Adaptations in equine axial movement and muscle activity occur during induced fore- and hindlimb lameness: a kinematic and electromyographic evaluation during in-hand trot.

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ABSTRACT

Background: The inter-relationship between equine thoracolumbar motion and muscle activation during normal locomotion and lameness is poorly understood.

Objectives: To compare thoracolumbar and pelvic kinematics and longissimus dorsi (longissimus) activity of trotting horses between baseline and induced forelimb (iFL) and hindlimb (iHL) lameness.

Study design: Controlled experimental cross-over study.

Methods: Three-dimensional kinematic data from the thoracolumbar vertebrae and pelvis, and bilateral surface electromyography (sEMG) data from longissimus at T14 and L1, were collected synchronously from clinically non-lame horses (n = 8) trotting overground during a baseline evaluation, and during iFL and iHL conditions (2-3/5 AAEP), induced on separate days using a lameness model (modified horseshoe). Motion asymmetry parameters, maximal thoracolumbar flexion/extension and lateral bending angles, and pelvis range of motion (ROM) were calculated from kinematic data. Normalised average rectified value (ARV) and muscle activation onset, offset and activity duration were calculated from sEMG signals. Mixed model analysis and statistical parametric mapping compared discrete and continuous variables between conditions (α=0.05).

Results: Asymmetry parameters reflected the degree of iFL and iHL. Maximal thoracolumbar flexion and pelvis pitch ROM increased significantly following iFL and iHL. During iHL, peak lateral bending increased towards the non-lame side (NLS) and decreased towards the lame side (LS). Longissimus ARV significantly increased bilaterally at T14 and L1 for iHL, but only at LS L1 for iFL. Longissimus activation was significantly delayed on the NLS and precipitated on the LS during iHL, but these clear phasic shifts were not observed in iFL.

Main limitations: Findings should be confirmed in clinical cases.

Conclusions: Distinctive, significant adaptations in thoracolumbar and pelvic motion and underlying longissimus activity occur during iFL and iHL and are detectable using combined motion capture and sEMG. For iFL, these adaptations occur primarily in a cranio-caudal direction, whereas for iHL, lateral bending and axial rotation are also involved.

1. INTRODUCTION

Lameness and back pain are common clinical issues in horses that are often interrelated; lame horses can exhibit adaptive thoracolumbar movement and horses with back pain can show clinical signs of forelimb (FL) and/or hindlimb (HL) lameness.[1] Lameness is one of the main reasons for veterinary consultation,[2] and the prevalence of back problems has been reported to be as high as 94% in ridden horses.[3] Despite this, the aetiology and clinical manifestation of equine back pain and the inter-relationship with FL and/or HL lameness, remain poorly understood, creating a diagnostic challenge.[4,5]

Quantitative equine gait analysis has been applied to measure axial motion in non-lame horses [6-9] and to quantify adaptive changes in axial motion in horses with induced lameness or back pain [10-13] during treadmill locomotion. Increased thoracolumbar range of motion (ROM) was observed in horses with induced unilateral back pain[12] and FL lameness,[10] but not during induced unilateral HL lameness.[11] These studies have advanced our understanding of adaptive axial movement associated with pain avoidance during treadmill locomotion, but clinical observations during overground locomotion indicate decreased thoracolumbar ROM during FL and/or HL lameness, which contradicts published findings.[11,12] Furthermore, the underlying neuromuscular mechanisms that ultimately facilitate these movement adaptations are poorly understood and have not been quantified during standardised lameness conditions.

Surface electromyography (sEMG) offers a solution to this shortcoming by quantifying isolated muscle activation through recordings of summated motor unit action potentials from electrodes placed on the skin over superficial muscles.[14] Zaneb et al.[15] used sEMG to quantify back muscle activity during treadmill trot and detected significantly lower amplitude ratios bilaterally from longissimus dorsi (longissimus) in a group of horses with chronic, unilateral HL lameness. They interpreted this finding as a “more distinct resting phase” between active contractions of longissimus.[15] Unfortunately, axial movement was not quantified to corroborate this interpretation and comparisons were drawn from horses with subjectively assessed and non-standardised lameness. In recognition of this, we have therefore initiated research to directly compare appendicular (St. George et al. under review) and

axial movement and muscle activity between non-lame and standardised lameness conditions during overground locomotion.

This study aimed to quantify and compare thoracolumbar and pelvic kinematics and longissimus activity in horses' thoracic and lumbar regions during overground trot in non-lame and induced forelimb (iFL) and hindlimb (iHL) lameness conditions. Based on previously reported findings and clinical observations, we hypothesised that there will be different adaptations during iFL and iHL, with the changes in ROM and longissimus activity being more localised to the thoracic and lumbar regions, respectively.

2. MATERIALS AND METHODS

Ethical approval for this study was obtained from Utrecht University (CCD: AVD108002015307) and the University of Central Lancashire (RE/17/08a_b).

2.1 Horses

Eight horses (Mean ± SD age: 9.2 ± 3.9 years, height: 161.3 ± 3.4 cm, body mass: 582.1 ± 39.4 kg, 7 mares, 1 stallion) were used. Horses were in regular ridden exercise, were accustomed to being walked and trotted in hand, and were deemed clinically non-lame (<1/5 AAEP Lameness Scale) through visual assessments by two equine veterinarians (T.S., F.S.B).

2.2 Kinematic instrumentation

Three-dimensional (3D) kinematic data were collected using an optical motion capture (OMC) system of 18 high-speed infrared cameras[a]. The OMC system was hardware synchronised to the sEMG system and recorded time series for both data types in one file for further processing. The calibrated volume for data collection was 56 m long and 10 m wide. Super-spherical, retro-reflective markers[b] (19 mm diameter) were attached over anatomical landmarks, as presented in Figure S1a. Individual markers and a marker cluster on the head were attached using double-sided adhesive tape, with an additional drop of cyanoacrylate glue used for the hoof and limb markers.

2.3 sEMG Instrumentation

sEMG data were collected bilaterally from longissimus using wireless sEMG sensors[c] with a fixed inter-electrode distance of 10 mm. Ultrasonography was used for the detection of the desired locations over longissimus, at the T14 and L1 vertebrae, 6 cm lateral to midline.[16] Once identified, each skin location was clipped of hair, then thoroughly cleaned using isopropyl alcohol. A small amount of electrolytic solution (0.9% saline) was applied to each electrode before attaching sensors to the prepared skin locations using double-sided adhesive interface strips[d], with the electrodes oriented perpendicular to the underlying muscle fibre direction.[17,18] Additional adhesion included a drop of cyanoacrylate glue on the double-sided tape, attached to the top and bottom of the sensor, above each electrode pair (Figure S1b).

2.4 Data collection

To simulate a real-world lameness examination, sEMG (2000 Hz) and 3D kinematic (200 Hz) data were synchronously collected from in-hand trot trials, conducted on a straight, hard, indoor runway during control and induced lameness (iFL, iHL) conditions. Four trials (passes down the runway) were conducted per condition. Data were initially collected from the control condition to determine the baseline gait pattern of each horse (baseline 1). Then, mild iFL (2-3/5 AAEP Lameness Scale) was temporarily induced by mechanical screw pressure applied to the sole of the hoof using a modified horseshoe.[19] Lameness induction was applied, graded, and monitored by veterinarians (T.S., F.S.B.). Horses were randomly divided into two groups (n=4) for right and (n=4) left iFL, in a cross-over design. Following iFL, trot trials were repeated. After a washout period of at least 24 hours, the same data collection process was repeated for baseline 2 and iHL conditions, where iHL was again randomised to the right (n=4) or left (n=4) HL.

2.5 Data Analysis

2.5.1 Kinematic processing and analysis

Stride segmentation was based on the detection of gait events using kinematic data as described by Roepstorff et al.[20] Upper body vertical displacement of poll, withers and pelvis were high-pass filtered (Butterworth 4th order) with the cut-off frequency adjusted to the stride frequency of each measurement.[21] Kinematic variables were calculated as previously described for upper body asymmetry[22] and for thoracolumbar and pelvic motion[6] and are described in detail in Supplementary Item 1. Briefly, the thoracolumbar angle was calculated using cranial and caudal segments, defined using markers located on the T6 and T13 vertebrae, and on the T13 vertebra and the tuber sacrale, respectively. Thoracolumbar flexion/extension angle was defined in the sagittal plane with flexion as positive and extension as negative, and lateral bending angle was defined in the transverse plane, with bending to the LS (lame side) as positive and NLS (non-lame side) as negative.[6] For the pelvic segment, pitch and yaw were defined relative to a line between the withers and tuber sacrale markers, with roll defined relative to the horizontal.[6] Pelvis pitching rotations were defined as negative during flexion and positive for extension and pelvis roll and yaw rotations were defined as downward (ventral) and forward (cranial) movements of the tuber coxae on the LS and NLS, respectively.[6]

In order to progress to further data analysis, the measured motion asymmetry differences between an individual horse's baseline and lameness induction had to exceed previously described reference values for upper body motion asymmetry of 13 mm for head movement (MinDiff Poll or MaxDiff Poll) and 5 mm for hindquarter (pelvic) motion (MinDiff Pelvis and/or MaxDiff Pelvis) and with standard deviations less than their respective means.[23]

2.5.2 sEMG data processing and analysis

Raw sEMG signals were DC-offset removed, high-pass filtered (Butterworth 4th order, 40 Hz cut-off),[24] and full-wave rectified. Discrete sEMG variables were calculated for each stride and included the average rectified value (ARV) and timings of sEMG activity onset, offset, and resultant activity duration for each muscle location.

ARV was calculated from full-wave rectified signals using stride duration as the temporal domain. As NLS and LS of longissimus were analysed separately, contralateral HL impact events were employed for stride segmentation for sEMG variables. Outliers in ARV data, defined as 2 standard deviations outside the mean ARV values within each horse, muscle location, and condition, were excluded from further analysis. To ensure that the same strides were analysed within the LS and NLS for each condition and muscle location, detected outlier strides were excluded for both muscle locations (T14 and L1) within that stride. To reduce inter-subject variability, within-horse ARV data were normalised to a reference voluntary contraction (RVC) defined as the maximum value observed for each muscle location relative to the corresponding baseline condition.[25] This permitted examination of the proportional change in muscle activity between baselines and the corresponding iFL/iHL conditions.

Muscle activity onset and offset events were calculated across strides, in accordance with the double threshold method.[26] Events were calculated from enveloped signals (Butterworth 4th order, low-pass filter, 10 Hz cut-off), with an amplitude threshold defined as 20% of the peak amplitude value of each individual sEMG signal and the timing threshold defined as 5% of the average gait cycle duration from the control condition across all horses.[26] Given the variation in baseline activity amplitude for longissimus signals and in accordance with St George et al.[26], the amplitude threshold was increased

or reduced by 5% to improve accuracy for certain horse/muscle combinations. Onset, offset, and resultant activity duration for each muscle were normalised to percentage stride duration.

To complement the discrete variables, continuous sEMG data, in the form of time and amplitude-normalised sEMG signals across all strides/conditions were prepared for analysis.[27] Within-horse, enveloped sEMG signals (Butterworth 4th order, low-pass filter, 25 Hz cut-off) were normalised to an RVC: the peak amplitude value of enveloped signals, observed for each muscle location across all strides (excluding detected outlier strides) from the corresponding baseline condition. As the RVC represents a submaximal contraction, it was possible for both normalised ARV and continuous data from the iFL/iHL conditions to exceed 100% of the RVC.

2.6 Statistical Analyses

To increase statistical power, asymmetry parameters from right iFL and iHL were multiplied by −1 to mirror the indices and thus categorise all data as if they were derived from left limb inductions only. For the remaining variables, including sEMG variables, data from right iFL and iHL, were also mirrored. Therefore, all results are reported as results of the lame side (LS) and the non-lame side (NLS). The original kinematic values, without the mirroring procedure applied, are presented in Table S1.

Linear mixed models were used to estimate the effect of lameness induction. iFL and iHL were modelled separately. Stride level data for discrete kinematic and sEMG variables were entered into the model for the baseline condition and the corresponding induced lameness conditions (baseline 1 and iFL, baseline 2 and iHL) from each horse. Models were calculated in open-source R-studio (version 3.6.3) using the package lme4 (version 1.1-15), with horse ID as a random effect and condition as fixed effect. Additionally, separate models were conducted to evaluate the impact of speed on results, using speed as a random slope to correct for this variable. Model fit was assessed using q-q plots and boxplots of the residuals. For each model, results are presented as estimated marginal means, standard error (SE) and 95% lower and upper confidence intervals calculated using the software package emmeans (version 1.7.1). Significance values were corrected for multiple comparisons using the false discovery rate method.

Statistical parametric mapping (SPM), a technique increasingly used to investigate differences in ambulatory behavior, was employed to analyse continuous kinematic and sEMG data, (i.e., complete time series of the normalised signals from one stride).[27,28,29] Time and amplitude normalised stride values for sEMG data and angle-time curves for kinematic data were assembled into 1*101*1 vector fields (median stride, 101 datapoints per stride and one dimension per data point) for each signal, condition, and horse. The open source spm1d package (version M.0.4.1) was used to conduct SPM analysis in Matlab (version 2020b).[28] For both sEMG and kinematic data, separate analyses were performed to compare signals between baseline and the corresponding iFL/iHL conditions. For group-level kinematic and sEMG data, paired samples t-tests were performed. For individual sEMG data, Hotelling's T2 tests were performed on T14 and L1 locations together, but separately for the LS and NLS. If significant results were found in a Hotelling's T2 test, paired samples t-tests were performed as post-hoc analyses. The two-tailed significance level was set at $\alpha = 0.05$ and p values were adjusted for multiple comparisons using the Bonferroni correction.

3. RESULTS

3.1 General Descriptive Findings

Thoracolumbar movement and longissimus activation patterns during trot are presented in a supplementary video (SV1), containing the moving 3D model and associated kinematic and sEMG signals from a representative horse during the baseline 1 condition. A total of 647 strides were used for kinematic analysis (163: baseline 1, 132: baseline 2, 189: iFL and 163: iHL). A total of 508 and 504 strides were employed for the separate sEMG analysis of the LS (138: baseline 1 and iFL, 116: baseline 2 and iHL) and NLS (136: baseline 1 and iFL, 116: baseline 2 and iHL), respectively. Across all horses, muscle locations and conditions, a biphasic activation pattern was observed for longissimus, with activation bursts consistently occurring between 33.1 ± 4.8% to 51.8 ± 4.7% and 84.3 ± 4.5% to 100.9 ± 4.6% of stride duration. Additional bursts or elongation of the bi-phasic pattern were observed, albeit less consistently, at 13.5 ± 4.0% to 24.6 ± 4.8% and 64.6 ± 3.9% to 75.5 ± 3.7% of stride duration. Linear mixed model results for iFL and iHL are presented in Tables 1 and 2, with sEMG activation timings presented separately in Table S2. To allow for comparison of the effect of speed on results, the following sections include data from both models, with (Table 1, S2) and without (Table 2, S2) statistical correction for speed. Unless otherwise stated, this section describes results from the speed-corrected model. Statistical correction for speed has not been applied to the continuous time-series data presented in Figures 1 – 6.

3.2 Effect of Forelimb Lameness Induction

3.2.1 Kinematic parameters

An increase in most asymmetry variables was found for iFL (Tables 1, 2, and S1), mainly Poll MinDiff (53.73 mm, $p<0.001$) and Withers MinDiff (13.14 mm, $p<0.001$). Changes in thoracolumbar motion for iFL were characterised by a significant decrease in peak flexion angle ($p<0.05$), and slight, but non-significant decreases in peak extension and peak left and right lateral bending angles (Table 1, Figures 1 and 2). Changes in pelvic motion were characterised by a significant increase in pitch ($p<0.0001$) and non-significant decreases in pelvis yaw and roll (Table 1, Figure 3). Non-speed corrected findings (Table 2) were similar except for pelvis yaw ROM, which increased significantly ($p<0.05$) without speed-correction. SPM results for kinematic data from the thoracolumbar and pelvic segments for the group of horses are presented in Figures 2 (a, b) and 3 (a– c), respectively, and showed no significant differences between conditions.

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256 *3.2.2 sEMG parameters*

257 Significant increases ($p<0.0001$) and decreases ($p<0.05$) in ARV were respectively observed at the
258 LS and NLS L1 sites during iFL, but changes in ARV at T14 locations were non-significant when
259 compared to baseline (Table 1). Activity duration of longissimus significantly increased ($p<0.0001$) at
260 the LS, T14 site, but was not significantly altered at the other locations. In general, onset/offset
261 timings were not significantly influenced by iFL (Figure 1, Table S2) and any significant alterations in
262 timings were not restricted to specific activation bursts, occurring both earlier and later across sensor
263 sites (Table S2). Contrasting sEMG results were observed between models with- and without
264 statistical correction for speed (Table 2, Table S2). For example, significant increases in ARV from the
265 T14 site on the NLS and LS were observed during iFL ($p>0.0001$) when speed was not corrected for
266 (Table S2). Significant differences in activity onset/offset timings were also observed more frequently
267 in the non-speed corrected model (Table S2).

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269 sEMG waveforms from individual horses showed significant differences between conditions when
270 analysed using SPM, as illustrated by “Horse 4” in Figure S2. SPM post-hoc analysis of LS sEMG
271 data revealed that significant differences between conditions are primarily influenced by significant
272 increases in amplitude at the L1 location (Figure S2). However, when sEMG data were grouped
273 across all horses, SPM results revealed that such differences were not significant (Figure 4).

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275 3.3 Effect of Hindlimb Lameness Induction

276 *3.3.1 Kinematic Parameters*

277 An increase in most asymmetry variables was found for iHL (Tables 1, 2 and S1), mainly pelvis
278 MinDiff (22.25mm, $p<0.001$), pelvis MaxDiff (27.87mm, $p<0.001$) and Hip Hike Swing (61.73mm,
279 $p<0.001$). Changes in thoracolumbar motion were characterised by a significantly larger peak
280 extension angle and significantly smaller peak flexion angle ($p<0.0001$) (Figure 5, Table 1). Peak
281 lateral bending angle significantly decreased ($p<0.001$) and increased ($p<0.0001$) on the LS and NLS,
282 respectively (Figure 5, Table 1). Changes in pelvic motion were characterised by a significant
283 increase in pitch and yaw ($p<0.0001$), and non-significant changes in roll ($p>0.05$) (Figures 3d– f,
284 Table 1). Results from the non-speed corrected model (Table 2), were congruent with results from the
285 speed corrected model (Table 1) except for pelvis yaw ROM, which was non-significant when speed
286 was not corrected for. SPM results showed no significant differences between conditions for
287 thoracolumbar motion (Figures 2c, d), but significant differences were observed for pelvis pitch and
288 roll during the lame diagonal stance (Figures 3d, e) ($p<0.05$).

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290 *3.3.2. sEMG Parameters*

291 Significant increases in ARV were observed bilaterally at T14 and L1 longissimus sites ($p<0.0001$)
292 (Table 1). At both T14 and L1, activation onset/offset events were generally detected significantly
293 earlier in the stride cycle on the LS, and later on the NLS ($p<0.05$) (Figures 5 and 6, Table S2). On
294 the LS, longissimus activity duration significantly increased at T14 ($p<0.0001$) and decreased at L1
295 ($p<0.0001$) (Table S2). ARV and sEMG activation timing results from the non-speed corrected model
296 (Table 2, S2), were congruent with results from the speed corrected model (Table 1, S2), except for
297 two activation events, which showed significant differences between conditions ($p<0.05$) when speed
298 was not corrected for (Table S2).

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300 sEMG waveforms from individual horses showed significant differences between conditions when
301 analysed using SPM, as illustrated in “Horse 6” (Figure S3), but when sEMG data were grouped
302 across all horses, SPM results revealed that such differences were not significant (Figure 6).

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4. DISCUSSION

This study combined motion capture and sEMG technology to quantify and compare thoracolumbar and pelvic kinematics and longissimus activity, between baseline and standardised iFL and iHL conditions. Kinematic asymmetry indices provided quantitative evidence for the successful induction of iFL and iHL across all horses, which resulted in different, significant changes in thoracolumbar and pelvic ROM, and longissimus muscle activity. iFL was characterised by significant decreases in peak thoracolumbar flexion and increases in pelvis pitching ROM (Figure 1). These adaptations were also observed during iHL, plus significant increases on LS and decreases on NLS in peak thoracolumbar lateral bending angle and increases in peak thoracolumbar extension angle and pelvis yaw ROM (Figure 5). Clear adaptations in longissimus activation patterns were observed during iHL, with significant bilateral increases in amplitude across T14 and L1 and distinct phasic shifts reflecting precipitated (LS) and delayed (NLS) muscle activation onset/offset within the stride cycle. In comparison, adaptations in longissimus activation patterns did not generally change during iFL, with no distinct phasic shifts in activation observed, but with significant changes in amplitude only observed at the L1 locations. Therefore, findings from this study support the hypothesis that iFL and iHL cause different adaptations in thoracolumbar and pelvic ROM and longissimus activity, but do not support the hypothesis that these changes are localised to the thoracic and lumbar areas during iFL and iHL, respectively.

4.1 Kinematic adaptations of thoracolumbar and pelvic motion during iFL and iHL

The overall finding that mild, reversible iFL and iHL result in different measurable adaptations in thoracolumbar and pelvic motion agrees with previous studies that reported increases in overall thoracolumbar flexion/extension ROM during iFL,[10] but no significant differences during iHL.[11] In contrast, horses in this study adapted to iFL by significantly decreasing peak thoracolumbar flexion during LS stance phase (Figures 1 and 2a), without significantly altering peak extension or lateral bending angles, and to iHL by significantly decreasing peak thoracolumbar flexion and increasing extension (Figures 2c and 5). Comparisons between studies are limited by methodological differences in data processing and analysis and the fact that horses were evaluated during treadmill locomotion, in which thoracolumbar motion differs from overground locomotion.[10,11,31,32] However, our findings are congruent with clinical observations of increased stiffness/decreased flexibility of the thoracolumbar region in horses presenting FL and HL lameness. Further, our findings for iHL (Figure 2c) agree with a descriptive study that reported decreased extension during LS stance and increased extension

during NLS stance in a single clinical hindlimb lameness case (right tarsal osteoarthritis) compared to a non-lame horse during overground trot.[30]

Gómez-Álvarez et al.[10] related compensatory “head nod” during iFL and its concurrent effects on thoracic flexion/extension to significant increases in extension angles of individual thoracic and lumbar vertebrae during lame diagonal stance. Indeed, an examination of group-averaged iFL time-angle curves in Figure 2a reveals a general, albeit non-significant, trend for increased extension and decreased flexion during lame diagonal stance. Thus, asymmetrical head and neck movement during iFL appears to affect the subtle, but largely non-significant, asymmetries observed in group-averaged thoracolumbar flexion/extension. Discrete data revealed that peak thoracolumbar flexion was significantly decreased during iFL and based on Figure 2a, this was attributed to the flexion peak bridging at the end of lame and non-lame diagonal stance phases (Figure 1). Significant increases in thoracic flexion, as observed by Gómez-Álvarez et al.[10] during non-lame diagonal stance, were not found in this study for group-averaged data, although individual kinematic data reveals that certain horses exhibited this movement pattern, particularly the two horses with the highest MinDiff Poll values (i.e., the highest degree of iFL) (Table S1). Significant increases in T10 and T13 lateral bending angles towards the LS during lame diagonal stance have been observed and interpreted as an attempt to shift the centre of mass towards the NLS.[10] Again, group-averaged lateral bending data from our study does not support this finding, but individual horses exhibited increased lateral bending towards the LS. Thus, in accordance with known inter-horse variance in back motion during non-lame locomotion[6,8,33], findings from this study suggest that individual horses adopt different adaptation strategies, most notably during iFL.

4.2 Electromyographic activity of the longissimus and adaptations during iFL and iHL

Longissimus is the largest equine epaxial muscle. Based on its anatomical location and attachments, it is thought to extend the spine when activated bilaterally in a concentric contraction, whereas unilateral concentric activation results in lateral bending and/or axial rotation.[16] Here, longissimus had a bilateral, biphasic activation pattern in each stride cycle, with each burst corresponding to the second half of HL stance, where thoracolumbar flexion occurs (Figures 1 and 5). This biphasic pattern is well-documented in sEMG studies of quadrupedal trot on a treadmill[16, 34-37], with longissimus

function generally attributed to eccentric activity that stabilises the thoracolumbar spine during passive flexion.[36-41] Across these studies, there are both inter-individual variation in activation timing[36,39,42] and variations in the number of activation bursts.[40,42,43] Our findings support inter-individual variation of longissimus activation, with some horses showing additional activation events in the first half of HL stance, producing additional bursts or elongation of the bi-phasic pattern. Von Scheven[43] explicitly described these additional bursts of longissimus activity in some horses during treadmill trot and, in the current study, they preceded peak thoracolumbar extension at approximately HL mid-stance (Figures 1 and 5). This is the first known study to acquire sEMG data from longissimus during overground quadrupedal trot on a hard surface, which is an important consideration given the known effect of both treadmill and surface type on locomotion, loading patterns, and workload.[31,32,44] Indeed, loading experiments to alter locomotor forces acting on the trunk and hindlimbs of dogs, have noted adaptations in longissimus activation.[40,41] Therefore, overground locomotion on a hard-surfaced runway, as studied here, may yield different longissimus activation patterns. However, further research comparing muscle function during overground vs. treadmill locomotion and examination of antagonist muscles (e.g., rectus abdominus) is required to confirm this.

Bilateral, significant increases in ARV observed at T14 and L1 during iHL support the theory posed by Barrey et al.[45] that bilateral adaptations in longissimus activity represent a stabilising function against compensatory sagittal plane forces during iHL, namely reduced vertical acceleration and displacement of the centre of mass during LS stance and vice versa during NLS stance.[13] Supporting this, observational analysis of Figures 5 and 6 depicts increases in sEMG amplitude during iHL that are most pronounced in longissimus activation bursts during the first half of HL stance, where significant adaptations in thoracolumbar extension occurred, albeit to varying degrees between horses, likely because of documented adaptations in vertical forces acting on the trunk.[13] These findings contrast with a study[15] reporting significantly lower bilateral longissimus amplitude in horses with chronic, unilateral HL lameness, which was interpreted as a “more distinct resting phase” between muscular activation bursts. Contrasting differences in longissimus activity could be related to chronicity of existing HL lameness compared to the acute, induced lameness evaluated in our study, but further comparative research is required to confirm this.[15] Interestingly, non-significant changes in sEMG amplitude were also reported by Fischer et al.[46] for the LS and NLS of longissimus activity at

L3/L4 sites in dogs with unilateral iHL during treadmill trot. Again, methodological differences make direct comparisons between studies difficult, particularly in relation to the type of locomotion (treadmill vs. overground), sEMG processing and analysis methods,[15,47] and lameness studied (acute/induced vs. chronic cases).[15]

Longissimus activation is affected by vertical and horizontal components of HL pro-retractor muscles.[41] Temporal adaptations in HL pro-retraction have been described during iHL[47], and in accordance with these changes, significantly delayed NLS longissimus activation timings were observed in our study and in Fischer et al.,[46] who also reported a non-significant trend for earlier activation on the LS, which was largely significant in our study. Trunk rotation towards the NLS has been described during iHL[11,13] as a means to unload the LS HL.[13] Significant changes in discrete lateral bending angles and continuous pelvic ROM data (Figures 3d, e), indicate that this compensatory mechanism was also observed in the current study. Lateral bending toward the NLS and pelvis roll and yaw rotations towards the LS were also found in this study, with significant differences in the SPM results for pelvic roll during LS stance (Figure 3d). It has been suggested that compensatory longitudinal rotations of the back and pelvis during iHL are driven by increased activity of NLS epaxial, as well as HL protractor muscles.[46] The significant increases in NLS longissimus amplitude observed in this study, as well as NLS superficial gluteal, biceps femoris and semitendinosus observed in St. George et al. (under review) support the realisation of increased lateral bending of the back towards the NLS and of the pelvis towards the LS. Taken together, these findings are the first to support postulated muscular adaptations for known compensatory weightbearing and movement patterns of the limbs, back, and pelvis during hindlimb lameness.

To our knowledge, this is the first study to examine equine muscle function during forelimb lameness. In contrast to iHL, longissimus amplitude and activation patterns at the sites evaluated remained largely unaltered during iFL, except for recordings at the LS L1 site, which significantly increased in amplitude (Figure 1). This finding appears to support the suggestion by Gómez-Álvarez et al.[10]: that increased activation of longissimus lumborum occurs during lame diagonal stance to produce lateral bending towards the LS in an attempt to shift the centre of mass towards the NLS in the horizontal plane.[48] It is possible that the pronounced increases in LS L1 activity reflect an active contraction to

aid lateral bending towards the LS. However, inter-individual differences in L1 activation were apparent in this study, further supporting the finding that horses adapt to iFL using individual compensatory movement patterns. Interestingly, significant changes were not observed in the thoracic recording sites, which were hypothesised to exhibit the greatest change during iFL, due to their closer proximity to the well-described compensatory "head-nod".[13] It is possible that the "head nod" produces subtle changes in thoracolumbar flexion/extension, but not enough to necessitate increased muscle activation of the longissimus at this region.

4.3 Clinical relevance and further considerations

The lameness induction model was considered ideal for this preliminary research, as it produces a highly reliable and standardised condition for study, but indeed lameness encountered clinically is variable and often chronic in nature. Further, inter-individual variation in the dataset from a small sample could be considered a limiting factor, but we argue that this finding reflects challenges within the clinical world, as well as previous research.[6,8,33] Prior to this study, only clinical perceptions about adaptations in epaxial muscle activation during equine lameness existed. Although findings from this study offer the first objective data on underlying muscular adaptations in the equine back during lameness, clinical extrapolation of preliminary data is challenging. Thus, further studies employing a larger sample of clinical lameness cases are required. Nevertheless, it is clear from our results that adaptation mechanisms to lameness are complex and single limb lameness can affect kinematic and muscle activation of the back in an acute lameness model.[49]

The known effect of speed on kinematic[36] and sEMG variables[50], was addressed in this study by presenting results from models with- and without a statistical correction for speed. This is especially relevant, as significant changes in stride velocity during equine lameness are known.[47] Therefore, it is only the adaptations in speed-corrected variables (Table 1, S2) that can be considered clinically relevant, as they are not confounded by the effects of speed and are thus the result of induced lameness. Finally, fewer group-averaged variables were found to be significantly altered during lameness conditions when analysed using SPM compared to linear mixed models. This discrepancy between the analysis of discrete and time series variables agrees with previous studies of equine biomechanics data.[26,27,29] As alluded to in previous studies, this is likely because alpha is more tightly

controlled when using SPM and the known variation in equine biomechanics data affects the level of significance using SPM.[27,29] Based on this, Smit et al.[27] and Hobbs et al.[29] suggest that reaching significance may not be as important when using SPM to evaluate clinical implications and that data from individual horses should be assessed to ensure that subtle changes are not overlooked when considering group-level data. Our findings agree with this, as the clusters of data points that reached significance following SPM post-hoc analysis of within-horse sEMG data (Figures S2 and S3), were often in accordance with the significant increases in discrete ARV and activation onset/offset variables (Table 1 and 2), suggesting that time series data from individual horses should be evaluated when clinically assessing the effects of equine lameness.

5. CONCLUSION

Distinctive differences in thoracolumbar and pelvic motion and underlying longissimus activity occur during iFL and iHL and have been measured here for the first time using combined motion capture and sEMG. iFL was characterised by significant decreases in peak thoracolumbar flexion angle, significant increases in pelvis pitching ROM, and significant changes in sEMG amplitude at L1 sites. In contrast, iHL was characterised by several significant adaptations including increases in thoracolumbar lateral bending towards the NLS and decreases towards the LS, decreased peak thoracolumbar flexion and increased peak extension angles, and increased pelvis yaw and pitching ROM. These kinematic changes during iHL occurred alongside significant bilateral increases in longissimus activity and clear phasic shifts in activation timings. These findings suggest that, during iFL, thoracolumbar and pelvic movement adaptations occur primarily in the cranio-caudal direction, but this seemingly does not necessitate significant adaptations in longissimus activation at the thoracic regions studied here. Instead, significant changes in longissimus activation at the lumbar regions were observed during iFL, but this was largely horse-specific and may reflect another compensatory mechanism of increasing LS lateral bending to horizontally shift the centre of mass away from the affected limb. Whereas findings suggest that compensation for iHL primarily involves lateral bending and axial rotation to shift the centre of mass horizontally, and that these adaptations are facilitated by significant phasic shifts and increases in longissimus activation at both of the thoracic and lumbar regions studied here. The subtle and often horse-specific nature of these adaptations drives home the importance of future research to determine whether the significant changes observed here constitute clinically meaningful changes and to develop further objective clinical evaluation techniques for the equine back. These future studies are particularly important because many of the kinematic adaptations, and certainly the underlying neuromuscular adaptations, to lameness, as observed here, are undetectable through human observation alone.

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Table 1. Speed corrected data as estimated marginal means (EM Mean) and standard error (SE) for discrete variables from baseline and iFL and iHL lameness conditions and estimated mean marginal differences (EM Mean Difference, EM Mean % Difference) between corresponding baseline and induced lameness conditions and associated p-values.

Variable	Induction	Baseline		Induction		EM Mean Difference	EM Mean % Difference	p-value
		EM Mean	SE	EM Mean	SE			
Stride Duration (s)	FL	0.77	0.01	0.75	0.01	-0.01	1.30	**<0.001**
	HL	0.73	0.01	0.71	0.01	-0.02	2.74	**<0.001**
Asymmetry Variables (mm)								
MinDiff Poll	FL	-3.36	5.30	-57.09	5.22	-53.73	n/a	**<0.001**
	HL	-5.72	5.17	-13.85	5.10	-8.13	n/a	**<0.001**
MaxDiff Poll	FL	-7.18	5.38	-29.47	5.72	-22.29	n/a	**<0.001**
	HL	-2.87	3.04	-11.95	2.92	-9.08	n/a	**<0.001**
MinDiff Withers	FL	-2.36	1.64	-15.51	1.75	-13.14	n/a	**<0.001**
	HL	-2.07	1.70	10.96	1.72	13.04	n/a	**<0.001**
MinDiff Pelvis	FL	1.03	1.14	3.25	1.29	2.22	n/a	**<0.001**
	HL	0.34	2.68	-21.91	2.67	-22.25	n/a	**<0.001**
MaxDiff Pelvis	FL	0.68	3.27	6.29	3.28	5.61	n/a	**<0.001**
	HL	4.78	1.37	-23.08	1.39	-27.87	n/a	**<0.001**
Hip Hike Swing	FL	0.81	4.22	13.98	4.23	13.17	n/a	**<0.001**
	HL	2.89	6.31	-58.84	6.33	-61.73	n/a	**<0.001**
Maximum Thoracolumbar Angle (degrees)								
Left/ LS Lateral Bending	FL	4.41	0.71	4.39	0.73	-0.02	0.45	0.9
	HL	3.54	1.00	2.91	1.00	-0.63	17.80	**<0.001**
Right/ NLS Lateral Bending	FL	-2.14	0.74	-2.04	0.77	0.11	5.14	0.7
	HL	-3.37	1.00	-4.26	1.00	-0.89	26.41	**<0.001**
Extension	FL	-23.75	1.13	-23.67	1.08	0.07	0.29	0.6
	HL	-21.03	1.10	-21.64	1.11	-0.61	2.90	**<0.001**
Flexion	FL	-16.36	0.85	-16.56	0.85	-0.20	1.22	**0.03**
	HL	-15.97	0.91	-16.34	0.91	-0.36	2.25	**<0.001**
Pelvic ROM (degrees)								
Pitch	FL	7.88	0.57	8.39	0.57	0.50	6.35	**<0.001**
	HL	8.49	0.66	9.27	0.66	0.77	9.07	**<0.001**
Roll	FL	7.53	0.78	7.40	0.77	-0.13	1.73	0.4
	HL	7.26	0.68	7.10	0.67	-0.16	2.20	0.4
Yaw	FL	3.20	0.33	3.10	0.33	-0.10	3.12	0.07
	HL	3.32	0.21	3.45	0.21	0.12	3.61	**<0.001**
Longissimus sEMG ARV (%)								
T14, NLS	FL	97.33	4.53	93.89	4.92	-3.44	3.5	0.09
	HL	82.29	6.83	108.23	6.78	25.94	31.52	**<0.001**
T14, LS	FL	91.19	1.78	88.78	1.96	-2.41	2.6	0.1
	HL	79.95	5.43	117.52	5.25	37.58	47	**<0.001**
L1, NLS	FL	93.05	1.53	89.35	1.76	-3.70	4.0	**0.03**

	HL	89.99	5.53	111.17	5.59	21.18	23.54	**<0.001**
L1, LS	FL	116.57	22.05	193.35	24.75	76.78	65.9	**<0.001**
	HL	84.73	4.35	97.23	4.31	12.50	14.75	**<0.001**

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509 Table 2. Non-speed corrected data as estimated marginal means (EM Mean) and standard error (SE)
510 for discrete variables from baseline and iFL and iHL lameness conditions and estimated mean
511 marginal differences (EM Mean Difference, EM Mean % Difference) between corresponding baseline
512 and induced lameness conditions and associated p-values.

Variable	Induction	Baseline		Induction		EM Mean Difference	EM Mean % Difference	p-value
		EM Mean	SE	EM Mean	SE			
Stride Speed (m/s)	FL	3.13	0.10	2.87	0.10	-0.26	8.31	**<0.001**
	HL	3.09	0.12	3.03	0.12	-0.06	1.94	**0.02**
Stride Duration (s)	FL	0.74	0.02	0.78	0.02	0.04	5.41	**<0.001**
	HL	0.75	0.01	0.74	0.01	-0.01	1.33	**<0.001**
Asymmetry Variables (mm)								
MinDiff Poll	FL	-3.99	4.42	-57.35	4.41	-53.36	n/a	**<0.001**
	HL	-3.04	3.19	-14.28	3.09	-11.25	n/a	**<0.001**
MaxDiff Poll	FL	-4.62	6.26	-24.27	6.25	-19.65	n/a	**<0.001**
	HL	-3.40	2.14	-13.07	2.04	-9.68	n/a	**<0.001**
MinDiff Withers	FL	-1.97	2.06	-14.13	2.06	-12.16	n/a	**<0.001**
	HL	-2.61	1.75	11.23	1.73	13.84	n/a	**<0.001**
MinDiff Pelvis	FL	-0.65	1.75	0.79	1.75	1.44	n/a	**0.05**
	HL	1.40	2.13	-21.24	2.11	-22.64	n/a	**<0.001**
MaxDiff Pelvis	FL	3.46	1.34	9.31	1.32	5.85	n/a	**<0.001**
	HL	5.60	2.55	-25.74	2.52	-31.34	n/a	**<0.001**
Hip Hike Swing	FL	3.92	2.33	16.72	2.32	12.80	n/a	**<0.001**
	HL	7.51	5.03	-56.32	4.98	-63.83	n/a	**<0.001**
Maximum Thoracolumbar Angle (degrees)								
Left/ LS Lateral Bending	FL	4.53	0.65	4.72	0.65	0.19	4.19	0.4
	HL	4.20	0.80	3.26	0.80	-0.94	22.38	**<0.001**
Right/ NLS Lateral Bending	FL	-2.10	0.70	-2.27	0.70	-0.17	8.10	0.4
	HL	-2.84	0.93	-3.93	0.93	-1.09	38.38	**<0.001**
Extension	FL	-22.54	0.96	-22.72	0.96	-0.18	0.80	0.2
	HL	-21.82	1.05	-22.50	1.05	-0.68	3.12	**<0.001**
Flexion	FL	-16.62	0.83	-16.91	0.83	-0.29	1.74	**<0.001**
	HL	-16.00	0.80	-16.33	0.80	-0.32	2.00	**<0.001**
Pelvis ROM (degrees)								
Pitch	FL	8.39	0.48	9.13	0.48	0.73	8.70	**<0.001**
	HL	8.63	0.42	9.68	0.42	1.05	12.17	**<0.001**
Roll	FL	7.12	0.66	7.25	0.66	0.12	1.69	0.4
	HL	7.25	0.63	7.21	0.63	-0.04	0.55	0.8
Yaw	FL	3.02	0.32	3.30	0.32	0.28	9.27	**0.02**
	HL	3.16	0.23	3.23	0.23	0.07	2.22	0.2
Longissimus sEMG ARV (%)								
T14, NLS	FL	86.73	6.09	80.30	6.09	-6.43	7.41	**<0.001**
	HL	85.08	4.83	109.81	4.81	24.73	29.07	**<0.001**
T14, LS	FL	88.01	3.31	82.65	3.31	-5.36	6.09	**<0.001**

	HL	86.66	5.73	118.28	5.72	31.62	36.49	**<0.001**
L1, NLS	FL	86.76	3.75	78.91	3.75	-7.85	9.05	**<0.001**
	HL	83.12	4.00	101.76	3.98	18.64	22.43	**<0.001**
L1, LS	FL	92.48	30.70	166.55	30.68	74.07	80.09	**<0.001**
	HL	86.28	2.77	95.08	2.77	8.81	10.21	**<0.001**

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LIST OF FIGURE LEGENDS

Figure 1: Graphs show mean (solid line) and standard deviation (shaded area) of amplitude-normalised, linear-enveloped sEMG signals from LS and NLS longissimus (L1 location) and time-angle curves for thoracolumbar flexion/extension and lateral bending from representative “horse 4” during baseline 1 (blue) and iFL (red) conditions. Within the sEMG graphs, upward and downward arrows demarcate sEMG activity onset and offset, respectively, for baseline 1 (blue arrows) and iFL (red arrows). Data are time-normalised between LS hindlimb impact events. Line drawings show the outline of the horse at different stages of the stride cycle, as illustrated by horizontal bars showing mean stance phase for each limb (baseline 1: blue bars, iFL: red bars). Within the line drawings, red arrows illustrate significant (solid arrows) and non-significant (outline arrows) decreases in thoracolumbar flexion/extension (vertical arrows) and lateral bending (horizontal arrows) following iFL. Significant increases in pelvis pitching are illustrated as curved, green arrows around the transverse axis.

Figure 2: SPM results for time-normalised thoracolumbar kinematic data across the group of horses for flexion/extension (a, c) and lateral bending (b, d) during baseline 1 and iFL (a, b) and baseline 2 and iHL (c, d). Upper graphs illustrate median (solid line) and standard deviation (shaded area) kinematic data for baseline (blue) and induced lameness (red) conditions. Lower graphs illustrate the paired samples t-test SPM result (black solid line) and the critical thresholds for significance (red dashed line). Data are time-normalised between impacts of the LS hindlimb.

Figure 3: SPM results for time-normalised pelvis segment kinematics across the group of horses for pitch (a, d), roll (b, e), yaw (c, f) during baseline 1 and iFL (a, c), and baseline 2 and iHL (d, f). Within each subpanel, upper graphs illustrate median (solid line) and standard deviation (shaded area) kinematic data for baseline (blue) and induced lamenss (red) conditions. Lower graphs illustrate the paired samples t-test SPM result (black solid line) and the critical thresholds for significance (red dashed line). Data are time-normalised between impacts of the LS hindlimb. Grey shaded areas indicate regions with statistically significant differences between conditions.

Figure 4: SPM results for time and amplitude-normalised longissimus sEMG data across the group of horses during baseline 1 (blue) and iFL (red) conditions for T14 (a, b) and L1 (c, d) locations on the LS (a, c) and NLS (b, d). Within each subpanel, upper graphs illustrate median (solid line) and standard deviation (shaded area) sEMG data and lower graphs illustrate the paired samples t-test SPM result (black solid lines) and the critical thresholds for significance (red dashed line). Data are time normalised between ipsilateral hindlimb impact events.

Figure 5: Graphs show mean (solid line) and standard deviation (shaded area) of amplitude-normalised, linear enveloped sEMG signals from LS and NLS longissimus (L1 location) and time-angle curves for thoracolumbar flexion/extension and lateral bending from representative “horse 2” during baseline 2 (blue) and iHL (red) conditions. Within the sEMG graphs, upward and downward arrows demarcate sEMG activity onset and offset, respectively, for baseline 2 (blue arrows) and iHL (red arrows). Data are time-normalised between LS hindlimb impact events. Line drawings show the outline of the horse at different stages of the stride cycle, as illustrated by horizontal bars showing mean stance phase for each limb (baseline 2: blue bars, iHL: red bars). Within the line drawings, arrows illustrate significant (solid arrows) and non-significant (outline arrows) increases (green arrow) and decreases (red arrow) in thoracolumbar flexion/extension (vertical arrows) and lateral bending (horizontal arrows) following iHL. Significant increases in pelvis pitch and yaw are illustrated as curved, green arrows around the transverse and vertical axes, respectively.

Figure 6: Results of SPM of time and amplitude normalised sEMG data from longissimus across the group of horses during baseline 2 (blue solid line/shaded area) and iHL (red solid line/shaded area) for T14 (a, b) and L1 (c, d) locations on the LS (a, c) and NLS (b, d). Within each subpanel, upper graphs illustrate median (solid line) and standard deviation (shaded area) sEMG data and lower graphs illustrate the paired samples t-test SPM result (black solid lines) and the critical thresholds for significance (red dashed line). Data are time normalised between ipsilateral limb impact events.

LIST OF LEGENDS FOR SUPPLEMENTARY ITEMS

Supplementary Video (SV1): Video showing axial movement and muscle activity from one representative horse (Horse 4) and stride at trot during the baseline 1 condition. In the first clip, thoracolumbar flexion/extension angle and sEMG signals from the left and right longissimus dorsi at T14 and L1 locations are presented as separate graphs in the right panel, alongside the moving three-dimensional model in the middle panel, to illustrate muscle activation in relation to movement during one trot stride. In a second clip, thoracolumbar lateral bending angle is presented alongside the sEMG signals and the moving three-dimensional model from the same representative horse and trot stride. Video clips were exported and adapted from Visual3D (C-motion Inc.) software.

Supplementary Item 1: detailed description of calculations for discrete kinematic variables are a separate document within the folder.

Figure S1: Retro-reflective markers and surface electromyography (sEMG) sensors attached to one subject (a) at the following anatomical locations: 1. Marker cluster attached to the head, 2. Poll, 3. Thoracic (T) 6, 4. T10, 5. T13, 6. Lumbar (L) 1, 7. L3, 8. L5, 9. Between the tuber sacrale, 10. Sacral vertebra (S) 3, 11. S5, and bilaterally over: 12. Proximal end spina scapula, 13. Greater tubercle of the humerus, 14. Lateral tuberosity radius, 15. Marker cluster attached to distal radius, 16. Marker cluster attached to mid 3rd metacarpus bone (MCIII), 17. Centre of rotation metacarpophalangeal joint (MCPJ), 18. Lateral hoof wall (at the centre of rotation of the distal interphalangeal joint (DIPJ), 19. Tuber coxae, 20. Greater trochanter femur, 21. Lateral tibia plateau, 22. Marker cluster attached to distal tibia, 23. Proximal end 4th metatarsal bone (MCIV), 24. Marker cluster attached to mid 3rd metatarsal bone (MTIII), 25. Centre of rotation metatarsophalangeal joint (MTPJ), 26. Lateral hoof wall. Bilateral sEMG sensor sites for Longissimus at T14 (27) and L1 (28). Inset (b): showing prepared skin sites and adhesion technique for markers and sensors at L1 (28).

Figure S2: Individual SPM results for horse 4 with induced forelimb lameness, showing the stride and amplitude normalised sEMG signal for T14, L1, LS and NLS. Results are compared between baseline 1 (BAS) (blue/ with shaded area) and iFL (red/ with shaded area). Graphs within the top two rows

show the median stride (solid lines) and their standard deviations (shaded areas). Graphs within the middle row show individual SPM results for hotelling T2 (black solid lines) with the respective critical thresholds (red dashed lines). Graphs within the bottom two rows show post-hoc analysis for each site separately (T14, L1) at LS and NLS, with the SPM t-statistic (black solid lines) and the critical threshold (red dashed lines). Statistically significant areas are indicated with the grey shaded areas, where $p < 0.05$. Data are time normalised between ipsilateral hindlimb impact events.

Figure S3. Individual SPM results for horse 6 with induced hindlimb lameness, showing the stride and amplitude normalised sEMG signal for T14, L1, LS and NLS. Results are compared between baseline 1 (BAS) (blue/ with shaded area) and iFL (red/ with shaded area). The top two panels show the median stride (solid lines) and their standard deviations (shaded areas). The middle graphs show individual SPM results for hotelling T2 (black solid lines) with the respective critical thresholds (red dashed lines). The bottom two rows show post-hoc analysis for each site separately (T14, L1) at LS and NLS, with the SPM t-statistic (black solid lines) and the critical threshold (red dashed lines). Statistically significant areas are indicated with the grey shaded areas, where $p < 0.05$. Data are time normalised between ipsilateral hindlimb impact events.

Table S1. Resume of kinematic variables of all horses for baseline 1,2 and after iFL and iHL – note that data is not mirrored and presented data is the original induced limb: MinDiff (difference between the two minima of the movement) and MaxDiff (difference between the two maxima of the movement) Hip Hike Swing (the difference between the upward movement of the left and right tuber coxae during swing phase) and Hip Hike Stance (difference between the upward movement of the left and right tuber coxae during stance).

Table S2: Estimated marginal means (EM Mean) and standard error (SE) for baseline and lameness induction conditions, and estimated differences (EM Mean Difference) between corresponding baseline and induction conditions and associated p-values for discrete sEMG activation onset, offset and activity duration variables (% stride) for longissimus dorsi. Data for iFL and iHL lameness conditions are presented from the models with- (speed corrected data) and without (non-speed corrected data) a speed*condition fixed effect. Bilateral sEMG data are presented for each muscle site (L1 and T14) from the NLS and LS, based on the side of induced lameness.

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