Feasability of An Eccentric Isokinetic Protocol to Induce Trunk Muscle Damage: A Pilot Study

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Abstract
Eccentric exercise is discussed as a treatment option for clinical populations, but specific responses in terms of muscle damage and systemic inflammation after repeated loading of large muscle groups have not been conclusively characterized. Therefore, this study tested the feasibility of an isokinetic protocol for repeated maximum eccentric loading of the trunk muscles. Nine asymptomatic participants (5 f/4 m; 34 ± 6 yrs; 175 ± 13 cm; 76 ± 17 kg) performed three isokinetic 2-minute all-out trunk strength tests (1x concentric (CON), 2x eccentric (ECC1, ECC2), 2 weeks apart; flexion/extension, 60 °/s, ROM 55 °). Outcomes were peak torque, torque decline, total work, and indicators of muscle damage and inflammation (over 168 h). Statistics were done using the Friedman test (Dunn's post-test). For ECC1 and ECC2, peak torque and total work were increased and torque decline reduced compared to CON. Repeated ECC bouts yielded unaltered torque and work outcomes. Muscle damage markers were highest after ECC1 (soreness 48 h, creatine kinase 72 h; $p < 0.05$). Their overall responses (area under the curve) were abolished post-ECC2 compared to post-ECC1 ($p < 0.05$). Interleukin-6 was higher post-ECC1 than CON, and attenuated post-ECC2 ($p > 0.05$). Interleukin-10 and tumor necrosis factor-α were not detectable. All markers showed high inter-individual variability. The protocol was feasible to induce muscle damage indicators after exercising a large muscle group, but the pilot results indicated only weak systemic inflammatory responses in asymptomatic adults.

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Introduction

Eccentric exercise is known for a variety of unique features, supporting its potential to serve as a highly efficient treatment strategy for various clinical conditions [1]. Since high mechanical loads can be applied, it is assumed that eccentric exercises can induce greater muscle hypertrophy than concentric loading [2, 3]. Also, it has been shown that eccentric exercises induce reduced cardiovascular load [4, 5] compared to concentric exercises. Eccentric exercises further resulted in improved glucose tolerance, insulin sensitivity, and serum lipid levels after exercises [6, 7]. More recently, some studies indicated that eccentric exercises might also be able to positively influence inflammatory processes [8, 9].

Eccentric exercises are often followed by a delayed-onset muscle soreness (DOMS), temporary loss of muscle strength, swelling, reduced range of motion, and the release of myocellular proteins [10, 11]. Repeated application of eccentric loading can diminish those symptoms, known as the repeated bout effect (RBE). Eccentric muscle-damaging exercise is also often followed by local and systemic pro- and anti-inflammatory processes [12]. To assess the potential applicability of eccentric exercises for individuals in different clinical populations, it is important to characterize the consequences of repeated exercise bouts regarding muscle damage as well as inflammatory parameters. Cytokines that are released in response to exercise seem to be rapidly excreted from the circulation and therefore often only minor changes could be detected in serum, especially after eccentric resistant exercises [13]. Some studies postulated that cytokine plasma levels after repeated bouts of muscle-damaging strength exercises do not follow typical RBE characteristics, at least for the cytokine interleukin (IL)-6 [14, 15]. It is suggested that the release of circulating pro- and anti-inflammatory parameters may depend on time and intensity of loading, but also on the amount of recruited muscle mass [16]. Previous studies often focused on unilateral loading of small muscle groups, e. g., elbow flexors [17, 18], or were limited to large muscles of the lower extremities, e. g., knee flexors and extensors [15, 19, 20]. Only recently, Chen et al. conducted a study investigating the effects of nine eccentric exercises that were performed in the same session and involved a variety of muscle groups simultaneously [21]. This approach allows comparing DOMS and strength parameters of different muscle groups, for example, but only the total cumulative effect on plasma parameters after loading of all targeted muscle groups can be measured and specific systemic reactions to loading of particular muscle groups remain unanswered. To the authors' knowledge, isolated eccentric trunk loading protocols investigating muscle damage characteristics and inflammation responses of the trunk encompassing musculature are lacking.

Therefore, the aim of the present pilot study was to test the feasibility of an isokinetic protocol for repeated maximum eccentric loading of the trunk muscles to induce muscle damage and a RBE under standardized conditions in a population of asymptomatic participants. To investigate whether the protocol is suitable and can be used in a subsequent main study to further characterize inflammatory responses in the circulation, the purpose was to determine, if clinically relevant changes of inflammatory parameters are measurable in serum samples and if there are preliminary indications of a RBE of these parameters under these conditions.

Materials & Methods

Participants

Nine asymptomatic volunteers were included from a university setting (▶ **Table 1**). All participants met the following inclusion criterion: being pain-free/asymptomatic prior to the first measurement. Exclusion criteria were acute infection/cold, acute/chronic injuries of the musculoskeletal system, or pathologies or diseases that contraindicate physical activity. Participants were recreationally active and were asked to abstain from any unaccustomed vigorous physical activity from the week before the first measurement day until the study end. All participants gave written informed consent for their participation. The pilot study was conducted in accordance with the ethical standards for scientific research [22] and was supervised by the medical board of the University Outpatient Clinic at the University of Potsdam.

Study design and procedure

All participants performed three isokinetic strength tests of the trunk, one concentric (CON) and two eccentric (ECC1, ECC2), each 2 weeks apart (▶ **Fig. 1a**). An isokinetic dynamometer (Con-Trex MJ, TP1000 trunk module; physiomed AG, Schnaittach, Germany) was used to assess resulting torque output during trunk flexion and extension movements. Participants were placed inside the dynamometer in a semi-seated posture, adjusted to align the axis of rotation to the intersection point of the mid-axillary line and the lumbosacral junction (L5–S1 level) [23]. All tests were performed in a movement range from 10 ° of trunk extension to 45 ° of trunk flexion (55 ° of total ROM), at a rotational velocity of 60 °/s (▶ **Fig. 1b**). The following experimental procedure was executed at each of the three measurement days (always performed at the same time of the day): a) a warm-up trial consisting of 30 concentric repetitions; b) a resting period of 3 minutes; and c) a testing trial consisting of a 2-minute all-out MVC task, performed in concentric mode (CON) on the first measurement day and in eccentric mode (ECC1 and ECC2) on the second and third measurement days (▶ **Fig. 1a**).

Participants provided subjective ratings of perceived exertion (RPE; Borg scale 6–20) immediately after strength testing. Capillary blood samples from the earlobe were taken to analyze blood lactate levels before and immediately after each protocol (Biosen Analyzer; EKF Diagnostics, Barleben, Germany). Muscle soreness of the trunk muscles was rated by the participants using a numeric rating scale (NRS) from 0 (no soreness) to 10 (worst imaginable soreness) before (0 h) and 4 h, 24 h, 48 h, 72 h and 168 h after each strength test. Venous blood samples were taken from an antecubital forearm vein using a disposable needle and vacutainer (S-Monovette Serum, Sarstedt AG & Co., Nümberecht, Germany). Blood was drawn at baseline (0 h) and 4 h, 24 h, 48 h, 72 h and 168 h after each strength test. Blood was immediately centrifuged (2000 xg, 10 min) and serum samples were stored in aliquots at –80 °C until analysis. An overview of the outcome assessment time points before and after the isokinetic loading protocol is presented in ▶ **Fig. 1c**.

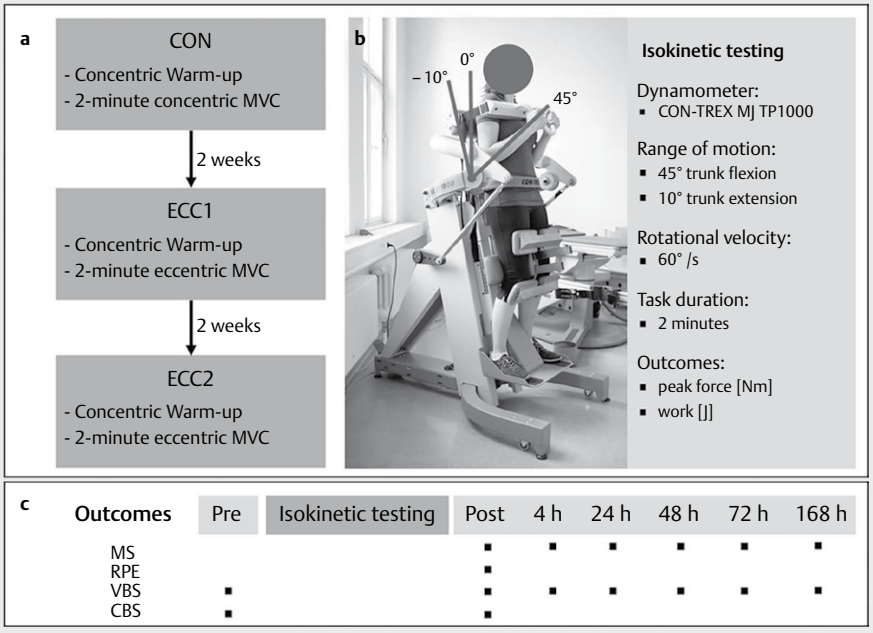
Blood analyses of markers of muscle damage and inflammation

Creatine kinase (CK), aspartate aminotransferase (AST), alanine aminotransferase (ALT), and C-reactive protein (CRP) concentra-

▶ **Table 1** Anthropometric data of participants.

	n	Age [years]	Weight [kg]	Height [m]	Phys. Activity [h/week] *
Overall	9	34 ± 6	76 ± 17	1.75 ± 0.13	2.1 ± 1.9
Females	5	35 ± 7	64 ± 13	1.65 ± 0.07	2.8 ± 2.1
Males	4	32 ± 5	90 ± 6	1.87 ± 0.08	1.2 ± 1.4

Data is presented as mean ± SD ; * Physical activity: hours of sport within 7 days before first measurement day.



▶ **Fig. 1** Measurement procedures with isokinetic trunk strength protocols and time points of assessed outcomes. **a**) General testing protocol including all three time points of measurement. **b**) Isokinetic testing conditions and exemplary participant within the isokinetic dynamometer. **c**) Time points of outcome assessments before and after isokinetic testing. CON, 2-minute concentric protocol; ECC1 and ECC2, 2-minute eccentric protocols; MVC, maximum voluntary contraction; MS, muscle soreness; RPE, rating of perceived exertion; VBS, venous blood sampling; CBS, capillary blood sampling; post, immediately after exercise protocol; 4 h–168 h hours after exercise protocol.

tions were measured of samples taken pre (0 h) and 4 h, 24 h, 48 h, 72 h, 168 h after each strength test. Analysis was done with the Pentra C400 clinical chemistry analyzer equipped with Pentra cuvette segments (Axon Lab AG, Baden, Switzerland) by using the calibrator reagents (Pentra CRP CAL, AXON MC) according to the manufacturer's instructions.

Cytokine concentrations were determined in serum samples taken before (0 h), 24 h, 48 h, 72 h and 168 h after each strength test. Commercially available high-sensitivity enzyme-linked immunosorbent assay (ELISA) kits (IBL International, Hamburg, Germany) were used. IL-6, IL-10 and tumor necrosis factor α (TNF-α) were analyzed using 50 µl of serum samples according to manufacturer's instructions.

Data analysis and statistics

Maximum peak torque [Nm] of trunk extensors (CON: trunk extension movement; ECC1, ECC2: trunk flexion movement) and trunk flexors (CON: trunk flexion movement; ECC1, ECC2: trunk extension movement) were derived from the mean value of the three highest out of five repetitions at the beginning (first five repetitions), midpoint (repetitions 23 to 27) and end (last five repetitions) of each trial [24]. Additionally, the resulting work [J] over the whole trial of CON, ECC1, and ECC2 was calculated during trunk flexion and extension separately. Peak torque reduction was calculated as peak torque difference [Nm] at mid- and endpoint of the trial in relation to the beginning. Overall responses of muscle damage and inflammation markers were stated as "area under the curve" (AUC), which was calculated for each parameter over time from pre-exercise (0 h) to 72 h after each loading trial.

Peak torque, work, RPE, blood lactate, and markers of muscle damage and inflammation were presented descriptively as mean ± standard deviation (SD). Statistical analyses were done for peak torque differences [Nm] between conditions (CON vs. ECC1 vs. ECC2) at the beginning as well as for torque reduction [Nm] within each condition (mid- vs. endpoints) for trunk flexor torque and trunk extensor torque separately. Further, comparisons between conditions (CON vs. ECC1 vs. ECC2) were done for total work, RPE (post-exercise), blood lactate (pre- and post-exercise) as well as muscle damage and inflammation markers for AUC and at the time point of post-ECC1 peak. All statistical analyses were done by

Friedman's ANOVA with Dunn's multiple comparisons test as data deviated from normal distribution (Shapiro Wilk test). The level of significance was set at $\alpha < 0.05$.

Results

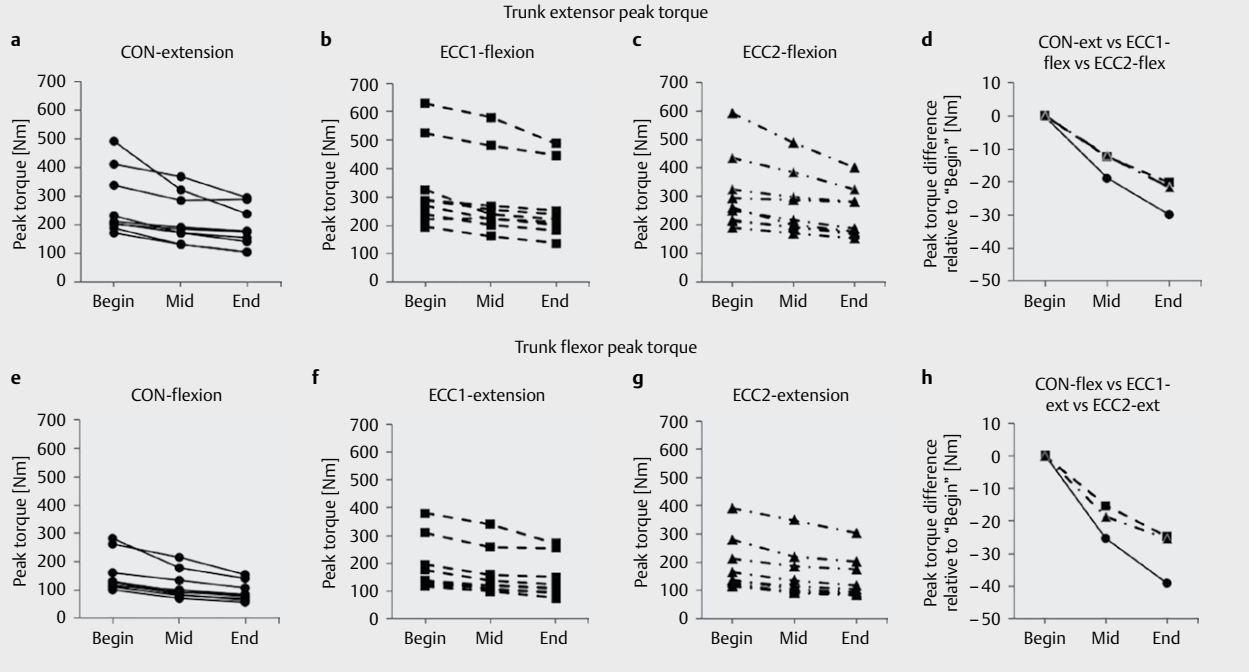
Strength measurements

CON resulted in 48 ± 1 repetitions of trunk flexion and extension during the 2-minute all-out protocol, ECC1 in 50 ± 0 and ECC2 in 50 ± 1 repetitions, respectively. Higher mean peak torques were found descriptively at the beginning for both flexor and extensor torque in ECC1 and ECC2 compared to CON (▶ **Table 2**) with statistically significant differences for extensor torque between CON and ECC1 (p = 0.003) and for flexor torque between CON and ECC1 (p = 0.003) as well as CON and ECC2 (p = 0.007). Peak torque reduction showed lower values during the mid- and endpoint for both ECC1 and ECC2 compared to CON (▶ **Fig. 2d/h**). Individual courses of flexor and extensor torque during CON, ECC1 and ECC2 for each participant are shown in ▶ **Fig. 2**. Total work differed statistically significantly between CON and ECC1 (extensor torque p = 0.007; flexor torque p = 0.014), and between CON and ECC2 (extensor torque p = 0.029) (▶ **Table 2**). Also, blood lactate accumulation post-exercise differed statistically significantly between CON and both eccentric bouts (ECC1: p = 0.029, ECC2: p = 0.049). In contrast, blood lactate pre-exercise and RPE post-exercise did not differ between the three protocols (▶ **Table 2**).

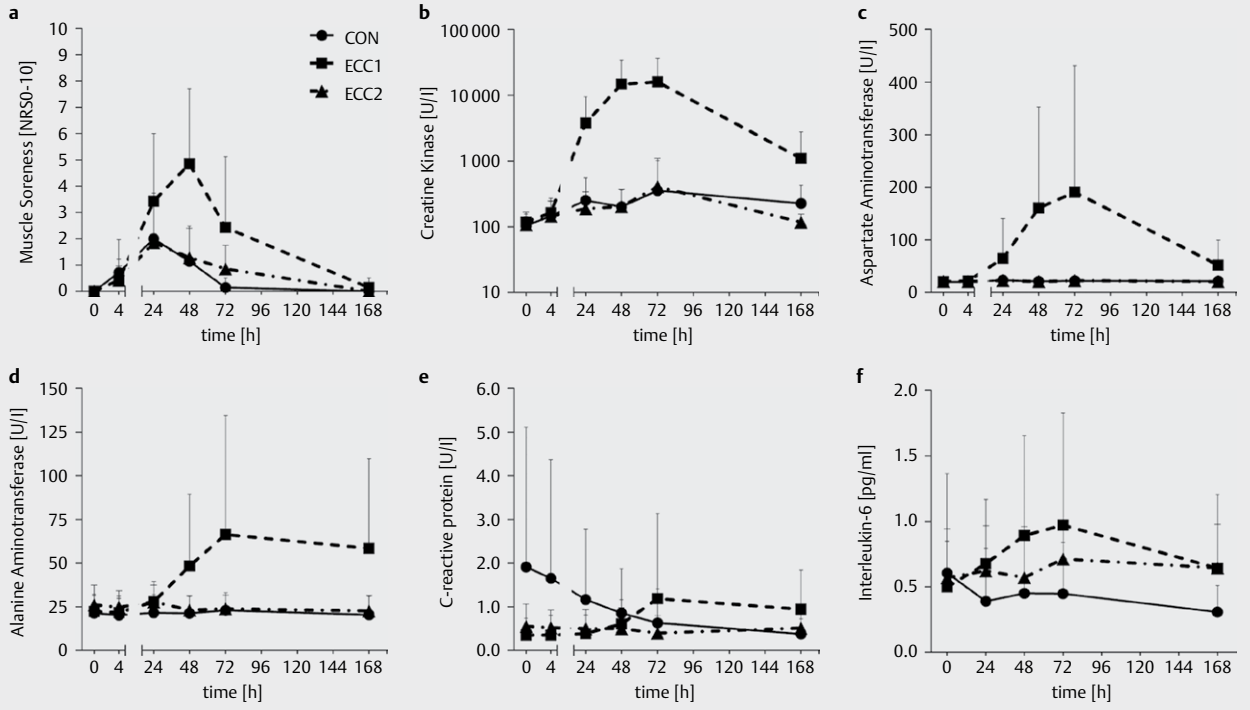
▶ **Table 2** Peak torque, total work, blood lactate, and RPE for the maximal concentric and repeated maximal eccentric loading of the trunk.

	Movement direction	Peak torque [Nm]			Work [J]	Blood Lactate [mmol/l]		RPE [Score]
		Begin	Mid	End	Begin to End	pre	post	post
CON	Extension	272 ± 114	218 ± 85	187 ± 71	7279 ± 2853	0.9 ± 0.4	6.9 ± 2.3	18.9 ± 1.2
	Flexion	156 ± 68	116 ± 50	93 ± 34	4273 ± 1920			
ECC1	Extension	189 ± 93#	161 ± 83	142 ± 72	5707 ± 3254#	1.0 ± 0.3	4.9 ± 2.3#	18.8 ± 1.3
	Flexion	331 ± 147	293 ± 141	265 ± 120	10369 ± 4638#			
ECC2	Extension	186 ± 94#	154 ± 86	140 ± 74	5368 ± 3154	0.9 ± 0.3	4.8 ± 2.3#	18.7 ± 1.1
	Flexion	309 ± 130	270 ± 107	239 ± 87	9311 ± 3487#			

Data are presented as mean ± SD for N = 9. CON, 2-minute concentric protocol; ECC1, ECC2, 2-minute eccentric protocol; RPE, ratings of perceived exertion. Statistically significant differences (p < 0.05) for CON vs. ECC1 or CON vs. ECC2 are indicated by (#); Friedman test with Dunn's multiple comparisons test.



▶ **Fig. 2** Trunk extensor and flexor peak torque during the 2-minute all-out protocols. CON, 2-minute concentric protocol (solid line); ECC1 and ECC2, 2-minute eccentric protocol (dotted line and dash-dotted line); Begin, beginning; Mid, midpoint; End, endpoint. **a–c)** Individual courses of trunk extensor peak torque for each participant. **e–g**) Individual courses of trunk flexor peak torque. **d**, **h**) Mean peak torque courses with differences relative to Begin.



▶ **Fig. 3** Changes in markers of muscle damage and inflammation after the 2-minute all-out protocols. **a**) Perceived muscle soreness was assessed using a Numeric Rating Scale (NRS 0–10). Concentrations of muscle enzymes (**b–d**) and inflammatory parameters (**e**, **f**) were assessed in serum samples. Data is presented as mean ± SD for N = 7 (A) and N = 7–9 (B-F). CON, 2-minute concentric protocol; ECC1, ECC2, 2-minute eccentric protocol. Note the logarithmic scale on the y axis of graph B.

Markers of muscle damage

Perceived muscle soreness increased following CON with a peak value at 24 h post-exercise (NRS 2.0 ± 1.7) and returned to baseline after 72 h (▶ **Fig. 3a**). ECC1 induced a delayed but higher increase of muscle soreness peaking at 48 h (NRS 4.9 ± 2.9) and reaching statistical significance in comparison to CON ($p = 0.049$; ▶ **Table 3**). Muscle soreness remained increased (NRS 2.4 ± 2.7) at 72 h post-ECC1. Following ECC2, muscle soreness increase was statistically significantly lower at 48 h compared to ECC1 ($p = 0.049$).

Serum concentration of the muscle enzyme CK increased after CON at 72 h post-exercise (▶ **Fig. 3b**, ▶ **Table 3**). ECC1 induced a high increase of CK serum level, which peaked at 72 h post-exercise and was statistically significantly higher compared to CON ($p = 0.007$). This increase had completely abated after ECC2 ($p = 0.029$) and reached a level comparable to post-CON. Overall, CK response showed a high variability between participants, indicated by high standard deviations. Two participants showed no increase of CK at all measured time points, whereas another showed a 375-fold increase in CK at 72 h post-ECC1. AST and ALT (▶ **Fig. 3c, d**) serum concentrations increased only after ECC1, peaked at 72 h post-exercise (▶ **Table 3**) and remained elevated for 7 days after muscle-damaging exercise. The overall response of the main markers of muscle damage 72 h after loading had significantly abated after ECC2 compared to ECC1 (AUC: muscle soreness: $p = 0.049$, CK: $p = 0.029$; ▶ **Table 3**).

Markers of inflammation

Before CON, baseline mean CRP was higher compared to all other measured time points (▶ **Fig. 3e**), caused by slightly increased CRP levels of two single participants. Mean serum CRP levels did not change over the observed period and were not statistically significant different 72 h after CON, ECC1, and ECC2 (▶ **Table 3**).

Although serum levels of IL-6 remained unchanged after CON, mean values increased after ECC1 by 2-fold compared to pre-exercise (pre: 0.5 ± 0.3 pg/ml, 72 h post: 1.0 ± 0.9 pg/ml; ▶ **Fig. 3f**). Overall IL-6 response was enhanced after ECC1 compared to CON, but differences did not reach statistical significance (AUC, $p = 0.102$; ▶ **Table 3**). After ECC2, IL-6 levels and overall IL-6 answers were, although not statistically significant, lower than post-ECC1 but higher than after CON. In total, IL-6 responses showed a high inter-individual variability. While three participants showed no change in systemic IL-6 levels after ECC1, the IL-6 levels in serum samples from the other participants increased post-ECC1 by 1.7-fold up to 8.8-fold compared to pre-ECC1.

Serum levels of cytokines TNF-α and IL-10 were mainly below the detection limits at all analyzed time points.

Discussion

The present pilot study investigated the feasibility of an isokinetic eccentric maximum trunk loading protocol to assess muscle dam-

▶ **Table 3** Changes in markers of muscle damage and inflammation over 72 h after the 2-minute all-out protocols. Markers of muscle damage and inflammation were assessed before and at different time points after each exercise bout. Peak time after ECC1 was selected (according to ▶ **Fig. 3**) and values of each parameter at this time point after CON, ECC1, and ECC2 were statistically analyzed. Area under the curve (AUC) was calculated for the period of pre- (0 h) to 72 h post-exercise.

	CON	ECC1	ECC2
Muscle Soreness			
[NRS] at peak (48 h post)	1.1 ± 1.3	4.9 ± 2.9#,$	1.3 ± 1.1
AUC [NRS * 72 h]	82 ± 61	226 ± 143#,$	87 ± 73
Creatine kinase (CK)			
[U/l] at peak (72 h post)	356.9 ± 657.5	15998.9 ± 20520.2#,$	408.7 ± 692.5
AUC [U/l * 72 h]	16651 ± 14282	595084 ± 808004$	15764 ± 16472
Aspartate aminotransferase (AST)			
[U/l] at peak (72 h post)	22.4 ± 5.1	190.9 ± 240.3#	22.8 ± 6.6
AUC [U/l * 72 h]	1580 ± 416	7513 ± 8371	1566 ± 265
Alanine aminotransferase (ALT)			
[U/l] at peak (72 h post)	23.0 ± 10.2	66.4 ± 68.1	24.0 ± 7.6
AUC [U/l * 72 h]	1552 ± 705	2803 ± 1901	1788 ± 579
C-reactive protein (CRP)			
[U/l] at peak (72 h post)	0.6 ± 0.8	1.2 ± 1.9	0.4 ± 0.4
AUC [U/l * 72 h]	78 ± 105	42 ± 40	35 ± 29
Interleukin-6 (IL-6)			
[pg/ml] at peak (72 h post)	0.4 ± 0.4	1.0 ± 0.9	0.7 ± 0.3
AUC [pg/ml * 72 h]	33 ± 32	58 ± 39	43 ± 25
Tumor necrosis factor-α (TNF-α)			
[pg/ml]	n.d.	n.d.	n.d.
Interleukin-10 (IL-10)			
[pg/ml]	n.d.	n.d.	n.d.

Data are presented as mean ± SD for N = 7 (muscle soreness) or N = 9. CON, 2-minute concentric protocol; ECC1, ECC2, 2-minute eccentric protocol; n.d., not detectable (high sensitivity ELISA, limit of detection (LoD) (TNF-α) = 0.13 pg/ml; LoD (IL-10) = 0.05 pg/ml). Statistically significant differences ($p < 0.05$) are indicated by (#) for CON vs. ECC1 and ($) for ECC1 vs. ECC2; Friedman test with Dunn's multiple comparisons test.

age and (anti-) inflammatory responses following single and repeated bouts of trunk muscle exercise. Comparison of assessed force parameters of the three applied protocols yielded an increased peak torque output, accompanied by a reduced decline of torque over the 2-minute all-out duration, and an increased total work for both eccentric protocols in comparison to the concentric protocol. Repeated bouts of eccentric loading resulted in unaltered torque and work-related outcomes. Despite the presence of highly increased markers of muscle damage, especially in CK, only a slight increase in serum IL-6 level after the first eccentric loading was observed, which was descriptively lowered after the second eccentric exercise session. Neither pro-inflammatory cytokine TNF-α nor anti-inflammatory cytokine IL-10 were detectable in serum samples taken before or after the protocols, raising the question as to whether there are systemically relevant inflammatory reactions after exercise-induced trunk muscle damage, at least in young asymptomatic participants.

Strength measurements

All trunk strength protocols under maximal effort condition were successfully performed, without any adverse events being detected throughout the study and thus were proven to be feasible for all participating volunteers. Comparable amounts of total load repetition were realized across all three testing protocols with minor differences being expected to be caused by alterations at the turning points of movement directions. In line with physiological anticipation, torque output was increased and torque decline over time (at mid- and endpoint of trial) was reduced in both eccentric protocols compared to the concentric protocol [2, 3, 25]. Intra-individual torque comparisons (▶ **Fig 2a–c, e–g**) revealed further a homogeneous torque decline over time for all participants. Decreased blood lactate in eccentric, at comparable levels of RPE across all protocols, further confirmed previously reported characteristics of eccentric loading protocols [2, 26–28]. Thus, the protocol proved to be able to elicit distinct loading situations, both in concentric and eccentric contraction modes to investigate related muscle damage.

Muscle damage markers

Delayed-onset muscle soreness and increased serum concentrations of muscle-derived enzymes (▶ **Fig. 2**, ▶ **Table 1**) were induced by the first eccentric protocol. Even so, there was a huge inter-individual variation that might be linked to the phenomenon of so-called high, moderate, or non-CK responders [29]. Unfortunately standardized definitions of responder categories are lacking [30, 31]. Therefore and because of the small sample size in this pilot

study, no subgroup analyses were performed. However, the overall abolished response in muscle damage markers after the repeated eccentric loading of the trunk were in line with similar findings after repeated eccentric exercises of other muscle groups [32, 33]. Accordingly, these findings provide evidence for the presence of a RBE after recurrent isolated maximal eccentric loading of the trunk muscles.

Inflammatory markers

Pro-inflammatory cytokine TNF-α was undetectable at all analyzed time points (▶ **Table 2**). Since TNF-α is relevant to local rather than systemic inflammatory responses after eccentric loading, no detectable serum levels or at least no changes were expected in asymptomatic participants [12]. IL-6 is the most commonly studied cytokine in terms of eccentric contractions, but acute eccentric loading of knee extensors and elbow flexors led to contradictory results regarding the response characteristics [17–19]. In this pilot study, no increase of serum IL-6 after CON, but a 2-fold increase 72 h after ECC1 (▶ **Fig. 3b**, ▶ **Table 2**) was detected. IL-6 is able to induce the acute-phase protein CRP, and the overall 3-fold CRP increase after ECC1 (versus pre-exercise, ▶ **Fig. 3a**) slightly reflected the observed IL-6 increase, albeit without reaching statistical significance and showing a high variability across participants. A study with obese elderly women identified high IL-6 responders after acute eccentric stress, which was not directly related to high CK responses, so that a causal relationship between IL-6 and CK increase remains questionable [30]. Additionally, it should be considered that the huge inter-individual variability seen in the change of these parameters might be caused by factors that were not controlled and tested for within this pilot study. CRP is involved in the innate immune signaling and therefore influenced by numerous immune-stimulating conditions like infections [34]. Although we excluded participants with upper respiratory tract infections, two participants started with a slightly increased CRP level probably caused by an unrecognized infection. Moreover, eccentric exercise-induced increases of CRP and IL-6 could be influenced by nutritional status [35, 36], which was not controlled or documented. Because of the overall minor and highly variable IL-6 response, an evaluation of the effects of repeated eccentric loadings is limited. Nevertheless, the IL-6 response after ECC2 tended to be lower than after ECC1 two weeks earlier, indicating the presence of an RBE for IL-6. Results from previous studies regarding effects of repeated eccentric bouts on systemic IL-6 level are limited and inconclusive. Willoughby et al. showed no differences in plasma IL-6 and mRNA levels between two eccentric bouts performed three weeks apart [15]. Deyhle et al. showed a decrease of intra-muscular IL-6 after a first eccentric bout, which was abolished after repetition [37]. As intra-muscular pro-inflammatory parameters increased and the anti-inflammatory mediator IL-4 decreased, the authors hypothesized that following a second bout of lengthening contractions an enhanced inflammatory response might occur [37]. In contrast, other studies showed a systemic increase of the anti-inflammatory cytokine IL-10, both in response to prolonged high-intensity downhill exercise and eccentric cycling [38, 39] as well as after isolated contraction of the elbow flexors [17, 18]. In our study investigating a large group of trunk muscles, IL-10 was not detectable before or after eccentric loading. Similarly, other studies loading large muscle groups (knee extensors, calf muscles) also reported unchanged [20] or even undetectable IL-10 levels [19]. These observations, in line with our findings, may indicate that not only intensity and duration of eccentric loading and extent of muscle damage are influencing factors for a systemic anti-inflammatory response [40, 41] but also the loaded muscle group. Particularly the lower extremities and the trunk are repeatedly exposed to high loads during decelerating movements and work against gravity during daily life activities. Therefore, these muscle groups may be pre-conditioned for eccentric loads leading to an attenuated inflammatory response following maximal eccentric bouts.

Clinical relevance

Although the present pilot study was conducted with young asymptomatic participants, potential influences of acute eccentric exercise bouts on systemic levels of inflammatory mediators in patients could be of clinical interest. For example, people with a low physical fitness level and overweight or obesity often show so-called low-grade systemic inflammation [42]. Exercise in general is known for its anti-inflammatory effects [43] and especially eccentric training was recently discussed as a promising intervention strategy for patients with obesity or metabolic disorders [44]. However, prior to application it needs to be clarified which eccentric exercise regimen (involved muscles, duration, intensity, and frequency), should be applied in different clinical populations and if acute eccentric loading of isolated muscle groups could be sufficient to induce anti-inflammatory responses. In previous studies, running downhill [6] and descending stairs [45], but also eccentric loading of individual muscle groups [46] showed an improvement in relevant metabolic parameters in older and/or overweight adults. It is particularly important to clarify whether loading of different (small and large) muscle groups triggers different inflammatory reactions and if loading of the trunk muscles is an appropriate approach to induce relevant systemic reactions in patients.

Limitations

Due to the asymptomatic young participants involved, no conclusion can be drawn for clinical populations. Moreover, the small sample size and heterogeneity of participant's CK responses after maximal eccentric loading limit the generalizability of the results. Although the chosen trunk protocol proved to be feasible and the assessed performance parameters revealed typical loading characteristics of an eccentric maximum load exercise, the duration of the eccentric loading may not have been sufficient to trigger an inflammatory response. These limitations must be considered for future studies.

The present pilot study proved that a 2-minute all-out eccentric strength protocol of the trunk is feasible to induce characteristic indicators of muscle damage in the trunk, which abate after a second maximal eccentric bout two weeks later. It further provided preliminary indications that despite the muscle-damaging exercise of a large muscle group, only weak systemic pro-inflammatory responses with little evidence for the presence of a repeated bout effect for IL-6, and no measurable changes in serum anti-inflammatory parameters might be elicited in asymptomatic adults. Further investigations are required to test whether these characteristics

are representative for a specific response of the trunk muscles being pre-conditioned by the frequent eccentric loading during daily life.

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Conflict of Interest

The authors declare that they have no conflict of interest.

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