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Differences in 10–20 Hz ‘shock’ attenuation emerge between injured and uninjured runners across a 30-minute run

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ABSTRACT

Running is associated with high rates of injury. One mechanism that has been proposed to contribute to running injury is greater exposure to 10–20 Hz frequencies. Typically, these frequencies are attenuated (decreased in power) as they travel up the body, however, attenuation may be compromised as runners become fatigued. Here, we report the first ever comparison of 10–20 Hz frequency attenuation in injured and uninjured runners across a fatiguing run. Thirty-one runners (16 uninjured; 15 previously injured; 14 female; 17 male; age 31.6 ± 12.6 years) ran overground for 30-minutes while accelerations were measured at the tibia, gastrocnemius, sacrum, and head using tri-axial inertial measurement units. Power spectral density was calculated at each location and transfer functions between locations were used to quantify attenuation. Across the 30-minute run, fatigue was recorded using Borg's rating of perceived exertion and percent maximum heart rate. Between-group differences in attenuation were analyzed using statistical parametric mapping and ANOVA. Injured and uninjured runners showed similar patterns of attenuation from the tibia to head but differed in how they achieved this attenuation: When compared to uninjured runners, injured runners had significantly greater 10–20 Hz attenuation with the lower body (between tibia and sacrum) and significantly less 10–20 Hz attenuation with the upper body (between the sacrum and head). Further, the magnitude of these differences increased across the duration of the run. These findings demonstrate that previously injured runners attenuate 10–20 Hz frequencies differently than uninjured participants, suggesting an altered neuromechanical control strategy.

1. Introduction

Running is associated with musculoskeletal overuse injury rates as high as 33 injuries per 1000 h (Vidbaek et al., 2015). With 54 million Americans running (Sports and Fitness Industry Association, 2017) developing approaches to prevent running-related injuries (RRIs) could have important health and economic impacts (Nielsen et al., 2014; Hespanhol Junior et al., 2016; Smits, et al., 2016). Given this motivation, a considerable amount of RRI prevention research has been conducted; however, risk factors remain difficult to identify, and RRI incidence remains high (van der Worp, et al., 2015; van der Worp et al., 2016; Hulme et al., 2017; Messier et al., 2018).

Fundamentally, overuse injuries like RRI are caused by mechanical fatigue, the accumulation of microstructural damage in response to repetitive loading (Edwards, 2018). In running, this repetitive loading originates from the millions of steps a runner takes across their lifetime

(e.g., > 58,000 running steps per week in collegiate runners; Kiernan, et al., 2018). With each step there is an exchange of energy between the foot and ground (Whittle, 1999). This exchange acts as a mechanical ‘input’ to the musculoskeletal system, applying a transient impulse that propagates upward like a ‘shockwave’ (Shorten & Winslow, 1992). The propagation of this shockwave is typically measured either as the ratio between proximal and distal acceleration peaks in the time domain or as the transfer of energy from distal to proximal locations in the frequency domain (Voloshin & Wosk, 1982; Shorten & Winslow, 1992). Both the magnitude and frequency of this shockwave are related to the force that causes it and the kinematics of the body but, when measured at the shank in rearfoot running, it typically has two distinct peaks in the frequency domain: A peak occurring in the 5–8 Hz range related to the motion of the leg during stance, and a peak occurring in the 10–20 Hz range related to the impact between the foot and the ground (Shorten & Winslow, 1992). As the shockwave propagates proximally, energy is

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dissipated and both its magnitude and frequency are reduced such that the 10–20 Hz impact peak is minimized and spectral power at the head is largely concentrated between 3–8 Hz (Hamill, Derrick, & Holt, 1995). This ~ 10–20 Hz ‘shock attenuation’ is thought to occur for two main reasons: (1) it minimizes disruptions to the visual and vestibular systems, and (2) it helps manage the loads that musculoskeletal structures are exposed to, decreasing their risk of injury (Pozzo et al., 1990; Derrick, 2004).

Shock attenuation can be achieved through both active and passive mechanisms. Passive shock attenuation is achieved through deformation of viscoelastic structures like the shoe, bones, muscles, and fat (Paul, et al., 1978; Xiang, et al., 2022). Shock attenuation may also be passively achieved by the ‘wobbling’ of soft tissue compartments (Challis & Pain, 2008). For example, when performing a karate strike, 70 % of kinetic energy in the forearm is dissipated by soft tissue deformation (Pain & Challis, 2002). It has been hypothesized, however, that if soft tissue ‘wobble’ is not completely damped between steps and the oscillations occur close to a tissue’s resonant frequency, it could increase tissue deformation and injury risk (Nigg & Wakeling, 2001). Resonant frequencies vary across structures: For a whole standing human they are ~ 12 Hz, for skeletal muscle they are typically ~ 15 Hz (but range from ~ 5–65 Hz), and for long bones they range between ~ 200–900 Hz (Nigg & Wakeling, 2001; Wakeling et al., 2002; Boyer & Nigg, 2006; Randall et al., 2010). The neuromuscular system may sense input frequencies in these ranges and, if these frequencies overlap with the resonant frequency of a limb, actively ‘tune’ muscle contraction to alter a limb’s resonant frequency (Wakeling & Nigg, 2001; Nigg et al., 2017).

Shock can also be actively attenuated through eccentric muscle contraction. Previous studies on running demonstrate that shock is reduced through increases in negative work associated with eccentric contraction at the ankle, knee, and hip (Derrick et al., 1998; Baggalety, et al., 2020). Studies using inline skating and human pendulum protocols further suggest that the neuromuscular system can actively filter out frequencies between 5–45 Hz through changes in knee flexion and joint stiffness associated with eccentric contraction (Edwards et al., 2012; Lafortune et al., 1996). Ultimately, the mechanism used to attenuate shock shifts the burden to the structures used by that mechanism: More reliance on passive attenuation means that more energy is transferred to and dissipated by bones, tendons, and ligaments; more reliance on active attenuation means that more energy is transferred to and dissipated by the muscles; and—in the absence of attenuation—more energy propagates up the body, imposing a greater burden on proximal structures. Thus, shock attenuation may provide an index of injury risk (Nigg et al., 1995).

Neuromuscular fatigue could play a role in this redistribution during running. As the neuromuscular system becomes fatigued the amount of attenuation possible by a given muscle may change, reducing the amount of attenuation in the system as a whole or shifting the mechanism by which attenuation occurs (i.e., shift from active to passive mechanisms and/or an alteration in recruitment patterns/kinematics to redistribute loading across muscles and joints). Although fatigue has demonstrated effects on many kinetic and kinematic variables in running, there have been few studies examining its effects on shock attenuation. The limited number of studies that have been conducted paint a conflicting picture of the relation between fatigue and shock attenuation: Two have found increases in attenuation (Voloshin et al., 1998; Derrick et al., 2002), one has found a decrease in attenuation (Mercer et al., 2003a), and one has found no change (Reenalda et al., 2019). Further, although shock has been suggested as a potential mechanism for RRI, to our knowledge there has been no direct investigation of the relation between attenuation and RRI (Vanwanseele et al., 2018; Hamill et al., 2018). If reduced shock attenuation does index RRI risk, then there may be differences between runners who have and have not been recently injured, and these differences may increase across a fatiguing run.

Here, we present the first experimental evidence directly

investigating whether 10–20 Hz shock attenuation is associated with RRI. We used a cross-sectional design to examine differences in shock attenuation across a 30-min fatiguing run between uninjured runners and runners that had been recently injured. We hypothesized that (a) runners would be unable to fully maintain shock attenuation throughout the entire run and thus, shock attenuation from the tibia to head would decrease over time, (b) previously injured runners would exhibit less shock attenuation between the tibia and head compared to uninjured runners, and (c) that this deficit would increase over time.

2. Methods

2.1. Participants

A priori sample size calculations in G*Power based on previous effect sizes, a power of 0.8, a correlation of 0.25, and an alpha of 0.05 indicated 15 participants per group were required (Erdfeldt et al., 1996; Mercer et al., 2003a; Guo et al., 2013). Participants were recruited from the local university, running groups, and physiotherapy clinics. Recruitment was discontinued once the threshold of 15 participants per group was met. Potential participants were included if they reported: (1) being aged ≥ 18 years, (2) running ≥ 16 km/week, (3) running with a rearfoot strike, and (4) details on any RRI that led to medical treatment. Injuries were categorized as ‘medical attention’ or ‘time-loss’ (Clarsen et al., 2013; Clarsen & Bahr, 2014). A total of 37 runners participated. Only rearfoot strikers were recruited due to findings that forefoot strikers have lower signal power in the 10–20 Hz bandwidth (Blackmore et al., 2016; Kiernan et al., 2017).

2.2. Participant characteristics

Experimental procedures were explained to participants and they provided written informed consent. Procedures complied with the Declaration of Helsinki and were Institutional Review Board approved. Participant height and weight were measured. Participants provided details on their sex, age, running history, injury history, and non-steroidal anti-inflammatory drug (NSAID) use. Participants were divided into *injured* (medical treatment for RRI 3–12 months before the study) and *uninjured* groups (no lower extremity or back injury within 12 months). Four participants were removed because they reported time-loss RRIs but had not sought medical treatment. Two were removed due to non-compliance with the protocol (one did not have a single support phase; one did not use a rearfoot strike). Ultimately, 16 participants were included in the uninjured group and 15 were included in the injured group (4 tendinopathies, 3 idiopathic pain, 2 IT band syndrome, 2 fasciitis, 1 bursitis, 1 torn meniscus, 1 chronic ankle sprain, and 1 fracture). Participants’ running exposure and performance were quantified (Honert et al., 2020; Rabello et al., 2025). Exposure was quantified as years experience, distance run per week, and number of runs per week. Performance was quantified as self-reported race time as a percentage of sex- and distance-matched world record time (<https://worldathletics.org/records/by-category/world-records>). Participants reported race distances from 0.80 to 160.93 km with 26 of 31 reporting at least one race. Using two-sample Kolmogorov-Smirnov (KS) and t-tests, uninjured and injured groups were compared across age, height, weight, experience, frequency, distance, mean performance across reported distances, and NSAID use. Results indicated that groups did not significantly differ and came from the same continuous distribution (Table 1).

2.3. Data collection

Participants were fit with a heart rate monitor (Polar H10, Polar Electro, Kempele, Finland). Inertial Measurement Units (IMUs) were attached to the anterior-medial aspect of the distal tibia, the muscle belly of the gastrocnemius, the sacrum, and the center of the forehead

Table 1
Participant characteristics across groups.

	Uninjured group	Injured group	t test (p)	KS test (p)	effect size (d)
Sample size	16	15			
Sex	8 male; 8 female	9 male; 6 female			
Age (years)	31.00 ± 11.96	31.20 ± 13.84	0.97	0.97	0.02
Height (m)	1.74 ± 0.09	1.75 ± 0.09	0.81	1.00	0.09
Weight (N)	676.67 ± 127.52	656.05 ± 92.10	0.61	0.69	0.19
Experience (years)	11.88 ± 11.37	13.13 ± 7.59	0.72	0.44	0.13
Frequency (runs/week)	5.00 ± 1.40	4.73 ± 2.23	0.69	0.77	0.14
Distance (km/week)	47.33 ± 24.26	47.44 ± 30.16	0.99	0.77	0.00
Performance (% record)	174.39 ± 24.78	155.01 ± 26.09	0.06	0.18	0.76
NSAID use/month	0.44 ± 0.51	0.57 ± 0.86	0.61	1.00	0.18

(Fig. 1A; ProMove MINI, Inertia Technology, Enschede, The Netherlands; ± 16 g range 49 μ g resolution ‘primary’ accelerometer, ± 100 g range 49 mg resolution ‘secondary’ accelerometer, ± 34.91 rad/s range 0.001 rad/s resolution gyroscope, 1000 Hz; 100 ns synchronization). The forehead IMU was placed in a custom-made water-resistant neoprene sleeve with an elastic strap with anti-slip silicone backer threaded through it (Fig. 1B). All other IMUs were attached to an elastic neoprene strap that had an anti-slip silicone inner using an adhesive-bonded hook-and-loop backer (Fig. 1C). IMU straps were tightened to the limit of participant comfort to minimize motion artefact. Participants wore their own shoes to maximize ecological validity and minimize any alterations to their gait caused by introducing novel footwear (Xiang, et al., 2022). Participants reviewed a paper copy of the 6–20 Borg scale of perceived exertion: 6 was described as “lying in bed completely at rest,” 7.5 as “slow walking,” 9 as “brisk walking,” 11 as a “light jog (the minimum we’ll be doing during testing),” and 20 as the “hardest work out you’ve ever done” (Borg, 1982; American College of Sports Medicine, 2016).

Participants left the lab and ran a self-paced 0.65 km warm up with

the experimenter biking beside them. After the warmup, all equipment was checked for security and comfort, the protocol was re-explained, and any questions were answered. Participants then ran overground for 30 min at the fastest pace they could maintain throughout the entire run. To constrain speeds within ± 10 % of each participant’s target speed, the experimenter used a cycling computer to continuously monitor participant speed and provide feedback (Edge 520 GPS Cycling Computer, Garmin, Olathe, KS; ± 3.65 m accuracy; ~ 0.15 Hz). The same running route was used for all participants and was selected to minimize changes in surface, elevation, wind, traffic, and stopping (Fig. 2). The running surface was asphalt with a small ~ 0.32 km section of dirt/gravel starting around the 1.29 km mark. At the 15-minute mark, participants turned around and ran back to the starting point. HR and IMU data were collected continuously. Perceived exertion was collected every 6 min.

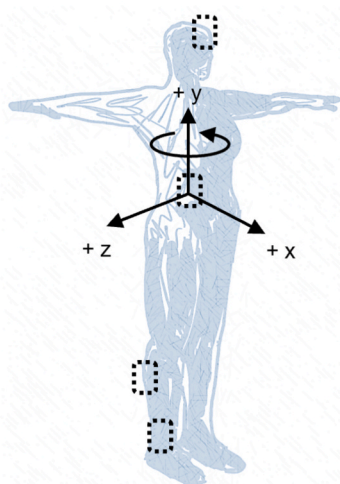
2.4. Data processing

Data were processed offline using custom MATLAB scripts (R2023a, The MathWorks, Natick, MA). Speed, elevation, and heart rate data clearly originating from sensor error were manually identified and removed (1.81 % of speed, 3.23 % of elevation, and 6.93 % of HR data). Linear interpolation replaced missing values and up-sampled from ~ 0.15 to 1 Hz. Heart rate was expressed as a percentage of age-predicted maximum (Tanaka, Monahan, & Seals, 2001):

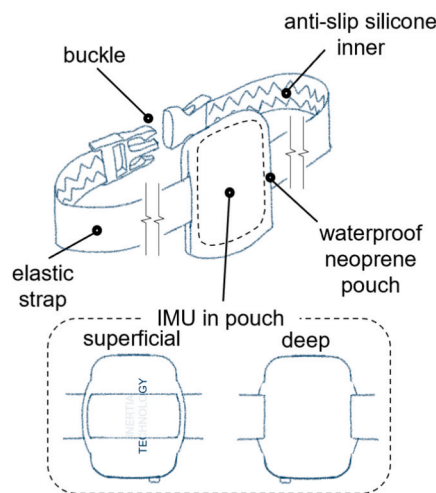
$$HR_{\%max} = \frac{HR}{208 - 0.7 \cdot age}$$

IMU data were processed to extract stance-by-stance resultant acceleration (Sup. 1; (Kiernan et al., 2023a; Kiernan et al., 2023; Kiernan et al., 2024). For each stance, the trend from the first to the last point was removed and the signal was demeaned (Hamill et al., 1995; Derrick et al., 1998; Mercer et al., 2003a; Gruber et al., 2014). The signal was zero-padded to 211 points to ensure periodicity. Power spectral density was calculated from 0 to 500 Hz in 1 Hz bins. Power was normalized such that the sum of the frequency domain was equal to the mean squared amplitude of the time domain. Finally, transfer functions describing the transmission of each frequency (in 1 Hz bins) from distal to proximal IMUs were calculated as the ratio of frequencies between locations:

1A. coordinate conventions



1B. forehead IMU attachment



1C. tibia, gastroc, and sacrum IMU attachment

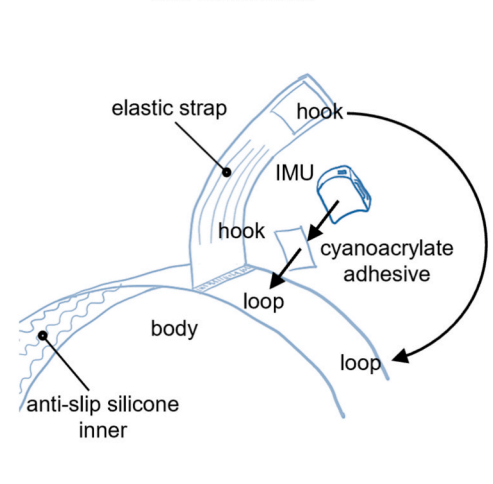


Fig. 1. (A) IMU placement and coordinate conventions. Coordinates were defined square to the IMU housing, which was roughly aligned with the direction of progression (+x), longitudinal axis (+y proximal), and right (+z). (B) Forehead IMU fixation. (C) Tibia, gastrocnemius, and sacrum IMU fixation.

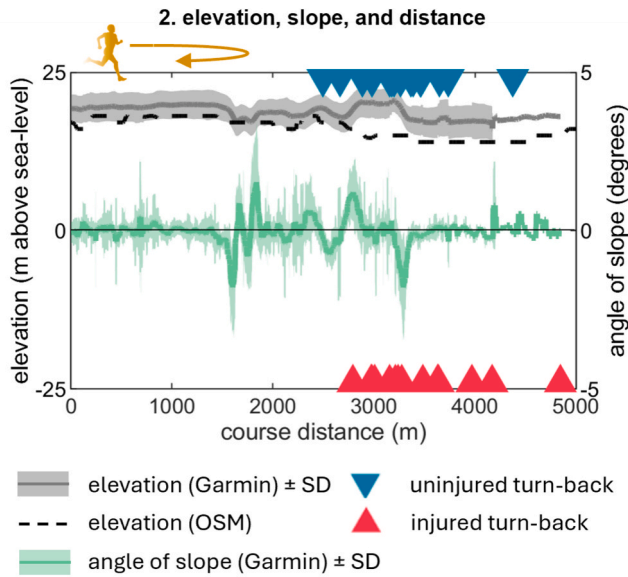


Fig. 2. Elevation data recorded with Garmin cycling computer (gray) and from OpenStreetMaps (<https://www.openstreetmap.org/#map=5/38.01/-95.84> OpenStreetMap. Retrieved 01 20, 2025; black dashed line). Angle of slope recorded with Garmin cycling computer (green). Distance run by each participant at 15-minutes when they turned back and returned to the starting point (blue triangles—uninjured participants; red triangles—injured participants). Note, Garmin elevation data for one participant were removed due to being an extreme outlier.

$$T_f = 10 \cdot \log_{10} \frac{P_{proximal,f}}{P_{distal,f}}$$

Where T_f is the transfer at frequency f , $P_{proximal,f}$ is the power of the proximal IMU (e.g., head) at frequency f , and $P_{distal,f}$ is the power of the distal IMU (e.g., tibia) at frequency f . To quantify transfer within the 10–20 Hz bin of interest, power was integrated across 10–20 Hz, then transfer between locations was calculated:

$$T_{10-20} = 10 \cdot \log_{10} \frac{\int_{f=10}^{f=20} P_{proximal,f}}{\int_{f=10}^{f=20} P_{distal,f}}$$

Thus, positive transfer indicates gain from the distal to proximal location at a specific frequency/bin (an increase in power), while negative transfer indicates attenuation from the distal to proximal location at a specific frequency/bin (a decrease in power).

2.5. Analysis

To test the hypothesis that injury status and fatigue interact to affect attenuation, a 2 (uninjured, injured) x 2 (start of run, end of run) mixed-measures ANOVA was used. To complement this analysis, Statistical Parametric Mapping (SPM) was used on the time- and frequency-series to identify if, and where, injured and uninjured runners significantly varied (Friston, Ashburner, Kiebel, Nichols, & Penny, 2007; Pataky, 2010). These tests allow continuous analysis of smooth scalar series based on the height and width of connected points in the series. The output is a statistic ‘field’ (e.g. the t-statistic as a function of time; Cao & Worsley, 1999) that identifies regions that differ across factors (Adler & Taylor, 2007). SPM analyses were conducted using the spm1D MATLAB package (M.O.4.10; Pataky, 2012; <https://spm1d.org/index.html>). Both parametric and non-parametric versions are presented to confirm test assumptions via qualitative agreement. Where appropriate, these analyses were supplemented with two sample KS- and t-tests and Spearman’s correlation. Significance for all tests was set to $p \leq 0.05$.

3. Results

3.1. Running speed

Running speeds were faster for injured (3.66 ± 0.60 m/s) than uninjured participants (3.33 ± 0.36 m/s; Fig. 3A). Although there was a medium effect size ($d = 0.67$), this difference was not significant (t -test; $p = 0.07$) and both groups came from the same continuous distribution (KS test; $D = 0.37$; $p = 0.18$). All participants maintained their speed within $\pm 10\%$ of their average speed throughout the entire 30-minute run (Fig. 3D); however, there were small but significant reductions in speed over time (uninjured: -0.06% of average speed per minute; Spearman’s correlation; $\rho = -0.47$; $p < 0.05$; injured: -0.12% average speed per minute; $\rho = -0.75$; $p < 0.05$). The rate of these reductions did not significantly differ between groups (t -test; $p = 0.09$, $d = 0.63$). SPM confirmed this finding, revealing that injured and uninjured runners did not significantly differ at any point throughout the run ($p > 0.05$; Fig. 3G). Separate analyses using absolute instead of percent speed revealed the same pattern of results.

3.2. Stride frequency

Stride frequency was similar for injured (1.42 ± 0.08 Hz) and uninjured participants (1.44 ± 0.09 Hz; Fig. 3B). There was a small effect size ($d = 0.27$), the difference was not significant (t -test; $p = 0.46$), and both groups came from the same continuous distribution (KS test; $D = 0.29$; $p = 0.45$). All participants maintained relatively consistent stride frequencies throughout the run (Fig. 3E); however, there were small but significant reductions over time (uninjured: < -0.01 Hz per minute; Spearman’s correlation; $\rho = -0.47$; $p < 0.05$; injured: < -0.01 Hz per minute; $\rho = -0.56$; $p < 0.05$). Rates of change did not significantly differ between groups (t -test; $p = 0.63$, $d = 0.18$). SPM confirmed this finding, revealing that stride frequencies did not significantly differ between groups at any time point ($p > 0.05$; Fig. 3H).

3.3. Stride length

Stride lengths were longer for injured (2.57 ± 0.35 m) than uninjured participants (2.31 ± 0.26 m; Fig. 3C). There was a large effect size ($d = 0.85$), the difference was significant (t -test; $p = 0.03$), and both groups came from the same continuous distribution (KS test; $D = 0.42$; $p = 0.10$). All participants maintained relatively consistent stride lengths throughout the run (Fig. 3F); however, there were small but significant reductions over time (uninjured: < -0.01 m per minute; Spearman’s correlation; $\rho = -0.33$; $p < 0.05$; injured: < -0.01 m per minute; $\rho = -0.65$; $p < 0.05$). Rates of change did not significantly differ between groups (t -test; $p = 0.12$, $d = 0.59$). Non-parametric SPM suggested that stride lengths significantly differed between groups at multiple points until minute 8 ($p < 0.03$); however, parametric SPM did not confirm this finding ($p > 0.05$; Fig. 3I).

3.4. Fatigue

Fatigue—as indexed by heart rate and perceived exertion—increased to near-maximal values across the run. After the first 2 min, heart rate increased significantly for both groups (uninjured: 0.14% of age-predicted max per minute; Spearman’s correlation; $\rho = 0.99$; $p < 0.05$; injured: 0.12% of age-predicted max per minute; Spearman’s correlation; $\rho = 0.99$; $p < 0.05$). The rate of increase did not differ between groups (t -test; $p = 0.19$, $d = 0.47$). By the last minute of the run, heart rate reached $98.10 \pm 6.36\%$ of age-predicted max for uninjured and $97.70 \pm 6.13\%$ for injured participants (Fig. 4A). Groups did not significantly differ during the last minute (t -test; $p = 0.86$, $d = 0.06$) or at any time point throughout the run (SPM; $p > 0.05$; Fig. 4B). Rating of perceived exertion also increased across time, starting from an average of 10.69 ± 2.02 for uninjured and 12.27 ± 2.19 for injured participants

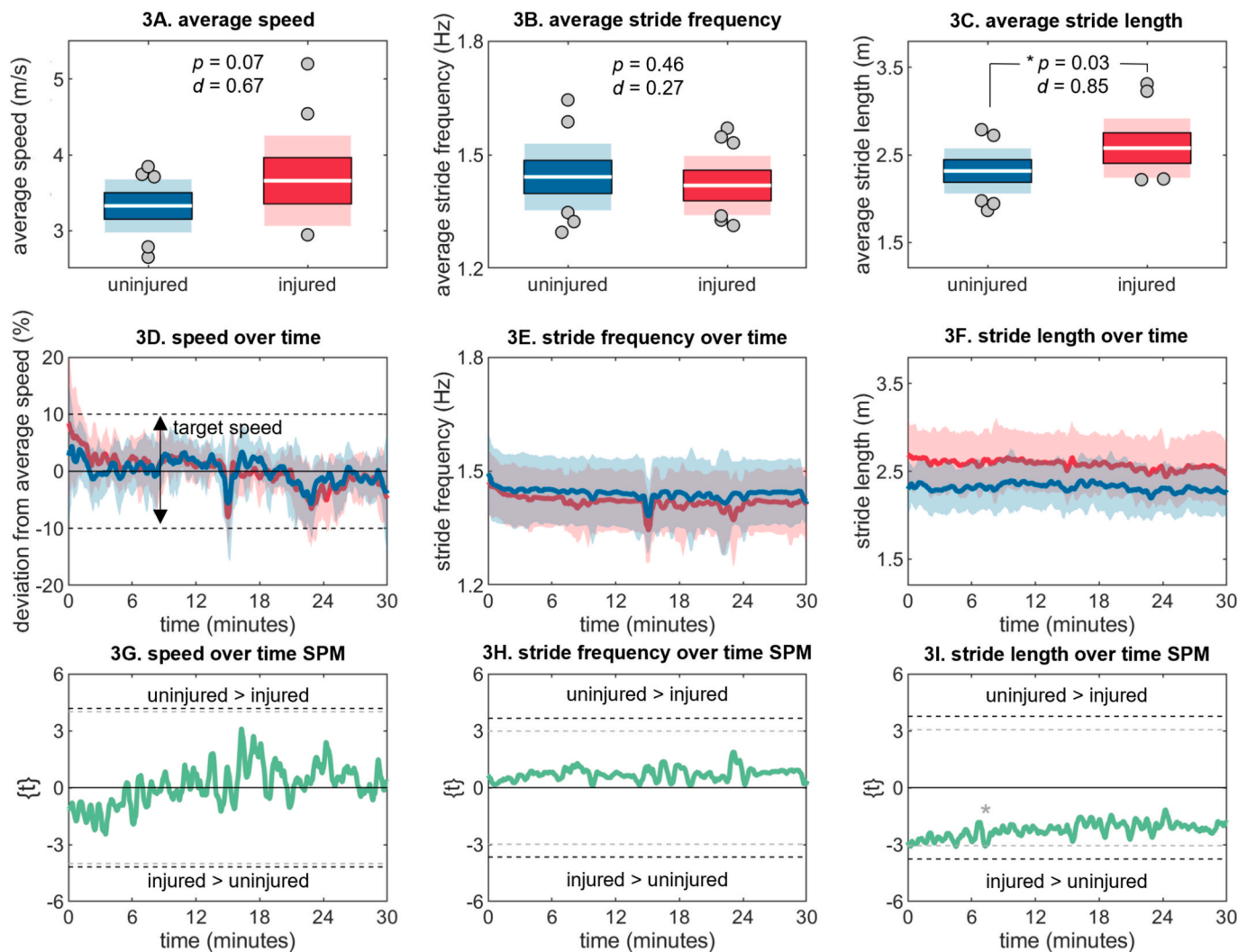


Fig. 3. **Top row** – Mean across 30-minutes for uninjured (blue) and injured (red) participants. The white horizontal line represents the mean; dark colors represent $\pm 95\%$ confidence interval (± 1.96 SEM) around the mean; light colors represent ± 1 SD around the mean; grey dots represent participants falling outside ± 1 SD. **Middle row** – 30-second moving average of uninjured (blue) and injured (red) groups across time. Dark red and blue lines represent the mean; shaded areas represent ± 1 SD around the mean. The brief decrease at 15-minutes occurs due to participants crossing the road and changing directions. **Bottom row** – SPM comparing deviations between groups. When the test statistic (green line) crosses the grey dashed lines (non-parametric) or black dashed lines (parametric), it represents a significant difference between injured and uninjured runners.

and finishing at an average of 17.94 ± 1.76 for uninjured and 18.73 ± 0.94 for injured participants (Fig. 4C). Perceived exertion did not significantly differ between groups at the end of the run (t -test; $p = 0.13$, $d = 0.84$) or at any other time point (SPM; $p > 0.05$; Fig. 4D).

3.5. Acceleration

When averaging stances across the 30-minute run, there were no significant differences between groups at any IMU location for acceleration ($p > 0.05$; Fig. 5), frequency content ($p > 0.05$; Fig. 6), or transfer function ($p > 0.05$; Fig. 7). In contrast, when examining 10–20 Hz transfer across the 30-minute run, between group differences emerged at the sacrum at later time points (Fig. 8). Non-parametric SPM suggested that tibia-sacrum 10–20 Hz transfer began to differ significantly at 27 min ($p = 0.02$) with injured runners demonstrating greater attenuation (more negative transfer). Parametric SPM qualitatively agreed but did not reach significance ($p > 0.05$). For sacrum-head transfer, both parametric and non-parametric SPM agreed that groups began to differ significantly (parametric at 27 min, $p = 0.04$; non-parametric at 15 min, $p < 0.01$) with injured runners demonstrating less attenuation (less

negative transfer).

Mixed-measures ANOVAs corroborated the SPM (Fig. 9). Significant differences were not observed in 10–20 Hz transfer from tibia-head ($F(1,29) < 0.06$, $p > 0.81$) or tibia-gastrocnemius ($F(1,29) < 0.72$, $p > 0.40$) but were observed in tibia-sacrum and sacrum-head transfer. For tibia-sacrum, there was an injury by time interaction ($F(1,29) = 6.97$, $p = 0.02$) and a main effect of injury ($F(1,29) = 4.24$, $p = 0.05$). Injured participants had greater attenuation from tibia-sacrum (more negative transfer) and this difference increased from minutes 2–7 to minutes 25–30 (1.90 dB greater at minutes 2–7 vs. 3.13 dB greater at minutes 25–30). Similarly, for sacrum-head, there was an injury by time interaction ($F(1,29) = 14.32$, $p < 0.01$) and a main effect of injury ($F(1,29) = 5.73$, $p = 0.02$). Injured participants had 1.70 dB less sacrum-head attenuation (less negative transfer) at minutes 2–7 and 3.03 dB less at minutes 25–30.

4. Discussion

Here, we present the first direct investigation of 10–20 Hz ‘shock’ attenuation in uninjured and previously injured runners across a 30-

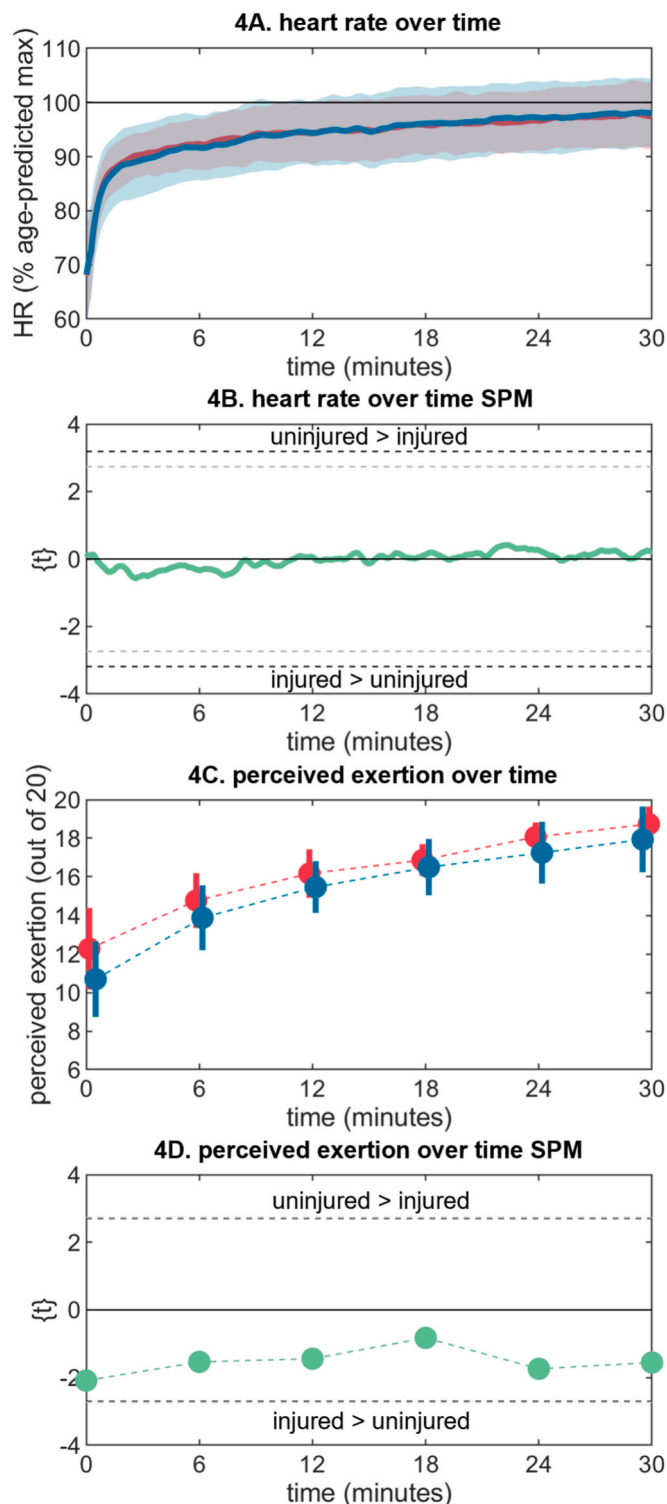


Fig. 4. A: 30-second moving average of uninjured (blue) and injured (red) participant heart rate across time, expressed as a percentage of age-predicted max. Dark red and blue lines represent the mean; shaded areas represent ± 1 SD around the mean. B: SPM comparing heart rate between groups. When the test statistic (green line) crosses the grey dashed lines (non-parametric) or black dashed lines (parametric), it represents a significant difference between injured and uninjured runners. C: Self-reported exertion across time. D: SPM comparing perceived exertion between groups. The grey dashed lines (non-parametric) and black dashed lines (parametric) nearly overlap, making them difficult to distinguish at this scale.

minute fatiguing run. We hypothesized that (a) runners would be unable to fully maintain shock attenuation throughout the entire run and therefore shock attenuation from the tibia to head would decrease over time, (b) previously injured runners would exhibit less shock attenuation between the tibia and head compared to uninjured runners, and (c) that this deficit would increase over time. These hypotheses were not supported: when comparing tibia-head attenuation there was (a) no main effect of time/fatigue, (b) no main effect of injury, and (c) no injury by time/fatigue interaction (Fig. 9C).

The observation that both groups maintained tibia-head attenuation across the run was contrary to our hypothesis that neuromuscular fatigue would compromise the ability to actively attenuate shock, allowing more 10–20 Hz content to propagate up the body. This finding is, however, consistent with previous observations that runners tightly regulate acceleration magnitudes and frequencies at the head even when distal inputs are systematically varied. For example, when tibial acceleration magnitudes and shock frequency power increase due to changes in foot strike, running speed, and stride length, acceleration magnitudes and frequencies at the head remain relatively stable (Shorten & Winslow, 1992; Hamill et al., 1995; Derrick et al., 1998; Mercer et al., 2002; Mercer et al., 2003b; Gruber et al., 2014). Although there may be exceptions to this regulation under certain constraints (Busa et al., 2016; Baggaley et al., 2020), collectively these studies suggest that minimizing energy transfer to the head is a priority of the neuromuscular system under normal running conditions (Pozzo et al., 1990).

Interestingly, although injured and uninjured participants demonstrated the same pattern of tibia-head attenuation, this attenuation was achieved differently across groups. Injured participants attenuated significantly more with their lower body (between tibia and sacrum) and significantly less with their upper body (between sacrum and head). What is more, the magnitude of these differences increased across time as participants became fatigued. Mechanistically, these differences must be caused by either a change in the input to the musculoskeletal system and/or a change in the neuromuscular system's response (e.g., a shift from active to passive shock attenuation). Factors such as increased running speed, body geometry at initial contact, and alterations in the foot-ground interface (i.e., shoes, running surface) can affect the input (as indexed by tibial acceleration). For example, Xiang et al. (2022) found that switching from maximalist to minimalist shoes increased tibial acceleration peak magnitudes and 9–20 Hz power during running. To address the possibility that systematic differences in footwear changed inputs across groups, we conducted a rough analysis using the make and model of each participant's shoes and third party-reported values for shoe weight, drop, flexion, traction, energy return, and softness (<https://runrepeat.com/>). We found that none of these parameters significantly differed between groups ($p \geq 0.12$; $d \leq 0.63$). Combined with the fact that every participant ran the same course, this analysis suggests that our findings are not attributable to the foot-ground interface.

Running speed could also influence inputs. Previous research demonstrates that tibial acceleration increases as a function of running speed. At running speeds from 3.2 to 6.4 m/s, Mercer et al. (2002) found that tibial acceleration magnitudes increased by 42 % for each 1 m/s increase in speed. Our injured participants ran faster than our uninjured participants (3.66 vs. 3.33 m/s) but, despite a medium effect size ($d = 0.67$), this difference was non-significant ($p = 0.07$; Fig. 3C). More critically, observed tibial acceleration 'inputs' were highly similar between groups in both the time and frequency domains (Fig. 5 and 6), suggesting that differences in shock attenuation are likely attributable to between-group differences in how inputs were attenuated as they propagated up the body rather than differences in the inputs themselves.

One factor established to affect attenuation is stride length (Hamill et al., 1995; Derrick et al., 1998). For example, Baggaley et al. (2020) demonstrated that longer stride lengths (a) increase tibial inputs (i.e., cause higher tibia acceleration peak magnitudes), and (b) increase attenuation between the tibia and sacrum. These observations are

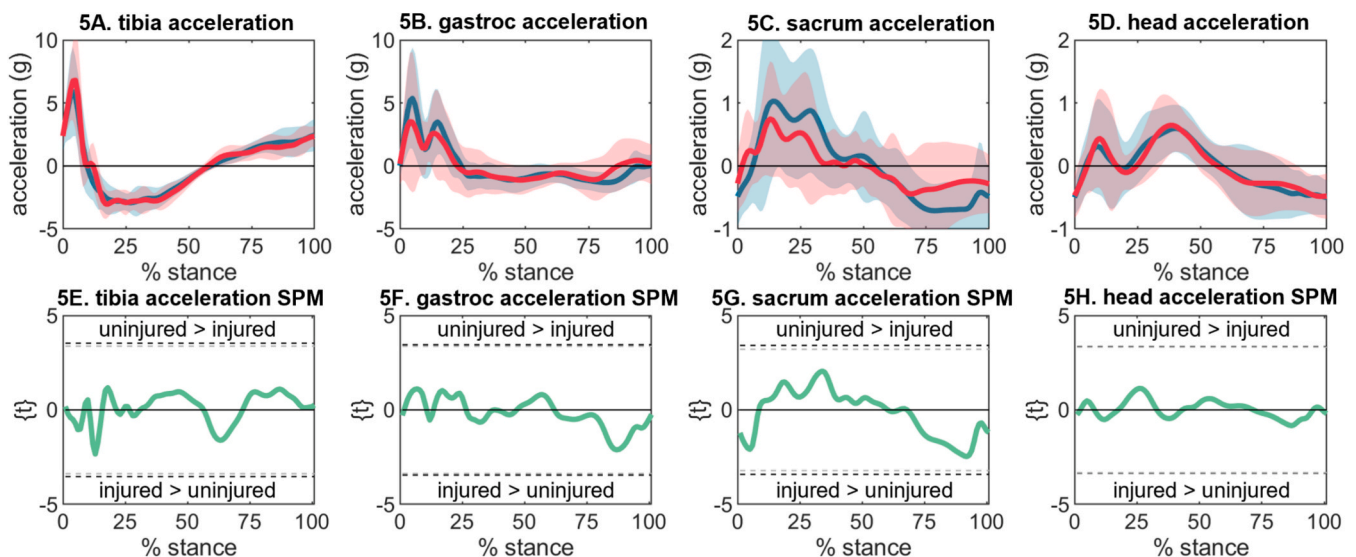


Fig. 5. Top row – uninjured (blue) and injured (red) acceleration time series measured at the tibia, gastrocnemius, sacrum, and head. Dark red and blue lines represent the mean; shaded areas represent ± 1 SD around the mean. Bottom row – SPM comparing accelerations between groups. When the test statistic (green line) crosses the grey dashed lines (non-parametric) or black dashed lines (parametric), it represents a significant difference between injured and uninjured runners. Note, parametric and non-parametric critical thresholds nearly overlap, making them difficult to distinguish at this scale.

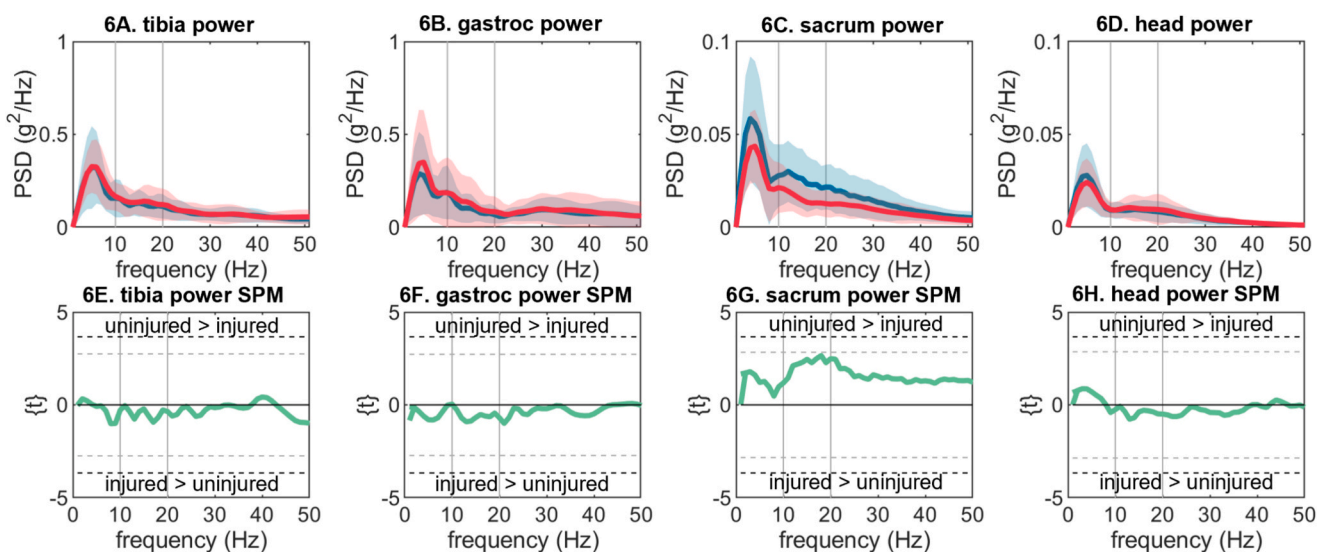


Fig. 6. Top row – uninjured (blue) and injured (red) acceleration frequencies measured at the tibia, gastrocnemius, sacrum, and head. Dark red and blue lines represent the mean; shaded areas represent ± 1 SD around the mean. Vertical grey lines denote the 10–20 Hz 'shock' frequency bin of interest. Bottom row – SPM comparing acceleration frequencies between groups. When the test statistic (green line) crosses the grey dashed lines (non-parametric) or black dashed lines (parametric), it represents a significant difference between injured and uninjured runners.

partially consistent with our findings: Injured participants had significantly longer stride lengths and greater lower limb attenuation, however, greater inputs at the tibia were not observed. The discrepancy in tibial inputs may be attributable to differences in ground reaction forces and effective mass. As the knee becomes less flexed at contact both the ground reaction force and effective mass increase, with the observed acceleration determined by the interplay of the two (Derrick, 2004). Given knee flexion tends to decrease as stride length increases (Heiderscheit et al., 2011), injured participants may have decreased knee flexion at contact, increasing both ground reaction forces and the effective mass of the lower limb, resulting in a negligible change to tibial accelerations. Despite the stability in tibial accelerations observed across groups and stride lengths, the injured group still demonstrated greater lower limb attenuation. This increase in attenuation with longer

stride lengths is consistent with previous work and has been associated with increased energy absorption at the ankle, knee, and hip suggesting that injured participants had greater eccentric muscle activity in their lower limbs (Baggaley et al., 2020).

These observations may be important for injury causation. The longer stride lengths and greater lower body attenuation observed in injured participants suggest that more energy is being absorbed/dissipated by their lower limbs, potentially exposing them to demands that could accumulate over time to exceed their capacity (Reenalda et al., 2019; Baggaley et al., 2020). These between-group differences in attenuation and stride length could be a mechanism that led to injury; however, given the cross-sectional and retrospective design of this study, it is impossible to establish whether these differences precipitated injury or were a response to injury. Indeed, injured participants may be

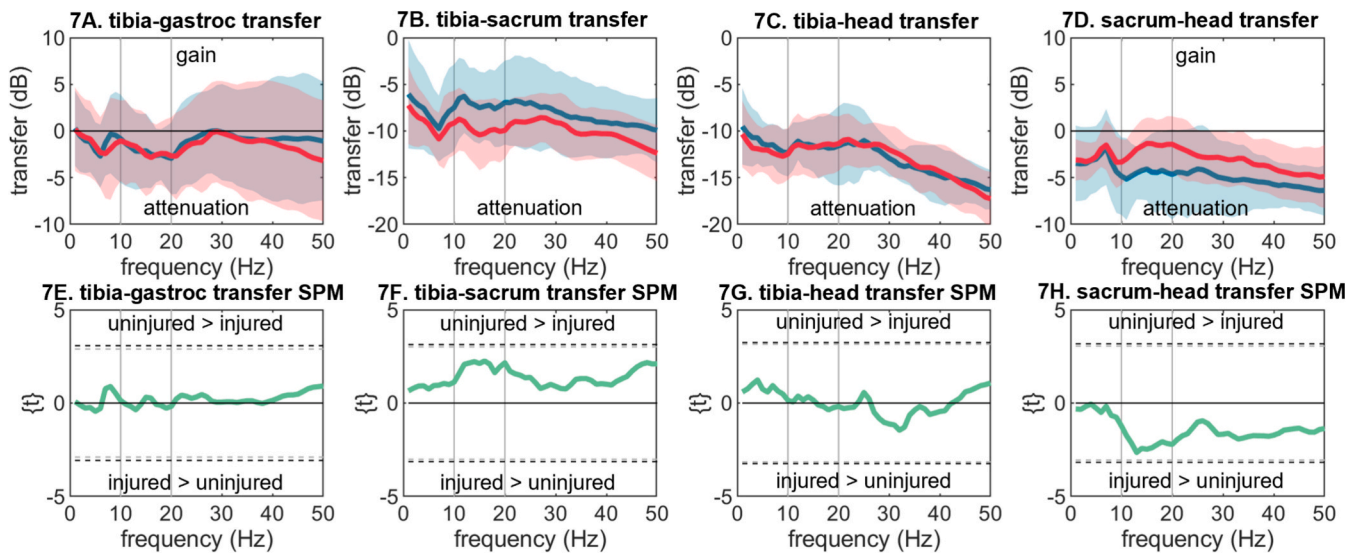


Fig. 7. **Top row** – uninjured (blue) and injured (red) transfer between each frequency. Calculated between the tibia and gastrocnemius, tibia and sacrum, tibia and head, and sacrum and head. Negative values reflect attenuation from proximal to distal while positive values reflect gain from proximal to distal. Dark red and blue lines represent the mean; shaded areas represent ± 1 SD around the mean. Vertical grey lines denote the 10–20 Hz ‘shock’ frequency bin of interest. **Bottom row** – SPM comparing transfer between groups. When the test statistic (green line) crosses the grey dashed lines (non-parametric) or black dashed lines (parametric), it represents a significant difference between injured and uninjured runners. Note, parametric and non-parametric critical thresholds nearly overlap, making them difficult to distinguish at this scale.

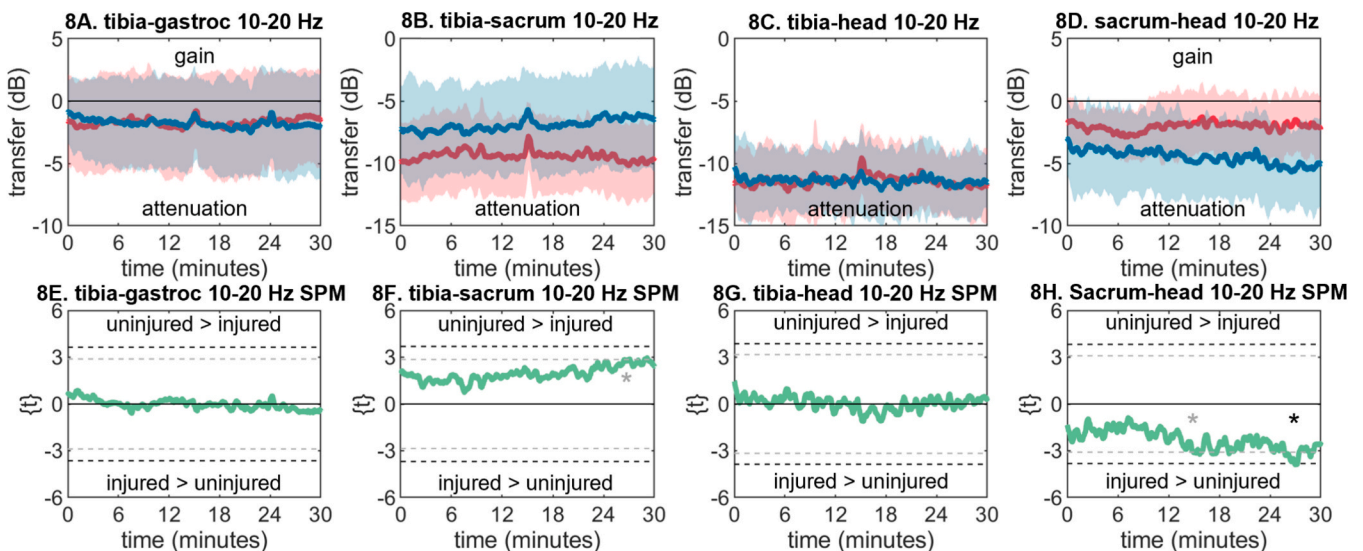


Fig. 8. **Top row** – 30 s moving average of uninjured (blue) and injured (red) transfer across the 10–20 Hz ‘shock’ frequency bin of interest. Calculated between the tibia and gastrocnemius, tibia and sacrum, tibia and head, and sacrum and head. Negative values reflect attenuation from proximal to distal while positive values reflect gain from proximal to distal. Dark red and blue lines represent the mean; shaded areas represent ± 1 SD around the mean. **Bottom row** – SPM comparing 10–20 Hz transfer across time between groups. When the test statistic (green line) crosses the grey dashed lines (non-parametric) or black dashed lines (parametric), it represents a significant difference between injured and uninjured runners; * $p < 0.05$.

responding to their injury by adopting a strategy that minimizes 10–20 Hz frequencies much earlier in the kinetic chain, protecting proximal structures. Thus, caution is warranted when interpreting these results and further research including more detailed kinematic, kinetic, and electromyographic analyses is needed to address these speculations and better explore the neuromechanics underlying observed between-group differences.

Another major limitation of this study is that a wide variety of injuries were grouped into a single ‘injured’ group. Although these were largely soft-tissue injuries, each injury likely has a different etiology and biomechanical sequelae. Despite this variability, we still found differences between groups, suggesting that monitoring 10–20 Hz attenuation

may be a robust index for injury risk/response. Further, although matching participants was a priority in this study and no differences were observed in the parameters we collected (Table 1), it is still possible that the groups represent different sub-populations of ‘runners’ (e.g., injured runners reported non-significantly faster race performances and ran non-significantly faster, suggesting they may have been higher performing and/or more fatigue-resistant; injured had a higher percentage of female participants and sex affects attenuation; Sinclair, 2016; Desai et al., 2024).

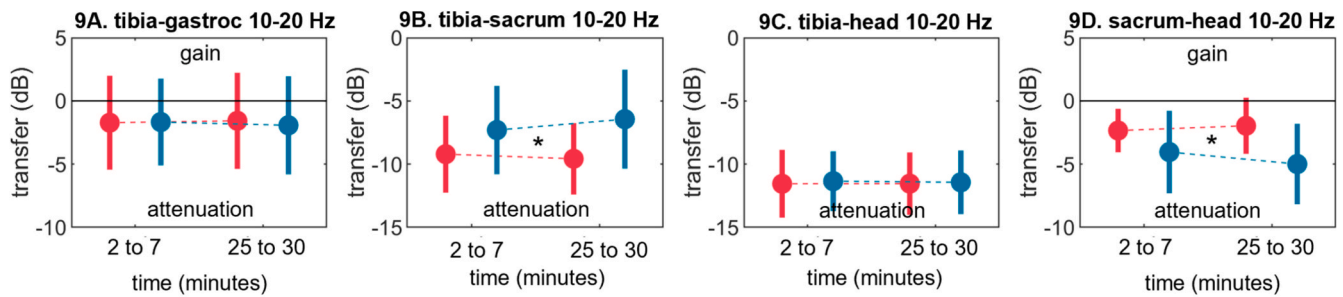


Fig. 9. Uninjured (blue) and injured (red) transfer across the 10–20 Hz ‘shock’ frequency bin of interest averaged for minutes 2–7 and minutes 25–30 of the run. Calculated between the tibia and gastrocnemius, tibia and sacrum, tibia and head, and sacrum and head. The circle represents the mean; solid lines represent ± 1 SD around the mean. * $p < 0.05$ for injury group by time interaction and main effect of injury group.

5. Conclusions

This study did not support our initial hypotheses that fatigue and injury would decrease tibia-head shock attenuation. It did, however, reveal interesting differences in how injured and uninjured runners achieve shock attenuation: Injured participants demonstrated significantly greater lower body (tibia to sacrum) attenuation and significantly less upper body (sacrum to head) attenuation than uninjured participants. This pattern was exacerbated by fatigue. These findings suggest that shock attenuation may serve as an index for injury risk or response, but additional prospective work is required to determine if these differences contribute to injury development or are simply a compensatory response. Additional work using comprehensive kinematic, kinetic, and electromyographic analyses should also be used to better characterize the neuromechanical mechanisms underlying the current findings. Finally, injured runners had significantly longer stride lengths; thus, these results join an emerging body of literature suggesting that decreased stride lengths may be protective against running injury (e.g., Edwards et al., 2009).

CRediT authorship contribution statement

Dovin Kiernan: Writing – original draft, Visualization, Validation, Software, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **David A. Hawkins:** Writing – review & editing, Supervision, Resources, Project administration, Methodology, Funding acquisition, Conceptualization. **Blaine A. Christiansen:** Writing – review & editing, Visualization, Supervision, Resources, Project administration, Formal analysis.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.jbiomech.2025.112955>.

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