



Original research

Movement profile influences systemic stress and biomechanical resilience to high training load exposure



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ABSTRACT

Objectives: Determine the influence of movement profile on systemic stress and mechanical loading before and after high training load exposure.

Design: Cross-sectional cohort study.

Methods: 43 physically active, college-aged field or court sport female athletes participated in this study. Participants were assigned to a “excellent” ($n=22$; age = 20.5 ± 1.9 yrs, height = 1.67 ± 0.67 m, mass = 64.5 ± 7.8 kg) or “poor” ($n=21$; age = 20.4 ± 1.3 yrs, height = 1.69 ± 0.67 m, mass = 60.9 ± 6.1 kg) movement group defined by The Landing Error Scoring System. Participants completed five cycles of high training load exercise of 5-min treadmill-running at a speed coincident with 100–120% ventilatory threshold and 10 jump-landings from a 30-cm box. Jump-landing vertical ground reaction force and serum cortisol were evaluated prior to and following exercise. Vertical ground reaction force ensemble averages and 95% confidence interval waveforms were generated for pre-exercise, post-exercise, and pre-post exercise changes. A two-way mixed model ANOVA was used to evaluate the effect of movement profile on systemic stress before and after exercise.

Results: There was no significant difference in changes in serum cortisol between the poor and excellent groups ($p=0.69$) in response to exercise. Overall, individuals in the poor group exhibited a higher serum cortisol level ($p<0.05$, $d=0.85$ [0.19,1.48]). The poor group exhibited higher magnitude vertical ground reaction force prior to ($d=1.02$ – 1.26) and after exercise ($d=1.15$) during a majority of the stance phase.

Conclusions: Individuals with poor movement profiles experience greater mechanical loads compared to individuals with excellent movement profiles. A poor movement profile is associated with greater overall concentrations of circulating cortisol, representative of greater systemic stress.

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1. Introduction

Rapid elevations or “spikes” in training load are associated with an increased risk of injury and illness.^{1,2} Recent evidence suggests individuals who elevate their weekly training load (acute training load) greater than $1.5\times$ their average training load from the month prior (chronic training load) are at an increased risk of injury.^{1,2} However, many individuals do not sustain subsequent injury even when training load exposure exceeds this acute-to-chronic threshold.^{1–3} Thus, it appears there are mediating factors

that influence an athlete’s response to high training load (HTL) exposure.^{1–3}

Movement quality or an individual’s movement profile is a modifiable risk factor for non-contact injury during sport and physical activity.^{4–6} Poor movement patterns such as excessive medial knee motion and/or stiff and rigid movement patterns are commonly reported mechanisms and risk factors for lower extremity injury.^{4–6} In contrast, those with excellent movement patterns, characterised by limited frontal plane motion and a “soft” sagittal plane movement strategy (greater knee and hip flexion motion), appear less prone to injury during training and competition.^{4–6} As such, movement quality may mediate the effects of HTL exposure and explain the variability in injury rates when individuals are exposed to HTLs during sport and physical activity.

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Movement quality influences vertical ground reaction force (vGRF), a biomechanical variable representative of the external force or load experienced by the body.⁷ Additionally, recent evidence suggests that movement quality influences time to fatigue, post-exercise biomechanics linked to lower extremity injury,^{8,9} and metabolic economy¹⁰ in healthy college-aged individuals. These findings further implicate that an individual's response to HTLs may be mediated by their movement profile.

An individual's response to HTLs can be quantified using objective biological markers such as circulating serum cortisol (sCORT).³ Elevations in sCORT suggest an increased stress response to an external stimulus or load.¹¹ Individuals with poor movement profiles may experience greater overall mechanical load exposure compared to those with excellent movement profiles, which may result in greater sCORT elevations following HTL exposure. However, the influence of an individual's movement profile on their response to HTLs has not been directly investigated. Therefore, the primary purpose of this study was to investigate the influence of movement profile on biomechanical and biological responses to HTL exposure. We hypothesized that individuals with a poor movement profile would experience greater vGRF and sCORT elevations compared to those with excellent profiles following HTL exposure. A secondary purpose of this study was to determine if there is an overall influence of movement profile and HTL exposure on biological and biomechanical markers of systemic stress and mechanical loading. We hypothesized that individuals with a poor movement profile would exhibit a greater overall level of sCORT and vGRF before and after HTL compared to individuals with an excellent movement profile, and that vGRF and sCORT levels would increase in response to HTL exposure across movement profile groups.

2. Methods

Inclusion criteria required that participants were current university students 18–25 years of age, actively participating in at least 30 min of moderate to high-intensity physical activity a minimum of three days per week and had a history of field and/or court sport participation at the secondary school varsity level. Individuals were excluded if they had history of lower extremity surgery within the past year, lower extremity joint surgery, prior ACL or meniscal injury, or lower extremity injury in the past six months, neuroendocrine, neurological or metabolic disease or condition, dysmenorrhea, or amenorrhea within the past six months. Participants were enrolled if they demonstrated a poor or excellent movement profile using the joint displacement and overall impression scoring item criteria previously described by Padua et al.'s Landing Error Scoring System (LESS) assessment.⁷ An excellent movement profile was defined during the jump-landing with a lack of medial knee displacement, with the center of the patella moving lateral to the great toe and an "average" or "soft" landing evidenced by "some" to "large" sagittal plane displacement of the trunk, hip, and knee.⁷ A poor movement profile was defined by presence of medial knee displacement, with the center of the patella moving in-line or medial to the great toe, and an "average" or "stiff" landing evidenced by "some" or "very little" sagittal plane displacement at the trunk, hip, and knee.⁷

A priori power analysis of previously published data revealed that a total sample size of 40 participants (excellent ($n=20$) & poor ($n=20$)) would permit investigators to detect a significant change in vGRF¹² and sCORT¹³ from pre- to post-HTL, with a power of at least 0.80 and $\alpha=0.05$.

All procedures (Fig. 1) were approved by the biomedical institutional review board (IRB# 14-3298) at The University of North Carolina at Chapel Hill. Pre-test guidelines are presented in Fig. 1. All participants reported to the research laboratory between 14:00 and

16:00 for their testing sessions to control for the diurnal variation of cortisol.¹⁴

Following informed consent, verification of pre-test guideline adherence, and a standardized 30-min supine rest period a sample of blood was drawn via antecubital venipuncture using a 20G 1½" BD PrecisionGlide™ vacutainer needle (Becton Dickinson, Franklin Lakes, New Jersey, USA). Participant blood samples were collected into 10 ml serum separator tubes with clot activator gel (BD SST Vacutainer, Becton Dickinson, Franklin Lakes, New Jersey, USA) and stored at 2–4 °C, then allowed to clot overnight. Samples underwent centrifugation (IEC Centra-8R Refrigerated Centrifuge, Thermo Fischer Scientific, Waltham, Massachusetts, USA) at 3000 RPM for 15 min at 4 °C. Serum was aliquoted into sterile 2.0 ml polypropylene long-term storage cryogenic vials (Nalgene–Thermo Fischer Scientific, Waltham Massachusetts, USA) and stored at –80 °C until thawing for ELISA procedures.

After the pre-HTL blood sample was collected, participants stood atop a 30-cm box located 50% body height from the leading edge of two conductive force plates.⁷ Participants were instructed to "face forward, and jump forward and down to the center of the force plates, and rebound upward for a maximal vertical jump" with their respective foot landing on a force plate.⁷ All participants completed three practice trials of the jump-landing task. Participants then completed three trials of the jump-landing task. vGRF data were sampled at 1000 Hz. The jump-landing assessment was completed prior to and immediately following HTL exposure.

For ventilatory threshold (VT) determination, participants completed a speed-only graded submaximal aerobic capacity assessment¹⁵ on a motorized treadmill (GE T2100 Exercise Stress System, General Electric–Healthcare, Little Chalfont, UK) using a metabolic cart (TrueOne 2400 Metabolic Measurement System, Parvo Medics, Sandy, Utah, USA) and chest-mounted heart rate monitor (A39 Exercise Monitor, Under Armour Inc., Baltimore, Maryland, USA). Following completion of the VT assessment, the participant's VT was determined using a modified V-slope method.¹⁶ After determination of VT, participants completed the acute HTL protocol (Fig. 1). The acute HTL protocol required that participants complete five cycles of five minutes of treadmill running at a speed coincident 100–120% of VT and 10 jump-landings from a 30-cm box placed half their height from a landing target line. To effectively control for physiological stress exposure during treadmill running, metabolic gas assessment, rate of perceived exertion, and heart rate were monitored to ensure the treadmill running speed was coincident with 100–120% of VT.

All vGRF data were analyzed for the dominant (kicking) limb during the stance phase of the jump-landing task; defined as the instant of initial ground contact (vGRF > 10 N) to toe-off (vGRF < 10 N).⁷ vGRF data were normalized to body weight (\times BW) and analyzed as continuous waveforms (Fig. 2) during the stance phase of the jump-landing.¹⁷ Ensemble average waveforms were time normalized to 201 data points (knots) over the stance phases of the three jump-landing task trials using a cubic spline function.¹⁷ 201 knot change score waveforms were calculated by subtracting the pre-HTL vGRF time series from the post-HTL vGRF time series for each participant.¹⁷

Pre- and post-HTL sCORT concentrations were analyzed using commercially available ELISA kits (Abcam Cortisol ELISA kit # ab108665, Cambridge, Massachusetts, USA). The results of the biomarker assays were assessed in duplicate using a 96 well, 8-channel microplate reader (ChroMate 4300, Awareness Technology Inc., Hauppauge, New York, USA). All samples from an individual participant were analyzed on a single ELISA plate. The intra-assay and inter-assay coefficients of variation were 1.57% and 4.48% respectively.

A custom computer program (MATLAB 2016a, MathWorks, Natick, Massachusetts, USA) was used to calculate Pre-HTL, post-HTL,

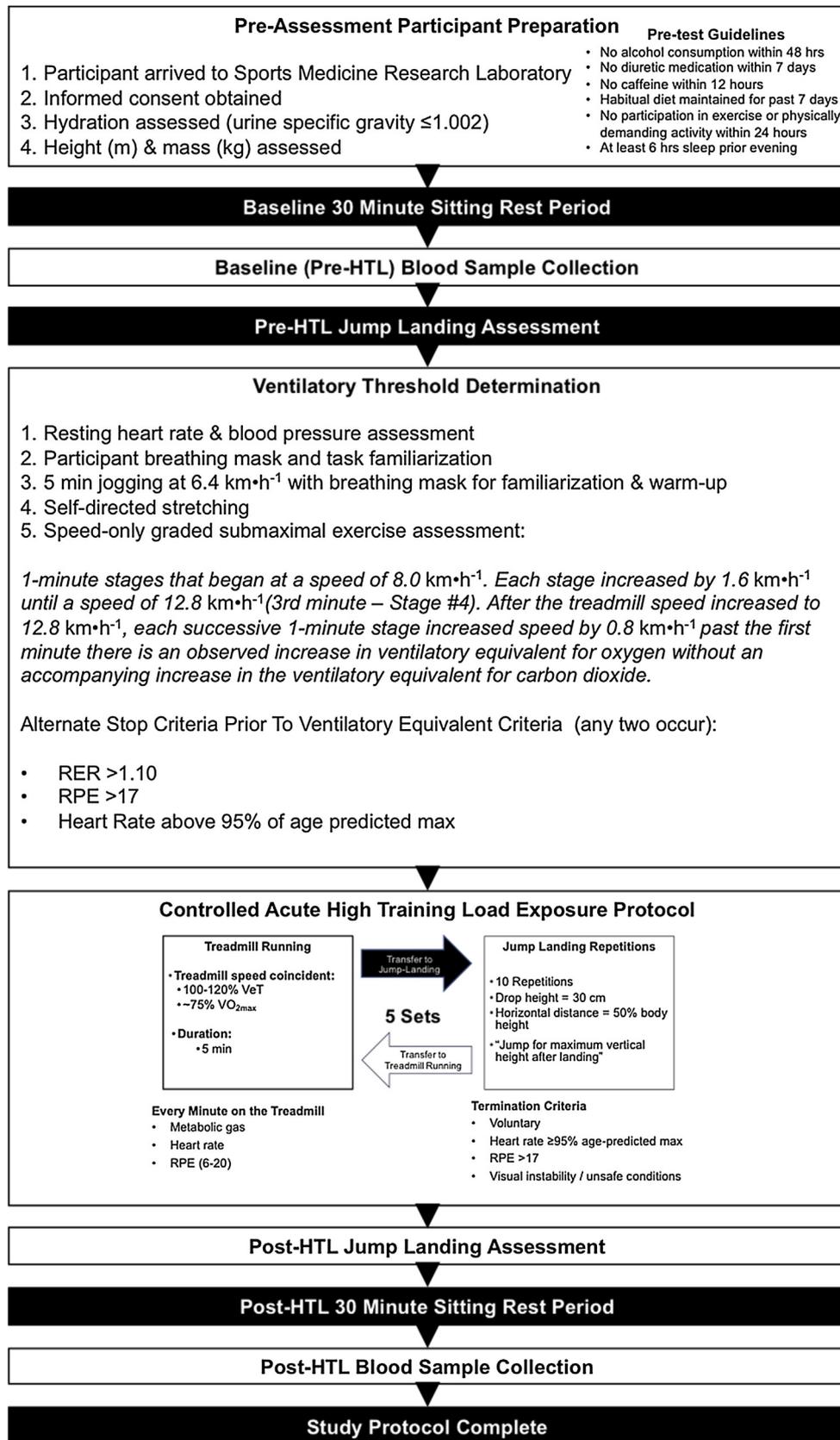


Fig. 1. Study methodology protocol.

Abbreviations: (RER) Respiratory exchange ratio, (RPE) Rate of perceived exertion

and change score vGRF ensemble means and associated 95% confidence intervals (CI) for each 0.5% of the stance phase of the jump-landing task. Pre- and Post-HTL differences in biomechanics

between the poor and excellent groups were identified as periods where there was no overlap in the 95% CI waveform between groups for a continuous period $>5\%$ of the stance phase.¹⁷ A vGRF

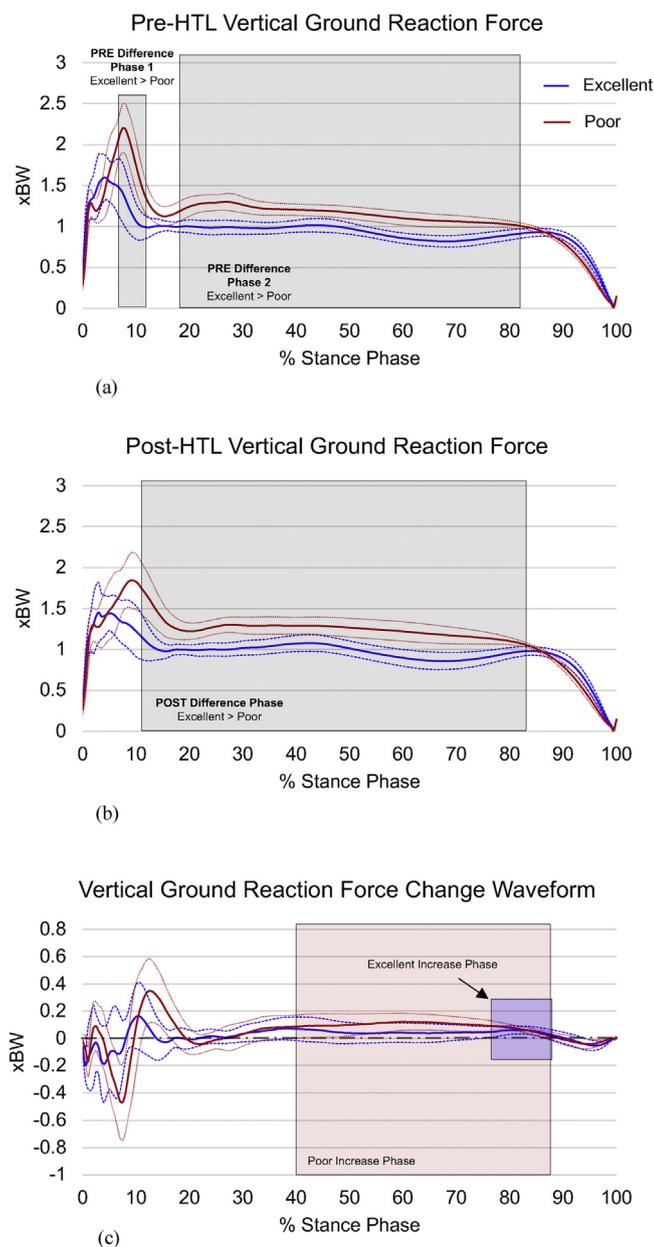


Fig. 2. Vertical ground reaction force (vGRF) ensemble curves for excellent and poor movement profiles during the stance phase of the jump-landing task. (a) Pre-HTL vGRF ensemble waveforms and associated 95% confidence intervals for excellent and poor groups during stance. (b) Post-HTL vGRF ensemble waveforms and associated 95% confidence intervals for excellent and poor groups during stance. (c) Excellent and poor group changes in vGRF ensemble waveforms and associated 95% confidence intervals during stance.

significant change following the HTL protocol was identified when a group's 95% CI waveform did not envelope zero for a continuous >5% of stance.¹⁷ Average effect sizes were calculated during phases of stance when ensemble waveforms and their associated 95% confidence intervals did not overlap to describe the magnitude of the effect of movement profile on vGRF at pre- and post-HTL exposure.

All sCORT concentrations were analyzed using SPSS statistics (Version 21 IBM, Armonk, New York, USA) and were natural log-transformed to establish normality for parametric statistical analyses using a 2×2 mixed model ANOVA to evaluate the effects of group and time on sCORT pre- and post-HTL.¹⁸ Cohen's d (d) and associated 95% confidence intervals were calculated for estimates of effect size for movement profile-by-time means and movement

profile and time marginal sCORT means.¹⁹ Effect size estimates were classified as small (0.2), medium (0.5), and large (0.8).¹⁹

Although sCORT levels were assessed for all participants, some samples presented with levels outside a physiological range, did not have viable sample pairs of pre- and post-HTL secondary to compromised sample integrity, or presented as statistical outliers >2 standard deviations outside the log-transformed group-by-time sample means.²⁰ A listwise deletion was applied such that the final number of participants with valid pre- and post-HTL values for sCORT was; nineteen ($n = 19$) individuals in the excellent and twenty-one ($n = 21$) individuals in the poor group.

3. Results

There were no significant ($p > 0.05$) differences between the movement profile groups for any demographic (excellent ($n = 22$): age = 20.5 ± 1.9 yrs, height = 1.67 ± 0.67 m, mass = 64.5 ± 7.8 kg & poor ($n = 21$): age = 20.4 ± 1.3 yrs, height = 1.69 ± 0.67 m, mass = 60.9 ± 6.1 kg) or fitness level variables (excellent: resting heart rate = 65.0 ± 9.8 bpm, resting diastolic blood pressure = 73.7 ± 9.6 mmHg, resting systolic blood pressure = 112.9 ± 6.4 mmHg, VO_2 at 100% VT = 33.2 ± 4.2 ml $kg^{-1} min^{-1}$ and poor: resting heart rate = 71.5 ± 14.5 bpm, resting diastolic blood pressure = 74.3 ± 14.2 mmHg, resting systolic blood pressure = 115.2 ± 7.4 mmHg, VO_2 at 100% VT = 34.0 ± 4.1 ml $kg^{-1} min^{-1}$).

There were no significant movement profile-by-time interaction effects ($p = 0.69$), suggesting there was no difference in sCORT response to HTL exposure between groups. There was a significant large overall effect for movement profile collapsed across time ($p < 0.05$, $d = 0.85$ [0.19, 1.48]) and a medium overall effect for time collapsed across movement profile ($p < 0.05$, $d = 0.50$, [0.05, 0.95]) on sCORT levels. Overall, the poor group exhibited significantly higher cortisol levels compared to the excellent group, and sCORT levels were significantly elevated following acute HTL exposure across both movement profile groups.

vGRF pre-HTL, post-HTL, and change ensemble waveforms and associated 95% confidence intervals are presented in Fig. 2. While both groups experienced increases in vGRF post-HTL exposure, there were no differences in the magnitude of increases between groups. vGRF magnitude was greater in the poor group compared to the excellent group pre- and post-HTL over the majority of the stance phase. During the pre-HTL condition there were large average effects from 7.0 to 12.5% ($d = 1.02$, Fig. 2a – PRE Difference Phase 1) and from 18.5 to 81.5% ($d = 1.26$, Fig. 2a – PRE Difference Phase 2) of stance for movement profile. Following the HTL protocol, a large effect for movement profile was observed, with the poor group continuing to exhibit greater vGRF from 11.5 to 82.5% ($d = 1.15$, Fig. 2b). In response to HTL exposure the poor group exhibited an increase in vGRF from 38 to 86.5% of stance ($d = 0.54$, Poor Increase Phase – Fig. 2c) whereas the excellent group experienced an increase in vGRF from 75.5 to 87% of stance ($d = 0.37$, Excellent Increase Phase – Fig. 2c).

4. Discussion

Contrary to our hypotheses, there were no differences in systemic stress and biomechanical loading responses to HTL exposure between individuals with a poor and excellent movement profile. Both movement profile groups experienced increases in sCORT levels following the acute HTL exposure ($d = 0.51$ [0.05, 0.95]). While there were no group differences in sCORT responses to the acute HTL bout, the poor group exhibited greater overall sCORT levels ($d = 0.85$ [0.19, 1.48]) compared to the excellent group across time. Our findings revealed that those with a poor movement profile

Table 1
Raw (ng ml⁻¹) serum cortisol concentration descriptive statistics.

	Pre-HTL exposure			Post-HTL exposure			Movement profile main effects		
	Mean	Standard deviation	95% Confidence interval	Mean	Standard deviation	95% Confidence interval	Mean	Standard deviation	95% Confidence interval
Excellent (n = 19)	96.62	(30.49)	[82.91, 110.32]	161.22	(144.55)	[96.23, 226.22]	128.92	(108.12)	[80.31, 177.53]
Poor (n = 21)	157.64	(65.49)	[129.63, 185.66]	252.52	(140.26)	[192.53, 312.51]	205.08 ^b	(118.30)	[154.49, 255.68]
Time main effects	128.66	(59.84)	[110.11, 147.20]	209.15 ^a	(147.87)	[163.33, 254.98]			

^a Main effect for time: Post-HTL > Pre-HTL ($p < 0.05$, $d = 0.50$ [0.05, 0.95]).

^b Main effect for movement profile: Poor > Excellent ($p < 0.05$, $d = 0.85$ [0.19, 1.48]).

exhibited greater overall vGRF compared to those with an excellent movement profile across time. During a controlled task, greater magnitude vGRF reflects greater vertical mechanical load exposure by the body,^{21,22} which may contribute to the overall mechanical load imposed upon by the body. A novel and impactful finding of this study is that an individual's movement profile appears to have a significantly strong association with circulating sCORT levels. The results of this study provide preliminary evidence for a link between biomechanical movement profile and systemic physiology in physically active females.

Movement profile significantly influences mechanical load exposure before and after HTL. A poor movement profile is associated with a greater vGRF profile both pre- and post-HTL compared to individuals with an excellent movement profile. Specifically, individuals in the poor group experienced vGRFs exceeding 1 × bodyweight for more than 4 × the duration of the excellent movement profile group's exposure (Fig. 2a & b). Furthermore, the poor group experienced an increase in vGRF for almost 50% of the stance phase (38–86.5% of stance) following HTL exposure in comparison to the excellent group who experienced an increase in late stance vGRF exposure for only ~10% of the entire stance phase (75.5–87% of stance) after HTL exposure. Thus, the poor group experienced an increase in vGRF for 40% more of the stance phase after being exposed to a HTL compared to the excellent group.

To our knowledge, this is the first study to investigate the link between movement profile and training load biomarkers associated with systemic stress response to an acute HTL. The observed increases in circulating sCORT post-HTL exposure are in agreement with previous studies that have reported acute HTL exposure to induce similar increases in sCORT in female field or court sport athletes during competition or training sessions.²³ These findings indicate that there is a significantly large difference ($d = 0.85$ [0.19, 1.48]) in systemic stress levels between physically active females with poor and excellent movement profiles, but there does not appear to be an influence of movement profile on the systemic stress response to acute HTL exposure, as both groups exhibited similar increases in sCORT (Table 1). While, there is no apparent interaction effect between movement profile and acute HTL exposure on markers of systemic stress, it is important to note the large difference in sCORT levels between the poor and excellent movement profile groups regardless of exercise exposure. The current study's findings suggest future research methodologies should deploy intervention designs aimed at improving poor movement profile characteristics to determine if there is a cause and effect link between movement profile and sCORT levels.

The results of this study are novel, comparing biomechanical loading profiles before and after an acute HTL exposure between those with excellent and poor movement profiles. Fatigue induces biomechanical changes associated with lower extremity injury; however, preceding work has focused on comparing the biomechanical response to fatiguing exercise in healthy^{12,24} or previously injured groups.²⁵ To date, the literature is conflicting regarding the

influence of fatigue and acute HTL exposure on vGRF. Interestingly, previous studies have observed increases,²⁶ decreases,¹² and no significant change²⁷ in peak vGRF during the early stance phase of landing tasks following fatigue or acute HTL protocols. Interestingly, our findings indicate that movement profile does not appear to influence the magnitude of the increase in vGRF from pre-to-post HTL exposure, but is associated with the length of time an individual is exposed to increases in vGRF early-to-mid stance during a jump-landing, with a poor movement profile being linked to a 40% greater duration increase in vGRF post-HTL.

The findings of this study implicate a movement profile linked to lower extremity musculoskeletal injury^{5,6} to be associated with an elevated marker of systemic stress response and biomechanical loading prior to and after HTL exposure in healthy college-aged female athletes. These results suggest poor biomechanics may predispose individuals to experience a greater overall systemic and mechanical stress. The current study's findings support the hypothesis that movement profile may explain some of the variability in injury patterns between individuals when exposed to HTLs. Higher vGRF mechanical load exposure may induce a greater stress hormone response which may shift the system into a greater catabolic state²⁸ with less resilience.

Cortisol is a downstream marker of hypothalamic–pituitary–adrenal axis activity responsible for maintaining homeostasis in response to stress both at rest and during exercise.^{11,28} It is possible that exposure to the same controlled metabolic stress (Appendix A of Supplementary material) resulted in a greater need for homeostatic regulation in the poor group compared to that of the excellent group, suggestive of a “mechanochemically inefficient” system.²⁹ The elevation in the poor group's sCORT may implicate a greater basal systemic stress level that may induce catabolism.³⁰ Thus, the poor movement profile may be linked to a blunted capacity to appropriately recover from repeated HTL exposure over time. Individuals with a poor movement profile may be predisposed to an elevated risk of sustaining a musculoskeletal injury during periods of HTL secondary to a decreased recovery capacity.

This study is not without limitations. The results of the current study lack generalization to other demographics within the physically active population. Our results are limited to describing the training load response profiles of healthy physically active college-aged females with a history of participation in field or court sports. While our results are not generalizable to the larger athlete and physically active population, college-aged female field and court sport athletes represent a population at high risk of non-contact, severe lower extremity injury such as ACL rupture.^{31,32}

Furthermore, we only evaluated the influence of baseline movement profile and HTL on biomechanical changes during a single sagittal plane dominant task. Changes in the landing biomechanics observed during the jump-landing cannot be immediately generalized to more complex athletic motions with greater multi-planar demands and changes in direction. However, previous literature has observed injury prevention programs aimed at increasing

sagittal plane motion at the trunk, hip, and knee to result in advantageous changes in side-step cutting tasks,³³ suggesting there is potential for biomechanical resilience transfer across tasks.

Additionally, our methodology lacked direct measures of inflammation and blood glucose or other circulating energy substrates such as lipoproteins. Thus, limiting our understanding of origin of the poor group's baseline elevation in cortisol. However, the control of our exercise protocol (Appendix A of Supplementary material) is supported by a similar cortisol response between groups, lending to the notion that while movement profile does not directly affect the stress response to HTL, it may influence the overall activity of the hypothalamic–adrenal–pituitary axis.^{11,28} Future investigations should implement intervention designs aimed at improving movement quality in individuals with inefficient poor movement profiles to determine if there is a cause-and-effect relationship between movement quality and resting systemic stress levels.

5. Conclusion

The results of the current study encourage clinicians to consider implementing corrective exercise paradigms that promote an excellent movement profile to enhance biomechanical resilience in response to HTL exposure. Greater levels of biomechanical resilience may buffer against elevated mechanical loading and lower systemic stress exposure during activities of daily life, physical activity, and sport participation. Reducing biomechanical load exposure may have implications for enhancing the recovery capacity of athletes during periods of HTL within an athletic season or training phase and prove an effective intervention route to mitigate musculoskeletal injury risk.

Practical implications

- A movement profile associated with an elevated risk of injury is associated with higher levels of systemic stress.
- Individuals with poor movement profiles experience higher biomechanical loads before and after acute HTL exposures.
- Corrective exercise programming aimed at improving poor movement patterns may enhance an athlete's biomechanical and systemic resilience, enhancing recovery during periods of HTL exposure.

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at <https://doi.org/10.1016/j.jsams.2018.05.017>.

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