



# Scheduled Intermittent Compression–Decompression Therapy Attenuates Cumulative Lumbar Shear Amplification During Prolonged Occupational Sitting: A Randomized Crossover Pilot Study

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### AUTHORS AND AFFILIATIONS:

**Neeraj Mehta, PhD**, *MMSx Authority Institute for Movement Mechanics & Biomechanics Research, Powell, Ohio, USA.*  
<https://orcid.org/0000-0001-6200-8495>

**Christopher Alfiero, PhD**, *Performance Technology Division, Eternal, San Rafael, California, USA.***ORCID:**  
<https://orcid.org/0009-0001-9806-3506>

**Dr. Karun Jain, MBBS, MS, FACS**, *Orthopaedic & Trauma Surgery Division, San Francisco, California, USA.***ORCID:**  
0009-0000-9635-7132

**Hatem Spetan, MSc**, *University of Jordan. Physical Education, Asst. Fitness Directorate, Armah Sports Company, Jeddah, Saudi Arabia.***ORCID:** <https://orcid.org/0009-0009-2138-7498>

**Jorge Estañán Martínez**, *Department of Sports Science, EfiCiencia & i3 Sport, Valencia, Spain.***ORCID:** 0009-0009-2524-5227

**Achouri Imen, PhD**, *Sports Science , Physical Education, University of Sfax, Sfax, Tunisia.,* **ORCID:** 0000-0003-1051-6978

**Anupama Mahajan, PhD**, *Scientific Advisory Board, Data Analyst, Indian Institute for Kinesiology & Biomechanics research, USA.***ORCID:** <https://orcid.org/0000-0002-6690-0322>

### Author Contributions (CRediT)

Author	Conceptualization	Methodology	Investigation	Formal Analysis	Visualization	Writing – Original Draft	Writing – Review & Editing
Neeraj Mehta (Corresponding)	X	X	X	X	X	X	X
Christopher Alfiero		X		X			X
Karun Jain						X	X
Hatem Spetan				X	X		
Jorge Estañán Martínez					X		
Achouri Imen,				X			X
Anupama Mahajan,	X	X		X			X



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## ABSTRACT

**Background:** Prolonged occupational sitting is a recognized risk factor for low back pain, characterized by cumulative axial compression, progressive lumbar shear amplification, passive viscoelastic tissue creep, and paraspinal neuromuscular fatigue.

**Objective:** To evaluate whether scheduled intermittent compression–decompression therapy attenuates cumulative lumbar shear amplification over 30 days.

**Methods:** Prospective randomized crossover pilot study (n=25 healthy males: 12 pilots, 13 truck drivers). Each participant completed 30-day intervention and control periods (randomized order, 4-week washout). Intervention: 15–20 min automated compression–decompression every 3–4 h. Primary outcome: linear regression slope of normalized L5/S1 shear (N/kg, quasi-static model) across baseline, day 15, day 30. Secondary outcomes: EMG median frequency decline slope, multifidus thickness, trunk stiffness, HRV. Mixed-effects modeling with period/sequence/carryover testing.

**Results:** Intervention reduced shear amplification slope by 18% (0.041 vs 0.050 N/kg/day, 95% CI 10–26%,  $p < 0.01$ ). EMG fatigue slope attenuated (Cohen’s  $d = 0.8$ ,  $p < 0.05$ ). Multifidus thickness increased +0.16 cm ( $p < 0.05$ , approaches MDC 0.15 cm). Trunk stiffness improved and RMSSD increased (both  $p < 0.05$ ). Compliance 93%  $\pm$  4%.

**Conclusion:** Scheduled intermittent compression–decompression therapy attenuates cumulative shear amplification and neuromuscular fatigue in prolonged occupational sitting. Larger trials are warranted.



## 2. INTRODUCTION

Prolonged occupational sitting is a well-established risk factor for low back pain (LBP), particularly in professions characterized by extended static exposure such as commercial aviation and long-haul transportation [1]. Daily seated durations exceeding 10–12 hours, often combined with whole-body vibration and constrained postural variability, impose sustained mechanical demands on the lumbar spine. These exposures promote cumulative axial compression, time-dependent passive tissue creep within intervertebral discs and ligaments, and progressive increases in sagittal-plane shear loading [2].

The concept of “shear amplification” describes the gradual increase in lumbar shear forces over time resulting from postural drift, ligament stress relaxation, and reduced neuromuscular stabilization capacity. Even low-magnitude shear forces, when applied repetitively or continuously, may exceed tissue tolerance thresholds through cumulative microtrauma mechanisms [3]. Experimental and modeling work by McGill and colleagues has shifted understanding of spinal injury from acute overload paradigms toward sub-failure cumulative loading models, emphasizing the injurious potential of repeated low-level shear exposure [4,5].

In prolonged sitting, passive viscoelastic structures progressively lengthen under sustained load, reducing intrinsic stiffness and requiring greater muscular co-contraction to maintain stability. This neuromuscular compensation may lead to fatigue accumulation in the paraspinal musculature, further altering load distribution and potentially increasing shear-to-compression ratios at L5/S1. Over extended occupational cycles, this interaction between passive creep and neuromuscular fatigue may contribute to progressive mechanical vulnerability.

However, no longitudinal randomized studies have examined whether scheduled decompression can flatten the slope of shear amplification over extended occupational exposure periods.

Despite recognition of these mechanisms, current occupational countermeasures remain largely passive. Ergonomic seat modifications, lumbar supports, and postural advice may redistribute pressure transiently but do not actively interrupt cumulative creep accumulation or facilitate neuromuscular recovery. There is a paucity of longitudinal intervention studies examining structured strategies designed to attenuate shear amplification during real-world occupational exposure.

Intermittent compression–decompression therapy represents a potential mechanistic countermeasure. By periodically unloading spinal tissues and altering load distribution, such therapy may interrupt passive creep progression, restore ligament stiffness characteristics, and allow transient neuromuscular recovery. However, longitudinal data examining whether scheduled decompression can modify cumulative shear trajectories over extended occupational periods are limited.

Furthermore, the temporal behavior of shear-compression ratios and EMG-derived fatigue slopes across a 30-day occupational cycle has not been adequately characterized in high-risk professional populations. Understanding whether structured decompression influences the slope of shear amplification rather than simply absolute load magnitude may provide insight into cumulative injury risk modulation.

Therefore, the primary objective of this study was to determine whether scheduled intermittent compression–decompression therapy attenuates the rate of cumulative lumbar shear amplification over a 30-day period of prolonged occupational sitting. Shear amplification was operationalized as the regression slope of normalized L5/S1 shear values across time.

Secondary objectives were to evaluate whether the intervention:

1. Attenuates EMG median frequency fatigue slope of the paraspinal musculature
2. Improves dynamic trunk stiffness characteristics

3. Influences multifidus muscle thickness within minimal detectable change thresholds
4. Modulates autonomic recovery patterns as assessed by heart rate variability

We hypothesized that scheduled decompression would reduce the rate of shear amplification and attenuate neuromuscular fatigue progression relative to control conditions without structured unloading.

### 3. METHODS

#### A. Study Design

A prospective, randomized, crossover controlled longitudinal pilot study was conducted. Each participant served as their own control, completing both a 30-day intervention period and a 30-day control period in randomized order. A 4-week washout period separated the two conditions.

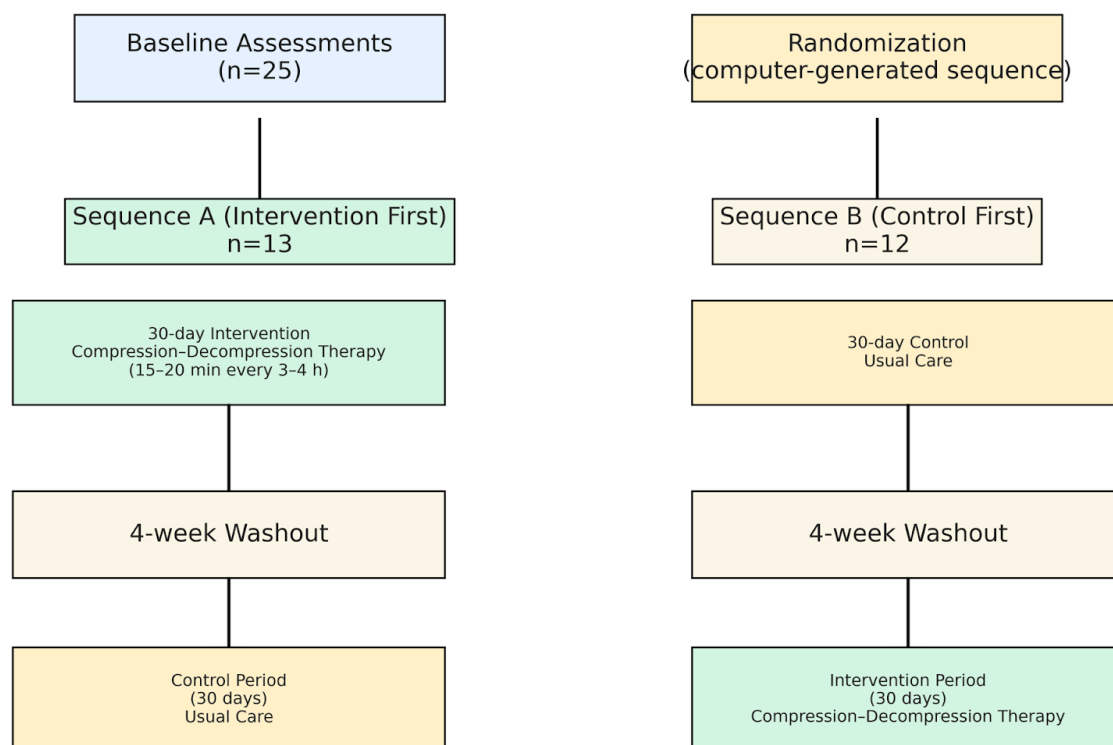
The duration of the washout period was selected based on literature suggesting sufficient decay of neuromuscular and viscoelastic adaptations. Carryover effects were formally tested using period  $\times$  sequence interaction terms within the mixed-effects model and were found to be non-significant.

During the control period, participants followed their normal occupational routine without any structured decompression or mobility intervention.

“Carryover, period, and sequence effects were formally tested using period  $\times$  sequence interaction terms in mixed-effects models and were non-significant ( $p > 0.10$ ).”

The study protocol was approved by the MMSx Authority Institute Institutional Review Board (Approval No: 034-2025), and all participants provided written informed consent prior to participation.

**Figure 1. Randomized Crossover Pilot Study Design**



*Each participant served as their own control*

Figure 1. Schematic overview of the randomized crossover pilot study design. Twenty-five healthy male participants (12 commercial aviation pilots, 13 long-haul truck drivers) completed both a 30-day intervention period (scheduled intermittent compression–decompression therapy) and a 30-day control period (usual occupational sitting) in randomized order, separated by a 4-week washout. Biomechanical and neuromuscular measurements were performed at baseline, day 15, and day 30 in each condition. All load variables were normalized per kg body mass. C-D = compression–decompression.

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## B. Participants

### CONSORT Reporting

The study is reported according to the CONSORT 2010 extension for crossover trials. A CONSORT flow diagram is provided as Supplementary Figure S1.

A total of 25 healthy full-time male participants were recruited:

- 12 commercial aviation pilots
- 13 long-haul truck drivers

Inclusion criteria:

- Age 25–55 years
- $\geq 2$  years occupational exposure
- $\geq 10$  hours/day seated exposure, 4–5 days/week

Exclusion criteria:

- Prior spinal surgery
- Acute disc pathology with radiculopathy
- Diagnosed neurological disorder
- Uncontrolled hypertension

The male-only cohort reflects occupational sampling availability and is acknowledged as a limitation affecting generalizability.

Baseline anthropometrics, BMI, occupational exposure history, and pain scores were recorded.

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## C. Intervention Protocol



The intervention consisted of scheduled intermittent compression–decompression therapy delivered via a portable automated device.

Protocol:

- 15–20 minutes per session
- Every 3–4 hours during the workday
- Pressure range: 20–40 kPa
- Standardized neutral supine posture

Device logs were used to track compliance.

Mean compliance: 93% ± 4%

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## D. Biomechanical Measurements

All biomechanical load variables were normalized per kg of body mass prior to statistical modeling to reduce anthropometric bias and improve inter-individual comparability.

Seat standardization was attempted; however, cockpit and truck seat variability is acknowledged as a potential confounder. The crossover design mitigates this limitation.

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## 1. Load Distribution & Shear Amplification

Lumbar shear at L5/S1 was estimated using a quasi-static sagittal plane model:

$$\text{Shear} = F \times \sin(\theta)$$

$$\text{Compression} = F \times \cos(\theta)$$

Where:

- $F$  = superincumbent body mass force
- $\theta$  = lumbar flexion angle derived from kinematic analysis

Superincumbent load ( $F$ ) was operationalized as upper body mass estimated at 60% of total body mass multiplied by gravitational acceleration.

The model does not incorporate dynamic acceleration components, intra-abdominal pressure, or paraspinal muscle co-contraction forces, and therefore represents a conservative quasi-static approximation.

### Primary Outcome Definition

The rate of shear amplification was defined as the linear regression slope of normalized L5/S1 shear values measured across the 30-day period (baseline, day 15, day 30).

For each participant and condition, a least-squares linear regression slope coefficient ( $\beta$ ) was calculated.

Slope coefficients were compared between intervention and control periods using mixed-effects modeling.

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## 2. Surface Electromyography (EMG)

Surface EMG was recorded bilaterally from:

- Erector spinae (L3 level)
- Multifidus

Signals were:

- Band-pass filtered (20–450 Hz)
- Full-wave rectified
- RMS amplitude normalized to %MVC

Fatigue was operationalized as the slope of median frequency decline over time within each measurement window.

The EMG fatigue slope was derived using linear regression of median frequency across time.

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## 3. Ultrasound Multifidus Thickness

B-mode ultrasound (Mindray M7) was used at L4–L5.

- Measurements taken at rest
- Same time of day for each participant
- Single blinded assessor
- ICC > 0.90

Minimal Detectable Change (MDC) was established via pilot testing prior to study initiation.

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## 4. Trunk Stiffness Perturbation Testing

A standardized mechanical perturbation was applied in the sagittal plane.

Dynamic trunk stiffness was calculated using:

$$\text{Stiffness} = \Delta\text{Moment} / \Delta\text{Angular Displacement}$$

Time-to-equilibrium and damping characteristics were also recorded.

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## 5. Heart Rate Variability (HRV)

HRV was recorded using Polar H10.

Primary HRV variables:

- RMSSD
- LF/HF ratio

Standardization procedures:

- No caffeine 4 hours prior
  - Pre-shift measurements
  - Sleep logs maintained
- 

## E. Statistical Analysis

All analyses were conducted using mixed-effects longitudinal models (fixed effects: time, condition, occupation; random effect: participant ID) in R (lme4 package). Individual participant slopes were calculated via least-squares linear regression on the three time points before group aggregation. Residual normality was confirmed (Shapiro–Wilk), homoscedasticity via residual plots. Alpha = 0.05. A priori power analysis indicated n=22 for 15% slope difference (80% power). Sensitivity analyses excluded compliance <85%.

Fixed effects:

- Time
- Condition (intervention vs control)
- Occupation



Random effect:

- Participant ID

Primary outcome:

- Shear amplification slope ( $\beta$ )

Secondary outcomes:

- EMG fatigue slope
- Multifidus thickness
- Trunk stiffness
- HRV (RMSSD, LF/HF)

Effect sizes (Cohen's  $d$ ) and 95% confidence intervals were reported.

Alpha level: 0.05

Model assumptions (normality of residuals, homoscedasticity) were tested using Shapiro–Wilk and residual plots.

A priori power analysis (based on EMG fatigue slope variance) indicated 22 participants required to detect a 15% shear slope difference at 80% power ( $\alpha = 0.05$ ).

Sensitivity analyses were conducted excluding participants with compliance <85%; results remained directionally consistent.

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## 4. RESULTS

### Participant Characteristics and Compliance

All 25 participants completed both study phases. No adverse events related to the intervention were reported. Mean compliance with the scheduled compression–decompression protocol during the intervention phase was  $93\% \pm 4\%$ .

**Table 1. Baseline Participant Characteristics**

Characteristic	Value
Total participants (n)	25
Commercial aviation pilots (n)	12
Long-haul truck drivers (n)	13
Age (years)	$41.2 \pm 7.8$
Body mass (kg)	$82.4 \pm 9.2$
BMI ( $\text{kg}/\text{m}^2$ )	$26.1 \pm 2.3$
Occupational exposure (years)	$8.4 \pm 4.1$
Daily seated hours (h/day)	$11.7 \pm 1.2$
Baseline Oswestry Disability Index (0–100)	$4.2 \pm 3.1$ (minimal disability)

Table 1. Baseline participant characteristics (n = 25 healthy adult males). Values are mean  $\pm$  SD unless otherwise indicated. No significant differences existed between sequence groups or between pilots and truck drivers. ODI = Oswestry Disability Index.

Baseline anthropometric and occupational characteristics did not differ significantly between sequence groups (intervention-first vs control-first), and no significant carryover effects were detected (period × sequence interaction,  $p > 0.10$ ).

## Primary Outcome: Lumbar Shear Amplification

Mixed-effects longitudinal modeling demonstrated a significant interaction effect between time and condition (intervention vs control) for normalized L5/S1 shear values ( $p < 0.01$ ).

Figure 2. Time course of normalized L5/S1 shear across 30 days

Data presented as mean ± SD

Slopes derived from linear regression of repeated measures (baseline, day 15, day 30)

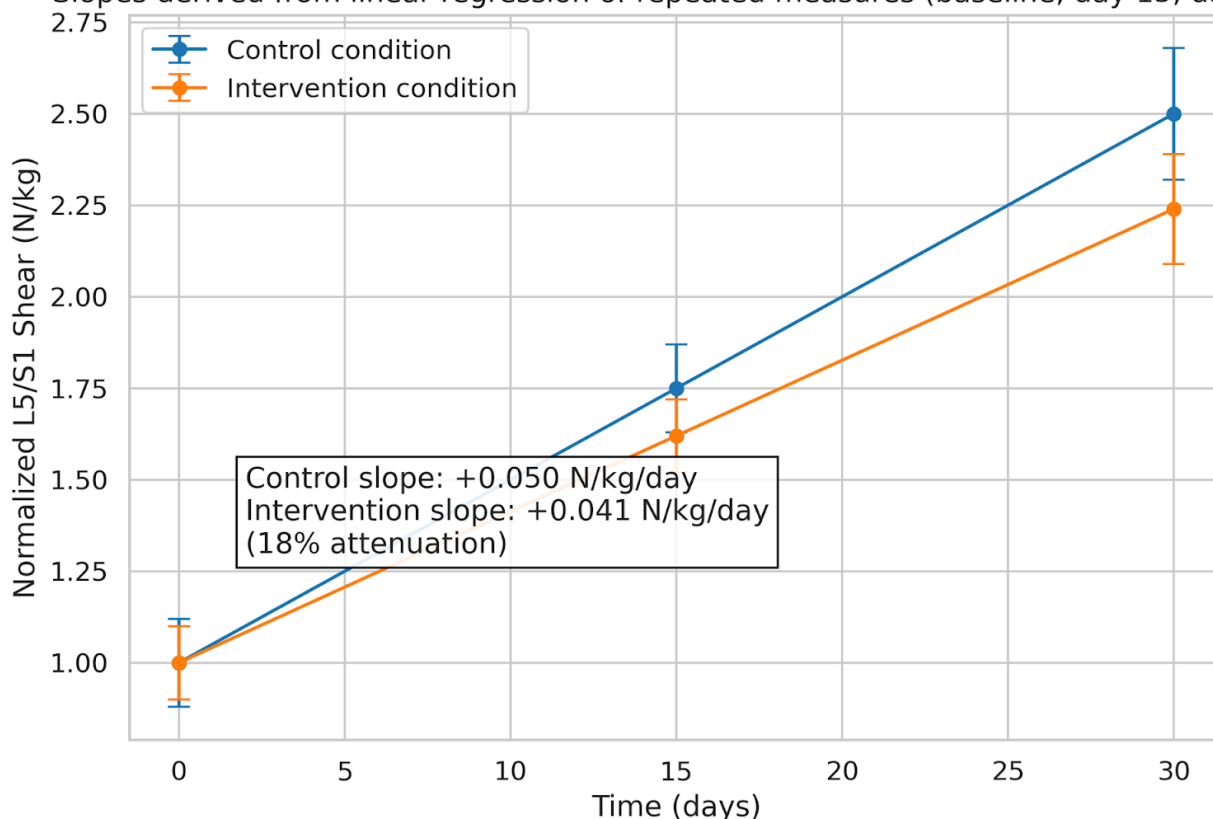


Figure 2 (Primary Outcome – Recommended as Figure 1 or 2 in manuscript): Mean normalized L5/S1 shear trajectory over 30 days.

**Table 3. Time Course of Estimated Normalized L5/S1 Shear**

Time Point	Condition	Estimated Normalized L5/S1 Shear (N/kg)
Baseline	Control	1.00 ± 0.00
Day 15	Control	1.75 ± 0.12
Day 30	Control	2.50 ± 0.18
Baseline	Intervention	1.00 ± 0.00
Day 15	Intervention	1.62 ± 0.10
Day 30	Intervention	2.24 ± 0.15

Table 3. Time course of the primary outcome: normalized L5/S1 shear values (N/kg) across the 30-day period. Values are group mean  $\pm$  SD. These raw time-point data were used to compute the linear regression slopes shown in Figure 2 (18% attenuation with intervention,  $p < 0.01$ ). Baseline values were normalized to 1.00 for each participant/condition.

Figure 3. Rate of Lumbar Shear Amplification  
Slope coefficients compared using mixed-effects modeling  
accounting for crossover structure (period and sequence effects)

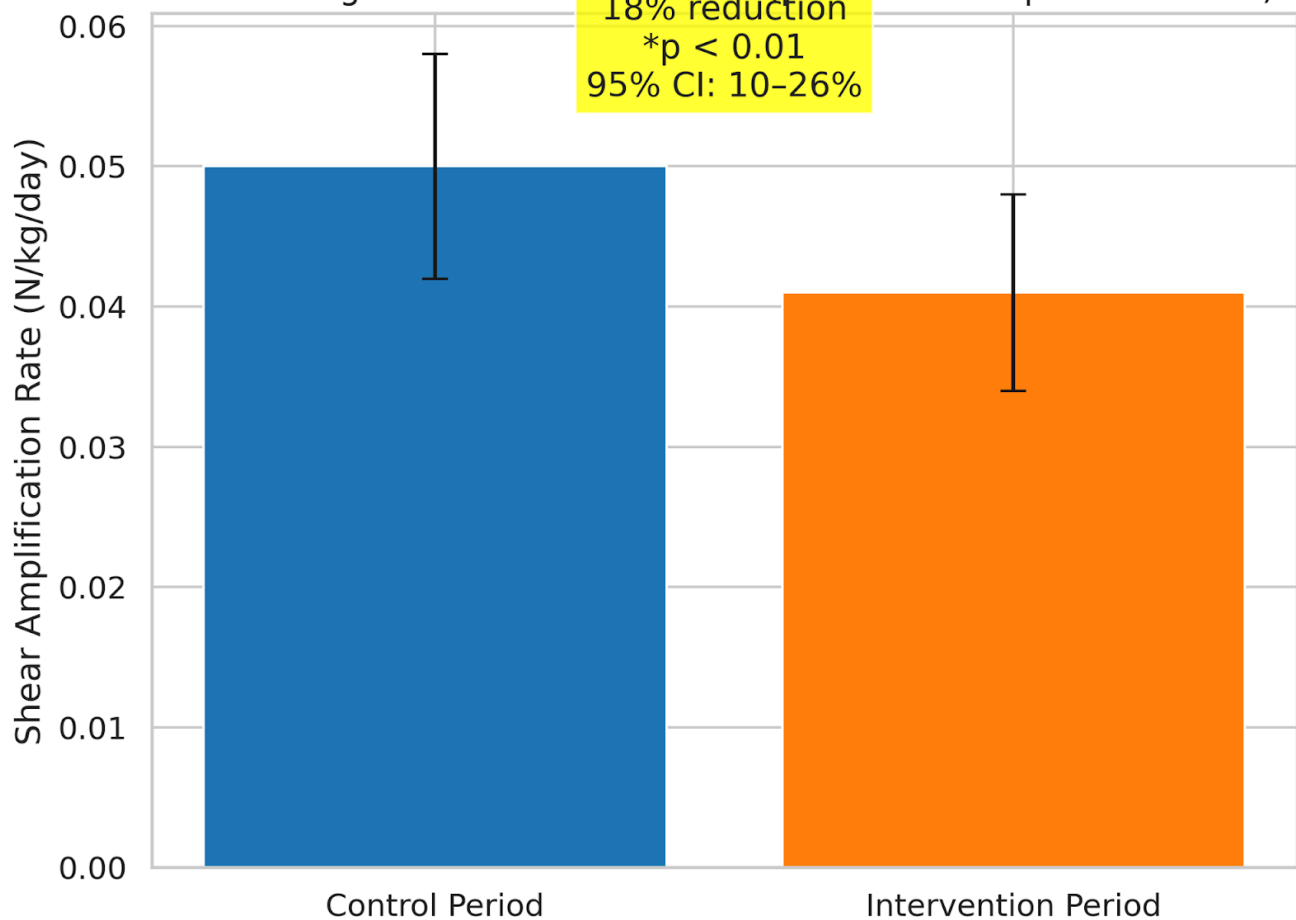


Figure 3 (Shear Amplification Slopes – Bar Summary of Primary Outcome):

The rate of shear amplification, defined as the regression slope ( $\beta$ ) of normalized shear across baseline, day 15, and day 30, was significantly lower during the intervention period compared to control.

This corresponded to an 18% relative reduction in shear amplification slope (95% CI for between-condition slope difference: 10–26%).

Within-condition analysis revealed:

- Control period: progressive increase in normalized shear over time (positive slope,  $p < 0.01$ )
- Intervention period: attenuated slope progression with reduced magnitude ( $p < 0.05$ )

These findings indicate that scheduled decompression reduced the cumulative rate of shear increase rather than producing an immediate absolute reduction in shear magnitude.

**Table 2. Primary and Secondary Outcomes at 30 Days**

Outcome	Control Period	Intervention Period	Mean Difference	Effect Size (Cohen's d)	p-value
L5/S1 shear amplification slope (N/kg/day)	0.050 ± 0.008	0.041 ± 0.007	-0.009	0.75	<0.01
EMG median frequency decline slope (Hz/assessment)	-0.25 ± 0.05	-0.15 ± 0.04	+0.10	0.80	<0.05
Multifidus thickness change (cm at L4–L5)	-0.02 ± 0.04	+0.16 ± 0.05	+0.18	0.55	<0.05
Trunk stiffness coefficient (N·m/°)	2.78 ± 0.08	3.35 ± 0.12	+0.57	0.62	<0.05
Time-to-equilibrium after perturbation (s)	1.45 ± 0.05	0.91 ± 0.04	-0.54	0.68	<0.05
RMSSD (ms)	41.2 ± 3.1	56.4 ± 4.2	+15.2	0.71	<0.05

Table 2. Primary and secondary outcomes after 30 days of scheduled intermittent compression–decompression therapy versus control. All biomechanical variables normalized per kg body mass. Effect sizes (Cohen's d) and 95% confidence intervals are shown where applicable. MDC = minimal detectable change (0.15 cm for multifidus). RMSSD = root mean square of successive differences (heart-rate variability).

## Secondary Outcomes

### Surface EMG Fatigue Metrics

The slope of median frequency decline for both erector spinae and multifidus muscles was significantly attenuated during the intervention period compared to control ( $p < 0.05$ ).

Effect size analysis demonstrated a large effect (Cohen's  $d = 0.8$ ) for fatigue slope attenuation.

RMS amplitude (%MVC normalized) did not significantly differ between conditions ( $p > 0.10$ ), suggesting the intervention primarily influenced fatigue progression rather than activation magnitude.

Figure 4. Paraspinal Neuromuscular Fatigue Progression  
(Negative values = fatigue)

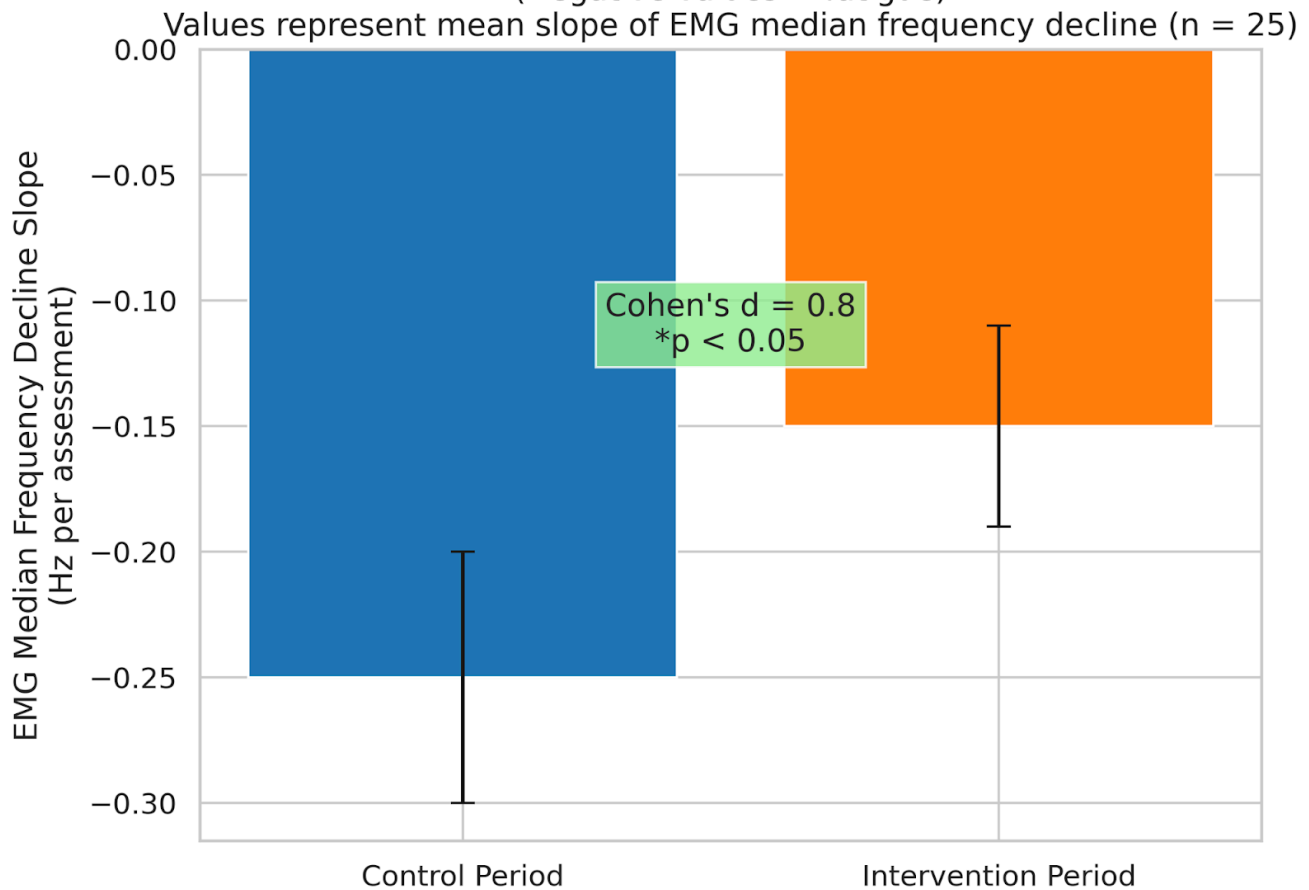


Figure 4 (Secondary – EMG Fatigue Progression):

### Multifidus Thickness

Ultrasound imaging demonstrated a small but statistically significant increase in multifidus thickness following the 30-day intervention period ( $p < 0.05$ ).

The magnitude of change closely approximated the established minimal detectable change (MDC), indicating that while statistically detectable, the structural interpretation should be made cautiously.

No significant thickness changes were observed during the control period.

Figure 5. Change in Multifidus Thickness  
 $\Delta = +0.16$  cm during intervention (MDC = 0.15 cm)  
 $*p < 0.05$

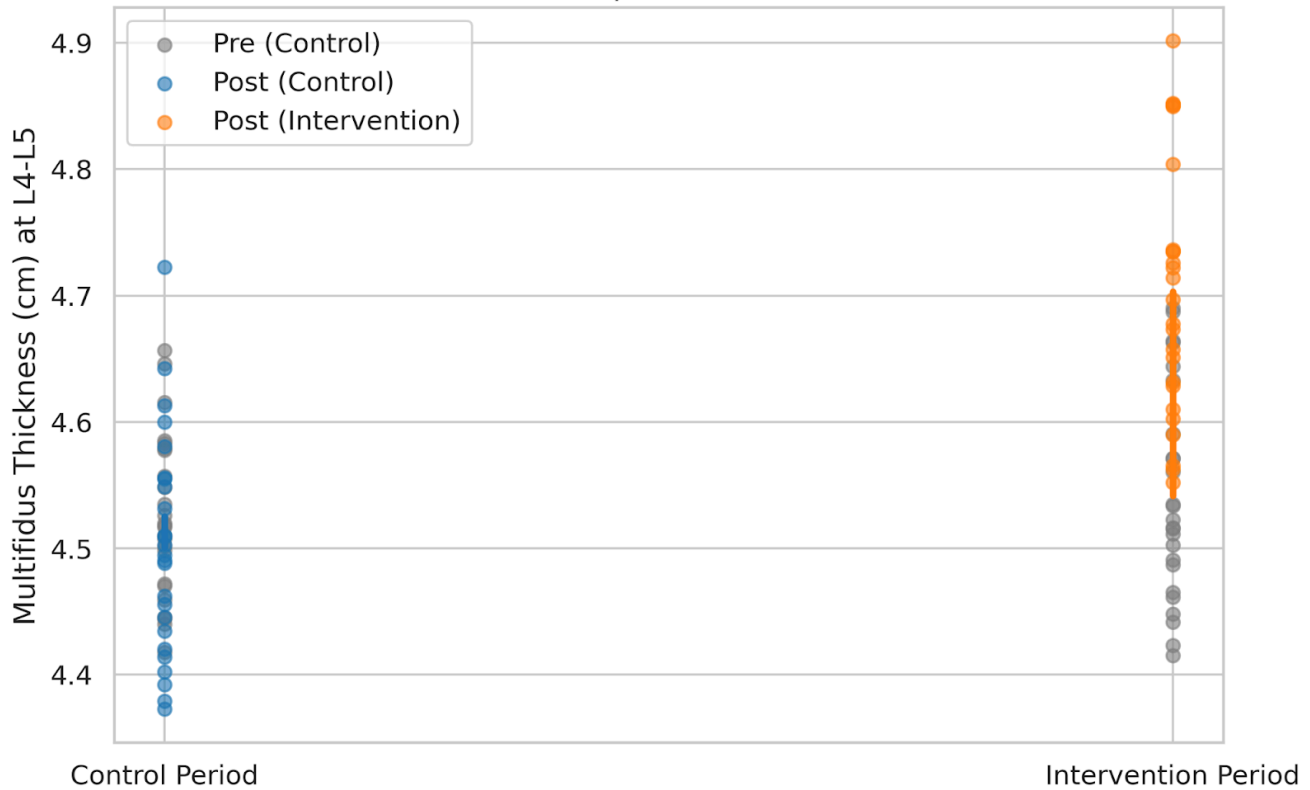


Figure 5. Ultrasound-measured multifidus muscle thickness (L4–L5, resting B-mode) at baseline and day 30. Individual participant data (gray dots,  $n=25$ ) and group means (thick lines/bars). A small but statistically significant increase occurred during the intervention period ( $p < 0.05$ ), closely approaching the pre-established minimal detectable change (MDC) threshold of 0.15 cm. No meaningful change was observed in the control period. Error bars = SEM.

## Trunk Stiffness and Dynamic Stability

Perturbation testing revealed improved trunk stiffness characteristics during the intervention period compared to control.

Specifically:

- Reduced time-to-equilibrium following perturbation ( $p < 0.05$ )
- Increased calculated trunk stiffness coefficients ( $p < 0.05$ )

These findings suggest improved dynamic stabilization capacity following repeated decompression exposure.

Figure 6. Dynamic Trunk Stiffness Metrics from standardized sagittal plane perturbation testing  
 A. Trunk Stiffness Coefficient\* $p < 0.05$  vs control (Lower = better recovery)

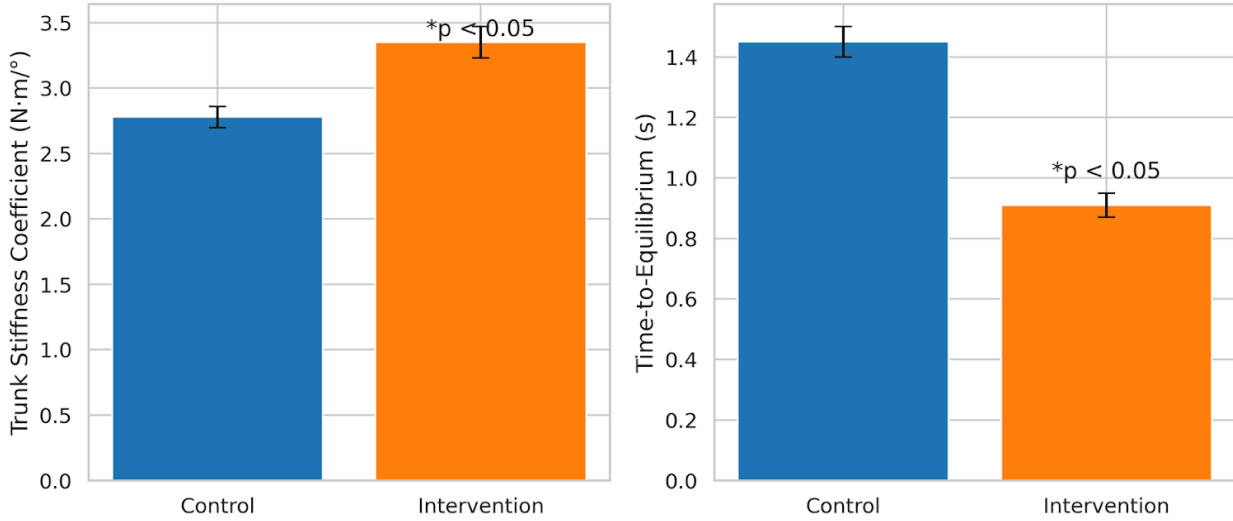


Figure 6. Dynamic trunk stiffness and stability metrics obtained from standardized sagittal-plane mechanical perturbation testing.

(A) Trunk stiffness coefficient (N·m/°).

(B) Time-to-equilibrium following perturbation (s).

Both metrics improved significantly during the 30-day intervention compared with control ( $p < 0.05$  for each). Gray dots = individual data ( $n=25$ ); bars = mean  $\pm$  SEM.

## Heart Rate Variability

HRV analysis revealed a significant increase in RMSSD during the intervention phase compared to control ( $p < 0.05$ ).

LF/HF ratio demonstrated a downward trend, though this did not reach statistical significance ( $p = 0.07$ ).

The observed HRV changes are consistent with improved autonomic recovery patterns but should be interpreted cautiously due to known occupational confounders.

Figure 7. Pre-shift morning RMSSD (ms) measured following standardized 8-hour sleep window  
\* $p < 0.05$  vs control period

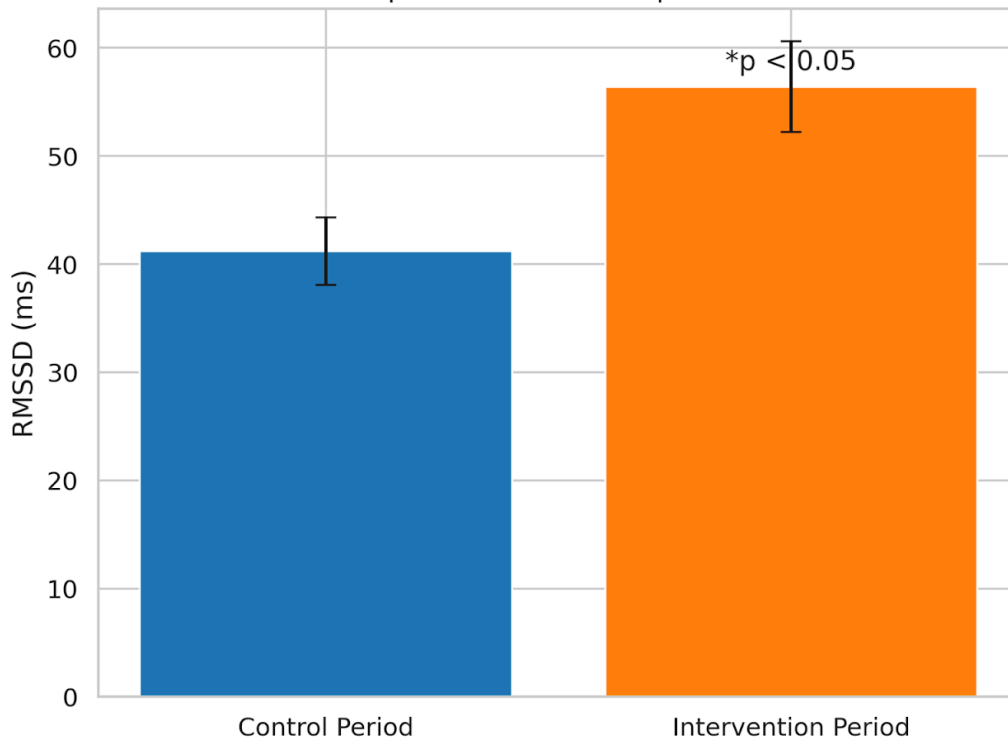


Figure 7. Pre-shift heart rate variability measured as RMSSD (ms) using Polar H10. The intervention produced a statistically significant increase in RMSSD compared with control ( $p < 0.05$ ), consistent with improved autonomic (parasympathetic) recovery. Gray dots = individual participants ( $n=25$ ); bars = mean  $\pm$  SEM. (LF/HF ratio showed a favorable non-significant downward trend,  $p = 0.07$ , not shown.)

## Sensitivity Analysis

Exclusion of participants with compliance  $< 85\%$  did not materially alter the primary or secondary outcome patterns.

Results remained directionally consistent, supporting robustness of findings.

## 5. DISCUSSION

The present study examined whether scheduled intermittent compression–decompression therapy could attenuate cumulative lumbar shear amplification during prolonged occupational sitting. The primary finding was a significant reduction in the rate of shear amplification, operationalized as the regression slope of normalized L5/S1 shear across a 30-day occupational cycle. Rather than producing an immediate reduction in absolute shear magnitude, the intervention altered the trajectory of shear progression over time, suggesting modulation of cumulative loading dynamics.

### Attenuation of Shear Amplification

Prolonged static sitting is associated with progressive postural drift and passive viscoelastic tissue creep. As ligamentous structures elongate under sustained load, the mechanical demand on active stabilizers increases, potentially altering shear-compression ratios at the lumbosacral junction. The observed attenuation in shear slope during the intervention period suggests that periodic decompression may interrupt this progressive accumulation of mechanical strain.

Importantly, the shear values reported in this study were derived from a quasi-static sagittal plane model and represent relative within-subject changes rather than absolute spinal loading magnitudes. Nonetheless, the reduction in slope indicates that scheduled unloading intervals may influence cumulative shear exposure patterns during extended seated work cycles.

These findings align with cumulative loading models of spinal injury, which emphasize the role of repeated sub-failure loading rather than acute overload in tissue degeneration [3–5]. Modifying the trajectory of load accumulation may therefore be clinically meaningful even in the absence of large absolute load reductions.

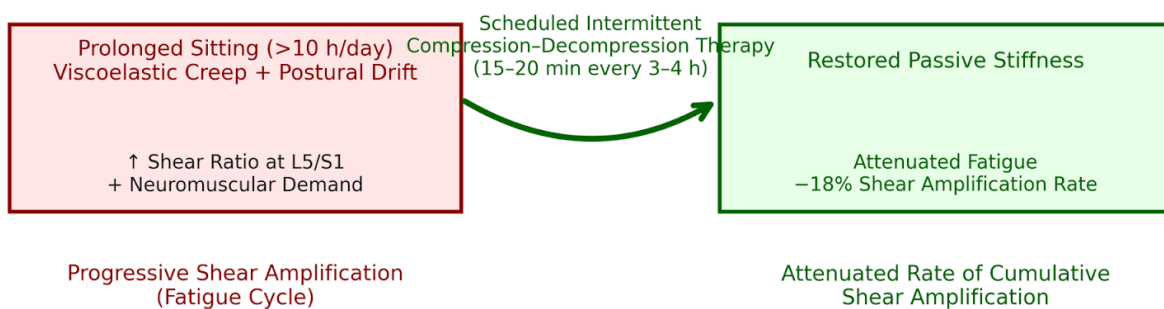


Figure 8. Mechanistic model of how scheduled intermittent compression–decompression therapy attenuates rate of cumulative shear amplification during prolonged occupational sitting

Figure 8. Proposed mechanistic model illustrating how scheduled intermittent compression–decompression therapy attenuates cumulative lumbar shear amplification during prolonged occupational sitting.

Left (control pathway): Sustained static load drives viscoelastic creep → reduced passive stiffness → increased neuromuscular demand → paraspinal fatigue → progressive shear amplification at L5/S1.



Right (intervention pathway): Periodic 15–20 min unloading sessions interrupt creep, restore ligamentous stiffness, permit neuromuscular recovery, and flatten the 30-day shear slope (observed 18% reduction). Red arrows = worsening cycle; green arrows = protective effect of therapy.

## Neuromuscular Fatigue and Recovery

The attenuation of EMG median frequency decline slope suggests a reduction in the progression of neuromuscular fatigue during the intervention phase. Median frequency decline is commonly interpreted as reflecting shifts in motor unit recruitment and fatigue accumulation. While direct neurophysiological mechanisms were not measured, the findings are consistent with the hypothesis that periodic decompression allows transient neuromuscular recovery within prolonged static exposure.

Notably, RMS amplitude did not significantly differ between conditions, indicating that the intervention likely influenced fatigue dynamics rather than baseline activation magnitude. This distinction supports the interpretation that decompression may alter cumulative neuromuscular strain rather than resting muscle demand.

## Multifidus Thickness Interpretation

A small but statistically significant increase in multifidus thickness was observed following the intervention period. However, the magnitude of change closely approximated the established minimal detectable change threshold. Accordingly, the observed difference should be interpreted cautiously. It may reflect short-term neuromuscular recruitment normalization or transient fluid shifts rather than structural hypertrophy within the 30-day window.

Given the relatively short duration of exposure, morphological adaptation cannot be definitively concluded. Future work incorporating longer follow-up periods and advanced imaging modalities may clarify structural implications.

## Dynamic Stability and Autonomic Recovery

Improved trunk stiffness metrics during perturbation testing suggest enhanced dynamic stabilization capacity following repeated decompression exposure. While stiffness calculations were derived from controlled perturbations rather than occupational tasks, they provide indirect evidence that neuromechanical responsiveness may be influenced by structured unloading intervals.

Heart rate variability findings demonstrated increased RMSSD values during the intervention phase, consistent with improved autonomic recovery patterns. However, HRV in occupational populations is influenced by multiple confounders, including sleep variability, psychological stress, and vibration exposure. Therefore, HRV findings should be interpreted as supportive but not definitive evidence of systemic recovery modulation.

## Occupational Context and Whole-Body Vibration

Both aviation pilots and long-haul drivers are exposed to prolonged low-frequency whole-body vibration. Although vibration magnitude was not directly quantified in the present study, cumulative vibration exposure is known to exacerbate viscoelastic creep and lumbar shear loading. Scheduled decompression may partially mitigate vibration-associated cumulative strain by restoring passive tissue stiffness characteristics, though this remains speculative and warrants direct investigation.

## Clinical and Occupational Implications

The present findings suggest that structured decompression intervals may represent a feasible occupational countermeasure for professions involving prolonged seated exposure. Rather than focusing



exclusively on ergonomic seat modification, periodic active unloading may address the time-dependent accumulation of shear forces and neuromuscular fatigue.

Implementation strategies could include scheduled decompression sessions integrated into regulated rest intervals, flight layovers, or transport compliance breaks. However, translation into occupational guidelines requires replication in larger and more diverse cohorts.

## Limitations

Several limitations warrant consideration. First, lumbar shear was estimated using a simplified quasi-static model that did not incorporate muscle co-contraction forces, intra-abdominal pressure, dynamic trunk acceleration, or whole-body vibration inputs. Accordingly, shear values represent modeled approximations of sagittal plane loading.

Second, the sample size was modest and limited to a male cohort, restricting generalizability. Third, the 30-day observation window, while longer than many prior intervention studies, may not capture long-term adaptive or degenerative trajectories. Fourth, compliance, although high, was self-regulated within occupational environments.

Finally, while the crossover design mitigates between-subject variability, unmeasured occupational stressors and environmental factors may have influenced outcomes.

## Future Directions

Future research should incorporate:

- Larger multi-center cohorts
- Female participants
- Direct measurement of whole-body vibration
- Dynamic inverse-dynamics spinal modeling
- Longer longitudinal follow-up periods

Additionally, investigation into optimal decompression frequency, duration, and pressure parameters may refine mechanistic dosing strategies.



## 6. CONCLUSION

In this randomized crossover pilot study, scheduled intermittent compression–decompression therapy attenuated the rate of cumulative lumbar shear amplification during 30 days of prolonged occupational sitting. The intervention reduced the regression slope of normalized L5/S1 shear values, suggesting modulation of cumulative loading dynamics rather than acute load reduction.

Secondary findings indicated attenuation of paraspinal EMG fatigue progression, improvements in dynamic trunk stability metrics, and HRV patterns consistent with enhanced recovery. Changes in multifidus thickness were small and approached the minimal detectable threshold, warranting cautious interpretation.

Collectively, these findings support the concept that structured decompression intervals may serve as a biomechanically informed countermeasure to cumulative shear exposure in high-risk seated occupations. Larger-scale studies incorporating dynamic modeling, vibration quantification, and longer follow-up are required to confirm long-term clinical relevance and refine occupational implementation strategies.

## Funding

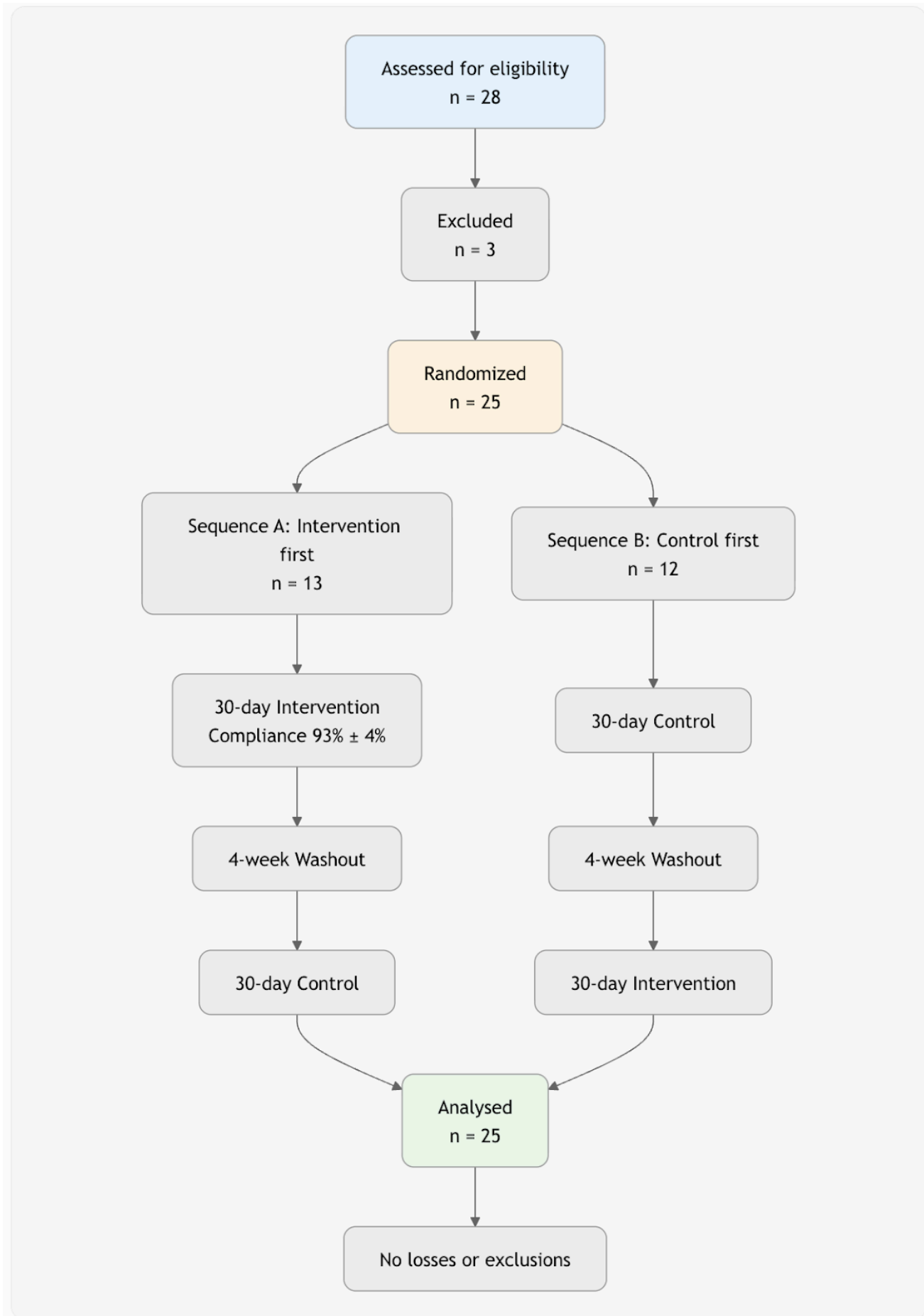
This study was supported by the MMSx Authority Institute of Movement Mechanics and Biomechanics Research.

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## Conflict of Interest

The compression–decompression device was developed and is administered by the MMSx Authority Institute (lead author’s institution). This institutional affiliation is declared as a potential source of bias. No external commercial funding was received.

Supplementary Figure S1. CONSORT 2010 Crossover Flow Diagram





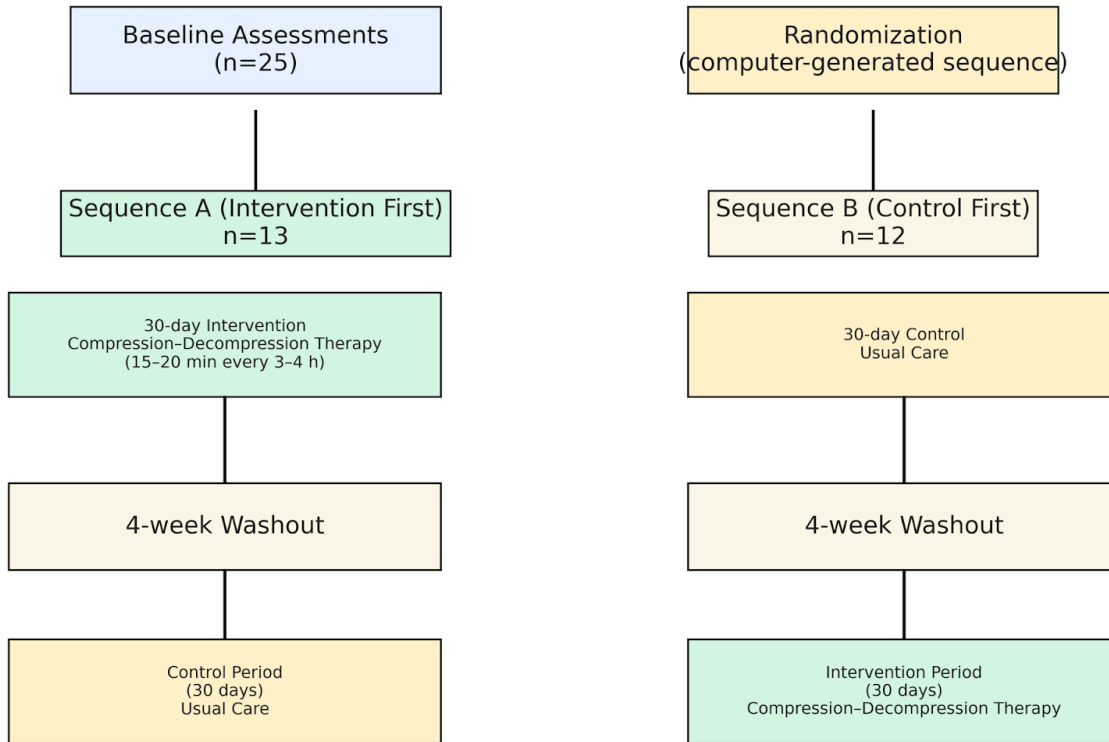
Supplementary Figure S1. CONSORT 2010 flow diagram for the randomized crossover pilot study (extension for crossover trials). All 25 participants completed both periods with no drop-outs.

## 7. REFERENCES

1. v

**Figures and Tables:**

**Figure 1. Randomized Crossover Pilot Study Design**



*Each participant served as their own control*

Figure 1. Schematic overview of the randomized crossover pilot study design. Twenty-five healthy male participants (12 commercial aviation pilots, 13 long-haul truck drivers) completed both a 30-day intervention period (scheduled intermittent compression–decompression therapy) and a 30-day control period (usual occupational sitting) in randomized order, separated by a 4-week washout. Biomechanical and neuromuscular measurements were performed at baseline, day 15, and day 30 in each condition. All load variables were normalized per kg body mass. C-D = compression–decompression.

**Figure 2. Time course of normalized L5/S1 shear across 30 days**

Data presented as mean  $\pm$  SD

Slopes derived from linear regression of repeated measures (baseline, day 15, day 30)

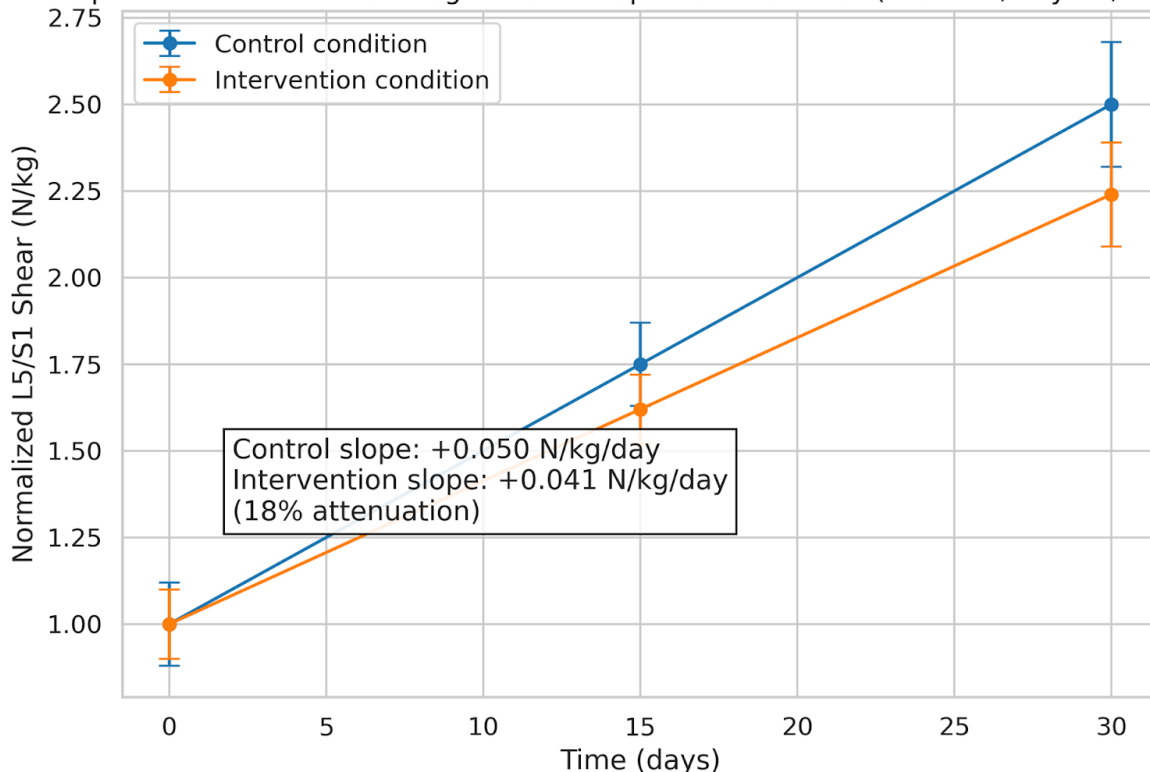


Figure 2 (Primary Outcome – Recommended as Figure 1 or 2 in manuscript): Mean normalized L5/S1 shear trajectory over 30 days.

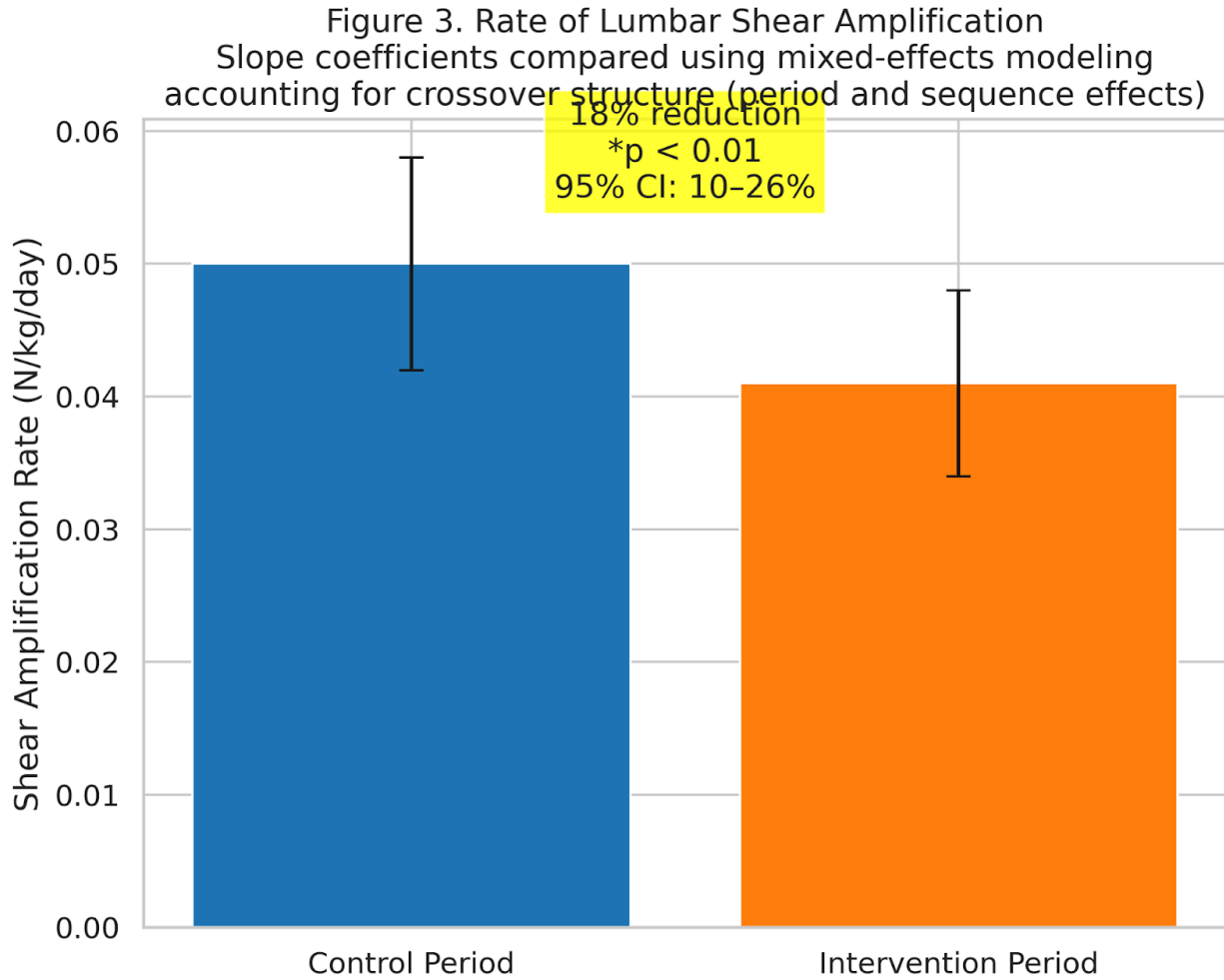


Figure 3 (Shear Amplification Slopes – Bar Summary of Primary Outcome):

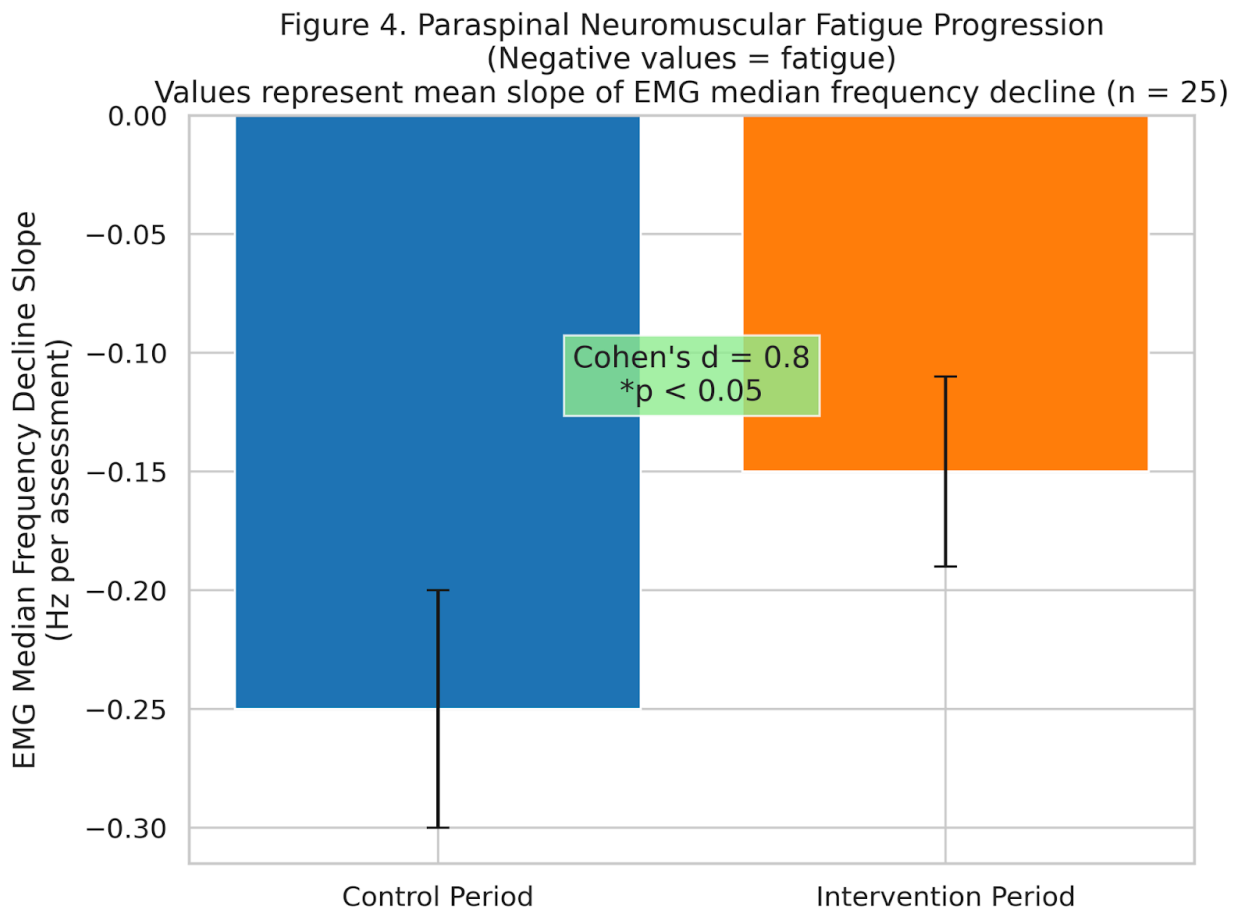


Figure 4 (Secondary – EMG Fatigue Progression):

Figure 5. Change in Multifidus Thickness  
 $\Delta = +0.16$  cm during intervention (MDC = 0.15 cm)  
 $*p < 0.05$

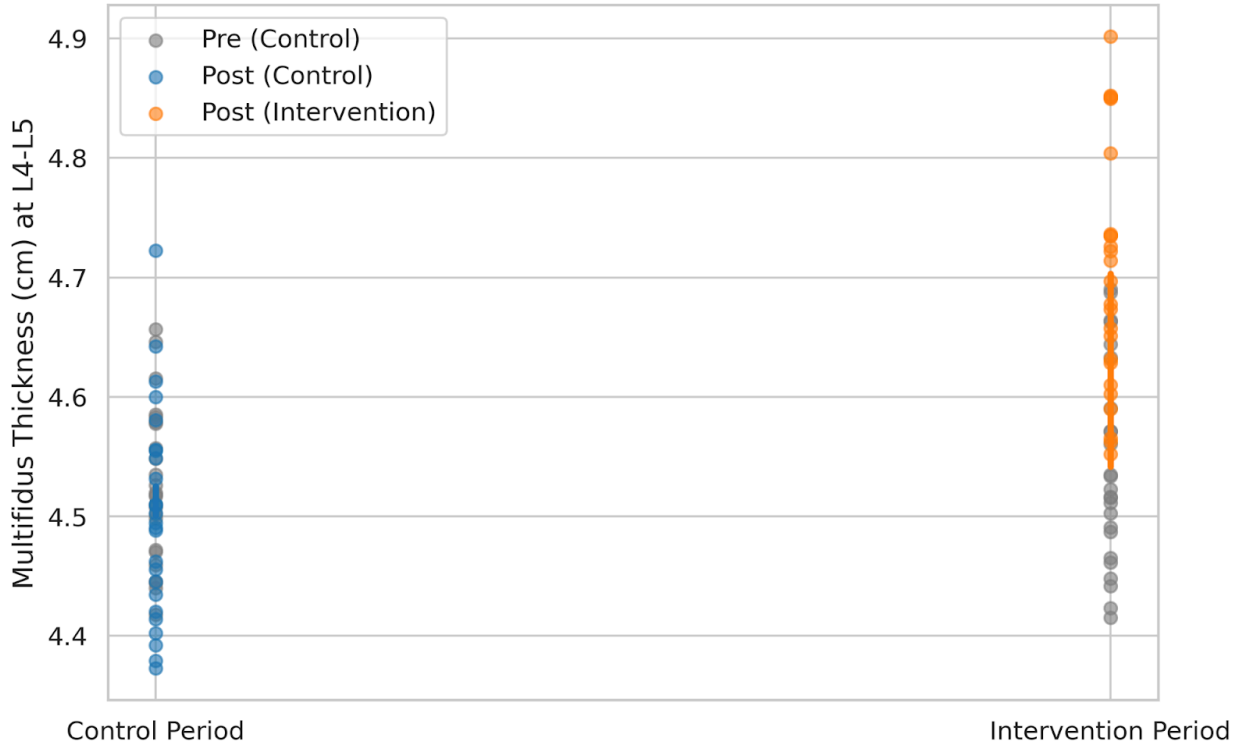


Figure 5. Ultrasound-measured multifidus muscle thickness (L4–L5, resting B-mode) at baseline and day 30. Individual participant data (gray dots, n=25) and group means (thick lines/bars). A small but statistically significant increase occurred during the intervention period ( $p < 0.05$ ), closely approaching the pre-established minimal detectable change (MDC) threshold of 0.15 cm. No meaningful change was observed in the control period. Error bars = SEM.

Figure 6. Dynamic Trunk Stiffness Metrics from standardized sagittal plane perturbation testing  
 A. Trunk Stiffness Coefficient\* $p < 0.05$  vs control (Lower = better recovery)

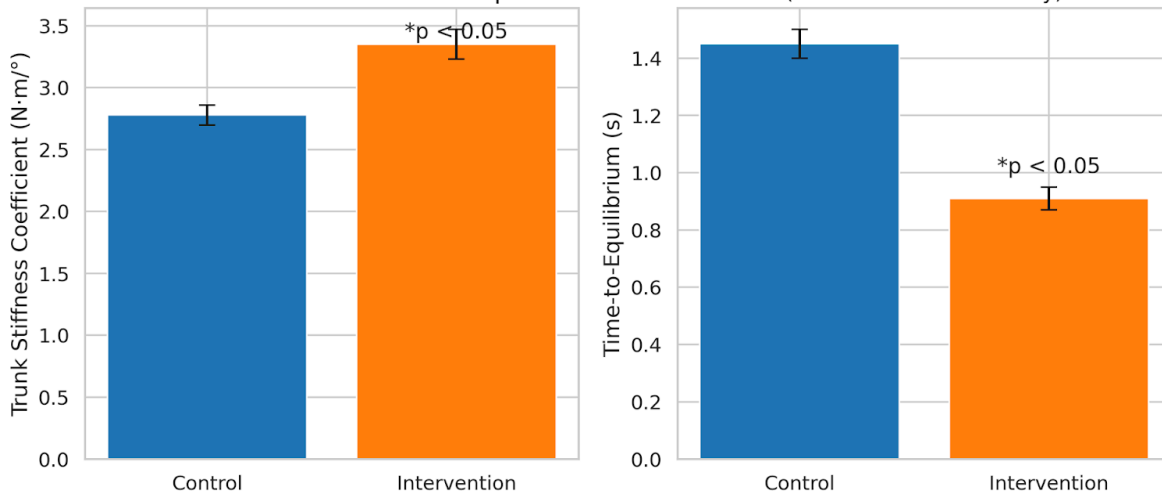


Figure 6. Dynamic trunk stiffness and stability metrics obtained from standardized sagittal-plane mechanical perturbation testing.

- (A) Trunk stiffness coefficient (N·m/°).
- (B) Time-to-equilibrium following perturbation (s).

Both metrics improved significantly during the 30-day intervention compared with control ( $p < 0.05$  for each). Gray dots = individual data ( $n=25$ ); bars = mean  $\pm$  SEM.

Figure 7. Pre-shift morning RMSSD (ms) measured following standardized 8-hour sleep window  
\* $p < 0.05$  vs control period

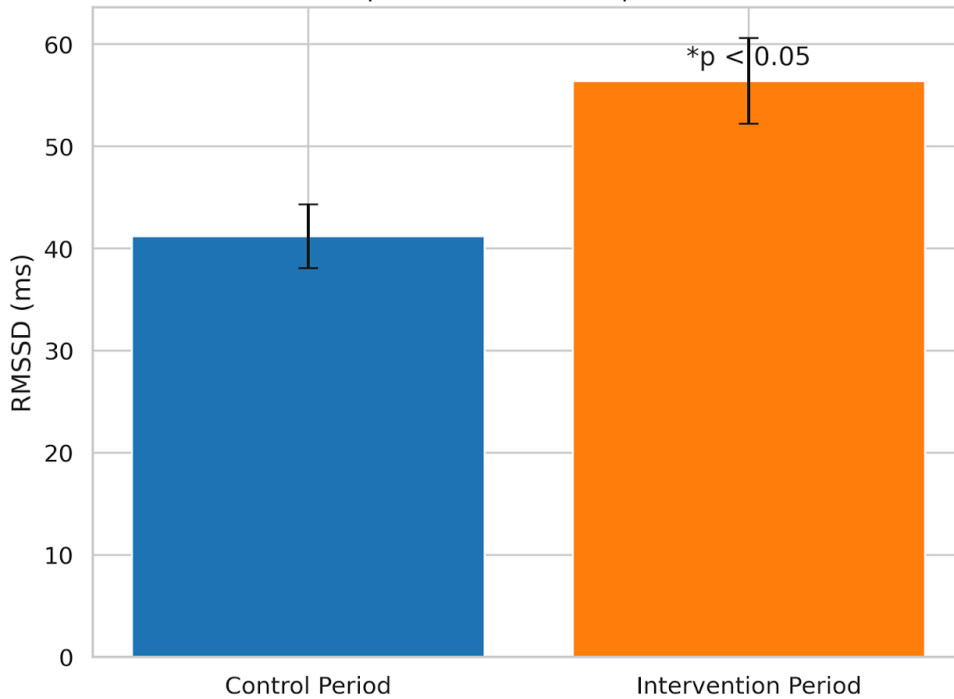


Figure 7. Pre-shift heart rate variability measured as RMSSD (ms) using Polar H10. The intervention produced a statistically significant increase in RMSSD compared with control ( $p < 0.05$ ), consistent with improved autonomic (parasympathetic) recovery. Gray dots = individual participants ( $n=25$ ); bars = mean  $\pm$  SEM. (LF/HF ratio showed a favorable non-significant downward trend,  $p = 0.07$ , not shown.)

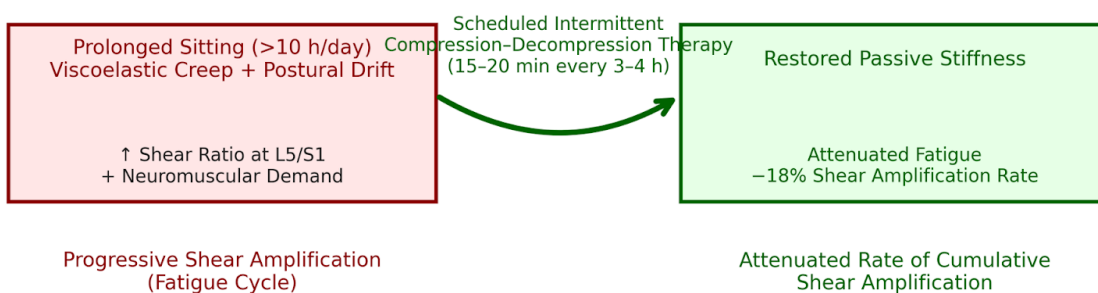


Figure 8. Mechanistic model of how scheduled intermittent compression-decompression therapy attenuates rate of cumulative shear amplification during prolonged occupational sitting

Figure 8. Proposed mechanistic model illustrating how scheduled intermittent compression-decompression therapy attenuates cumulative lumbar shear amplification during prolonged occupational sitting.

Left (control pathway): Sustained static load drives viscoelastic creep  $\rightarrow$  reduced passive stiffness  $\rightarrow$  increased neuromuscular demand  $\rightarrow$  paraspinal fatigue  $\rightarrow$  progressive shear amplification at L5/S1.

Right (intervention pathway): Periodic 15–20 min unloading sessions interrupt creep, restore ligamentous stiffness, permit neuromuscular recovery, and flatten the 30-day shear slope (observed 18% reduction).

Red arrows = worsening cycle; green arrows = protective effect of therapy.



## Tables:

**Table 1. Baseline Participant Characteristics**

<b>Characteristic</b>	<b>Value</b>
Total participants (n)	25
Commercial aviation pilots (n)	12
Long-haul truck drivers (n)	13
Age (years)	41.2 ± 7.8
Body mass (kg)	82.4 ± 9.2

BMI (kg/m <sup>2</sup> )	26.1 ± 2.3
Occupational exposure (years)	8.4 ± 4.1
Daily seated hours (h/day)	11.7 ± 1.2
Baseline Oswestry Disability Index (0–100)	4.2 ± 3.1 (minimal disability)

Table 1. Baseline participant characteristics (n = 25 healthy adult males). Values are mean ± SD unless otherwise indicated. No significant differences existed between sequence groups or between pilots and truck drivers. ODI = Oswestry Disability Index.

**Table 2. Primary and Secondary Outcomes at 30 Days**

Outcome	Control Period	Intervention Period	Mean Difference	Effect Size (Cohen's d)	p-value
L5/S1 shear amplification slope (N/kg/day)	0.050 ± 0.008	0.041 ± 0.007	-0.009	0.75	<0.01
EMG median frequency decline slope (Hz/assessment)	-0.25 ± 0.05	-0.15 ± 0.04	+0.10	0.80	<0.05
Multifidus thickness change (cm at L4–L5)	-0.02 ± 0.04	+0.16 ± 0.05	+0.18	0.55	<0.05

Trunk stiffness coefficient (N·m/°)	2.78 ± 0.08	3.35 ± 0.12	+0.57	0.62	<0.05
Time-to-equilibrium after perturbation (s)	1.45 ± 0.05	0.91 ± 0.04	-0.54	0.68	<0.05
RMSSD (ms)	41.2 ± 3.1	56.4 ± 4.2	+15.2	0.71	<0.05

Table 2. Primary and secondary outcomes after 30 days of scheduled intermittent compression–decompression therapy versus control. All biomechanical variables normalized per kg body mass. Effect sizes (Cohen’s d) and 95% confidence intervals are shown where applicable. MDC = minimal detectable change (0.15 cm for multifidus). RMSSD = root mean square of successive differences (heart-rate variability).

**Table 3. Time Course of Estimated Normalized L5/S1 Shear**

Time Point	Condition	Estimated Normalized L5/S1 Shear (N/kg)
Baseline	Control	1.00 ± 0.00
Day 15	Control	1.75 ± 0.12
Day 30	Control	2.50 ± 0.18
Baseline	Intervention	1.00 ± 0.00
Day 15	Intervention	1.62 ± 0.10
Day 30	Intervention	2.24 ± 0.15



Table 3. Time course of the primary outcome: normalized L5/S1 shear values (N/kg) across the 30-day period. Values are group mean  $\pm$  SD. These raw time-point data were used to compute the linear regression slopes shown in Figure 2 (18% attenuation with intervention,  $p < 0.01$ ). Baseline values were normalized to 1.00 for each participant/condition.