 274 s). The quality assurance procedure was performed before each test using a cuboid calibration phantom (200 × 130 × 60 mm). DXA provides a measure of appendicular lean soft tissue (ALST) which includes skeletal muscle mass and other components of bone free lean mass (i.e. ligaments, tendons, joint capsula and meniscal tissue) (Kim et al., 2002). A large proportion of ALST is skeletal muscle mass. Levine et al. (2000) found a high correlation between DXA thigh lean mass and CT-determined skeletal muscle mass ($r = 0.86$, $p < 0.001$). Thus, ALST can be considered as an estimate of skeletal muscle mass.

2.6. Data analysis

2.6.1. IMVT, explosive voluntary strength and voluntary activation

The highest maximal voluntary contraction was retained for analysis. The following parameters were calculated: iMVT and MRTD, as well as RTD and IMP in the early contraction phase. MRTD was defined as the maximal slope of the torque-time curve. The early rise in muscle strength was obtained from the average slope of the torque-time curve in different time intervals after torque onset: 0–50 ms (RTD_{50}), 50–100 ms (RTD_{100}), 100–150 ms (RTD_{150}), 150–200 ms (RTD_{200}). Onset of contraction was determined when the torque increased 3.5 Nm above the resting baseline level (Suetta et al., 2007). RTD was normalized to iMVT (RTD/iMVT). Absolute and normalized RTD values were considered for analysis. IMP was defined as the area under the torque-time curve.

The RMS–EMG for the vastus medialis, vastus lateralis and rectus femoris was calculated during a 200 ms epoch at iMVT ($RMS–EMG_{iMVT}$), this means prior to the electrical stimuli when the maximal torque level was achieved at the moment when the superimposed stimuli were delivered or 100 ms preceding and 100 ms following the iMVT if the maximal torque level was reached before the superimposed stimuli were administered. The RMS–EMG was also calculated at MRTD ($RMS–EMG_{MRTD}$), (i.e., 50 ms on either side of MRTD). The $RMS–EMG_{iMVT}$ and $RMS–EMG_{MRTD}$ of each muscle were normalized to their respective M_{max} value and averaged across the vastus medialis, vastus lateralis and rectus femoris to provide a global index of total quadriceps activity ($\overline{X}RMS–EMG_{iMVT}/M_{max}$, $\overline{X}RMS–EMG_{MRTD}/M_{max}$).

Furthermore, the RMS–EMG was calculated in the early phase of contraction in time windows of 0–50, 50–100, 100–150 and 150–200 ms relative to the onset of the EMG signal ($RMS–EMG_{RTD}$). The $RMS–EMG_{RTD}$ values were normalized to M_{max} ($RMS–EMG_{RTD}/M_{max}$) as well as to the RMS–EMG during iMVT ($RMS–EMG_{RTD}/RMS–EMG_{iMVT}$). Absolute and normalized $RMS–EMG_{RTD}$ values were averaged across the three muscles ($\overline{X}RMS–EMG_{RTD}$, $\overline{X}RMS–EMG_{RTD}/M_{max}$, $\overline{X}RMS–EMG_{RTD}/RMS–EMG_{iMVT}$).

Voluntary activation was calculated using the formula: %VA = (1 − superimposed twitch × (Tb/iMVT)/control twitch) × 100, whereby Tb represents the torque level just before the superimposed twitch (Strojnik and Komi, 1998).

2.6.2. Spinal excitability

The amplitudes of H_{max} and M_{max} were measured peak-to-peak. H_{max} was normalized to M_{max} (H_{max}/M_{max}-ratio) in order to assess alterations in α-motoneuron excitability and/or presynaptic inhibition of primary muscle spindle afferents (Zehr, 2002). Furthermore, the latencies of *H* and *M* waves were calculated by determining the time interval between the stimulus artefact and the first deflection of the amplitudes. The latencies of *H* and *M* waves were measured to detect modulations in the signal conduction speed through the reflex arc and the direct motor pathway. The latencies were normalised to the height of the subjects (Δt_H/height, Δt_M/height) since the length of the reflex pathway is related to the subjects' height (Guiheneuc and Bathien, 1976).

2.6.3. Contractile properties and lean mass

The resting twitch torques were analyzed with regard (1) peak torque (PT), the highest value of the twitch torque signal, (2) maximal rate of torque development ($MRTD_{TT}$), the highest value of the first derivative of the twitch toque signal, (3) maximal rate of torque relaxation ($MRTR_{TT}$), the lowest value of the first derivative of the twitch toque signal, (4) twitch contraction time (TCT_{TT}), the time from the onset to the maximal twitch torque, (5) twitch half-relaxation time ($THRT_{TT}$), the time from the maximal twitch torque to one-half of its peak value and (6) total twitch area (TTA_{TT}), the area under the torque-time curve.

Lean mass of the dominant leg was calculated by the software Lunar *enCORE*™ 2007 (version 11.40.004). It has been shown that the reliability of regional ALST is high when machine-made regions of interest are manual adjusted (Lohman et al., 2009). In order to avoid measurement errors, the calculated regions were checked manually to make corrective adjustments if necessary.

2.7. Statistical analysis

Data were checked for normal distribution using a Shapiro–Wilk *W* test. Age-related differences between the groups were tested for significance by the unpaired Student's *t* test or Mann–Whitney *U* test. All data were analysed using the SPSS statistical package 19.0 (SPSS Inc., Chicago, IL, USA). The level of significance was established at $p < 0.05$. Data are presented as mean ± standard deviation in the tables and displayed as boxplots in the figures.

3. Results

3.1. IMVT, explosive voluntary strength and voluntary activation

IMVT and voluntary activation were decreased by 37.9% ($p < 0.001$) and 6.1% ($p = 0.034$), respectively, in the elderly subjects. $\overline{X}RMS–EMG_{iMVT}/M_{max}$ was significantly reduced by 16.4% ($p = 0.036$) (Fig. 2A–C).

Absolute MRTD and MRTD/iMVT were significantly lower (66.1%, $p < 0.001$ and 48.0%, $p < 0.001$) in the elderly compared with young subjects (Fig. 2D and E). No differences were found for normalized muscle activity during MRTD (Fig. 2F).

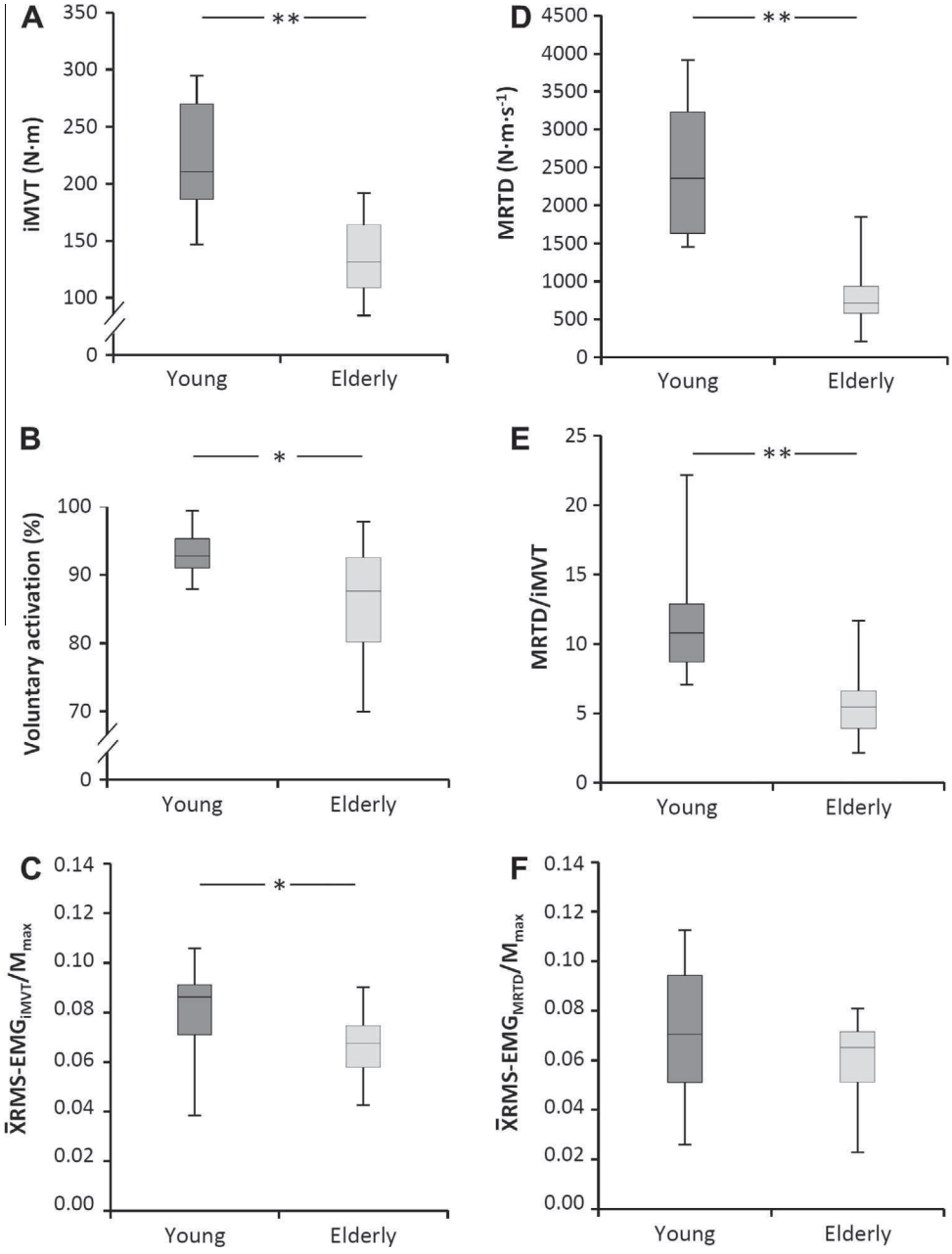

Fig. 2. Comparisons between groups. (A) isometric maximal voluntary torque (iMVT), (B) voluntary activation, (C) normalized and averaged root mean square of the EMG signal of vastus medialis, rectus femoris and vastus lateralis during iMVT ($\overline{X}$RMS–EMG$_{iMVT}$/M_{max}), (D) maximal rate of torque development (MRTD), (E) MRTD normalized to iMVT (MRTD/iMVT) and (F) normalized and averaged root mean square of the EMG signal of the three muscles during MRTD ($\overline{X}$RMS–EMG$_{MRTD}$/M_{max}). *Denotes a significant difference between the groups (* <0.05, ** <0.01).

Absolute RTD and IMP were significantly reduced during all of the four 50 ms time windows (Fig. 3A and C). The highest reduction of absolute RTD (67.6%, $p = 0.003$) and IMP (73.2%, $p = 0.007$) was observed for the first 50 ms after torque onset. Afterwards, the difference in absolute RTD (45.8% RTD_{100}, $p = 0.002$, 34.4% RTD_{150}, $p = 0.015$, 35.8% RTD_{200}, $p = 0.001$) and IMP (60.6% IMP_{100}, $p < 0.001$, 53.8% IMP_{150}, $p < 0.001$, 52.6% IMP_{200} $p < 0.001$) between the two groups decreased. By contrast, a decline in RTD/iMVT (48.18%, $p = 0.015$) was only observed in the first time window (0–50 ms) after torque onset (Fig. 3B).

$\overline{X}$RMS–EMG$_{RTD}$ was significantly lower during the 50–100 ms (44.1%, $p = 0.030$), 100–150 ms (43.0%, $p = 0.027$) and 150–200 ms (43.3%, $p = 0.010$) of the contraction phase in the elderly (Fig. 4A). On the contrary, there was a significant age-related increase of $\overline{X}$RMS–EMG$_{RTD}$/RMS–EMG$_{iMVT}$ by 27.0% ($p = 0.015$) and 34.8% ($p = 0.001$) during 100–150 ms and 150–200 ms after onset of the EMG signal (Fig. 4C). No differences for $\overline{X}$RMS–EMG$_{RTD}$/M_{max} were found during any of the four time windows (Fig. 4B).

3.2. Spinal excitability

The *H* reflex could be elicited in 10 young and 11 elderly subjects. H_{max} and the H_{max}/M_{max}-ratio of the vastus medialis revealed no significant difference between groups. M_{max} was significantly reduced in the vastus lateralis (24.7%) and vastus medialis (35.6%). Elderly subjects revealed a longer Δt_M/height in the vastus medialis (7.17%) while no differences were observed in the vastus lateralis and rectus femoris. Δt_H/height re-

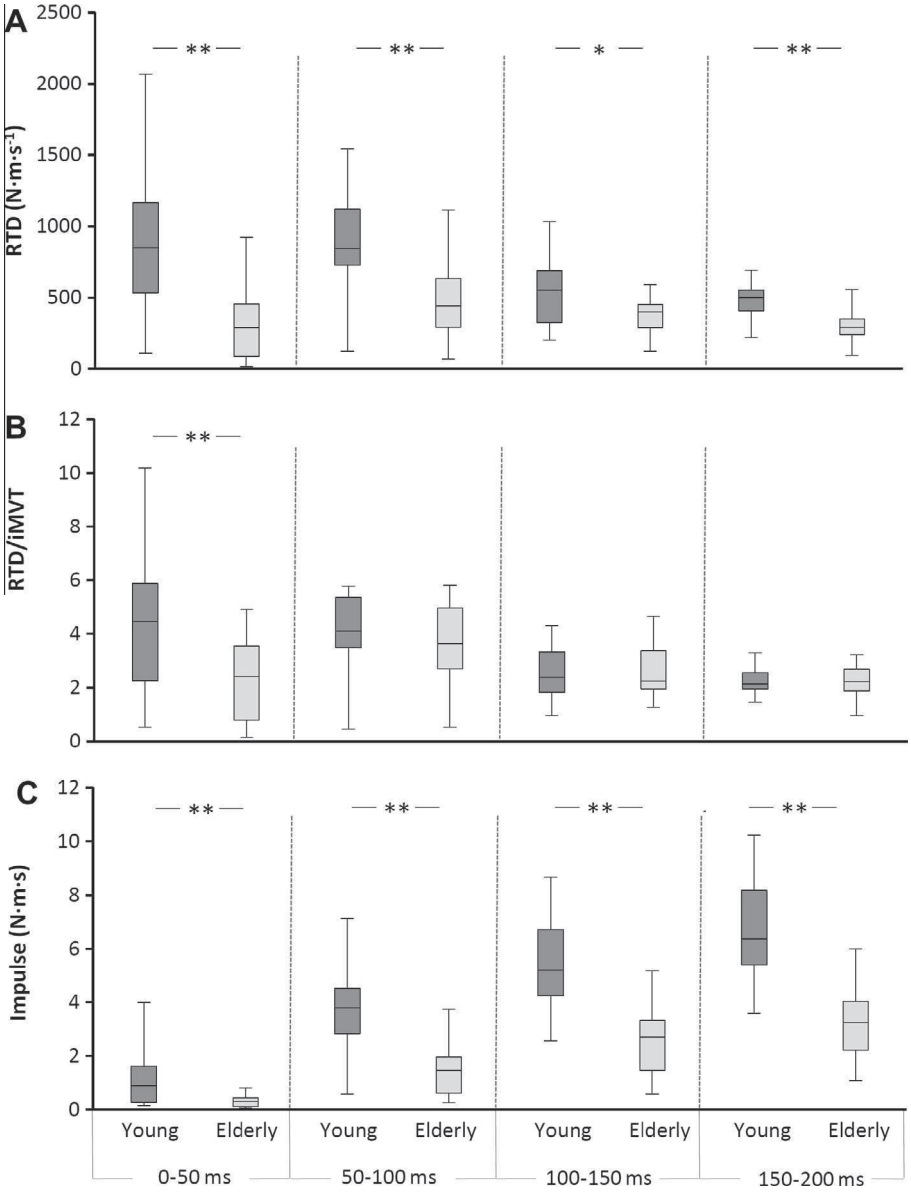

Fig. 3. Comparisons between groups: (A) rate of torque development (RTD), (B) RTD normalized to iMVT (RTD/iMVT) and (C) impulse (IMP) during the early phase of contraction in time windows of 0–50, 50–100, 100–150 and 150–200 ms after torque onset. *Denotes a significant difference between the groups (*<0.05, **<0.01).

mained unaffected by aging. The mean values ± standard deviations of evoked potentials are displayed in Table 2.

3.3. Contractile properties and lean mass

The PT, $MRTD_{TT}$ and $MRTR_{TT}$ of the resting twitch responses of the quadriceps were significantly reduced in elderly subjects (single: 61.04%, 62.9%, 64.9%, doublet: 39.9%, 53.7%, 60.3%). The TCT_{TT} of the resting twitch responses was significantly longer in elderly subjects when doublet stimulation was used (27.1%). No significant differences between the groups could be determined for $THRT_{TT}$. The TTA_{TT} of the resting twitch responses was reduced in elderly subjects (single: 50.9%, doublet: 33.6%). The mean values ± standard deviations and *p*-values of contractile properties are displayed in Table 3.

Lean body mass of the leg was significantly reduced by 19.1% in elderly subjects (Table 1).

4. Discussion

The present study was designed to provide further insight into the neuromuscular mechanisms that determine the decrease in iMVT and explosive voluntary strength of the quadriceps muscle with aging. IMVT and MRTD as well as RTD and IMP in the early phase of contraction were reduced in healthy, active elderly men and women. Voluntary activation and normalized muscle activity during iMVT were decreased indicating a reduced neural drive to the muscle. The decline in MRTD and RTD during the early phase of contraction might be more affected by modulations of muscle properties than neural changes, as corresponding muscle activity remained primarily unchanged. There were no differences in the H_{max} and H_{max}/M_{max}-ratio of the vastus medialis, but a longer normalized M_{max} latency (Δt_M/height) was observed, indicating a decline in efferent conduction velocity. In addition, the age-related decreases in iMVT and explosive voluntary strength were accompanied by changes at the muscle level, as evidenced by a decline in twitch contractile properties and leg lean mass.

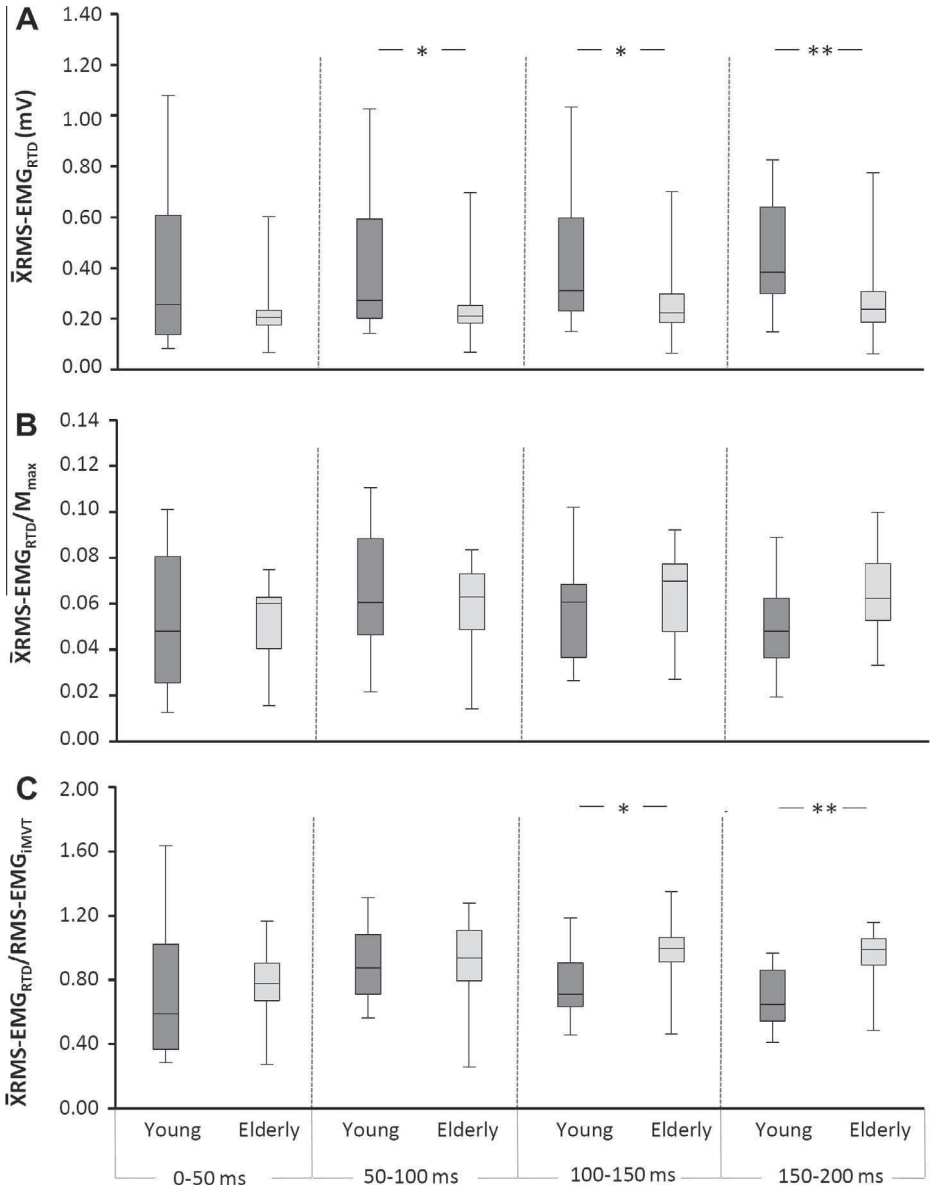

Fig. 4. Comparisons between groups of averaged root mean square of the EMG signal of vastus medialis, rectus femoris and vastus lateralis in four different time intervals after onset of the EMG signal (i.e., 0–50, 50–100, 100–150 and 150–200 ms): (A) absolute value ($\bar{X}$RMS–EMG$_{RTD}$), (B) normalized to M_{max} ($\bar{X}$RMS–EMG$_{RTD}$/M_{max}) and (C) normalized to RMS of the EMG signal during iMVT ($\bar{X}$RMS–EMG$_{RTD}$/RMS–EMG$_{iMVT}$). *Denotes a significant difference between the groups (*<0.05, **<0.01).

Table 2
Maximal *M* wave (M_{max}), maximal *H* reflex (H_{max}), H_{max}/M_{max}-ratio and height-normalized H_{max} and M_{max} latency (Δt_H/height, Δt_M/height). Values are presented as mean ± standard deviation. *Denotes a significant difference between the groups (*<0.05, **<0.01).

	Young	Elderly	*p*
Vastus medialis			
M_{max} (mV)	8.55 (3.04)	5.59 (3.33)	0.015*
H_{max} (mV)	1.75 (0.90)	1.57 (2.61)	0.695
H_{max}/M_{max}-ratio	0.23 (0.13)	0.34 (0.16)	0.129
Δt_H/height (ms cm^{-1})	0.105 (0.009)	0.111 (0.006)	0.065
Δt_M/height (ms cm^{-1})	0.033 (0.003)	0.036 (0.003)	0.038*
Vastus lateralis			
M_{max} (mV)	9.59 (3.27)	6.18 (3.75)	0.026*
Δt_M/height (ms cm^{-1})	0.035 (0.003)	0.037 (0.004)	0.075
Rectus femoris			
M_{max} (mV)	3.17 (0.95)	3.69 (2.01)	0.694
Δt_M/height (ms cm^{-1})	0.024 (0.003)	0.025 (0.004)	0.434

4.1. Voluntary activation

The decline in iMVT might be due to a reduced efferent motoneuron output, as confirmed by a decrease in voluntary activation and normalized muscle activity during iMVT. According to the literature, the extent to which a decrease in voluntary activation contributes to dynapenia of the quadriceps under isometric conditions remains less clear. Some authors have shown a decrease in voluntary activation (Harridge et al., 1999; Stackhouse et al., 2001; Stevens et al., 2003), whereas other investigations have observed no age-related differences (Cannon et al., 2007; Knight and Kamen, 2001; Miller et al., 2006; Roos et al., 1999; Suetta et al., 2009; Wilder and Cannon, 2009). The contrary findings might be related to divergent stimulation methods, testing procedures and the heterogeneity of the population. An age-related decrease in voluntary activation was detected when the central activation ratio was calculated (Knight and Kamen, 2001; Stackhouse et al., 2001;

Table 3
Contractile properties evaluated by single and doublet stimulation at supramaximal intensity. Peak torque (PT), maximal rate of torque development ($MRTD_{TT}$), maximal rate of torque relaxation ($MRTR_{TT}$), twitch contraction time (TCT_{TT}), twitch half relaxation time ($THRT_{TT}$) and total twitch area (TTA_{TT}) of the resting twitch torque. Values are presented as mean ± standard deviation. *Denotes a significant difference between the groups (*<0.05, **<0.01).

	Single		p	Doublet		p
	Young	Elderly		Young	Elderly	
PT (Nm)	39.39 (10.55)	15.35 (7.20)	0.000*	69.05 (14.67)	41.47 (13.91)	0.000**
$MRTD_{TT}$ (Nm s^{-1})	1814.41 (449.65)	673.54 (359.21)	0.000**	3220.57 (117.83)	1491.53 (173.61)	0.000**
$MRTR_{TT}$ (Nm s^{-1})	828.08 (171.78)	292.97 (163.94)	0.000**	1326.27 (80.82)	526.93 (42.76)	0.000**
TCT_{TT} (s)	0.064 (0.017)	0.069 (0.022)	0.101	0.067 (0.004)	0.085 (0.006)	0.013*
$THRT_{TT}$ (s)	0.084 (0.015)	0.073 (0.019)	0.106	0.090 (0.004)	0.083 (0.005)	0.290
TTA_{TT} (Nm s)	4.37 (0.55)	2.15 (0.44)	0.001**	8.70 (0.60)	5.78 (0.63)	0.006**

Stevens et al., 2003) whereas no differences could be identified when the interpolated twitch ratio was used (Cannon et al., 2007; Roos et al., 1999; Suetta et al., 2009; Wilder and Cannon, 2009). This discrepancy in results could be associated with the numbers of stimuli that were applied. The central activation ratio is classically derived by the torque generated with a pulse train. Pulse-train stimulation is more likely to cause a torque increment, and is thus regarded as more sensitive for detecting activation deficits (Miller et al., 1999). However, the present study showed an activation deficit by using paired pulses and calculating the interpolated twitch ratio whereby the standard formula (Allen et al., 1995) was corrected in order to bypass methodological limitations of the interpolated twitch technique (Folland and Williams, 2007). IMVT and the torque level just before the superimposed twitch were included in the original equation to correct the variations in the torque level (Strojnik and Komi, 1998).

The reduction of muscle activity during iMVT provided further evidence for the decreasing efferent motoneuron output to the quadriceps muscle. The only study available that has evaluated age-related changes in neural drive to the knee extensors by measuring voluntary activation and surface EMG under isometric condition was carried out by Cannon et al. (2007). These authors showed no age-related differences in either parameter between young and elderly women. However, comparing the present results with those of Cannon et al. is critical as possible gender differences might have affected the results.

In the present study, it was further shown that absolute and normalized MRTD (MRTD/iMVT) as well as absolute RTD and IMP in the early phases of torque development were reduced with aging. Absolute muscle activity was significantly decreased in elderly subjects. However, the analysis of absolute surface EMG signals is regarded as risky because various factors influence the signal and limit the comparison of data with young subjects (Klass et al., 2008). Therefore, muscle activity was normalized to M_{max} ($\overline{X}RMS–EMG_{RTD}/M_{max}$) and to muscle activity during iMVT ($\overline{X}RMS–EMG_{RTD}/RMS–EMG_{iMVT}$). Normalized EMG data suggest that the decline in explosive voluntary strength might be unrelated to changes in efferent neural drive to the quadriceps muscle. $\overline{X}RMS–EMG_{RTD}/M_{max}$ remained unchanged whereas $\overline{X}RMS–EMG_{RTD}/RMS–EMG_{iMVT}$ was significantly higher in elderly subjects in the later phases of contraction (100–150, 150–200 ms). In summary, the present data suggest that changes in skeletal muscle properties rather than neural adaptations contribute to the decrease in MRTD and RTD. This is an unexpected result, as it has been shown that aging is related to a decrease in RTD accompanied by a reduction in neural drive to the muscle (Ojanen et al., 2007). Klass et al. (2008) found that a slower RTD during the first 50 ms of torque rise was related to a reduction in motor unit discharge frequency and number of doublet discharges of the soleus muscle whereas no differences in normalized surface EMG data were observed. It was concluded that surface EMG may not always detect changes in the motor unit discharge rate. Smith et al. (1995) have shown that discharge frequency is not linearly related to surface EMG. Thus, estimating the neural drive to the muscle by surface EMG can be regarded critically (Farina et al., 2010).

Furthermore, Andersen and Aagaard (2006) observed a moderate correlation of voluntary rate of force development to twitch contractile properties. Klass et al. (2008) suggested that a greater slowing of RTD during fast voluntary contractions compared with electrically evoked twitch torques might be an indication of reduced neural activation of the muscle. The present data showed only a slight difference between MRTD and $MRTD_{TT}$ (4.9%) which indicates no age-related differences in muscular activation during RTD and thus supports the findings of normalized EMG data.

4.2. Spinal excitability

The H_{max} and the H_{max}/M_{max}-ratio of the vastus medialis remained unchanged which suggests that aging was not accompanied by differences in α-motoneuron excitability and/or presynaptic inhibition of primary muscle spindle afferents. Most studies of age-related modulations of the *H* reflex response have been carried out on the soleus muscle and have demonstrated contrary results (Kido et al., 2004; Koceja et al., 1995; Scaglioni et al., 2003). The discrepancies in results might be ascribed to differences and limitations of test conditions and stimulation protocols as well as to the variability of the reflex response which seems to enhance with increasing age (Falco et al., 1994). The measured decrease in M_{max} amplitudes is in line with findings in the literature and might be related to changes in the effectiveness of the neuromuscular junction and modifications at the muscle tissue level (Scaglioni et al., 2003). Furthermore, the analyses of normalized H_{max} latency (Δt_H/height) and Δt_M/height indicated a decrease in conduction velocity whereby only the efferent motor axons seemed to be affected. Thus, age-related changes in conduction speed, possibly due to a loss of largest axonal fibers, reduced myelination and a decreased internodal length of axons in the peripheral nerve, might contribute to the decrease in iMVT and explosive voluntary strength (Scaglioni et al., 2003).

4.3. Contractile properties and lean mass

The present results further suggest that the decrease in iMVT and explosive voluntary strength might be partially related to changes within the skeletal muscle system, since lean mass and twitch contractile properties were reduced. These results are in line with findings in the literature (Doherty, 2003; Roos et al., 1999). However, it has been demonstrated that the relationship between dynapenia and age-related loss of muscle mass (sacopenia) is weak. Thus, the contribution of other mechanisms, such as the slowing of contractile kinetics and/or neural modulations, seems to be greater in mediating age-related loss in muscle strength (Manini and Clark, 2011).

In conclusion, the data from this study confirm that the age-related decline in iMVT of the quadriceps muscle might be due to modulations of the nervous system and changes in skeletal muscle properties. Surprisingly, the reduction in MRTD as well as RTD and IMP during the early contraction phases might be more affected by modulations at the muscle level than by neural changes.

Acknowledgement

This study was supported by a scholarship of the doctorate program "Aging of individuals and society" of the Interdisciplinary Faculty, University of Rostock.

References

Allen GM, Gandevia SC, McKenzie DK. Reliability of measurements of muscle strength and voluntary activation using twitch interpolation. Muscle Nerve 1995;18(6):593–600.
Andersen LL, Aagaard P. Influence of maximal muscle strength and intrinsic muscle contractile properties on contractile rate of force development. Eur J Appl Physiol 2006;96(1):46–52.
Barbeau H, Marchand-Pauvert V, Meunier S, Nicolas G, Pierrot-Deseilligny E. Posture-related changes in heteronymous recurrent inhibition from quadriceps to ankle muscles in humans. Exp Brain Res 2000;130(3):345–61.
Behrens M, Mau-Moeller A, Bruhn S. Effect of exercise-induced muscle damage on neuromuscular function of the quadriceps muscle. Int J Sports Med 2012;33(8):600–6.
Cannon J, Kay D, Tarpenning KM, Marino FE. Comparative effects of resistance training on peak isometric torque, muscle hypertrophy, voluntary activation and surface EMG between young and elderly women. Clin Physiol Funct Imaging 2007;27(2):91–100.
Clark BC, Taylor JL. Age-related changes in motor cortical properties and voluntary activation of skeletal muscle. Curr Aging Sci 2011;4(3):192–9.
Doherty TJ. Invited review: aging and sarcopenia. J Appl Physiol 2003;95(4):1717–27.
Falco FJ, Hennessey WJ, Goldberg G, Braddom RL. H reflex latency in the healthy elderly. Muscle Nerve 1994;17(2):161–7.
Farina D, Holobar A, Merletti R, Enoka RM. Decoding the neural drive to muscles from the surface electromyogram. Clin Neurophysiol 2010;121(10):1616–23.
Folland JP, Williams AG. Methodological issues with the interpolated twitch technique. J Electromyogr Kinesiol 2007;17(3):317–27.
Folland JP, Wakamatsu T, Fimland MS. The influence of maximal isometric activity on twitch and *H*-reflex potentiation, and quadriceps femoris performance. Eur J Appl Physiol 2008;104(4):739–48.
Guiheneuc P, Bathien N. Two patterns of results in polyneuropathies investigated with the *H* reflex. correlation between proximal and distal conduction velocities. J Neurol Sci 1976;30(1):83–94.
Harridge SD, Kryger A, Stensgaard A. Knee extensor strength, activation, and size in very elderly people following strength training. Muscle Nerve 1999;22(7):831–9.
Kido A, Tanaka N, Stein RB. Spinal excitation and inhibition decrease as humans age. Can J Physiol Pharmacol 2004;82(4):238–48.
Kim J, Wang Z, Heymsfield SB, Baumgartner RN, Gallagher D. Total-body skeletal muscle mass: estimation by a new dual-energy X-ray absorptiometry method. Am J Clin Nutr 2002;76(2):378–83.
Klass M, Baudry S, Duchateau J. Voluntary activation during maximal contraction with advancing age: a brief review. Eur J Appl Physiol 2007;100(5):543–51.
Klass M, Baudry S, Duchateau J. Age-related decline in rate of torque development is accompanied by lower maximal motor unit discharge frequency during fast contractions. J Appl Physiol 2008;104(3):739–46.
Knight CA, Kamen G. Adaptations in muscular activation of the knee extensor muscles with strength training in young and older adults. J Electromyogr Kinesiol 2001;11(6):405–12.
Koceja DM, Markus CA, Trimble MH. Postural modulation of the soleus *H* reflex in young and old subjects. Electroencephalogr Clin Neurophysiol 1995;97(6):387–93.
Levine JA, Abboud L, Barry M, Reed JE, Sheedy PF, Jensen MD. Measuring leg muscle and fat mass in humans: comparison of CT and dual-energy X-ray absorptiometry. J Appl Physiol 2000;88(2):452–6.
Lohman M, Tallroth K, Kettunen JA, Marttinen MT. Reproducibility of dual-energy X-ray absorptiometry total and regional body composition measurements using different scanning positions and definitions of regions. Metabolism 2009;58(11):1663–8.
Lord SR, Ward JA, Williams P, Anstey KJ. Physiological factors associated with falls in older community-dwelling women. J Am Geriatr Soc 1994;42(10):1110–7.
Manini TM, Clark BC. Dynapenia and aging: an update. J Gerontol: Biol Sci Med Sci 2011;67(1):28–40.
Miller M, Downham D, Lexell J. Superimposed single impulse and pulse train electrical stimulation: a quantitative assessment during submaximal isometric knee extension in young, healthy men. Muscle Nerve 1999;22(8):1038–46.
Miller M, Flansbjer UB, Downham D, Lexell J. Superimposed electrical stimulation: assessment of voluntary activation and perceived discomfort in healthy, moderately active older and younger women and men. Am J Phys Med Rehabil 2006;85(12):945–50.
Murdock GH, Hubley-Kozey CL. Effect of a high intensity quadriceps fatigue protocol on knee joint mechanics and muscle activation during gait in young adults. Eur J Appl Physiol 2012;112(2):439–49.
Newman AB, Kupelian V, Visser M, Simonsick EM, Goodpaster BH, Kritchevsky SB, et al. Strength, but not muscle mass, is associated with mortality in the health, aging and body composition study cohort. J Gerontol: Biol Sci Med Sci 2006;61(1):72–7.
Ojanen T, Rauhala T, Hakkinen K. Strength and power profiles of the lower and upper extremities in master throwers at different ages. J Strength Cond Res 2007;21(1):216–22.
Porter MM, Myint A, Kramer JF, Vandervoort AA. Concentric and eccentric knee extension strength in older and younger men and women. Can J Appl Physiol 1995;20(4):429–39.
Roos MR, Rice CL, Connelly DM, Vandervoort AA. Quadriceps muscle strength, contractile properties, and motor unit firing rates in young and old men. Muscle Nerve 1999;22(8):1094–103.
Scaglioni G, Narici MV, Maffiuletti NA, Pensini M, Martin A. Effect of ageing on the electrical and mechanical properties of human soleus motor units activated by the *H* reflex and *M* wave. J Physiol 2003;548(Pt 2):649–61.
Schultz AB. Muscle function and mobility biomechanics in the elderly: an overview of some recent research. J Gerontol: Biol Sci Med Sci 1995;50 [Spec No60-3].
Smith L, Zhong T, Bawa P. Nonlinear behaviour of human motoneurons. Can J Physiol Pharmacol 1995;73(1):113–23.
Stackhouse SK, Stevens JE, Lee SC, Pearce KM, Snyder-Mackler L, Binder-Macleod SA. Maximum voluntary activation in nonfatigued and fatigued muscle of young and elderly individuals. Phys Ther 2001;81(5):1102–9.
Stevens JE, Stackhouse SK, Binder-Macleod SA, Snyder-Mackler L. Are voluntary muscle activation deficits in older adults meaningful? Muscle Nerve 2003;27(1):99–101.
Strojnik V, Komi PV. Neuromuscular fatigue after maximal stretch-shortening cycle exercise. J Appl Physiol 1998;84(1):344–50.
Suetta C, Aagaard P, Magnusson SP, Andersen LL, Sipila S, Rosted A, et al. Muscle size, neuromuscular activation, and rapid force characteristics in elderly men and women: effects of unilateral long-term disuse due to hip-osteoarthritis. J Appl Physiol 2007;102(3):942–8.
Suetta C, Hvid LG, Justesen L, Christensen U, Neergaard K, Simonsen L, et al. Effects of aging on human skeletal muscle after immobilization and retraining. J Appl Physiol 2009;107(4):1172–80.
Wilder MR, Cannon J. Effect of age on muscle activation and twitch properties during static and dynamic actions. Muscle Nerve 2009;39(5):683–91.
Zehr PE. Considerations for use of the Hoffmann reflex in exercise studies. Eur J Appl Physiol 2002;86(6):455–68.

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