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Myofascial Release Versus Conventional Exercise on Pain and Pain Sensitivity in Chronic Low Back Pain

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ABSTRACT: Chronic non-specific low back pain (CLBP) is a leading cause of disability, and it is unclear whether myofascial release (MFR) and exercise differ in their effects on pain and on pain sensitivity. This assessor-blinded randomized controlled trial compared MFR with conventional core-stabilization exercise in adults aged 20–40 years with CLBP and pain of at least 4 on the Numeric Pain Rating Scale (NPRS). Sixty-four participants were randomized to 24 supervised sessions of MFR or exercise over 8 weeks. The primary outcome was NPRS at week 8, analysed by intention-to-treat ANCOVA with multiple imputation. Secondary outcomes were local (lumbar) and remote (tibialis anterior) pressure pain thresholds (PPT), the Central Sensitization Inventory (CSI), the Pain Catastrophizing Scale (PCS) and responder status. Fifty-nine participants (92%) completed the trial. At week 8, pain was 1.10 points lower after MFR than after exercise (95% CI -1.86 to -0.35; p = 0.004; d = -0.74), and 90.0% versus 51.7% of participants achieved a clinically important improvement (number needed to treat 2.6). MFR increased local PPT by 54.2 kPa more than exercise (Bonferroni-adjusted p = 0.025), whereas exercise produced a modest increase in remote PPT; CSI and PCS improved similarly in both groups. The between-group difference in pain was no longer significant at the week-12 follow-up, and adverse events were mild in both groups. MFR provided greater short-term pain relief and local pain-threshold improvement than conventional exercise, suggesting a predominantly local mechanism; strategies to maintain its benefit warrant investigation.

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1. INTRODUCTION

Low back pain is the most disabling condition in terms of years lived with disability globally with 619 million individuals affected in 2020 and predicted to increase to 2050 (GBD 2021 Low Back Pain Collaborators, 2023). A pooled estimate for India demonstrates high prevalence in working age individuals (Shetty et al., 2022). Approximately 90% of low back pain cases do not have a specific pathoanatomical origin and are called non-specific low back pain. If pain persists past 12 weeks this is called chronic non-specific low back pain (CLBP). Chronic pain is the main reason individuals with CLBP seek treatment but most non-surgical treatments have shown only short and modest improvements in pain (Foster et al., 2018).

Exercise therapy is considered first line treatment for CLBP with small to moderate pain improvement compared to minimal care (Hayden et al., 2021). Core stabilization and motor control exercises appear to be among the most effective (Saragiotto et al., 2016; Owen et al., 2020). Analgesic effects appear to come from improved motor control as well as pain modulation through exercise induced hypoalgesia. Hypoalgesia is a systemic decrease in pain sensitivity that can also affect body areas that are remote from where the exercise is performed (Naugle et al., 2012; Rice et al., 2019).

The thoracolumbar fascia (TLF) can be a source of pain in CLBP. The TLF is innervated by nociceptive free nerve endings (Tesarz et al., 2011; Willard et al., 2012). Individuals with CLBP have been found to have decreases in shear strain in the TLF suggesting a change in connective tissue mechanics (Langevin et al., 2011). Myofascial release (MFR) uses sustained low intensity pressure and stretch to fascial tissue and has been proposed to reduce pain through both local tissue effect and modulation of nociceptive processing in the central and peripheral nervous systems (Schleip, 2003; Bialosky et al., 2009). A meta-analysis and randomized controlled trials have found MFR to have pain reduction effects in individuals with CLBP (Ajimsha et al., 2014; Arguisuelas et al., 2017; Wu et al., 2021). Most of the studies however compared MFR to usual care or sham treatment instead of to an exercise treatment with evidence for efficacy.

The mechanisms proposed have differing implications for pain sensitivity. MFR may have increased pain thresholds at local treated spinal tissue but may not increase pain thresholds at remote areas while exercise may increase thresholds in both local and remote areas. Pressure pain threshold (PPT) is lower in both local and remote areas in individuals with CLBP (Giesecke et al., 2004; den Bandt et al., 2019). However, most studies only report a pain score and there is a lack of data to determine if either MFR or exercise affects pressure pain sensitivity in either local or remote areas of the body. There has not been a head to head comparison between MFR and conventional exercise on pressure pain sensitivity (local and remote), pain intensity and central sensitization in young individuals with CLBP. The purpose of this study was therefore to compare pain intensity, pressure pain sensitivity (local and remote), pain catastrophizing and central sensitization between individuals undergoing 8 weeks of MFR to those undergoing 8 weeks of conventional exercise. It was hypothesized that after 8 weeks MFR would have greater reduction in pain intensity (H_1) and greater increase in local lumbar PPT (H_2) compared to conventional exercise.

2. MATERIALS AND METHODS

2.1. Study design, setting and ethics

This study is a randomized controlled trial, two arms (MFR and exercise), parallel, assessor blinded with 1:1 allocation. The study was conducted in the Advanced Musculoskeletal Rehabilitation Unit, ACS Medical College and Hospital in Chennai, India and has been reported in accordance with the CONSORT 2010 recommendations (Schulz et al., 2010). The study protocol was approved by the Institutional Review Board and was conducted in accordance with the Declaration of Helsinki with prior informed consent from the participants.

2.2. Participants

Individuals 20-40 years old of either gender with non-specific low back pain for at least 12 weeks, pain intensity of at least 4 on the NPRS at screening and ability to attend three times per week for eight weeks are eligible. Excluded individuals include those with specific spinal pathology (infection, malignancy, inflammation of arthritis or spinal fracture), radiculopathy with neurological deficit, spinal surgery within 12 months prior to screening, pregnancy, skin abnormalities or bleeding disorders that would contraindicate MFR, or anticoagulant therapy, individuals who have had injections or manual therapy for low back pain within the past three months and individuals with serious comorbidities (cardiac disease, psychiatric illness or uncontrolled diabetes). BMI was based on Asian cut offs (WHO Expert Consultation, 2004).

2.3. Sample size

We calculated a difference between groups of 2 on the NPRS which corresponds to the minimal clinically important difference (Ostelo et al., 2008) with a standard deviation of 2.5. This results in an effect size of d = 0.8. With an alpha level of 0.05 (two tailed) and 80% power we needed 26 individuals per group. Accounting for a 15% attrition rate we calculated we needed at least 30 individuals per group. 32 individuals were recruited per group for a total of 64 individuals. All participants were randomized.

2.4. Random assignment, allocation secrecy, and masking

An autonomous investigator developed a computer-generated randomization sequence with blocks of sizes 4 and 6. Allocations were hidden inside consecutively numbered, non-transparent sealed envelopes that were unsealed after the baseline. The evaluator and statistician were unaware of the assignment. The participants and physiotherapists were not blinded to the interventions because of the inherent characteristics of the therapies.

2.5. Interventions

Both interventions consisted of 24 sessions (three times per week for eight weeks) of approximately 20 minutes. Both also received the same standardized education on staying active and good posture. Participants were allowed paracetamol for rescue pain and use of paracetamol was recorded. For the MFR group individuals received direct and indirect MFR techniques from a physiotherapist who had experience in MFR. These included cross-hand release of TL fascia in prone (3 × 90-120 s), longitudinal release along erector spinae (three passes per side), release of quadratus lumborum in side-lying (90-120 s per side), sustained pressure release of gluteal fascia (90 s per side) and lumbosacral decompression in supine (2 min). For the exercise group a warm up of pelvic tilts and cat-camel (10 repetitions) was performed. Following this the abdominal drawing-in exercise (10 × 10-s holds) was performed. Supine bridging, bird dog, curl up and side plank (2 sets × 10 repetitions or 10-s holds) and prone back extension (2 sets × 10 repetitions) were also performed. Every two weeks these exercises progressed through repetition, hold time or through the use of an unstable surface. Attendance and any adverse event were recorded for all sessions.

2.6. Outcome measures

All outcome measures were assessed by the assessor. The primary outcome was pain intensity using the 11 point NPRS (average over past 24 h) at week 8 and NPRS was also recorded at baseline, week 4 and week 12 follow up (Childs et al., 2005). Local pressure pain thresholds (PPT) were measured over bilateral L3 and L5 paraspinal muscles using a digital algometer with a 1 cm^2 probe at baseline, week 4 and week 8. Remote (widespread) pain sensitivity was measured over bilateral tibialis anterior at baseline and week 8. At each site the mean of three trials was used (kPa). Local and remote PPT were calculated as the mean of the four lumbar and two tibialis anterior sites respectively (Balaguier et al., 2016). Central sensitization was assessed using the Central Sensitization Inventory Part A (CSI; 0-100; ≥40 indicating clinically relevant sensitization) (Mayer et al., 2012; Neblett et al., 2013) and pain cognitions using the Pain Catastrophizing Scale (PCS; 0-52 with subscales of rumination, magnification and helplessness) (Sullivan et al., 1995) at baseline and week 8. A responder was defined as a participant with a pain intensity reduction of at least 2 points or 30% at week 8 (Farrar et al., 2001; Ostelo et al., 2008). Adverse events were recorded at every session.

2.7. Statistical analysis

Analyses were done on the full dataset (intention-to-treat). Missing values at follow up were analyzed using multiple imputation with 50 data sets created through Bayesian linear regression imputation using baseline pain, group, age, sex, BMI, pain duration and attendance as predictors. Imputed data were then combined using Rubin's rules (Rubin, 1987; Sterne et al., 2009). Pain intensity (NPRS) at week 8 was compared between groups using analysis of covariance (ANCOVA) with adjustment for baseline pain intensity. NPRS was also analyzed at weeks 4, 8 and 12 using a linear mixed model with adjustment for baseline pain intensity, time, group and group × time interaction with a random participant intercept. Secondary continuous outcomes (local PPT, remote PPT, CSI and PCS at week 8) were also analyzed in the same way. Bonferroni correction was applied to these four outcomes. Pain intensity at weeks 4 and 12, local PPT at week 4 and PCS subscales were analyzed as exploratory. Effect sizes were shown using Cohen's d accompanied by 95% confidence intervals (CI). The Shapiro-Wilk test was used to evaluate normality. Baseline disparities were evaluated using independent t tests or Mann-Whitney U tests for continuous variables, and χ^2 or Fisher's exact tests for categorical variables. The assumptions of ANCOVA regarding the homogeneity of regression slopes, homogeneity of variances, and normality of residuals were evaluated for each study. Sensitivity evaluations for the primary outcome including full case ANCOVA, baseline observation carried forward, and analysis of per protocol participants

(those who attended a minimum of 80% of sessions). Additional sensitivity analyses included adjustment for additional covariates, robust (HC3) standard errors and non-parametric methods. Proportions of responders were assessed using the χ^2 test and number needed to treat (NNT) and relative risk (RR) were provided with 95% CI. Group alterations were evaluated using the Wilcoxon signed-rank test. The dependability of the three trial pressure pain threshold strategy was assessed using intraclass correlation coefficients (ICC 3,1) (Koo and Li, 2016). The subgroup impact was evaluated using the interaction terms of group and subgroup. The threshold for significance was established at a two-tailed $p < 0.05$. Analyzes were conducted using Python 3 (pandas, SciPy, and statsmodels).

3. RESULTS AND DISCUSSION

3.1. Baseline characteristics and participant flow

There were two dropouts in the MFR group (muscle soreness post-treatment and relocation) and three in the exercise group (change in exercise times and pain intensifying with exercise and loss to follow-up). Thus data was available for week 8 in 59 participants (MFR 30, exercise 29; 92.2%) and 57 for follow-up at week 12 (Figure 1). No significant differences were found between groups at baseline in age, sex, pain level, duration of pain, pain catastrophising, sensory or clinical characteristics (Table 1). Most participants were young adults (mean age approximately 31 years), had moderate pain (mean NPRS of approximately 6) with a median duration of pain of 18-19 months and 37.5% of both groups had clinically relevant central sensitisation (CSI ≥40). All six PPT sites showed high reliability (ICC 3,1 = 0.95-0.96; SEM 26-35 kPa) and it is therefore likely the sensory results were accurate.

3.2. Primary outcome: pain intensity

Pain intensity reduced significantly more in the MFR group than in the exercise group (Table 2; Figure 2). In the intention to treat analysis, pain intensity at week 8 was 1.10 points less in the MFR group compared to the exercise group (95% CI -1.86 to -0.35; $p = 0.004$), which represents a moderate to large effect size ($d = -0.74$; 95% CI -1.25 to -0.23). There was also an advantage in week 4 (mixed model difference of -0.86; $p = 0.015$) and the difference between the two groups was consistent across the complete-case, per-protocol, covariate adjusted, non-parametric, baseline-observation-carried-forward and mixed model analyses ($p \leq 0.041$). Individual responses also demonstrated that almost all subjects in the MFR group improved whilst there was greater heterogeneity in responses to the exercise group (Figure 3). Therefore the null hypothesis was rejected in favour of the alternative hypothesis.

Clinically relevant pain reduction was also observed. At week 8 90.0% (27/30) of those in the MFR group were responders (compared to 51.7% (15/29) of those in the exercise group) (RR 1.74; 95% CI 1.20-2.52; NNT 2.6; 95% CI 1.7-5.8; $p = 0.001$). 56.7% (17/30) of MFR patients also achieved at least 50% pain reduction compared to 27.6% (8/29) in the exercise group ($p = 0.024$) (Table 4; Figure 4). Adjusted difference between groups is approximately 1.1 points. Whilst this is less than the 2 point individual MCID used for responder status, it also represents a moderate to large effect size. The study was also adequately powered to detect this effect.

Results are consistent with previous studies and a recent meta-analysis showing that MFR can reduce pain in CLBP (Ajimsha et al., 2014; Arguisuelas et al., 2017; Wu et al., 2021). In addition, this study demonstrated that MFR is effective compared to an active, guideline recommended, exercise programme of the same intensity. Within group changes with exercise (-1.6 points) are consistent with moderate effect sizes demonstrated in systematic reviews of exercise programmes (Owen et al., 2020; Hayden et al., 2021). However the effect of MFR was not sustained. At 4 weeks post-treatment, pain had increased in the MFR group and the between group difference was no longer significant (mixed model difference -0.51; $p = 0.159$). This is typical of passive manual therapies (Bialosky et al., 2009) suggesting MFR might be useful for providing short term pain relief followed by a maintenance strategy (such as home exercise).

3.3. Local and widespread pressure pain sensitivity

Local lumbar PPT increased significantly following MFR (+76.7 kPa; 15-22% increase at the four sites; all $p < 0.001$) and there was a small but not significant increase with exercise (+20.2 kPa). After adjustment local PPT at week 8 was 54.2 kPa higher in the MFR group (95% CI 15.3 to 93.2; Bonferroni-adjusted $p = 0.025$; $d = 0.72$) (Table 3; Figure 5). The between group difference was larger than the measurement error specified in the protocol and therefore H_2 was supported. No significant difference was seen at week 4 ($p = 0.323$) suggesting this change in PPT accumulated over the treatment period. At remote

sites (tibialis anterior) the pattern was reversed with exercise increasing remote PPT (+28.9 kPa; p = 0.009) with the largest remote increase in right tibialis anterior (+9.9%), whereas with MFR there was no significant difference in remote PPT (+13.1 kPa; p = 0.248). The between group difference in remote PPT was not significant (Bonferroni-adjusted p = 1.000) (Figure 6)

A comparison of local versus widespread changes in pressure pain sensitivity can be seen in Figure 7. MFR participants showed a significant increase in local PPT compared to no change in remote PPT and exercise participants demonstrated a small change with some increased remote PPT. This supports the hypothesised differences between the mechanisms of MFR and exercise. MFR targets densely innervated paraspinal and TLF muscles (Tesarz et al., 2011; Willard et al., 2012) and may decrease local mechanical hyperalgesia and peripheral nociceptive input. This would be consistent with increased local PPT observed following other manual therapies (Coronado et al., 2012). Exercise is hypothesised to produce endogenous descending pain inhibition and this results in exercise induced hypoalgesia that is also detected at remote sites (Naugle et al., 2012; Rice et al., 2019). In CLBP both local and remote PPT are reduced (den Bandt et al., 2019) and so combining MFR and exercise could provide improvements in both local and widespread pain sensitivity and this should be investigated.

3.4. Central sensitization and pain catastrophising

Within group improvements in central sensitisation (CSI) and pain catastrophising (PCS) were observed (CSI -7.2 vs -3.9 points; PCS -4.7 vs -2.8 points; all $p \leq 0.015$). However no significant between group difference was observed (Table 3). The proportion of participants with clinically relevant central sensitisation (CSI ≥40) decreased from 36.7% to 6.7% in the MFR group and from 34.5% to 27.6% in the exercise group. In the MFR group 10 of the 11 participants with moderate to extreme levels of central sensitisation at baseline also moved to a lower level of central sensitisation (Figure 8). Among the PCS subscales the largest change in favour of MFR was seen with helplessness although this was exploratory and did not reach significance (p = 0.056). The CSI is not a direct measure of central nociceptive processing but instead is a measure of subjective symptoms related to central sensitisation (Mayer et al., 2012; Nijs et al., 2010). The lack of a significant between group difference in remote PPT also suggests that MFR did not result in a significant change in widespread pain sensitivity over 8 weeks. Changes in pain were weakly associated with changes in local PPT ($\rho = -0.20$) but not with changes in CSI or PCS suggesting pain reduction and changes in pain sensitivity and pain cognitions were not fully correlated.

3.5. Attendance and safety

Attendance was equivalent in both groups (median 83% of sessions) and 56.2% of each group attended at least 80% of sessions. There was no association between attendance and pain reduction therefore it is not likely that increased exposure to treatment resulted in an increase in the effect of MFR (Table 4). There was also a slightly lower use of rescue paracetamol in the MFR group (80 tablets in total compared to 99 tablets in the exercise group; p = 0.293). Adverse events occurred in 25.0% of MFR participants and 28.1% of exercise participants (p = 1.000), all adverse events were mild and transient. With MFR the adverse events were soreness or tenderness following treatment and a transient increase in pain after the first week. With exercise the adverse events were hamstring soreness, knee discomfort during squats and a flare in pain after progression. One participant from each group withdrew due to discomfort associated with treatment. There were no serious events.

3.6. Exploratory subgroup analysis

The direction of the effect of MFR was consistent across all subgroups of sex, pain duration, baseline pain intensity, pain catastrophising, central sensitisation, BMI and occupation as well as age and occupation (all interaction $p \geq 0.46$). The only significant interaction was found for age with a larger effect in patients under 32 years of age (-1.75 vs -0.35 points; interaction p = 0.047). This result should be treated as hypothesis generating due to the number of subgroups that were assessed.

3.7. Strengths and limitations

This study has several strengths including concealed randomisation, blinding of data collection and analysis, use of an active dose equivalent exercise programme, use of quantitative measures of pain with high reliability at both local and remote sites, low drop out and use of multiple imputation in an ITT analysis, and consistency in sensitivity analyses. Limitations included the use of a single site study, age range of only 20-40 years which limits generalisability to older adults, inability to blind participants or therapists which could have affected self reported measures, lack of a no treatment or sham condition and lack of disability measures. The follow up at week 12 only used NPRS and the durability of the changes in psychological and sensory measures is unknown.

4. CONCLUSIONS

Eight weeks of myofascial release resulted in a significant reduction in pain intensity compared to conventional core-stabilisation exercise with a higher proportion of clinically relevant responders and significant increase in local lumbar PPT compared to a modest increase in widespread PPT with exercise in young adults with CLBP. Both MFR and exercise resulted in similar improvements in measures of catastrophising and central sensitisation and were safe. However the effect of MFR was not sustained at 4 weeks after treatment ended. MFR may be useful in patients with prominent local pain tenderness and in combination with or after exercise to maintain the effect of MFR. Further multicentre studies should investigate combined MFR and exercise, include longer follow up and disability measures and should also include older adults.

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Dr Ajay Kumar: supervision, data collection, writing – review and editing

Dr. Vijayakumar Palaniswamy: supervision, methodology, writing – review and editing.

Dr Praveen B M: Statistical analysis and visualization, review and editing.

Dr. S Jeyakumar: Statistical analysis, data curation and visualization

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Dr. S. Veenakirthika: writing – review and editing and visualization

All authors read and approved the final manuscript.

Conflict of interest

The authors declare that they have no potential conflict of interest.

Ethics statement

The study was approved by the Institutional Review Board of Faculty of Physiotherapy (0372/MSK/PHYSIO/IRB/2026) conducted in accordance with the ICMR

National Ethical Guidelines (2017) and the Declaration of Helsinki. Written informed consent was obtained from all participants.

Data availability statement

The dataset supporting the findings of this study is available from the corresponding author on reasonable request.

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Natural Resources for Human Health
eISSN: 2583-1194

Table 1. Baseline characteristics of randomized participants by group

Characteristic	MFR (n = 32)	Exercise (n = 32)	p value
Demographic and clinical			
Age (years)	30.5 ± 5.6	31.8 ± 4.3	0.291
BMI (kg/m²)	24.1 ± 3.7	23.9 ± 3.5	0.815
Female sex, n (%)	18 (56.2)	17 (53.1)	1.000
BMI category (Asian), n (%)			0.727
Underweight	1 (3.1)	2 (6.2)	
Normal	13 (40.6)	11 (34.4)	
Overweight	5 (15.6)	3 (9.4)	
Obese	13 (40.6)	16 (50.0)	
Occupation, n (%)			0.172
Mainly sedentary	18 (56.2)	16 (50.0)	
Mainly standing	7 (21.9)	13 (40.6)	
Manual / heavy work	7 (21.9)	3 (9.4)	
Duration of LBP (months), median (IQR)	19 (10–30)	18 (12–30)	0.609
Pain medication at entry, n (%)	13 (40.6)	9 (28.1)	0.430
Pain and sensitivity			
NPRS (0–10)	6.09 ± 1.40	5.91 ± 1.30	0.468
Local PPT, L3–L5 (kPa)	412.3 ± 130.5	412.0 ± 141.0	0.993
Remote PPT, tibialis anterior (kPa)	380.2 ± 124.5	385.1 ± 145.4	0.886
CSI Part A (0–100)	34.1 ± 13.6	32.5 ± 12.4	0.614
CSI ≥40, n (%)	12 (37.5)	12 (37.5)	1.000
PCS total (0–52)	20.8 ± 9.4	20.4 ± 9.9	0.887
PCS >30, n (%)	6 (18.8)	4 (12.5)	0.732

Values are mean ± SD unless otherwise stated. p values from independent t-test or Mann–Whitney U test (continuous) and χ^2 or Fisher's exact test (categorical). BMI, body mass index; CSI, Central Sensitization Inventory; LBP, low back pain; NPRS, Numeric Pain Rating Scale; PCS, Pain Catastrophizing Scale; PPT, pressure pain threshold.

Table 2. Pain intensity (NPRS, 0–10) at each time point and between-group effects

Time point / analysis	**MFR mean ± SD (n)**	**Exercise mean ± SD (n)**	**Adjusted difference MFR − exercise (95% CI)**	**p value**
Baseline	6.09 ± 1.40 (32)	5.91 ± 1.30 (32)	—	—
Week 4	4.42 ± 1.54 (31)	5.19 ± 1.70 (31)	-0.86 (-1.55 to -0.17)	0.015
Week 8 (primary end point)	3.37 ± 1.43 (30)	4.21 ± 1.80 (29)	-1.08 (-1.79 to -0.37)	0.003
Week 12 (follow-up)	4.21 ± 1.63 (29)	4.46 ± 1.67 (28)	-0.51 (-1.23 to 0.20)	0.159
Week-8 between-group analyses	***n***	***Cohen's d (95% CI)***	***Adjusted difference (95% CI)***	***p value***
ITT ANCOVA, multiple imputation (primary)	**64**	**-0.74 (-1.25 to -0.23)**	**-1.10 (-1.86 to -0.35)**	**0.004**
ITT linear mixed model	64		-1.08 (-1.79 to -0.37)	0.003
ITT baseline observation carried forward	64		-1.09 (-1.84 to -0.33)	0.005
Complete-case ANCOVA	59		-1.04 (-1.80 to -0.29)	0.008
Per-protocol (attendance ≥80%)	36		-1.06 (-2.08 to -0.05)	0.041
Adjusted for age, sex, BMI, pain duration, medication	59		-1.06 (-1.80 to -0.31)	0.007
Mann–Whitney U on change scores	59		-1.18 (-1.93 to -0.40)	0.005

Upper panel: observed values; adjusted differences from the linear mixed model (baseline NPRS, time, group and group × time; random intercept; n = 64); group × time interaction p = 0.249. Lower panel: week-8 analyses adjusted for baseline NPRS; per-

protocol analysis included 18 participants per group; for the Mann–Whitney row the difference is in mean change with a bootstrap 95% CI. Negative values favour MFR. ITT, intention-to-treat.

Table 3. Secondary outcomes at baseline and week 8 and adjusted between-group differences

Outcome	MFR baseline	MFR week 8	Exercise baseline	Exercise week 8	Adjusted difference (95% CI)	p value	Bonferroni p	Cohen's d (95% CI)
Local PPT (kPa)	**418.2 ± 132.0**	**495.0 ± 168.6**	**415.2 ± 144.9**	**435.4 ± 167.0**	**54.2 (15.3 to 93.2)**	**0.006**	**0.025**	**0.72 (0.20 to 1.24)**
Remote PPT (kPa)	385.9 ± 125.7	399.0 ± 130.5	389.6 ± 147.2	418.5 ± 166.7	-16.6 (-48.4 to 15.2)	0.306	1.000	-0.27 (-0.79 to 0.25)
CSI Part A (0–100)	34.4 ± 13.7	27.3 ± 10.6	31.7 ± 11.8	27.8 ± 12.0	-2.5 (-6.9 to 1.9)	0.260	1.000	-0.29 (-0.80 to 0.22)
PCS total (0–52)	20.8 ± 9.6	16.1 ± 10.5	19.9 ± 9.4	17.1 ± 9.9	-1.8 (-4.5 to 1.0)	0.205	0.821	-0.33 (-0.85 to 0.18)
PCS rumination (0–16) ‡	8.30 ± 3.02	7.13 ± 3.77	7.83 ± 3.56	7.00 ± 3.79	-0.34 (-1.49 to 0.81)	0.561	—	-0.16 (-0.68 to 0.37)
PCS magnification (0–12) ‡	3.83 ± 2.45	3.07 ± 2.53	4.41 ± 2.43	3.34 ± 2.48	0.08 (-0.96 to 1.12)	0.883	—	0.04 (-0.48 to 0.56)
PCS helplessness (0–24) ‡	8.67 ± 4.95	5.93 ± 4.78	7.62 ± 3.99	6.72 ± 4.33	-1.52 (-3.09 to 0.04)	0.056	—	-0.50 (-1.02 to 0.01)

Baseline and week-8 values are mean ± SD in completers (MFR n = 30, exercise n = 29). Adjusted differences (MFR − exercise) are from intention-to-treat ANCOVA with multiple imputation (n = 64) adjusted for the baseline value; for PPT a positive difference favours MFR, for other outcomes a negative difference favours MFR. Bonferroni correction across the four

secondary outcomes; ‡ exploratory outcome. CSI, Central Sensitization Inventory; PCS, Pain Catastrophizing Scale; PPT, pressure pain threshold.

Table 4. Responder analysis, attendance, rescue medication and adverse events

Outcome	MFR	Exercise	Effect (95% CI)	p value
Responders				
NPRS reduction ≥2 points or ≥30% at week 8	27/30 (90.0)	15/29 (51.7)	RR 1.74 (1.20–2.52); NNT 2.6 (1.7 to 5.8)	0.001
NPRS reduction ≥50% at week 8	17/30 (56.7)	8/29 (27.6)	RR 2.05 (1.05–4.00); NNT 3.4 (1.9 to 19.9)	0.024
NPRS reduction ≥2 points at week 12	17/29 (58.6)	14/28 (50.0)	RR 1.17 (0.73–1.90); NNT 11.6 (NS)	0.514
Attendance and rescue medication				
Attendance (%), median (IQR)	83 (75–88)	83 (75–88)		0.797
Attendance ≥80%, n (%)	18 (56.2)	18 (56.2)		1.000
Rescue paracetamol, tablets per participant, median (IQR)	2 (1–4)	3 (1–5)		0.293
Adverse events				
Participants with ≥1 adverse event, n (%)	8 (25.0)	9 (28.1)		1.000
Withdrawal due to adverse event, n	1	1		—
Serious adverse events, n	0	0		—

Responder denominators are participants assessed at that time point; attendance, medication and adverse events include all randomized participants (n = 32 per group). p values from χ^2 test (responders), Mann–Whitney U test or Fisher's exact test. NNT, number needed to treat; NS, not significant (CI includes infinity); RR, relative risk.

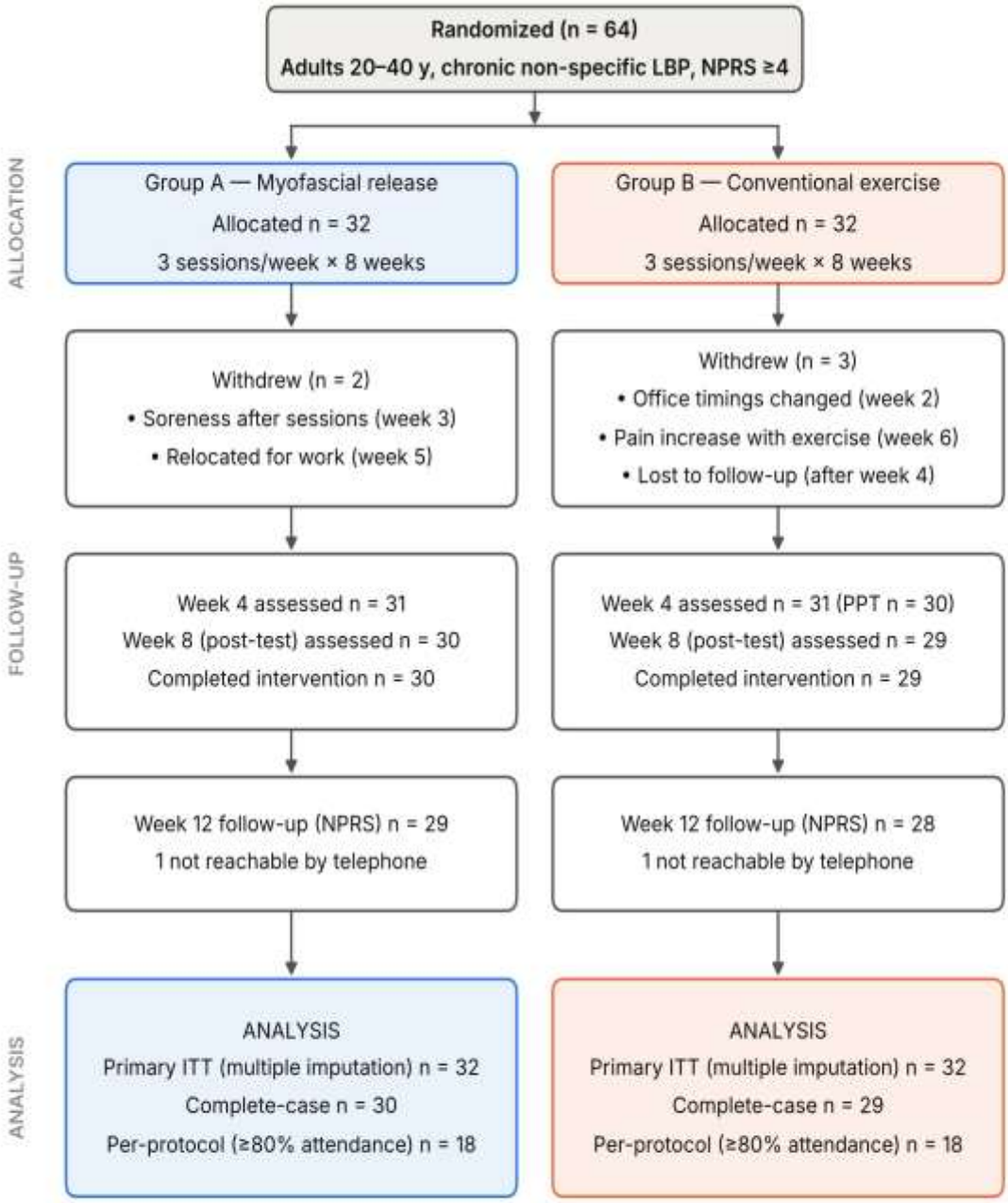


Figure 1. CONSORT flow chart of participants through the study. Number of participants assessed at each time point, number included in each analysis and reason for withdrawal for each group are shown. ITT, intention-to-treat.

Natural Resources for Human Health
eISSN: 2583-1194

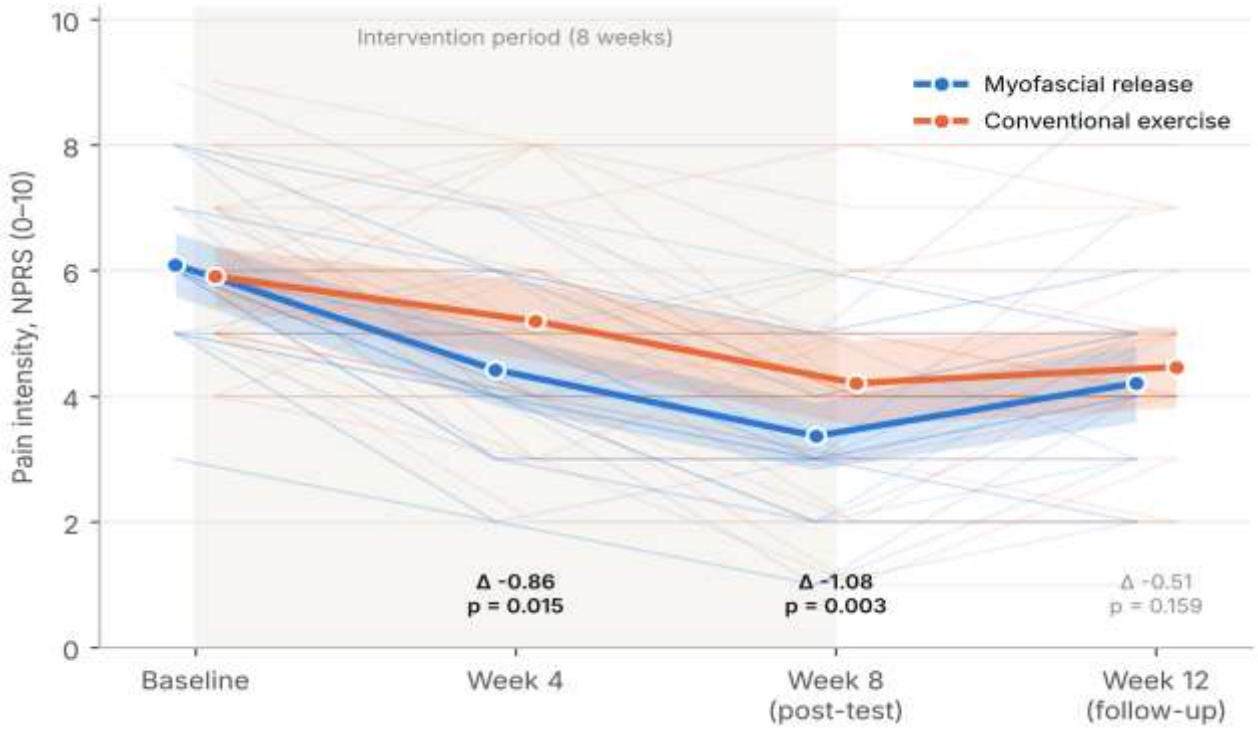


Figure 2. Pain intensity (NPRS) from baseline to week 12 follow-up. Individual data points are shown by thin lines and group means by thick lines with 95% confidence bands shaded in. The points have been slightly displaced to improve clarity. The shaded area represents the duration of the intervention (8 weeks). Values at the bottom represent linear mixed-model adjusted differences (MFR − exercise) and associated p values.

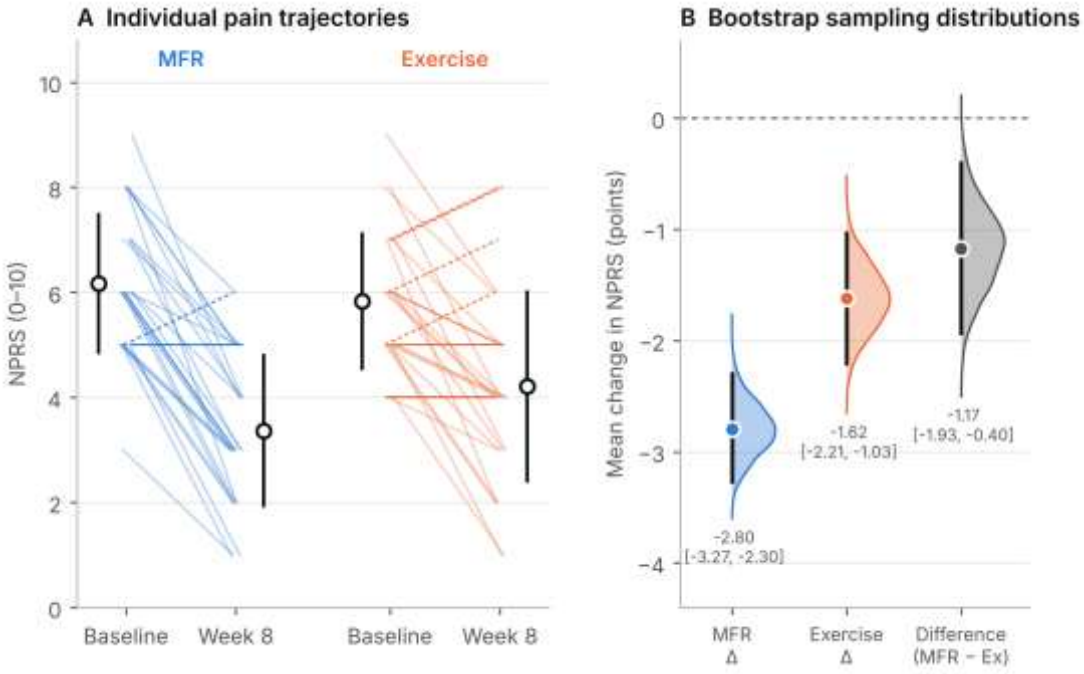


Figure 3. Change in pain intensity from baseline to week 8. (A) Individual changes in participants with data available at week 8 (MFR n = 30, exercise n = 29). Solid lines represent falls in pain intensity and dashed lines represent rises. Open bars with lines represent mean ± SD. (B) Bootstrap distributions (5,000 samples) of within-group mean change and the between-

group difference in mean change. Means and 95% confidence intervals are shown.

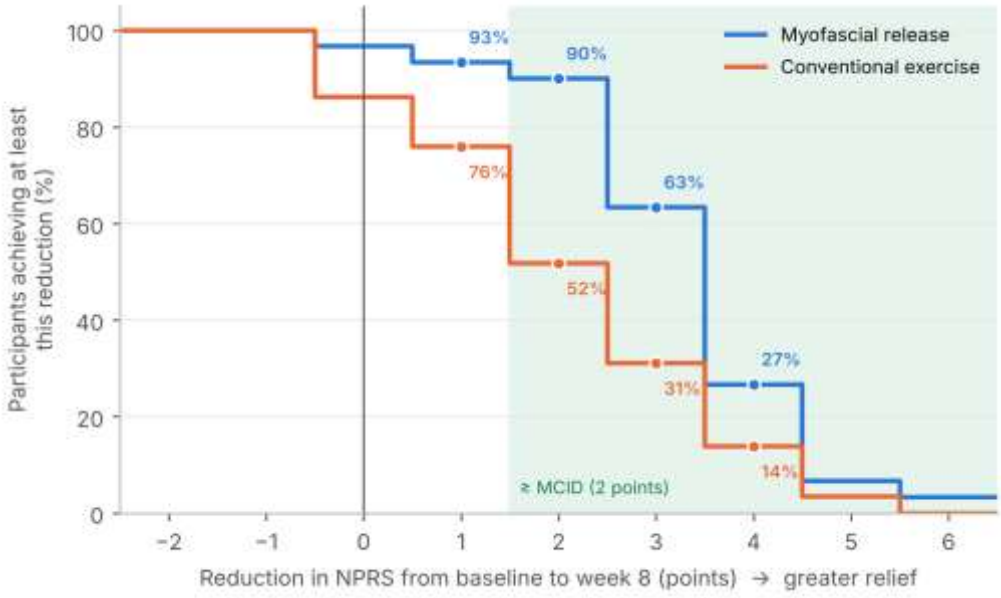


Figure 4. Cumulative responder curve for change in pain intensity at week 8. The horizontal axis represents a change in pain intensity (NPRS) and the curve represents the percentage of participants with a change of at least this size in pain intensity. The green area represents changes of at least the minimal clinically important difference of 2 points.

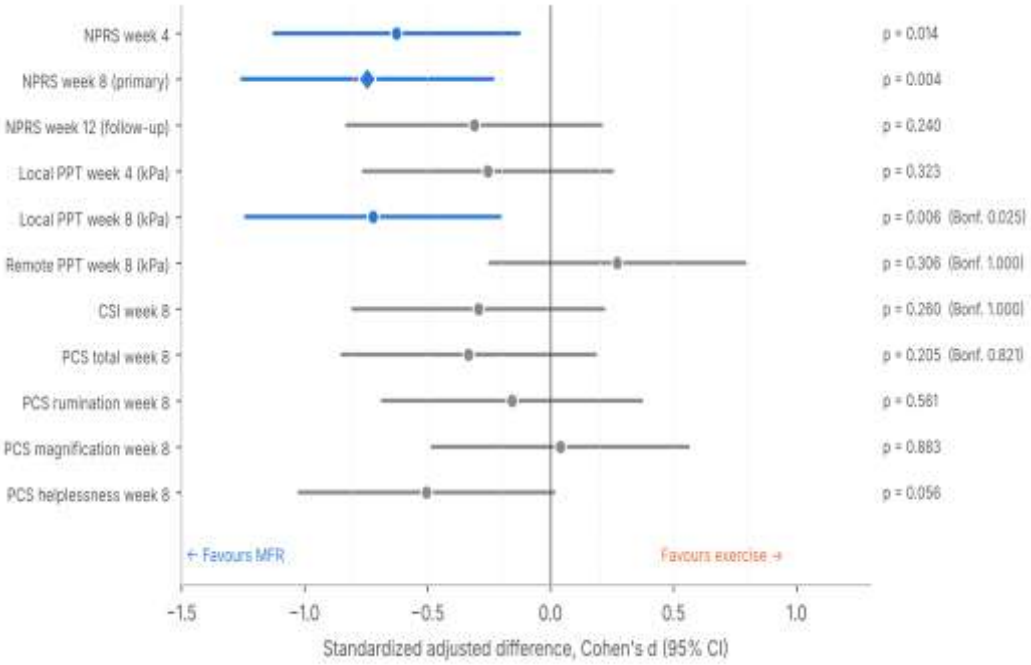


Figure 5. Standardized effects across all outcomes. Cohen's d with 95% confidence intervals from intention-to-treat ANCOVA (multiple imputation, m = 50) adjusted for baseline measures. Effects are oriented such that values to the left of zero are in favour of MFR (except for pressure pain thresholds where the sign is reversed). Blue symbols represent $p < 0.05$. Bonferroni adjusted p values are shown for the secondary outcomes. All other outcomes were exploratory.

Natural Resources for Human Health
eISSN: 2583-1194

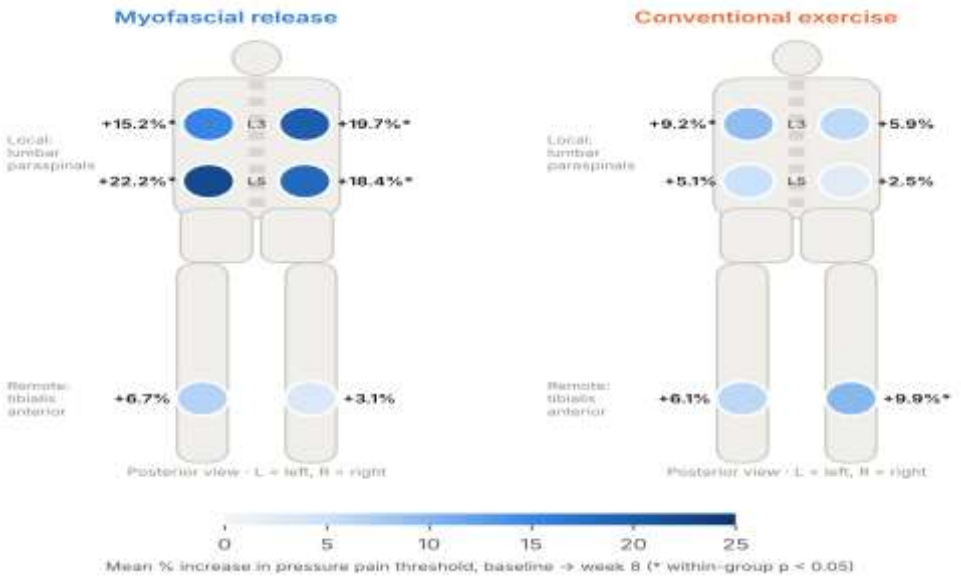


Figure 6. Change in pressure pain threshold at each assessment site. A posterior view diagram showing the mean % increase in pressure pain threshold from baseline to week 8 at the local (paraspinal at L3 and L5) and remote (tibialis anterior) sites for each group. The darker the shade the greater the increase. * $p < 0.05$ within group.

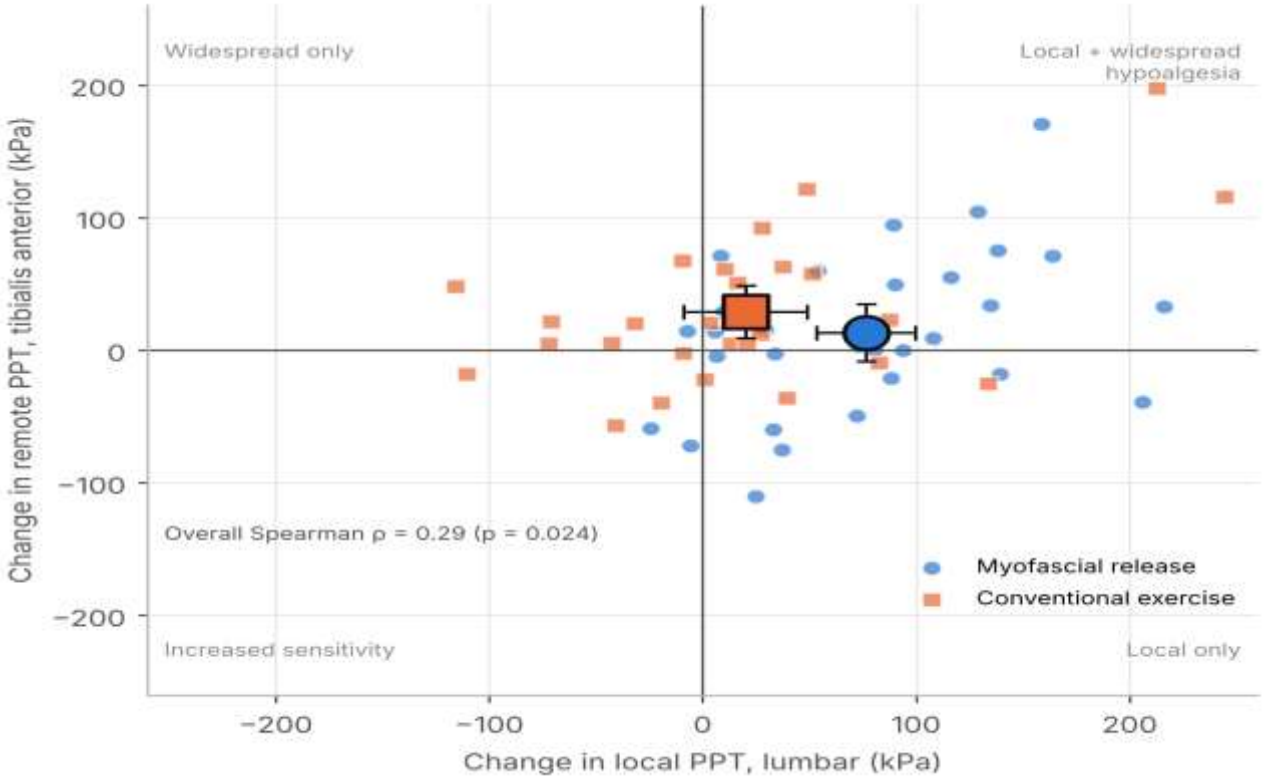


Figure 7. Change in local and remote pressure pain thresholds. Each point represents the change in local (lumbar) pressure pain threshold and remote (tibialis anterior) pressure pain threshold from baseline to week 8 for one participant. Large symbols represent group means with 95% confidence intervals. Quadrants represent no hypoalgesia, widespread hypoalgesia, combined hypoalgesia or local hypoalgesia.

Natural Resources for Human Health
eISSN: 2583-1194

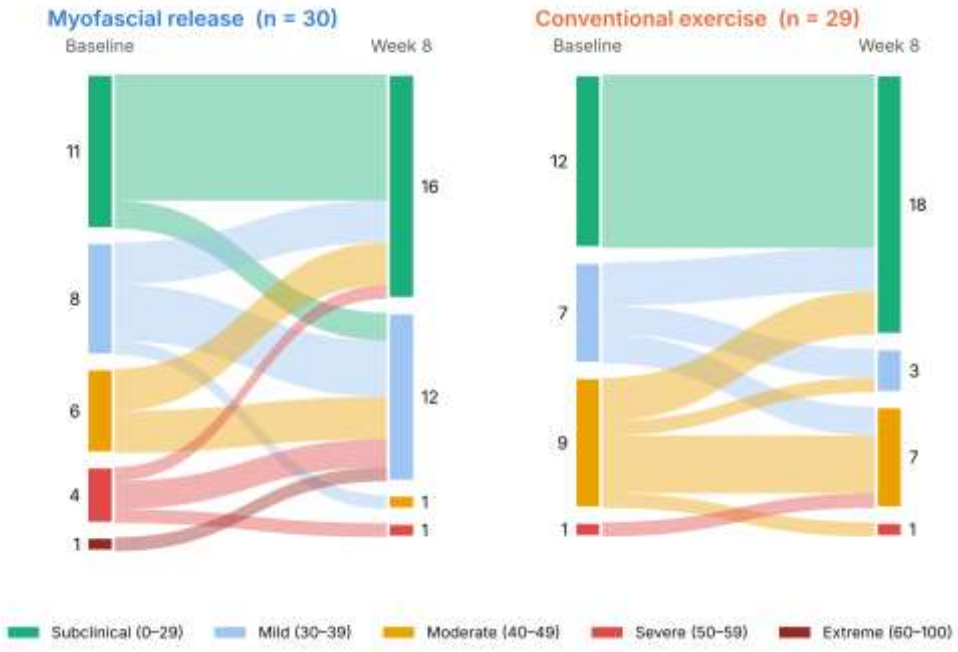


Figure 8. Change in severity category of the Central Sensitization Inventory from baseline to week 8. Blocks represent the number of participants in each severity category of the CSI at baseline (left) and at week 8 (right). Ribbons represent individual transitions between severity categories and are coloured according to baseline category. (MFR n = 30, exercise n = 29.)