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Original research

Altered spinal-level sensorimotor control related to pain and perceived instability in people with chronic ankle instability

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ABSTRACT

Objectives: To compare soleus spinal reflex excitability, presynaptic inhibition and recurrent inhibition between chronic ankle instability (CAI), acute Lateral Ankle Sprain coper (LAS-coper) and healthy populations. The relationship between spinal reflex excitability and pain and perceived instability in people with CAI was also examined.
Design: Cross-sectional laboratory experiment.
Methods: Twelve individuals with CAI, twelve 'copers' and twelve healthy age, limb and gender-matched controls participated. Soleus H-reflex recruitment curves, pre-synaptic excitability and recurrent inhibition of the spinal-reflex pathway were examined during static double- and single-leg stance. Reporting of pain and perceived instability were used to perform a regression analysis on measures of soleus spinal excitability in people with CAI, LAS-coper and healthy controls.
Results: Soleus spinal reflex excitability was greater during single-leg stance in CAI compared to healthy and coper individuals ($p = <0.001$). Pre-synaptic inhibition was three-times less in CAI participants compared to both healthy controls and copers ($p = <0.001$). There were no differences between healthy and coper participants in spinal-level measures of sensorimotor control. Reports of pain explained 15–16% of the variance in soleus spinal reflex excitability and presynaptic inhibition during single and double-leg stance, while perceived instability explained 20% of the variance in spinal reflex during single leg stance only.
Conclusions: CAI participants presented with an inability to suppress soleus spinal reflexes during tasks with increased postural threat; likely due to disinhibition of pre-synaptic mechanisms. Pain and perceived instability may contribute to changes in spinal-level sensorimotor control in CAI.

Practical implications

- Individuals with CAI demonstrate a greater reliance upon, or re-weighting of sensorimotor control toward, soleus spinal reflex responses during stance; perhaps in the absence of joint receptor information, antagonistic input and/or feed-forward anticipatory drive.
- Higher soleus spinal reflex excitability during stance was also partially explained by pain and perceived instability.
- Our findings may support the use of sensory-targeted rehabilitation strategies in individuals with CAI.

1. Introduction

After sustaining a lateral ankle sprain (LAS), up to 70% of individuals experience residual pain, feelings of instability and recurrent ankle sprain injuries.[1–3] Such characteristics are symptomatic of chronic ankle instability (CAI) – a condition associated with reduced quality of life and early onset osteoarthritis.[4] A large body of evidence has shown increased postural sway, prolonged peroneal reaction time and reduced eversion strength likely contribute to ankle sprain injuries.[5] As these factors are dependent on the ability of the central nervous system to drive and appropriately respond to a perturbation, impaired sensorimotor integration is thought to contribute to the development of CAI.[6]

Sensorimotor integration occurs through complex neural networks that combine sensory input from multiple sources to generate an appropriate motor response.[7] During functional tasks,

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the first sensorimotor response to a change in postural position is mediated at the spinal cord.[8] These spinal reflexes are the body's first opportunity to respond to a de-stabilising task, and occur at latencies consistent with the onset of LAS injuries.[9] Centre of mass displacement and postural sway size are primarily controlled gastrocnemius-soleus activity during stance.[10] Individuals with CAI show reduced maximal spinal reflex responses of the soleus (assessed using H-reflexes) in seated postures,[11,12] and no difference during stance[13,14] compared to LAS-coper (individuals who have incurred an ankle sprain, but have report no chronic symptoms) and healthy populations (no history of lateral ankle sprain). Inconsistent reports of altered soleus H-reflex responses based on postural position limits interpretation regarding the importance of this mechanism in CAI. However, recent research has shown inter-individual variance in soleus H-reflex excitability can be partially explained by self-report perceived instability and recurrent ankle sprain injury in those with CAI, LAS-coper and healthy controls.[11,15] Differences in self-report pain also explained a proportion of variance in soleus H-reflex excitability in those with an acute LAS injury and healthy controls.[16] Together, these studies suggest that altered spinal reflex excitability is associated with self-report function in CAI.

Further investigation into the mechanistic control of the spinal reflex pathway has shown that individuals with CAI are unable to modulate pre-synaptic inhibition, and have greater recurrent inhibition, of soleus spinal reflex excitability when compared to healthy controls in single and double-leg stance.[13,14] However, comparisons of presynaptic and recurrent inhibition in CAI have yet to include a LAS-coper population and it is unknown whether impaired regulation of the spinal reflex pathway, through presynaptic and recurrent inhibition, is characteristic of injury history (common to CAI and LAS-copers), or symptomatic of chronic ankle instability (CAI alone). Further, the relationship between pain, perceived instability and variance in presynaptic and recurrent inhibition of the soleus muscle has not been compared between the CAI, LAS-coper and healthy populations. If differences in presynaptic and recurrent inhibition are a distinguishing feature of CAI compared with LAS-coper and healthy controls, it is possible that self-reported function might explain individual variance. This study aimed to compare spinal reflex excitability, presynaptic and recurrent inhibition between individuals with CAI, healthy controls, and LAS-copers during single and double-leg stance. A secondary aim was to examine the relationship between spinal reflex excitability and differences in self-report measures of pain and perceived instability between groups.

2. Material and methods

Twelve individuals with CAI (4 females, 8 males; age, 25.2 ± 3.7yr; ht., 177.7 ± 8.1 cm; wt., 75.8 ± 14.8 kg), twelve 'copers' (4 females, 8 males; age, 24.2 ± 4.7 cm; ht., 172.1 ± 8.2 cm; wt., 71.4 ± 6.9 kg) and twelve healthy (4 females, 8 males; age, 23.3 ± 4.5 year; ht., 171.6 ± 6.2 cm; wt., 74.3 ± 10.2 kg) age, limb and sex-matched controls participated. All participants were recreationally physically active according to their own operational definition.

To be eligible, CAI participants needed to meet the minimum standardised inclusion criteria endorsed by the International Ankle Consortium[17] including: (i) a history of at least one significant lateral ankle sprain (at least 12 months prior to study enrolment) that caused inflammatory symptoms and disrupted activity, (ii) the most recent ankle sprain occurred >3months prior to study participation, (iii) reports of the previously injured joint "giving way" and/or recurrent sprain and/or "feelings of instability", (iv) answering "yes" to $\geq$5 questions on the Ankle Instability Instrument (AII), and (v) scoring <24 on the Cumberland Ankle Instability Tool (CAIT). Individual's in the LAS-coper group presented with a history of ankle sprain (at least one ankle sprain >6months prior to study participation) but did not report recurrent injuries, "giving way" and/or associated "feelings of instability", in accordance with previous methods.[11,12] To be considered healthy, individual's presented without a history of ankle sprain injury in either limb. Participants with a history of unilateral or bilateral ankle sprains were recruited. In the case of bilateral CAI and bilateral LAS-coper individuals, the limb with the worst perceived stability (CAIT score) was selected as the 'testing' limb. History of ankle injury is reported in Table 1.

All individuals presented without a history of neurological or orthopaedic impairment. Individuals with a history of surgery, fracture requiring realignment and/or acute injury to the musculoskeletal structures (bone, joint structure and/or nerve) in either lower limb were excluded. All research was conducted according to ethical standards outlined in the Helsinki statement and approved by the local human research ethics committee (Human Research Ethics Committee approval number: H11324).

A detailed explanation and familiarisation of the peripheral nerve stimulation procedure and postural conditions was provided prior to testing. Lighting was kept consistent throughout the trial and minimal auditory input was provided to control for potential attentional and anticipatory influences on spinal reflex excitability. Soleus H-reflex responses (mono-synaptic, spinal reflex) were elicited by percutaneous electrical stimulation of the posterior tibial nerve during two postural conditions: double-leg and single-leg stance. Participants were instructed to focus on a target with their hands resting by their sides and body-weight evenly distributed to keep the standing procedure consistent between trials. First, the soleus H-reflex recruitment curve and maximal motor response (M_{-max}) were determined during quiet, double-leg stance. A minimum of fifty-six stimuli were then elicited during double-leg stance and fifty-six stimuli during single-leg stance in each participant across three different stimulation protocols: assessment of 50% of the maximal H-reflex response ($H_{50\%}$), pre-synaptic inhibition and recurrent inhibition. A ten to fifteen second rest was provided between stimuli to avoid influences of post-activation depression.

Electromyographic (EMG) data from the soleus, tibialis anterior and peroneus longus muscles were amplified ($\times$1000), band-pass filtered (20–500 Hz) and sampled at 4 kHz using a 16-channel biological signal acquisition system (Powerlab, ADInstruments AUS). EMG signals were collected using bipolar, disposable 10 mm Ag/AgCl adhesive surface electrodes (Maxensor, Medimax Global UK). Electrode sites were prepared by shaving the area or abrading the skin prior to sanitising with isopropyl alcohol swabs. Soleus EMG electrodes were placed 2/3 of the distance between the medial condyle of the femur and medial malleolus. Tibialis and peroneal electrodes were placed 1/3 of the distance between the fibula head and medial malleolus and 1/4 of the distance between the fibula head and lateral malleolus, respectively. All electrode placements were chosen according to the surface EMG for non-invasive assessment of assessment of muscles (SENIAM) recommendations[18] and were aligned with the presumed orientation of the underlying muscle fibres. A reference electrode was placed on the patella. Activity of tibialis anterior and peroneus longus was monitored during testing to ensure electrical stimulation did not activate the common peroneal nerve and minimise confounding by reciprocal muscle activation.

A large diameter anode (10×7 cm) constructed of aluminum foil and conductance gel was fixed anteriorly and superior to the patella. The optimal cathodal stimulation site was determined by probing the popliteal fossa for the largest soleus H-reflex amplitude response at 10–15 mA. Electrical stimulation of the posterior tibial nerve was delivered using a 400 V, 1-ms square-wave pulse (Digitimer, DS7A UK).

Table 1
Ankle injury history.

Injury history	Group		
	CAI	LAS-coper	Healthy
CAIT score	16.22 ± 5.97	26.75 ± 2.22	30.00 ± 0.00
AII score	6.33 ± 1.66	1.92 ± 0.67	0.00 ± 0.00
No. of ankle sprains	5.00 ± 1.41	1.17 ± 0.39	–
Time since last sprain (years)	1.29 ± 0.89	4.23 ± 3.61	–
Frequency of giving way (last 6 months)	3.33 ± 1.97	0	0
Frequency of feelings of instability (per week)	3 ± 1.55	0	0

Data represents means ± standard deviation. AII, Ankle Instability Instrument; CAIT, Cumberland Ankle Instability Tool.

Soleus H-reflex recruitment curve and $M_{\text{-max}}$ were plotted during double-leg stance. H-threshold was determined by systematically altering the stimulus intensity (mA) until the minimum single-pulse stimulator output to elicit an H-reflex in at least one of three trials was identified. Soleus $M_{\text{-max}}$ was defined by a plateau in M-wave amplitude despite increasing stimulus intensity. A fifteen-point logarithmic scale was calculated based on stimulator output intensities for H-threshold and $M_{\text{-max}}$ responses in the soleus and used to plot the ascending portion of the H-reflex recruitment curve and $H_{50\%}$. The participant's raw H-reflex recruitment curve (ascending portion only) was fit using a sigmoidal function and general least squares model, as described in previous methodologies.[19] The predicted sigmoidal function was calculated using: $y = H_{max}/1 + e\hat{}Hslope(s50\text{-}x)$; where y is the predicted H-reflex amplitude, x is the given stimulus intensity, $H_{\text{-}max}$ is the maximal H-reflex amplitude response, H_{slope} is the gradient of the tangential slope at y, and *s50* is the estimated stimulator output 50% of $H_{\text{-max}}$. Three participants were excluded from the study and data analysis as the predicted stimulator output at $H_{50\%}$ elicited an H-reflex and concomitantly triggered an M-wave response.

Ten stimuli at $H_{50\%}$ during double-leg stance, ten stimuli at $H_{50\%}$ during single-leg stance and four $M_{\text{-max}}$ stimulations were then delivered in blocks of six stimuli in a pseudorandom order: five $H_{50\%}$, one $M_{\text{-max}}$, repeated four times. Randomisation was used to control for potential confounding due to sweat causing a shift in cathode placement.

Paired pulse stimuli of the same intensity ($H_{50\%}$) were delivered to the posterior tibial nerve, at an inter-stimulus interval of 100 ms, and counter-balanced with an unconditioned stimulus to circumvent any neural adaptation. A total of ten paired-pulse stimulations were completed in double-leg and single-leg stance conditions. The depression of the second H-reflex represents the influence of prior activation of the sensory afferent (also termed post-activation depression) and is mediated by gamma-aminobutyric acid release at the afferent terminal. Presynaptic inhibition (PSI) was calculated as the percent decrease of the conditioned soleus H-reflex amplitude (H_2) relative to that of the first, unconditioned H-reflex (H_1): $\%PSI = \{H_2/H_1\}*100$.

Intrinsic post-synaptic modulation of the soleus H-reflex was assessed using a recurrent inhibition (RI) protocol. This conditioning protocol involved eliciting a stimulus equal to 35% of soleus M_{max} (S_1) 10 ms prior to an M_{max} (S_2) stimulus. A total of twenty counterbalanced trials were obtained: ten unconditioned (test) S1 stimuli and ten S2 responses conditioned by S1. Recurrent inhibition was calculated as the percent difference between test and conditioned reflexes: $\%RI = 100 - \{(Conditioned/Test)*100\}$.

All analyses were performed using the Statistical Package for the Social Sciences (v24, SPSS Inc., Chicago, IL). Kolmogorov–Smirnov tests for normality indicated that $H{:}M_{\text{-max}\%}$, H_{slope} and single-leg $H_{50\%}$ were normally distributed. A log transformation was applied to all non-normally distributed dependant variables: double and single-leg $PSI_{\%}$, double and single-leg $RI_{\%}$ and double-leg $H_{50\%}$. A one-way Analysis Of Variance (ANOVA) was used to test for differences between groups (CAI, LAS-coper, healthy control) in dependent variables during double-leg and single-leg stance: H-reflex recordings ($H{:}M_{\text{-max}\%}$, H_{slope} and $H_{50\%}{:}M_{\text{-max}\%}$), intrinsic pre-synaptic inhibition and intrinsic recurrent inhibition. Soleus background EMG 100 ms prior to stimulation was compared between groups in single and double-leg stance. Post-hoc tests were performed using the Tukey HSD method. Significance was established at $p < 0.05$. A binary, logistic regression was used to determine the influence of pain on variables which were significantly different between groups; data were extracted from question 1 of the CAIT regarding presence of ankle pain (Pain; YES/NO). Linear regression analyses were used to determine the influence of perceived instability on variables which were significant; data were extracted from questions 2 to 7 of the CAIT regarding perceived instability. Regression analyses were performed on the combined population sample. Please see Supplementary material file 1 (Table 3) for scores assigned to each question of the CAIT used in regression analysis. Normality, homoscedasticity and independency of the errors were assessed for all regression analyses.

Table 2
Group data for measures of spinal excitability.

	Group		
	CAI	LAS-coper	Healthy
Double-leg			
$H{:}M\text{-}max_{\%}$	25.7 ± 13.7	34.0 ± 15.6	24.0 ± 11.9
H_{slope}	0.8 ± 0.4	1.2 ± 0.7	0.9 ± 0.3
$H_{50\%}{:}M\text{-}max_{\%}$	10.5 ± 3.0	15.0 ± 4.3	12.5 ± 3.6
$iPSI_{\%}$	97.9 ± 33.7†,‡	20.0 ± 8.0	29.5 ± 10.5
$iRI_{\%}$	77.2 ± 9.4	87.1 ± 5.3	81.3 ± 8.1
Single-leg			
$H_{50\%}{:}M\text{-}max_{\%}$	16.9 ± 3.5†,‡	7.8 ± 1.6	10.36 ± 3.7
$iPSI_{\%}$	83.0 ± 17.2†,‡	33.3 ± 7.7	50.82 ± 11.0
$iRI_{\%}$	72.6 ± 18.5	85.8 ± 9.6	82.86 ± 12.2

Data represents means ± 95% confidence intervals. Superscript characters represent significantly different from LAS-coper†, different from healthy‡ ($p \le 0.001$).

3. Results

Means and 95% confidence intervals are provided for each dependant variable in Table 2. Differences were observed between CAI, LAS-coper and healthy controls for measures of single-leg $H_{50\%}$, double-leg $iPSI_{\%}$ and single-leg $iPSI_{\%}$. No differences were observed for measures of $H{:}M_{\text{-max}\%}$, H_{slope}, double-leg $H_{50\%}$ or $iRI_{\%}$ in either stance condition.

No differences in $H{:}M_{\text{-max}\%}$ and H_{slope} recruitment curve parameters [$H{:}M_{\text{-max}\%}$, $F(2, 35) = 0.584$, $p = 0.565$; H_{slope}, $F(2, 35) = 1.271$, $p = 0.298$] or double-leg $H_{50\%}$ excitability [$F(2, 35) = 1.312$, $p = 0.290$] were observed between groups. However, $H_{50\%}$ excitability was greater in the single-leg stance condition in individuals with CAI compared to healthy and LAS-coper individuals [$F(2, 35) = 13.930$, $p = <0.001$]. There were no differences in bEMG between groups

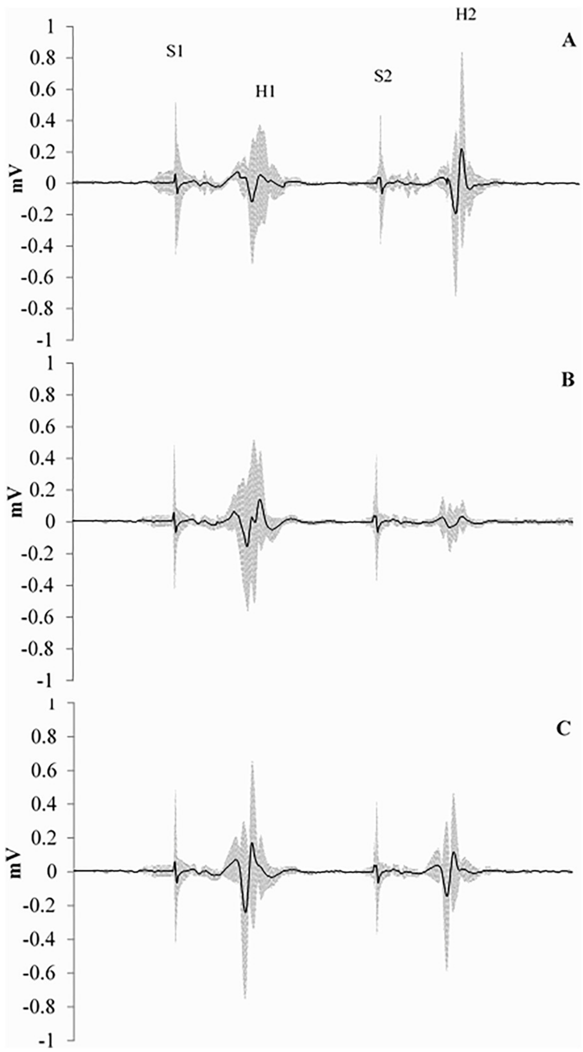

Fig. 1. Pre-synaptic inhibition in the CAI (A), LAS-coper (B) and healthy (C) groups. (B) Solid black line indicates the mean soleus H-reflex response trace in the double-leg, paired-reflex iPSI protocol. No difference observed responses between healthy and LAS-coper groups. Transparent grey shading indicates standard deviation of responses. Legend: S1, stimulation at 0 ms; H1, first H-reflex response; S2, stimulation at 100 ms; H2, second H-reflex response.

in double [bEMG, $F(2, 35) = 2.630$, $p = 0.461$] or single-leg stance [bEMG, $F(2, 35) = 1.550$, $p = 0.566$].

Relative H2 reflexes for the CAI group were 3.3 times greater during double-leg and 1.6 times greater during single-leg stance conditions, compared to LAS-coper and healthy controls [double-leg $PSI_{\%}$, $F(2,35) = 11.30$, $p = <0.001$; single-leg $PSI_{\%}$ $F(2,35) = 12.434$, $p = <0.001$]. A visual representation of mean intrinsic presynaptic inhibition observed in CAI, LAS-coper and healthy participants during double-leg stance is provided in Fig. 1.

No difference was observed in $RI_{\%}$ between groups [double-leg $RI_{\%}$ $F(2, 35) = 2.89$, $p = 0.07$; single-leg $RI_{\%}$ $F(2, 35) = 0.19$, $p = 0.19$].

Binary logistic regression indicates the presence of pain explained 18.4% of the variance in single-leg $H_{50\%}$ excitability [χ^2 $(1, 36) = 5.353$, $p = 0.021$]. The presence of pain also explained 13.6% of $PSI_{\%}$ variance in single-leg stance, and 22.6% in double-leg stance [single-leg χ^2 $(1, 36) = 3.87$, $p = 0.049$; double-leg χ^2 $(1, 36) = 6.489$, $p = 0.024$]. Differences in spinal excitability and presynaptic inhibition correctly predicted 61.1% of cases where there was no presence of pain and 82.4% of cases where pain was involved, giving an overall percentage correct prediction rate of 71.8% [Hosmer and Lemeshow test χ^2 $(1, 36) = 6.817$, $p = 0.458$]. Linear regression indicate perceived instability predicted and explained 21.5% of the variance in single-leg $H_{50\%}$ excitability [$R = 0.464$, $\beta = 0.46$, $t(1, 229) = 2.819$, $F(1, 299) = 7.948$, $p = 0.009$], and 15.7% in single-leg $PSI_{\%}$ [$R = 0.397$, $\beta = 0.40$, $t(1, 229) = 2.327$, $F(1, 299) = 7.948$, $p = 0.009$]. However, perceived instability did not explain double-leg $PSI_{\%}$.

4. Discussion

This study is the first to compare pre- and post-synaptic inhibition between CAI, LAS-coper and healthy populations. Our data demonstrate that LAS-coper individuals have similar spinal-level sensorimotor control (excitability and inhibition) to healthy individuals. In contrast, individuals with CAI exhibit disinhibition of presynaptic mechanisms. Specifically, H-reflex excitability was increased during single-leg, but not double-leg, stance when compared to healthy and LAS-coper individuals, suggesting CAI individuals utilise an alternate strategy to maintain postural control in challenging postures. Further, presynaptic inhibition was reduced by 330% in double-leg, and 160% in single-leg, stance in those with CAI compared to healthy controls and copers. Reports of pain explained 15–16% of the variance in spinal reflex excitability and presynaptic inhibition during single and double-leg stance, while perceived instability explained 20% of the variance in spinal reflex during single leg stance only.

Our data demonstrate that soleus spinal reflex excitability at 50% of the maximal spinal reflex response ($H_{50\%}$) is facilitated in individuals with CAI compared to LAS-copers and healthy controls during single-leg stance, without changes in maximal H-reflex excitability ($H:M_{max}$). Probing spinal reflex excitability using $H_{50\%}$ may therefore be more sensitiove measure to detect changes in CAI compared to the maximal $H:M_{max}$ response. Compared to LAS-coper and healthy participants, individuals with CAI also showed reduced presynaptic inhibition (disinhibition) regardless of the stance condition. By comparison, previous research has reported increased presynaptic inhibition during single-leg stance,[14] impaired modulation of pre-synaptic inhibition between single and double-leg stance,[13] or increased recurrent inhibition.[13] These discrepancies are likely explained by several variances in methodology.

First, the current study ensured that spinal reflex responses were measured on the ascending portion of the Ia-afferent recruitment curve ($H_{50\%}$). Earlier measures of pre- and post-synaptic mechanisms of spinal reflex excitability have controlled the stimulus intensity at 25–35% of the maximal direct motor, or M-max response.[13,14] Methods using 10–30% of M-max have been shown to elicit 'small M-wave' responses[20,21] and concomitant activation of sensory and motor nerve fibres; confounding measures of presynaptic inhibition with post-synaptic inhibition and anti-dromic collision. Thus, we contend that the findings of the current study likely provide a more accurate reflection of disinhibition of the Ia-afferent, spinal reflex pathway in individuals with CAI. Second, a 100 ms condition-test stimulus interval was used. While an inter-stimulus interval of 80 ms produces the greatest post-activation depression (reduced availability of neurotransmitters at the Ia-afferent terminal); intervals of 100 ms elicit responses that are sensitive to both post-activation depression and heteronymous facilitation,[22] and therefore, are arguably a more functional measure of integrative sensorimotor control via presynaptic inhibition than measures at 80 ms.

Disinhibition of the conditioned response, as observed in CAI individuals, indicates the contribution of an external excitatory mechanism, most likely through compensatory, heteronymous facilitation.[21] Considering the mechanism of ankle sprain injuries and the role of soleus during stance, compensatory soleus spinal reflex excitability may be driven by impaired feedback from damaged articular receptors, altered antagonistic activation and/or insufficient anticipatory drive. However, our findings also indicate that perceived instability, can at least in part, explain differences in soleus spinal reflex excitability during single-leg stance, whilst pain can predict excitability and (dis)inhibition regardless of stance condition. These findings align with previous research reporting pathways mediating pain and perceived instability drive disinhibition of spinal mechanisms.[22–24] Thus pain, and anxiety associated with increased postural threat, could also contribute to the dynamic re-weighting of sensory information in CAI. Sensory-targeted rehabilitation strategies (massage, stretching, joint mobilisation) reduce giving-way episodes at the ankle,[25] however the mechanisms mediating this benefit are not well-understood. It is plausible that such strategies benefit CAI individuals by reducing pain and re-orienting sensory information for the maintenance of postural control.

There are limitations of the current study. First, soleus spinal reflexes were examined during a simple postural task. It is unclear how (if at all) altered soleus spinal reflex excitability during stance relates to giving way in dynamic tasks. Further research which examines excitability during dynamic activities is needed to clarify the functional significance of altered spinal reflex control in CAI. Second, due to the retrospective design of the study, it is still unclear whether differences in soleus spinal reflex excitability manifest from a history of injury, or inherent mechanisms which cause injury. Further research may consider relationship between altered spinal excitability and influence of injury history and severity.

5. Conclusions

Disinhibition of spinal reflexes by pre-synaptic mechanisms, and during tasks with increased postural threat, are a characteristic specific to the CAI population when compared to individuals with a history of acute lateral ankle sprain (copers) and those without a history of sprain (healthy). As these changes were present in the CAI group and not copers, it is possible that altered spinal excitability contributes to an individual's inability to recover from a lateral ankle sprain injury. Pain and perceived instability explained a significant proportion of changes in spinal-level sensorimotor control. These two factors may be important outcomes in future research and rehabilitation.

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