BMC Sports Science, Medicine and Rehabilitation

RESEARCH

Open Access

Effects of high- versus moderate-intensity multimodal whole-body training to improve inspiratory muscle function in persons with chronic nonspecific low back pain

Sim Klaps[1*], Jonas Verbrugghe[1], Nina Goossens[1], Sofie Van Wesemael[1], Bjorn Van Eecke[1], Albère Köke[2,3], Jeanine Verbunt[2,3], Daniel Langer[4,5], Lotte Janssens[1] and Annick Timmermans[1]

Abstract

Background To compare the effects of a multimodal whole-body high-intensity training (HIT) program versus a moderate-intensity training (MIT) program on inspiratory muscle function in persons with chronic nonspecific low back pain (CNSLBP), and to investigate whether changes in inspiratory muscle function relate to changes in disability, pain intensity, anxiety, depression, and exercise capacity.

Methods In this randomized controlled trial, persons with CNSLBP followed a 12-week HIT or MIT program consisting of cardiorespiratory, general resistance, and trunk muscle strength training. Inspiratory muscle function outcomes, assessed pre- and post-intervention, included maximal inspiratory pressure (MIP), inspiratory muscle endurance (IME) time, exercise-induced inspiratory muscle fatigue (IMF), and surface electromyography amplitude of the sternocleidomastoid and scalene muscles during postural control tasks.

Results Sixty-four participants were included (mean age: 44.9 ± 14.5 years, 38 males). $MIP_{predicted}$ increased after both HIT (Δ9.0 ± 9.9%, p= 0.002) and MIT (Δ10.8 ± 13.2%, p< 0.0001), with no significant between-group difference (p= 0.558). IME time increased more after HIT (Δ69.6 ± 78.6s, p< 0.0001) compared to MIT (Δ20.0 ± 60.9s, p= 0.414; p= 0.010 for between-group difference). Exercise-induced IMF and inspiratory muscle activation during postural control did not improve in either group (p> 0.05). Larger increases in MIP related to larger decreases in depressive symptoms (Adj R^2= 0.07, p= 0.047) and smaller increases in exercise capacity (Adj R^2= 0.08, p= 0.018), while larger increases in exercise-induced IMF related to larger decreases in trait anxiety (Adj R^2= 0.10, p= 0.019).

Conclusion This study showed that both multimodal HIT and MIT improved MIP in persons with CNSLBP, while only HIT enhanced IME time. Exercise-induced IMF or inspiratory muscle activation during postural control did not improve after HIT or MIT.

Trial registration This trial was registered at ClinicalTrials.gov (NCT05384457) on May 11, 2022.

Keywords Chronic nonspecific low back pain, High-intensity training, Exercise therapy, Inspiratory muscles

*Correspondence:
Sim Klaps
sim.klaps@uhasselt.be

Full list of author information is available at the end of the article

Background

Low back pain is the leading cause of disability globally, affecting over 600 million people in 2020 [1, 2]. Up to 90 percent of the low back pain cases are nonspecific, meaning there is no clear or identifiable source for the pain [3]. While most people recover within weeks, up to 24% develop chronic nonspecific low back pain (CNSLBP), which is characterized by persistent pain lasting at least three months [3, 4]. CNSLBP is a complex, multifactorial condition, involving a dynamic interplay of biological, psychological, and social factors [1, 5].

Clinical guidelines recommend exercise therapy as the primary treatment for CNSLBP [6]. However, its effectiveness in improving pain intensity and disability remains insufficient [7], which may be partly explained by the large heterogeneity in exercise prescriptions, including differences in training modality (e.g., aerobic, resistance, trunk strength, or multimodal training) and training intensity [6]. While no single exercise modality has proven to be clearly superior, multimodal exercise therapy, typically combining cardiorespiratory training, resistance training, and trunk-strength exercises, is commonly applied in both research and clinical practice [8].

In the absence of a clearly superior training modality, identifying key training parameters such as intensity becomes essential to optimize outcomes in persons with CNSLBP. Training intensity is a key determinant of physiological adaptation, with high-intensity training (HIT) inducing greater cardiorespiratory, neuromuscular, and metabolic responses compared to moderate-intensity training (MIT) [9–12]. From a clinical perspective, this is highly relevant, as optimizing training intensity may enhance treatment efficiency and lead to greater improvements in clinical and psychosocial functioning [13]. Indeed, previous studies in persons with CNSLBP have shown that multimodal whole-body HIT consisting of cardiorespiratory, general resistance, and trunk muscle strength training improved disability, exercise capacity, and psychosocial outcomes more compared to multimodal MIT [14, 15]. Despite this, it remains unclear why multimodal HIT is superior to MIT. A deeper understanding of the underlying mechanisms could lead to more precise and mechanism-based interventions for CNSLBP, potentially improving their effectiveness.

One potential mechanism of multimodal HIT being superior to MIT is that HIT may improve inspiratory muscle function more than MIT. Inspiratory muscle dysfunctions are increasingly recognized as modifiable impairments in CNSLBP [16]. The diaphragm has a dual role as it is not only the primary inspiratory muscle but also contributes to postural control by generating intra-abdominal pressure and controlling the spine [17, 18]. Importantly, this postural function is partly mediated through anticipatory (feedforward) activation of the diaphragm, which occurs independently of respiration and contributes to spinal stability during movement [19, 20]. Inspiratory muscle dysfunctions observed in CNSLBP include a smaller diaphragm excursion and an elevated diaphragm position during postural control [21–23], reduced inspiratory muscle strength [24], reduced inspiratory muscle endurance (IME) [25], increased diaphragm fatigue [26], and altered diaphragm activation [27] compared to persons without pain.

These dysfunctions may impair postural control by diminishing intra-abdominal pressure generation by the diaphragm [28]. Impaired postural control, in turn, has been identified as an important contributor to both the development and recurrence of nonspecific low back pain [29, 30]. Experimental studies further support this link, showing that increased respiratory demand in healthy controls leads to increased postural sway [31], altered postural strategies [28], and reduced postural activation of the diaphragm [32]. Therefore, assessing inspiratory muscle function, such as diaphragm activation, during postural control tasks may provide important insights into its postural role in persons with CNSLBP.

Moreover, the clinical characteristics of persons with CNSLBP are associated with these inspiratory muscle dysfunctions. For instance, higher perceived disability correlated with lower inspiratory muscle strength [33]. Moreover, higher pain intensity correlated with a more elevated diaphragm position [21]. Pain can also lead to compensatory breathing strategies, such as shallow or rapid breathing, limiting diaphragm movement and reducing intra-abdominal pressure generation by the diaphragm [34]. Furthermore, psychological factors, such as anxiety and depression, were found to correlate with lower inspiratory muscle strength [35]. Anxiety and depression may also negatively impact inspiratory muscle function through their relationship with dyspnea and dysfunctional breathing [36]. Finally, research revealed a significant correlation between lower exercise capacity and lower inspiratory muscle strength in persons with CNSLBP [35].

Interestingly, previous studies have shown that whole-body exercise can enhance inspiratory muscle function. For example, high-intensity cardiorespiratory interval training on a cycle ergometer in healthy adults [37], general resistance training in elderly women [38], and trunk muscle strength training combined with upper limb resistance training in healthy persons [39] all resulted in improvements in inspiratory muscle strength. Therefore, we can assume that multimodal training can also improve inspiratory muscle function in CNSLBP. However, to date, it remains unclear whether multimodal whole-body HIT has superior effects on inspiratory muscle function compared to multimodal whole-body MIT in persons with CNSLBP. Furthermore, it is unknown

whether improvements in inspiratory muscle function are related to improvements in disability, pain intensity, anxiety, depression, and exercise capacity.

Therefore, the primary objective of this study was to evaluate the effects of multimodal HIT compared to multimodal MIT on inspiratory muscle strength in persons with CNSLBP. We hypothesized that HIT would lead to greater increases in inspiratory muscle strength compared to MIT in persons with CNSLBP.

Secondary objectives were to evaluate the effects of multimodal HIT compared to multimodal MIT on other dimensions of inspiratory muscle function, including inspiratory muscle endurance (IME), exercise-induced inspiratory muscle fatigue (IMF), and inspiratory muscle activation during postural control. We hypothesized that HIT would lead to greater increases in IME, and inspiratory muscle activation during postural control, while leading to a greater reduction in exercise-induced IMF compared to MIT in persons with CNSLBP.

An additional secondary objective was to investigate the extent to which changes in inspiratory muscle function are related to changes in disability, pain intensity, anxiety, depression, and exercise capacity. We hypothesized that greater improvements in inspiratory muscle strength, IME, IMF, and activation during postural control are related to greater improvements in disability, pain intensity, anxiety, depression, and exercise capacity.

Methods

This study is reported according to the CONSORT guidelines and the intervention is described following the Consensus on Exercise Reporting Template guidelines [40].

Design

Persons with CNSLBP participated in this randomized controlled trial involving a 12-week training program. Participants were randomly assigned to an experimental group (HIT) or a control group (MIT). We refer to the MIT group as the control group because it represents the treatment recommended in clinical guidelines [6]. Assessments were conducted pre-intervention and post-intervention. The training program and measurements were organized at the REVAL Rehabilitation Research Center (Hasselt University, Diepenbeek, Belgium). The study was conducted in accordance with the Declaration of Helsinki. This study was approved by the Medical Ethics Committee of Hasselt University (B1152021000029) and registered at clinicaltrials.gov (NCT05384457) on May 11, 2022.

Participants and recruitment

Between August 2022 and February 2025, persons with CNSLBP were recruited through local distribution of flyers (e.g., in libraries, pharmacies, and university facilities) in Limburg (BE), and adverts on social media. Dutch-speaking males and females with CNSLBP, aged 18 to 65 years, were eligible for study participation. CNSLBP was defined as pain located below the costal margin and above the inferior gluteal folds, with or without referred leg pain, persisting for at least 12 weeks, and not linked to any specific underlying pathology [3]. Proof of diagnosis was required from a general practitioner or specialist. Participants were required to report an average pain intensity of 3–8/10 over the past week. This range was selected to include participants with clinically relevant pain levels while excluding those with minimal symptoms or severe pain that could limit participation in exercise therapy. This range is consistent with previous trials in nonspecific low back pain [41]. Exclusion criteria included prior invasive spinal surgery, medical conditions that could affect the study outcomes (e.g., respiratory disorders, neurological deficits, diabetes mellitus, rheumatoid arthritis, pneumothorax, pacemaker, or balance disorders), other musculoskeletal conditions interfering with training, participation in exercise therapy for low back pain within the previous three months, contraindications for exercise according to a physician, inability to attend therapy sessions, or pregnancy. Interested individuals received a study information letter and attended an intake session during which eligibility criteria were assessed and written informed consent was obtained.

Randomization and blinding

After the baseline measurements were performed, participants were randomly assigned to either the HIT or MIT group. To ensure concealment of allocation, a research assistant not involved in the study picked a sealed, opaque, sequentially numbered envelope containing an abbreviation related to the allocated group. These envelopes were prepared in advance and kept by a research assistant who was not involved in participant recruitment, assessment, or intervention delivery. The envelopes were only opened after completion of baseline measurements. The researcher responsible for enrolling participants did not have access to the random allocation sequence and was therefore unable to predict or influence group assignment. A permuted block randomization with a block size of two was used to ensure equal group division (1:1 allocation ratio, n=32/group). Blinding of the participants and therapists regarding group assignment was not possible due to the nature of the training program. To minimize performance bias, participants were informed that both groups could expect similar progression. Additionally, to reduce motivation bias, they were blinded to their test results until the study protocol was finalized. Lastly, the outcome assessor was not blinded.

Interventions

Participants performed 24 training sessions (2 x 1.5 hours/week) under one-to-one, face-to-face supervision by trained therapists. As several therapists supervised the training sessions, precautions were taken to ensure the consistency of training content and mode of delivery. All therapists received extensive training on the training protocol for at least 15 hours (by S.K.). Before starting to supervise participants, all therapists had to pass an exam regarding training content, mode of delivery, and safety. Additionally, a detailed manual with the training protocol, progressions, and instructions was provided to all therapists. Furthermore, the therapists completed an exercise fidelity checklist after each training session. This checklist, which is described in Appendix 1, covers four key dimensions of exercise fidelity: adherence, dosage, quality of intervention delivery, and participant responsiveness [42].

Participants were instructed to conduct only the exercises assigned to their group and not make any significant lifestyle or exercise regimen changes during the intervention period. Sessions missed by participants (e.g., due to work-related obligations, familial issues, …) were postponed as long as the training program was finalized within 16 weeks after the first training session. Participants who did not complete the training program within 16 weeks were categorized as having missing data. Both the HIT and the MIT group followed a multimodal protocol consisting of cardiorespiratory training, general resistance training, and trunk muscle strength training [15]. The difference between the two groups was the training intensity and is described in Appendix 2. There were no non-exercise components in the training program, nor was there a home program component.

High-intensity training

Cardiorespiratory training consisted of an interval training on a cycle ergometer (Technogym, Gambettola, Italy). After a five-minute warm-up, participants performed an interval protocol, consisting of five one-minute bouts (110 revolutions per minute at 100% of the maximal oxygen uptake (VO_{2max}) workload), separated by one minute of active rest (75 revolutions per minute at 50% VO_{2max} workload). General resistance training included a circuit of three lower-body exercises and three upper-body exercises performed on fitness devices (Technogym, Gambettola, Italy; Inspire Fitness, Corona, CA, USA) (Figure 1), performed at 80% of the one-repetition maximum (1RM). Trunk muscle strength training consisted of six static trunk exercises executed on an Airex mat (Figure 2). Trunk muscle strength training with their estimated activation intensities and progressions are described in Appendix 3. A detailed description of the training protocol has been published previously [15].

Moderate-intensity training

Cardiorespiratory training consisted of a continuous training on a cycle ergometer (Technogym). After a

Fig. 1 General resistance training. 1: leg press; 2: leg extension; 3: leg curl; 4: vertical traction; 5: arm curl; 6: chest press

Fig. 2 Trunk muscle strength training. 1: glute bridge; 2: glute clam with elastic band; 3: shoulder retraction with hip hinge; 4: superman back extension; 5: adapted knee plank; 6: adapted knee side plank

five-minute warm-up, participants cycled continuously for 14 minutes (90 revolutions per minute at 60% VO_{2max} workload). General resistance training was identical to the protocol described in 'HIT' with the exception of the training intensity. Exercises were performed at 60% of the 1RM. Trunk muscle strength training was identical to the protocol described in 'HIT' with the exception of the training intensity. A detailed description of the training protocol has been published previously [15].

Outcomes

Primary outcome

Inspiratory muscle strength Maximal inspiratory pressure (MIP) is a reliable and valid measure of maximal inspiratory muscle strength [43]. MIP was measured at residual volume with a portable electronic mouth pressure transducer (POWERbreathe International Ltd., type KH2, Warwickshire, UK). For each MIP measurement, participants performed a minimum of five (before CPET) or a minimum of three (after CPET) MIP attempts. The highest value obtained from repeated attempts was used for analysis [44]. Both absolute MIP (MIP_{abs}) values and predicted MIP (MIP_{pred}) values were analyzed. MIP_{pred} was calculated using sex-, age-, and body mass index (BMI)-specific reference equations [45]. MIP_{abs} was then expressed as a percentage of MIP_{pred}. Inspiratory muscle weakness was defined as < 80% MIP_{pred}, low-to-moderate strength as 80%–99% MIP_{pred}, and normal-to-high strength as ≥ 100% MIP_{pred} [46]. The detailed measurement procedure has been described previously [35].

Secondary outcomes

Inspiratory muscle endurance IME was measured using a pressure threshold load set at 60% of MIP_{abs} obtained during the baseline measurement [26, 28]. Participants performed the resistive breathing task while seated, wearing a nose clip, and breathing through the device for a maximum of 900 seconds. Participants were instructed to inhale maximally and as rapidly as possible at a frequency of 15 breaths per minute. The trial ended when the participant was unable to achieve the target flow of 0.6 L/s despite verbal encouragement from the investigator.

Exercise-induced inspiratory muscle fatigue IMF is defined as a temporary reduction in the ability to produce inspiratory muscle force following contractile activity that can be reversed through rest [43]. Exercise-induced IMF was assessed by calculating the differences in MIP_{pred} before and immediately (IMF_{0min}), 15 minutes (IMF_{15min}), and 30 minutes (IMF_{30min}) post-CPET. A reduction of ≥ 10% in MIP_{pred} was considered as IMF [47].

Inspiratory muscle activation during postural control Inspiratory muscle activation was measured during two postural control tasks using surface electromyography (EMG). The outcomes were normalized mean EMG amplitude of the sternocleidomastoid ($sEMG_{scm}$) and scalene ($sEMG_{scal}$) muscles, as they are non-invasive physiological markers for diaphragm EMG [48].

EMG procedure EMG electrode placement

$sEMG_{scm}$ and $sEMG_{scal}$ were measured using a wireless transmission system (Delsys, Inc., MA, USA). Specifically,

two bipolar electrodes (Delsys Trigno™ Mini sensor) were used. Before electrode placement, the skin was prepared by shaving the necessary areas and thoroughly scrubbing them with alcohol-soaked cotton pads. The sensing head of the Trigno™ Mini sensor (25x12x7mm, 10 mm inter-electrode distance, 99.99% silver) was positioned as follows [49]: (1) right at the midpoint along the long axis of the sternocleidomastoid muscle between the mastoid process and the medial clavicle, and (2) left at the posterior triangle of the neck at the level of the cricoid process for the scalene muscle.

Maximal voluntary contractions

After placing the electrodes, three maximal voluntary contraction (MVC) maneuvers were performed for the sternocleidomastoid and the scalene muscles. The MVC maneuvers involved cervical lateral flexion against manual resistance [48]. Maximal voluntary contractions were performed solely for EMG normalization purposes. Normalization to MVC is necessary to allow valid comparisons of EMG amplitudes across time points, as raw EMG signals are influenced by non-physiological factors such as electrode placement and skin impedance [50].

Postural control tasks

After performing the MVCs, participants performed two postural control tasks in a standardized order. The tasks involved three sets of repetitive ballistic arm movements, performed as fast as possible, in upright, bipedal standing without vision (i.e., by wearing non-transparent goggles) on an unstable support surface (i.e., standing on an Airex balance pad placed on top of the force plate). Both postural trials had a total duration of 90 seconds and consisted of a 20-second rest period, followed by three sets of repetitive ballistic shoulder flexion-extension movements (10-degree range of motion during 10 seconds), with elbows and wrists extended and executed as rapidly as possible, and a final 20-second rest period.

The only difference between both tasks was the breathing mode during the arm movements: either normal breathing ($sEMG_{scm\_NB}$ and $sEMG_{scal\_NB}$) or breath holding at end-expiration ($sEMG_{scm\_EE}$ and $sEMG_{scal\_EE}$). The breath hold at end-expiration was used in an attempt to isolate the postural function from the inspiratory function to obtain a more accurate result of the postural role of the inspiratory muscles [20]. These tasks were chosen based on previous research showing that the diaphragm is activated during postural control tasks with rapid arm movements [19].

EMG sampling and processing

EMG was sampled at 2000 Hz by Delsys Trigno Discover software (Delsys, Inc., MA, USA) and then processed with Matlab 6.5 (Mathworks, Natick, MA, USA). Raw EMG signals were filtered and processed, including electrocardiogram artifact removal, as previously described by Klaps et al. [48]. EMG activity was converted into mean amplitudes and expressed as a percentage of the highest EMG amplitude recorded during the MVC maneuvers (peak value over a three-second period).

The difference in normalized mean EMG amplitude between the rest position and the average mean EMG amplitude across the three sets of ballistic arm movements was calculated. The zones for these calculations were defined based on the EMG activity from the deltoid muscle. The first five seconds and the last five seconds were excluded from the rest position to ensure a stable baseline measurement.

Disability Disability related to CNSLBP was evaluated using the Modified Oswestry Disability Index (MODI), which measures the impact of low back pain on a person's daily activities [51]. The MODI includes 10 items scored on a five-point scale, with the total score represented as a percentage. Scores from 0% to 20% indicate minimal disability; 20% to 40%, moderate disability; 40% to 60%, severe disability; 60% to 80%, crippled; and 80% to 100%, bedbound or exaggerating [51].

Pain Pain intensity was evaluated using a numeric pain rating scale from 0 to 10, where participants rated their average low back pain over the past week. This scale was derived from the Brief Pain Inventory [52]. Scores of 5 or less signify mild pain, scores of 6 to 7 represent moderate pain, and scores of 8 or greater indicate severe pain [53].

Anxiety Anxiety was assessed using the State Trait Anxiety Inventory (STAI) [54]. The STAI assesses both state-anxiety (i.e., a temporary emotional state) and trait-anxiety (i.e., anxiety as a personality trait). Each subscale consists of 20 items scored on a four-point Likert scale. STAI scores are classified as "no or low anxiety" (20–37), "moderate anxiety" (38–44), and "high anxiety" (45–80) [54].

Depression Depression was evaluated using the Beck Depression Inventory (BDI) [55]. This BDI contains 21 items, each rated on a four-point scale. The level of depression severity is classified as "minimal" (0–13), "mild" (14–19), "moderate" (20–28), and "severe" (29–63) [55].

Exercise capacity Exercise capacity was evaluated through a maximal CPET on an electronically braked cycle ergometer (eBike Basic, General Electric GmbH, Bitz, Germany). The CPET started at a low workload, which was incrementally increased every minute (starting at 30 Watts + 15 Watts/minute; 75 revolutions per minute). A minimum respiratory exchange ratio threshold of 1.10 was used to ensure maximal effort [56]. VO_{2max} was used as the outcome for exercise capacity, which was

assessed through breath-by-breath gas exchange analysis (Cortex, Metamax 3B).

Statistical analysis

An a priori sample size calculation was performed for the primary research objective. This calculation was based on the therapeutic effects of (a) a 12-week multimodal whole-body HIT program on VO_{2max} [15] and (b) an 8-week high-intensity inspiratory muscle training program on MIP [57] in persons with CNSLBP. These outcomes were chosen as they relate most to the respiratory system and are thus most fitting to indicate possible effects on inspiratory muscle function. For inspiratory muscle endurance, fatigue, and activation, no established minimal clinically important differences are currently available and prior intervention studies in persons with CNSLBP are lacking, making a reliable sample size calculation for these outcomes not feasible. The sample size calculation considered a minimal clinically important difference of 3.5ml/kg/min for VO_{2max} [58] and 17.2 cmH_2O for MIP_{abs} [59]. As VO_{2max} required the largest sample size to detect a clinically meaningful between-group difference, the final calculation was based on this outcome. A significance level of 0.05 and a power level of 0.80, resulted in a required sample size 38 patients (n= 19 per group). Additionally, we expected a 30% loss to follow-up during rehabilitation and a further 10% dropout for the long-term follow-up assessments. Consequently, the total sample size was 64 participants (n= 32 per group).

Data analysis was performed using JMP Pro (17.2 SAS Institute Inc, Cary, USA). Demographic characteristics (age, sex, BMI, duration of low back pain) and baseline results for each group were presented using descriptive statistics. Normality was assessed via visual inspection of Q-Q plots, while homoscedasticity was evaluated using the Brown-Forsythe test. The two-sample t-test was used to compare demographic characteristics and baseline results of both the primary and secondary outcomes between the two groups, and the chi-square test was used to compare sex.

To assess the effects of HIT compared to MIT on inspiratory muscle function, a linear mixed model was used to analyze within- and between-group differences. Fixed effects included group, time, and their interaction (group x time), while participant was included as a random effect. Normality of residuals was assessed through Q-Q plot inspection, and homoscedasticity was assessed using the Brown-Forsythe test and residual vs. fitted plots. Post-hoc analysis for multiple comparisons was conducted using the Tukey honest significant difference test. In addition to statistical significance, effect sizes were calculated using Cohen's d to quantify the magnitude of differences. Effect sizes were interpreted as small (0.2), medium (0.5), and large (0.8) [60]. No data imputation was performed, assuming that data were missing at random. Linear mixed models are valid under missing at random assumptions based on the ignorability property.

To assess the extent to which changes in inspiratory muscle function are related to changes in disability, pain intensity, anxiety, depression, and exercise capacity, the following statistical approach was used: First, Pearson correlation tests were performed to assess bivariate correlations between changes in independent variables (MIP, IME time, exercise-induced IMF, and normalized mean amplitude of $sEMG_{scm}$ and $sEMG_{scal}$) and changes in dependent variables (MODI, BPI, STAI STATE, STAI TRAIT, BDI, and VO_{2max}). Independent variables with a p-value < 0.2 were selected for inclusion in the regression model to evaluate relationships between the dependent and independent variables. A p-value < 0.2 was used as an initial screening threshold to avoid excluding potentially relevant variables, consistent with recommendations for variable selection in multivariable modelling [61]. Second, a standard least squares regression model was performed for each dependent variable separately, including the selected independent variables from the correlation analyses. A stepwise backward elimination approach was applied, starting with all candidate variables in the model. Independent variables with the highest non-significant p-value (p > 0.05) were sequentially removed until only significant independent variables remained.

Results

Participants

A total of 103 persons were screened for eligibility. Of these, 39 did not meet the inclusion criteria. As such, 64 persons were included (HIT: n= 32, MIT: n= 32). Of the 64 included participants, 57 completed the intervention as planned; seven participants discontinued the program between session 7 and 18. *All 57 completers attended all prescribed training sessions within the 16-week period, as recorded using supervised attendance logs, resulting in a 100% session completion rate among completers. Adherence, defined as attending training sessions on the originally scheduled day, was 94%. When participants were unable to attend a scheduled session, it was rescheduled within the same week to ensure full completion of the protocol.* Both groups had similar demographic characteristics and baseline results (p> 0.05). A comprehensive research design flowchart is displayed in Figure 3. An overview of the demographic characteristics and baseline results is displayed in Table 1.

Effects of HIT and MIT on inspiratory muscle function

MIP increased significantly in both the HIT (Δ9.0 ± 9.9%, p= 0.002) and MIT (Δ10.8 ± 13.2%, p< 0.0001) groups. However, the difference between groups was not significant (p= 0.558). IME time increased only in the HIT

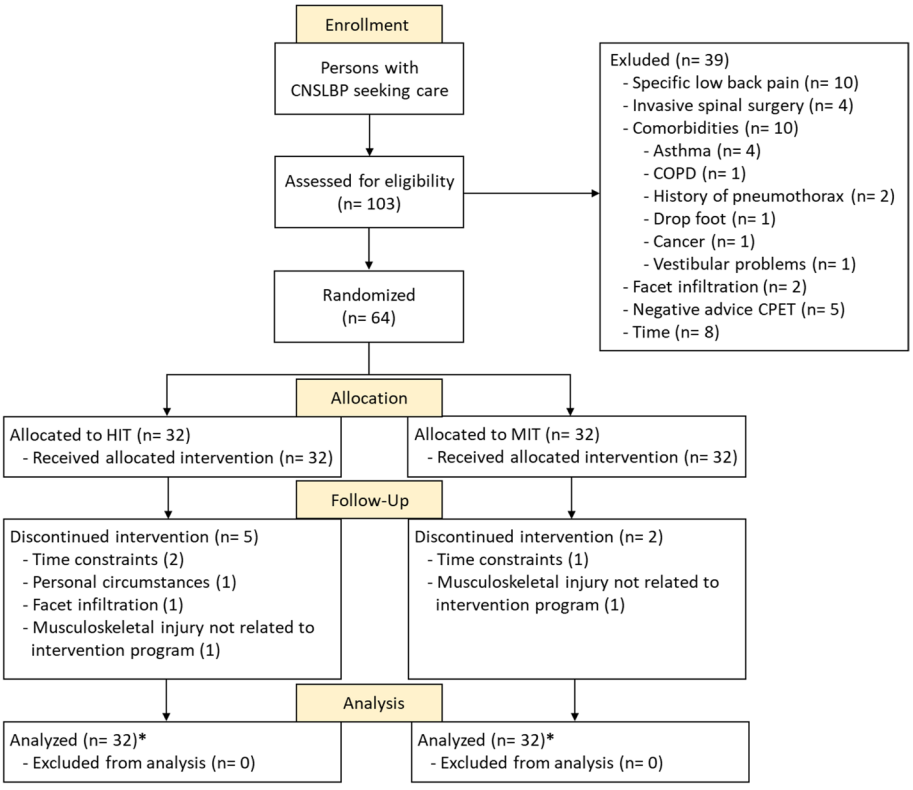

Fig. 3 Comprehensive research design flowchart. *Abbreviations: CNSLBP, chronic nonspecific low back pain; COPD, chronic obstructive pulmonary disease; HIT, high-intensity training; MIT, moderate-intensity training.* *All participants were included in the linear mixed model analysis according to the ignorability assumption

group (Δ69.6 ± 78.6s, $p< 0.0001$), with no improvement in the MIT group (Δ20.0 ± 60.9s, $p = 0.414$), resulting in a significant between-group difference of 49.6 seconds in favor of the HIT group ($p= 0.010$). In contrast, no improvements were observed in exercise-induced IMF at any time point post-CPET in either group ($0.301\leq p\leq 0.999$). Furthermore, no improvements were observed in the normalized mean amplitude of $sEMG_{scm}$ and $sEMG_{scal}$ during any postural control task in either group ($0.764\leq p\leq 1.000$). The results are shown in Table 2 and Figure 4.

Effects of HIT and MIT on disability, pain intensity, anxiety, depression, and exercise capacity

Both groups showed improvements in disability (MODI: HIT −6.8 ± 8.9%, $p= 0.001$; MIT −6.9 ± 7.9%, $p= 0.001$), but no significant between-group difference was found ($p= 0.966$). Similarly, pain intensity (BPI: HIT −2.1 ± 2.5 points, $p= 0.0001$; MIT −1.5 ± 2.0 points, $p= 0.005$) decreased in both groups, without a significant between-group difference ($p= 0.376$). For psychological outcomes, only the HIT group showed significant reductions in state anxiety (STAI-State: −4.7 ± 11.2 points, $p= 0.025$), trait anxiety (STAI-Trait: −5.2 ± 8.4 points, $p= 0.007$), and BDI (−3.2 ± 5.6 points, $p= 0.005$), while no significant changes were observed in the MIT group ($0.611\leq p\leq 0.771$). However, between-group differences were not significant for any of these measures ($0.070\leq p\leq 0.172$). VO_{2max} increased only in the HIT group (5.4 ± 3.6 $mL^{*}kg^{-1}{*}min^{-1}$, $p= 0.001$), with no improvement in the MIT group (2.5 ± 4.0 $mL^{*}kg^{-1}{*}min^{-1}$, $p= 0.169$), resulting in a significant between-group difference of 2.9 $mL^{*}kg^{-1}{*}min^{-1}$ in favor of the HIT group ($p= 0.006$). The results are shown in Table 2.

The relationship between changes in inspiratory muscle function and changes in disability, pain intensity, anxiety, depression, and exercise capacity

Only correlations with $p< 0.2$ were considered relevant and subsequently included in the regression models. For the MODI, IME time ($r= -0.20$, $p= 0.132$) was added. For the BPI, IME time ($r= -0.25$, $p= 0.064$), and IMF_{15min} ($r= 0.18$, $p= 0.197$) were added. For STAI-State, normalized mean $sEMG_{scm\_EE}$ amplitude ($r= 0.18$, $p= 0.187$) was added. For STAI-Trait, IMF_{0min} ($r= -0.21$, $p= 0.128$), IMF_{15min} ($r= -0.28$, $p= 0.040$), IMF_{30min} ($r= -0.31$, $p= 0.031$), normalized mean $sEMG_{scm\_NB}$ amplitude ($r= 0.26$, $p= 0.052$), and normalized mean $sEMG_{scm\_EE}$ amplitude ($r= 0.18$, $p= 0.189$) were added. For BDI, MIP_{pred} ($r= -0.27$, $p= 0.045$), IMF_{0min} ($r= -0.26$, $p= 0.056$), IMF_{15min}

Table 1 Demographic characteristics and baseline results

	HIT group (*n*= 32)	MIT group (*n*= 32)	*P*-value
Demographic outcomes			
Age (years)	44.4 ± 13.8	45.3 ± 15.3	0.824
Sex (male/female)	17/15	21/11	0.308
BMI (kg/m^2)	25.5 ± 4.1	26.3 ± 4.1	0.446
LBP duration (years)	11.7 ± 7.4	10.4 ± 8.0	0.480
Baseline inspiratory muscle function outcomes			
MIP_{abs} (cmH_2O)	105.7 ± 29.7	106.7 ± 25.2	0.886
MIP_{pred} (%)	89.3 ± 23.3	89.1 ± 17.0	0.979
MIP_{pred} < 80% (n)	13	10	0.816
80% ≤ MIP_{pred} < 100%	9	10	
MIP_{pred} ≥ 100%	10	12	
IME time (s)	235.8 ± 99.6	248.6 ± 125.3	0.675
IMF_{0min} (%)	2.1 ± 9.7	−1.7 ± 7.5	0.110
IMF_{15min} (%)	5.2 ± 7.5	3.4 ± 9.8	0.461
IMF_{30min} (%)	4.5 ± 6.0	2.9 ± 9.4	0.453
$sEMG_{scm\_NB}$ (%)	19.2 ± 20.8	18.8 ± 15.9	0.923
$sEMG_{scm\_EE}$ (%)	15.8 ± 14.5	17.9 ± 14.6	0.560
$sEMG_{scal\_NB}$ (%)	30.9 ± 19.7	26.4 ± 14.3	0.454
$sEMG_{scal\_EE}$ (%)	26.9 ± 15.9	26.6 ± 13.5	0.817
Baseline secondary outcomes			
MODI (0–100)	19.5 ± 11.3	20.2 ± 9.0	0.787
BPI (0–10)	4.8 ± 1.8	4.3 ± 1.6	0.281
STAI STATE (20–80)	35.7 ± 9.7	32.9 ± 10.4	0.301
STAI TRAIT (20–80)	38.7 ± 10.2	36.7 ± 12.7	0.532
BDI (0–63)	7.8 ± 6.8	8.4 ± 6.1	0.734
VO_{2max} (mL*kg^{-1}*min^{-1})	36.4 ± 9.5	35.3 ± 8.0	0.589

Abbreviations: *BMI* Body Mass Index, *LBP* low back pain, MIP_{abs} maximal inspiratory pressure absolute values, MIP_{pred} maximal inspiratory pressure predicted values, *IME* inspiratory muscle endurance, *IMF* inspiratory muscle fatigue, $sEMG_{scm\_NB}$ surface electromyography of Sternocleidomastoid muscle during normal breathing, $sEMG_{scm\_EE}$ surface electromyography of Sternocleidomastoid muscle end-expiration, $sEMG_{scal\_NB}$ surface electromyography of Scalene muscles during normal breathing, $sEMG_{scal\_EE}$ surface electromyography of Scalene muscles end-expiration, *MODI* Modified Oswestry Disability Index, *BPI* Brief Pain Inventory, *STAI* State Trait Anxiety Inventory, *BDI* Beck Depression Inventory, VO_{2max} maximal oxygen uptake, *HIT* high-intensity training, *MIT* moderate-intensity training

(*r*= −0.27, *p*= 0.047), and IMF_{30min} (*r*= −0.22, *p*= 0.105) were added. For VO_{2max}, MIP_{pred} (*r*= −0.31, *p*= 0.018), and IME time (*r*= 0.26, *p*= 0.050) were added. Table 3 displays all correlations.

None of the models included more than four factors, adhering to the commonly used rule of thumb of at least 15 participants per factor. This suggests that the models were sufficiently powered [62]. Changes in IMF_{30min} accounted for 8% of the variance in changes in STAI-Trait (*p*= 0.019), with larger increases in IMF_{30min} being related to larger decreases in STAI TRAIT. Changes in MIP_{pred} accounted for 7% of the variance in changes in BDI (p= 0.047), with larger increases in MIP being related to larger decreases in BDI. Changes in MIP_{pred} accounted for 10% of the variance in changes in VO_{2max} (*p*= 0.018), with larger increases in MIP_{pred} being related to smaller increases in VO_{2max}. An overview of each final model is displayed in Table 4.

Discussion

Main findings

This study compared the effects of a multimodal whole-body HIT program to a multimodal whole-body MIT program on maximal inspiratory pressure (MIP), the primary outcome measure, in persons with CNSLBP. Secondary outcomes included other dimensions of inspiratory muscle function, namely inspiratory muscle endurance time, exercise-induced inspiratory muscle fatigue (IMF), and normalized mean $sEMG_{scm}$ and $sEMG_{scal}$ amplitude during postural control. In addition, this study explored whether changes in inspiratory muscle function outcomes were related to changes in disability, pain intensity, anxiety, depression, and exercise capacity.

The results showed that maximal inspiratory pressure (MIP) increased in both the HIT and MIT groups, with no significant difference between groups. In contrast, inspiratory muscle endurance time increased only in the HIT group. Neither group showed improvements in exercise-induced inspiratory muscle fatigue (IMF) or normalized mean $sEMG_{scm}$ and $sEMG_{scal}$ amplitude during postural control. Additionally, exploratory analyses showed that larger increases in MIP were related to larger decreases in depressive symptoms and to smaller increases in exercise capacity, while larger increases in exercise-induced IMF were related to larger decreases in trait anxiety. Furthermore, changes in inspiratory muscle function outcomes were not related to changes in disability or pain intensity. These exploratory relationships should be interpreted with caution.

Maximal inspiratory pressure (MIP)

With regard to inspiratory muscle strength, both groups showed significant increases in MIP indicating that multimodal training, independent of its intensity, can improve inspiratory muscle strength in persons with CNSLBP. The magnitude of these increases is similar to those observed in a previous study involving general resistance training in older persons [38]. However, the increases in MIP in our study are modest compared to those from high-intensity inspiratory muscle training, which has shown increases in MIP as high as 42 cmH_2O [57]. This may be explained by the fact that MIP gains from multimodal training are primarily driven by neuromuscular adaptations, such as increased motor unit recruitment and improved synchronization of muscle contraction [63, 64]. In contrast, inspiratory muscle training applies the principles of training specificity and overload more directly to the inspiratory muscles [63], resulting not only in greater strength gains but also in structural adaptations, such

Table 2 Within- and between-group differences for HIT and MIT

	HIT (*n*= 32)				MIT (*n*= 32)				HIT vs MIT		
Outcome measure	**Pre**	**Post**	**Delta (post-pre)**	***p*-value**	**Pre**	**Post**	**Delta (post-pre)**	***p*-value**	**Dif**	***p*-value**	**Cohen's d**
MIP_{abs} (cmH2O)	105.7 ± 29.7	116.1 ± 25.6	10.4 ± 11.5	**0.002***	106.7 ± 25.2	119.3 ± 23.1	12.5 ± 15.5	**<0.0001***	−2.1	0.575	−0.15
MIP_{pred} (%)	89.3 ± 23.3	98.3 ± 19.7	9.0 ± 9.9	**0.002***	89.1 ± 17.0	100.0 ± 15.5	10.8 ± 13.2	**<0.0001***	−1.9	0.558	−0.16
IM endurance time (s)	235.8 ± 99.6	305.3 ± 117.8	69.6 ± 78.6	**<0.0001***	248.6 ± 125.3	268.7 ± 138.5	20.0 ± 60.9	0.414	49.6	**0.010***	0.71
IMF_{0min} (%)	2.1 ± 9.7	3.9 ± 11.5	1.9 ± 12.5	0.847	−1.7 ± 7.5	1.9 ± 6.1	3.6 ± 10.6	0.301	−1.8	0.574	−0.15
IMF_{15min} (%)	5.2 ± 7.5	7.7 ± 10.4	2.5 ± 12.6	0.763	3.4 ± 9.8	4.0 ± 7.6	0.6 ± 13.2	0.999	1.9	0.587	0.15
IMF_{30min} (%)	4.5 ± 6.0	8.3 ± 8.7	3.8 ± 10.5	0.317	2.9 ± 9.4	5.1 ± 6.5	2.3 ± 12.0	0.770	1.6	0.614	0.13
sEMGscm											
FoamFLEXT_NB	19.2 ± 20.8	19.2 ± 13.0	0.0 ± 16.0	1.000	18.8 ± 15.9	18.6 ± 16.6	−0.2 ± 20.1	0.999	0.2	0.895	0.01
FoamFLEXT_EE	15.8 ± 14.5	15.9 ± 11.7	0.1 ± 10.5	1.000	17.9 ± 14.6	17.5 ± 13.3	−0.4 ± 18.7	0.997	0.5	0.872	004
sEMGscal											
FoamFLEXT_NB	30.9 ± 19.7	32.6 ± 24.0	1.7 ± 18.1	0.982	26.4 ± 14.3	31.1 ± 17.8	4.7 ± 23.1	0.764	−3.0	0.609	−0.15
FoamFLEXT_EE	26.9 ± 15.9	28.5 ± 24.3	1.6 ± 24.6	0.983	26.6 ± 13.5	28.6 ± 14.7	2.0 ± 19.4	0.977	−0.4	0.991	−0.02
MODI	19.5 ± 11.3	12.7 ± 9.1	−6.8 ± 8.9	**0.001***	20.2 ± 9.0	13.3 ± 10.8	−6.9 ± 7.9	**0.001***	0.1	0.966	0.01
BPI	4.8 ± 1.8	2.7 ± 2.4	−2.1 ± 2.5	**0.0001***	4.3 ± 1.6	2.7 ± 1.7	−1.5 ± 2.0	**0.005***	−0.5	0.376	−0.27
BDI	7.8 ± 6.8	4.6 ± 4.9	−3.2 ± 5.6	**0.005***	8.4 ± 6.1	7.2 ± 6.2	−1.2 ± 3.5	0.611	−2.0	0.107	−0.44
STAI STATE	35.7 ± 9.7	31.0 ± 8.7	−4.7 ± 11.2	**0.025***	32.9 ± 10.4	32.0 ± 10.7	−0.9 ± 9.7	0.672	−3.9	0.172	−0.38
STAIT TRAIT	38.7 ± 10.2	33.5 ± 9.2	−5.2 ± 8.4	**0.007***	36.7 ± 12.7	35.3 ± 11.3	−1.4 ± 7.1	0.771	−3.8	0.070	−0.51
$VO2_{max}$	36.4 ± 9.5	41.8 ± 9.4	5.4 ± 3.6	**0.001***	35.3 ± 8.0	37.8 ± 8.7	2.5 ± 4.0	0.169	2.9	**0.006***	0.76

Bold values indicate statistical significance

Abbreviations: MIP_{abs} maximal inspiratory pressure absolute values, MIP_{pred} maximal inspiratory pressure predicted values, *IME* inspiratory muscle endurance, *IMF* inspiratory muscle fatigue, $sEMG_{scm\_NB}$ surface electromyography of Sternocleidomastoid muscle during normal breathing, $sEMG_{scm\_EE}$ surface electromyography of Sternocleidomastoid muscle end-expiration, $sEMG_{scal\_NB}$ surface electromyography of Scalene muscles during normal breathing, $sEMG_{scal\_EE}$ surface electromyography of Scalene muscles end-expiration, *MODI* Modified Oswestry Disability Index, *BPI* Brief Pain Inventory, *STAI* State Trait Anxiety Inventory, *BDI* Beck Depression Inventory, VO_{2max} maximal oxygen uptake, *HIT* high-intensity training, *MIT* moderate-intensity training, *Dif* difference

as increased diaphragm thickness and muscle hypertrophy [65]. Additionally, task-specific effects of inspiratory muscle training may further contribute to these larger gains, as training with the same device and protocol used for testing can improve performance on that test independently of structural adaptations [66]. Although the improvements in MIP we observed were smaller than those typically reported after inspiratory muscle training, they could still hold clinical relevance. The gains in our study exceeded the estimated learning effect of repeated MIP measurements, which is around 7 cmH2O [67]. However, while increases of 17.2 cmH_2O [59] have been proposed as clinically meaningful in patients with chronic obstructive pulmonary disease, no minimal clinically important difference has been established for persons with CNSLBP. In addition, in our study, changes in MIP were not significantly related to changes in disability or pain intensity. Taken together, the clinical significance of these strength gains in the context of CNSLBP remains uncertain and warrants further investigation.

Contrary to our hypothesis, HIT did not result in superior improvements in MIP compared to MIT. A plausible explanation is that both training protocols provided sufficient intensity, frequency, and duration to stimulate comparable neuromuscular adaptations in inspiratory muscle strength [68]. Additionally, a ceiling effect may have limited further MIP gains in participants with normal-to-high baseline MIP values, who represented 34% of the sample. Another 30% of participants had reduced baseline MIP, while 36% met the criteria for inspiratory muscle weakness at baseline [46]. This heterogeneity likely contributed to interindividual variability in training response and may have masked potential group differences. No subgroup analyses were performed due to the limited sample size. Therefore, the potential influence of baseline inspiratory muscle function on training response remains speculative. Future studies should investigate whether predefined subgroups with inspiratory muscle weakness show different group effects following training, as has been demonstrated in other populations such as chronic obstructive pulmonary disease, where those with low baseline MIP values achieved the largest improvements in MIP [69].

Exercise-induced inspiratory muscle fatigue (IMF)

Exercise-induced IMF did not improve after HIT or MIT. At baseline, neither group showed IMF. Several factors may explain the absence of IMF at baseline. First,

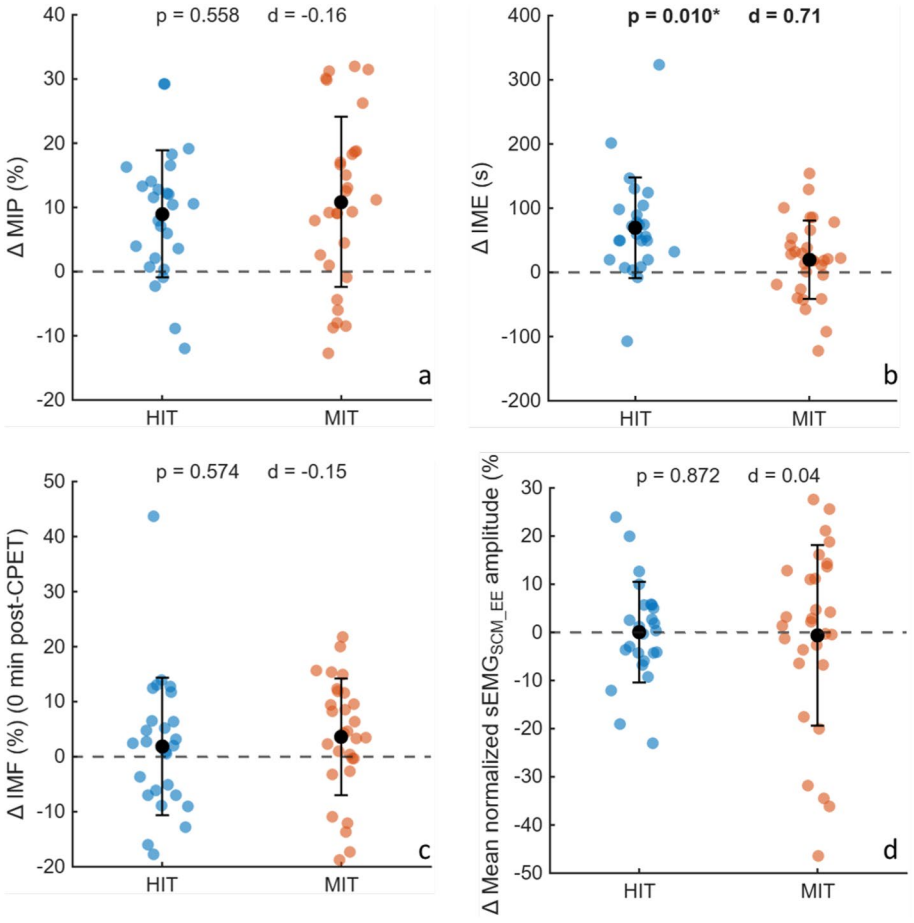

Fig. 4 Effects of high- and moderate-intensity training on inspiratory muscle function. Individual change and between-group differences following high- and moderate-intensity training in (**a**) maximal inspiratory pressure, (**b**) inspiratory muscle endurance time, (**c**) inspiratory muscle fatigue measured 0 minutes post-CPET, and (**d**) normalized mean amplitude of the Sternocleidomastoid muscle end-expiration. The black dot represents the group mean, and the error bars indicate the standard deviation. Abbreviations: MIP, maximal inspiratory pressure; IME, inspiratory muscle endurance; IMF, inspiratory muscle fatigue; CPET, cardiopulmonary exercise test; $sEMG_{scm\_EE}$, surface electromyography of Sternocleidomastoid muscle end-expiration; HIT, high-intensity training; MIT, moderate-intensity training

Table 3 Pearson correlation coefficients between changes (Δ) in independent and dependent variables

	Δ dependent variables					
	MODI	**BPI**	**STAI State**	**STAI Trait**	**BDI**	**VO_{2max}**
Δ independent variables						
MIP_{pred}	−0.02 (0.893)	−0.15 (0.260)	0.04 (0.743)	−0.15 (0.286)	**−0.27 (0.045)**	**−0.31 (0.018)**
IME time	**−0.20 (0.132)**	**−0.25 (0.064)**	−0.06 (0.650)	−0.13 (0.347)	−0.15 (0.255)	**0.26 (0.050)**
IMF_{0min}	−0.09 (0.510)	0.06 (0.686)	−0.05 (0.739)	**−0.21 (0.128)**	**−0.26 (0.056)**	−0.08 (0.581)
IMF_{15min}	0.08 (0.558)	**0.18 (0.197)**	0.04 (0.752)	**−0.28 (0.040)**	**−0.27 (0.047)**	−0.06 (0.646)
IMF_{30min}	0.12 (0.368)	0.11 (0.415)	0.09 (0.518)	**−0.31 (0.019)**	**−0.22 (0.105)**	−0.16 (0.256)
$sEMG_{scm\_NB}$ (%)	−0.09 (0.528)	−0.01 (0.920)	0.14 (0.324)	**0.26 (0.052)**	0.02 (0.906)	−0.16 (0.243)
$sEMG_{scm\_EE}$ (%)	−0.12 (0.368)	−0.12 (0.396)	**0.18 (0.187)**	**0.18 (0.189)**	0.07 (0.633)	−0.09 (0.471)
$sEMG_{scal\_NB}$ (%)	0.07 (0.639)	−0.09 (0.521)	−0.03 (0.838)	0.02 (0.902)	−0.13 (0.343)	0.06 (0.681)
$sEMG_{scal\_EE}$ (%)	0.13 (0.345)	−0.01 (0.922)	−0.05 (0.698)	−0.06 (0.654)	−0.07 (0.606)	0.07 (0.596)

Values are reported as Pearson's r (*p*-value). Bold values indicate correlations with $p < 0.2$, considered as candidates for inclusion in the regression models

Abbreviations: MIP_{pred} maximal inspiratory pressure predicted values, *IME* inspiratory muscle endurance, *IMF* inspiratory muscle fatigue, $sEMG_{scm\_NB}$ surface electromyography of Sternocleidomastoid muscle during normal breathing, $sEMG_{scm\_EE}$ surface electromyography of Sternocleidomastoid muscle end-expiration, $sEMG_{scal\_NB}$ surface electromyography of Scalene muscles during normal breathing, $sEMG_{scal\_EE}$ surface electromyography of Scalene muscles end-expiration, *MODI* Modified Oswestry Disability Index, *BPI* Brief Pain Inventory, *STAI* State Trait Anxiety Inventory, *BDI* Beck Depression Inventory, VO_{2max} maximal oxygen uptake

Table 4 Regression analyses between changes (Δ) in independent and dependent variables

		p. est.	95% CI	st. β	Total adjusted R^2	F-ratio	*p*-value
ΔIMF_{30min}	ΔSTAI TRAIT	−0.222	−0.40;−0.04	−0.315	0.08	5.82	**0.019***
ΔMIP_{pred}	ΔBDI	−0.107	−0.21;−0.01	−0.269	0.07	4.14	**0.047***
ΔMIP_{pred}	ΔVO_{2max}	−0.109	−0.20;−0.02	−0.314	0.10	5.91	**0.018***

Bold values indicate statistical significance

Abbreviations: MIP_{pred} maximal inspiratory pressure predicted values, *IMF* inspiratory muscle fatigue, *STAI* State Trait Anxiety Inventory, *BDI* Beck Depression Inventory, VO_{2max} maximal oxygen uptake, *p. est.* parameter estimate, *CI* confidence interval, *st. β* standardized beta

the participants had relatively high baseline inspiratory muscle strength, which likely limited the development of IMF, as stronger inspiratory muscles tend to experience less fatigue during exercise [70]. From a methodological perspective, IMF was assessed through MIP, a voluntary measure influenced by motivation and accessory muscle recruitment. Non-volitional methods, such as EMG responses to phrenic nerve stimulation, are more accurate measurements for IMF assessment [26, 71]. Furthermore, the duration of the cardiopulmonary exercise test, particularly the time spent above 90% of VO_{2max}, may have been insufficient to induce IMF [72]. Moreover, the exact proportion of persons who develop exercise-induced IMF in this population remains unclear.

Furthermore, no significant changes in exercise-induced IMF were found at the group level after the training program, although substantial interindividual variability was observed. Since previous research showed that greater exercise-induced IMF correlates with higher VO_{2max}, higher workload achieved during the cardiopulmonary exercise test, and longer duration of the cardiopulmonary exercise test [35], it could be expected that those with larger improvements in VO_{2max} would also exhibit more IMF. However, our study found no relationship between changes in IMF and VO_{2max}. Interestingly, we did find that larger increases in exercise-induced IMF were related to larger decreases in trait anxiety (i.e. anxiety as a personal characteristic), and that larger increases in MIP were related to larger decreases in the severity of depressive symptoms. These exploratory associations suggest that psychological factors may be related to inspiratory muscle performance during maximal exercise. Despite all participants reaching an RER≥ 1.10, indicating maximal effort during the cardiopulmonary exercise test, anxiety and depression could still have affected the voluntary drive to the inspiratory muscles and the efficiency of their activation [73]. Anxiety has been shown to alter respiratory control by increasing both the central neural drive to respiratory muscles and modifying breathing patterns, often leading to faster and more superficial breathing at rest or during anticipation of dyspnea [73]. This can result in increased inspiratory muscle recruitment and heightened perception of breathlessness, even in the absence of physiological necessity, likely mediated by limbic system activation influencing brainstem respiratory centers [74]. However, during exercise, decreases in anxiety or depression could potentially facilitate improved breathlessness tolerance and greater willingness to sustain effort [75], potentially resulting in greater inspiratory muscle recruitment and fatigue. Nevertheless, given the low explained variance of changes in trait anxiety and depressive symptoms, and the limited number of participants with high levels of trait anxiety and depression, these findings should be interpreted with caution. It is likely that additional physical and psychological factors also contribute to changes in IMF.

Inspiratory muscle endurance

Inspiratory muscle endurance time increased significantly in the HIT group, but not in the MIT group, indicating that HIT is more effective in inducing endurance-related adaptations. HIT likely imposes a greater ventilatory load and respiratory effort than MIT. Previous research suggests that this may lead to more substantial diaphragm activation and fatigue during training [76], potentially driving peripheral adaptations such as increased mitochondrial density and capillarization, which improve endurance capacity independently of muscle hypertrophy [77]. Consistent with this, efforts sustained above a certain ventilatory threshold (e.g., ≥90% VO_{2max} for 8–10 minutes) are required to elicit these peripheral adaptations [72]. Our MIT group trained below this threshold, which may explain the absence of improvements in inspiratory muscle endurance. Additionally, because inspiratory muscle endurance was assessed using a voluntary task, motivational and perceptual factors may have influenced the performance [43]. Participants in the HIT group may have developed greater tolerance to discomfort, stronger perceived competence, and more positive affective responses to exercise, all of which could improve task engagement and effort [78]. Moreover, repeated exposure to high breathing loads during HIT may reduce the perceived difficulty of respiratory effort [79], further supporting the potential advantage of HIT in improving inspiratory muscle endurance.

Inspiratory muscle activation

Normalized mean $sEMG_{scm}$ and $sEMG_{scal}$ amplitude during postural control did not change in either group. To date, no studies have specifically examined treatment effects on EMG activity of these inspiratory muscles during postural

control in persons with CNSLBP, limiting direct comparisons with existing literature. However, previous research suggests that EMG amplitude changes are most often observed following interventions that specifically target the muscles in question. For example, inspiratory muscle training has been shown to increase inspiratory muscle EMG amplitude [80]. Our multimodal exercise therapy program may therefore not have been sufficient to induce such amplitude changes. Furthermore, this study focused on EMG amplitude of inspiratory muscles during postural control. However, since the diaphragm plays a crucial role in postural control via feedforward mechanisms [19], it is important to consider other EMG parameters as well. Persons with CNSLBP often show impairments in anticipatory postural adjustments [81], which may be due to delayed muscle activation during postural control tasks. Interestingly, previous research has shown that both specific and general exercise interventions can improve timing-related EMG parameters in persons with chronic low back pain [82]. Therefore, future studies should investigate whether different exercise modalities or intensities affect timing aspects of inspiratory muscle activation during postural tasks.

Pain and disability

Disability and pain intensity improved in both groups, but substantial interindividual variability in training responses was observed. Notably, improvements in disability and pain were not significantly associated with improvements in inspiratory muscle function. One possible explanation is that the magnitude of change in inspiratory muscle function achieved through multimodal training may not have been sufficient to establish such a relationship. In contrast, previous research demonstrated that high-intensity inspiratory muscle training led to substantially greater improvements in inspiratory muscle strength, which were associated with reductions in pain intensity in persons with recurrent low back pain [57]. A recent systematic review and meta-analysis found that combining inspiratory muscle training with whole-body exercise does not provide extra benefits regarding disability or pain intensity [83]. This supports the notion that improvements in inspiratory muscle function alone may not be a key driver of improvements in disability and pain intensity after multimodal whole-body HIT and MIT, suggesting that other underlying mechanisms might contribute to these benefits.

Interindividual variability and future directions

We observed considerable variability in individual responses to the training program, both in terms of changes in inspiratory muscle function and in secondary outcomes, with the standard deviation of the change scores exceeding the mean change for all outcome measures. Such interindividual variability is a well-recognized challenge in exercise therapy research and contributes to the modest average treatment effects commonly reported in the literature [7]. One possible explanation is that our study did not have specific inclusion criteria related to inspiratory muscle dysfunction or disability levels, which may have contributed to the heterogeneity of the sample and may have influenced the potential for change. Given the heterogeneous nature of CNSLBP, it is important that future studies move beyond group-level analyses and focus more explicitly on individual response patterns. Identifying the characteristics of persons who respond well to HIT could provide valuable insights into patient-specific treatment planning. Larger studies using methods such as cluster analysis could help to reveal meaningful subgroups with distinct clinical profiles and therapy responses, ultimately contributing to more personalized and effective exercise interventions. In our study on multimodal whole-body HIT, the observed variability in changes in inspiratory muscle function and in secondary outcomes suggests that certain profiles, such as persons with predominantly inspiratory muscle dysfunctions or psychological comorbidities, might benefit more from this intervention. Such individualized strategies are in line with precision rehabilitation, which aims to match interventions to patient-specific characteristics to optimize outcomes.

Limitations

Several limitations must be considered when interpreting our findings. The recruitment strategy may have introduced selection bias, as persons with positive beliefs about physical activity were possibly more likely to participate. To reduce motivational bias, participants were blinded to the study hypotheses and outcomes. In addition, therapists and outcome assessors were not blinded, which may have introduced performance and detection bias, particularly for outcomes that depend on participant effort and interaction with the assessor (e.g., inspiratory muscle strength and endurance). However, standardized testing protocols, verbal instructions, and exercise fidelity checklists were implemented to minimize variability and reduce potential bias. Finally, the absence of a no-treatment control group limits our ability to account for the natural course of symptoms, although the long symptom duration of our sample (11.0 ± 7.8 years) suggests that major spontaneous recovery was unlikely. However, the primary aim of this study was to investigate the specific effect of training intensity within exercise therapy rather than the overall effectiveness of exercise compared to no treatment. Therefore, a MIT group was selected as the comparator, as it reflects current standard care and allows for a clinically meaningful comparison. In addition, ethical and feasibility considerations were taken into account, as withholding active treatment in persons with CNSLBP may reduce acceptability and increase dropout rates.

Conclusion

This study suggests that both multimodal whole-body HIT and MIT improve MIP, the primary outcome measure, whereas only HIT appeared to improve inspiratory muscle endurance time. Neither HIT nor MIT improved exercise-induced IMF or normalized mean $sEMG_{scm}$ and $sEMG_{scal}$ amplitude during postural control. Furthermore, increases in MIP were related to decreases in the severity of depressive symptoms and to smaller increases in exercise capacity, and increases in exercise-induced IMF related to decreases in trait anxiety. These relationships should be interpreted with caution due to the exploratory nature of these analyses and require confirmation in larger adequately powered studies. Future research should focus on optimizing the effects of HIT in persons with CNSLBP, particularly by including participants with inspiratory muscle dysfunctions and a higher disorder profile (i.e., higher pain intensity, disability, and psychological burden). It is essential to move beyond group-level analyses and investigate individual response patterns to identify characteristics of responders to HIT, enabling more personalized treatment. Larger studies employing methods such as cluster analysis could uncover clinically meaningful subgroups with distinct profiles and therapy responses.

Abbreviations

BDI	Beck Depression Inventory
BMI	Body mass index
CNSLBP	Chronic nonspecific low back pain
CPET	Cardiopulmonary exercise test
EMG	Electromyography
HIT	High-intensity training
IME	Inspiratory muscle endurance
IMF	Inspiratory muscle fatigue
MIP	Maximal inspiratory pressure
MIPabs	Absolute maximal inspiratory pressure values
MIPpred	Predicted maximal inspiratory pressure values
MIT	Moderate-intensity training
MODI	Modified Oswestry Disability Index
MVC	Maximal voluntary contraction
NPRS	Numeric Pain Rating Scale
$sEMG_{scm}$	Electromyography of sternocleidomastoid muscle
$sEMG_{scm}$	Electromyography of scalene muscles
STAI	State-Trait Anxiety Inventory
VO_{2max}	Maximal oxygen uptake
1RM	One-repetition maximum

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s13102-026-01773-y.

Additional file 1.

Additional file 2.

Additional file 3.

Acknowledgements

The authors wish to thank the participants for their time and effort. The results of the study are presented clearly, honestly and without fabrication, falsification, or inappropriate data manipulation.

Research support

This work was supported by the Special Research Fund of Hasselt University (grant number: BOF21DOC15), and by Research Foundation Flanders (FWO-TBM T000822N, FWO-grant G0A1N24N).

Relationships

There are no additional relationships to disclose.

Patents and Intellectual Property

There are no patents to disclose.

Other activities

There are no additional activities to disclose.

Authors' contributions

The authors' responsibilities were as follows: **Sim Klaps:** Conceptualization, Methodology, Data collection, Data analysis, Writing - original draft preparation. **Jonas Verbrugghe:** Conceptualization, Methodology, Writing - reviewing and editing. **Nina Goossens:** Conceptualization, Methodology, Writing - reviewing and editing. **Sofie Van Wesemael:** Data collection, Writing -reviewing and editing. **Bjorn Van Eecke:** Data processing, Writing - reviewing and editing. **Albère Köke:** Conceptualization, Writing - reviewing and editing. **Jeanine Verbunt:** Conceptualization, Writing - reviewing and editing. **Daniel Langer:** Conceptualization, Writing - reviewing and editing. **Lotte Janssens:** Conceptualization, Methodology, Supervision, Writing - reviewing and editing. **Annick Timmermans:** Conceptualization, Methodology, Supervision, Writing - reviewing and editing.

Funding

This work was supported by the Special Research Fund of Hasselt University (grant number: BOF21DOC15), and by Research Foundation Flanders (FWO-TBM T000822N, FWO-grant G0A1N24N). The funding body had no role in the study design; data collection, analysis, or interpretation; or in the writing of the manuscript.

Data availability

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Human ethics and consent to participate

The study was approved by the Medical Ethical Committee of Hasselt University (B1152021000029), and written informed consent to participate was obtained from all participants before the start of the study.

Consent for publication

Written informed consent for publication of anonymized images was obtained. All images were blinded to prevent individual identification.

Competing interests

The authors declare that they have no competing interest

Author details

[1]UHasselt, REVAL Rehabilitation Research Center, Diepenbeek, Belgium
[2]Adelante Centre of Expertise in Rehabilitation and Audiology, Hoensbroek, The Netherlands
[3]Department of Rehabilitation Medicine, Research school CAPHRI, Maastricht University, Maastricht, The Netherlands
[4]Department of Rehabilitation Sciences, Faculty of Movement and Rehabilitation Sciences, Research Group for Rehabilitation in Internal Disorders, KU Leuven, Leuven, Belgium
[5]Respiratory Rehabilitation and Respiratory Division, University Hospital Leuven, Leuven, Belgium

Received: 26 February 2026 / Accepted: 25 May 2026
Published online: 09 June 2026

References

1. Hartvigsen J, et al. What low back pain is and why we need to pay attention. Lancet. 2018;391:2356–67.
2. Ferreira ML, et al. Global, regional, and national burden of low back pain, 1990–2020, its attributable risk factors, and projections to 2050: a systematic analysis of the Global Burden of Disease Study 2021. Lancet Rheumatol. 2023;5:e316–29.
3. Maher C, Underwood M, Buchbinder R. Non-specific low back pain. Lancet. 2017;389:736–47.
4. Meucci RD, Fassa AG, Faria NM. Prevalence of chronic low back pain: systematic review. Rev Saude Publica. 2015;49:73.
5. Green BN, et al. A scoping review of biopsychosocial risk factors and co-morbidities for common spinal disorders. PLoS ONE. 2018;13:e0197987.
6. Oliveira CB, et al. Clinical practice guidelines for the management of non-specific low back pain in primary care: an updated overview. Eur Spine J. 2018;27:2791–803.
7. Hayden JA, Ellis J, Ogilvie R, Malmivaara A, van Tulder MW. Exercise therapy for chronic low back pain. Cochrane Database of Systematic Reviews. 2021.
8. Owen PJ, et al. Which specific modes of exercise training are most effective for treating low back pain? Network meta-analysis. Br J Sports Med. 2020;54:1279–87.
9. Batacan RB, Duncan MJ, Dalbo VJ, Tucker PS, Fenning AS. Effects of high-intensity interval training on cardiometabolic health: a systematic review and meta-analysis of intervention studies. Br J Sports Med. 2017;51:494–503.
10. MacInnis MJ, Gibala MJ. Physiological adaptations to interval training and the role of exercise intensity. J Physiol. 2017;595:2915–30.
11. Wewege M, van den Berg R, Ward RE, Keech A. The effects of high-intensity interval training vs. moderate-intensity continuous training on body composition in overweight and obese adults: a systematic review and meta-analysis. Obesity Reviews. 2017;18 635–646 Preprint at https://doi.org/10.1111/obr.12532.
12. Lopez P. et al. Resistance Training Load Effects on Muscle Hypertrophy and Strength Gain: Systematic Review and Network Meta-analysis. Medicine and Science in Sports and Exercise. 2021. vol. 53 1206–1216 Preprint at https://doi.org/10.1249/MSS.0000000000002585.
13. Ram AK, Summers SJ, Booth J, Gibbs MT, Jones MD. Higher intensity exercise reduces disability more than lower intensity exercise in adults with chronic low back pain: a systematic review and meta-analysis. Musculoskelet Care. 2023. https://doi.org/10.1002/msc.1734.
14. Klaps S. et al. The influence of exercise intensity on psychosocial outcomes in musculoskeletal disorders: a systematic review. Sports Health. 2022. 19417381221075350.
15. Verbrugghe J, et al. Exercise intensity matters in chronic nonspecific low back pain rehabilitation. Med Sci Sports Exerc. 2019;51:2434–42.
16. O'Sullivan PB, et al. Cognitive functional therapy: an integrated behavioral approach for the targeted management of disabling low back pain. Phys Ther. 2018;98:408–23.
17. Kocjan J, Adamek M, Gzik-Zroska B, Czyżewski D, Rydel M. Network of breathing. Multifunctional role of the diaphragm: a review. Adv Respir Med. 2017;85:224–32.
18. Hodges PW, Eriksson AEM, Shirley D, Gandevia SC. Intra-abdominal pressure increases stiffness of the lumbar spine. J Biomech. 2005;38:1873–80.
19. Hodges PW, Butler JE, McKenzie DK, Gandevia SC. Contraction of the human diaphragm during rapid postural adjustments. J Physiol. 1997;505:539.
20. Hodges PW, Gandevia SC. Activation of the human diaphragm during a repetitive postural task. J Physiol. 2000;522:165–75.
21. Vostatek P, Novak D, Rychnovský T, Rychnovská Š. Diaphragm postural function analysis using magnetic resonance imaging. PLoS ONE. 2013;8:e56724.
22. Kolář P, et al. Postural function of the diaphragm in persons with and without chronic low back pain. J Orthop Sports Phys Ther. 2012;42:352–62.
23. Bunphrom W, et al. Diaphragm excursion and thickness in patients with chronic low back pain with and without lumbar instability. Sci Rep. 2025;15:9353.
24. Vermeersch A, et al. Preliminaire studie over het belang van de opsporing van ademhalingsproblemen bij patiënten met lage rugpijn. Tijdschr Geneeskd. 2017;73:529–40.
25. Shah SG, Choezom T, Raja GP. Comparison of respiratory parameters in participants with and without chronic low back pain. J Bodyw Mov Ther. 2019;23:894–900.
26. Janssens L, et al. Greater diaphragm fatigability in individuals with recurrent low back pain. Respir Physiol Neurobiol. 2013;188:119–23.
27. Perez AM, et al. Diaphragmatic activation correlated with lumbar multifidus muscles and thoracolumbar fascia by B-mode and M-mode ultrasonography in subjects with and without non-specific low back pain: a pilot study. Medicina (B Aires). 2023;59:315.
28. Janssens L, Brumagne S, Polspoel K, Troosters T, McConnell A. The effect of inspiratory muscles fatigue on postural control in people with and without recurrent low back pain. Spine (Phila Pa 1976). 2010;35:1088–94.
29. Ruhe A, Fejer R, Walker B. Center of pressure excursion as a measure of balance performance in patients with non-specific low back pain compared to healthy controls: a systematic review of the literature. Eur Spine J. 2011;20:358–68.
30. Claeys K, et al. Young individuals with a more ankle-steered proprioceptive control strategy may develop mild non-specific low back pain. J Electromyogr Kinesiol. 2015;25:329–38.
31. Janssens L, et al. The effect of acute respiratory demand on postural control: a systematic review. Gait Posture. 2024;113:310–8.
32. Hodges PW, Heijnen I, Gandevia SC. Postural activity of the diaphragm is reduced in humans when respiratory demand increases. J Physiol. 2001;537:999–1008.
33. Wong M, Formiga M, Spielholz N, Cheng S, Cahalin L. Respiratory muscle strength is significantly reduced and related to perceived disability in patients with low back pain. Preprint at 2017.
34. Jafari H, Courtois I, Van den Bergh O, Vlaeyen JWS, Van Diest I. Pain and respiration: a systematic review. Pain. 2017;158:995–1006.
35. Klaps S, et al. Maximal inspiratory pressure and exercise-induced inspiratory muscle fatigue in chronic nonspecific low back pain. Respir Med. 2025;236:107887.
36. Vlemincx, E. Dysfunctional breathing: a dimensional, transdiagnostic perspective. 2023;61 Preprint at.
37. Dunham C, Harms CA. Effects of high-intensity interval training on pulmonary function. Eur J Appl Physiol. 2012;112:3061–8.
38. Abrahin O, et al. Single-and multiple-set resistance training improves skeletal and respiratory muscle strength in elderly women. Clin Interv Aging. 2014;16:1775–82.
39. DePalo VA, Parker AL, Al-Bilbeisi F, McCool FD. Respiratory muscle strength training with nonrespiratory maneuvers. J Appl Physiol. 2004;96:731–4.
40. Slade SC, Dionne CE, Underwood M, Buchbinder R. Consensus on exercise reporting template (CERT): explanation and elaboration statement. Br J Sports Med. 2016;50:1428–37.
41. Amundsen PA, et al. Inclusion and exclusion criteria used in non-specific low back pain trials: a review of randomised controlled trials published between 2006 and 2012. BMC Musculoskelet Disord. 2018;19:1–13.
42. An M, Dusing SC, Harbourne RT, Sheridan SM, START-Play Consortium. What really works in intervention? Using fidelity measures to support optimal outcomes. Phys Ther. 2020;100:757–65.
43. Laveneziana P, et al. ERS statement on respiratory muscle testing at rest and during exercise. Eur Respir J. 2019;53:1801214.
44. Black LF, Hyatt RE. Maximal respiratory pressures: normal values and relationship to age and sex. Am Rev Respir Dis. 1969;99:696–702.
45. Lista-Paz A, et al. Maximal respiratory pressure reference equations in healthy adults and cut-off points for defining respiratory muscle weakness. Arch Bronconeumol. 2023;59(12):813–20.
46. Azevedo IG, et al. Relationship between inspiratory muscle strength and balance in women: a cross-sectional study. PLoS ONE. 2023;18:e0280465.
47. Verges S, Lenherr O, Haner AC, Schulz C, Spengler CM. Increased fatigue resistance of respiratory muscles during exercise after respiratory muscle endurance training. Am J Physiol Regul Integr Comp Physiol. 2007;292:R1246–53.
48. Klaps S, et al. The value of extra-diaphragmatic inspiratory muscle surface electromyography during postural control tasks in patients with chronic obstructive pulmonary disease. Respir Med. 2025;243:108127.
49. Gallina A, et al. Consensus for experimental design in electromyography (CEDE) project: high-density surface electromyography matrix. J Electromyogr Kinesiol. 2022;64:102656.
50. Besomi M, et al. Consensus for experimental design in electromyography (CEDE) project: amplitude normalization matrix. J Electromyogr Kinesiol. 2020;53:102438.
51. Denteneer L, et al. The modified low back pain disability questionnaire: reliability, validity, and responsiveness of a Dutch language version. Spine (Phila Pa 1976). 2018;43:E292–8.
52. Stanhope J. Brief pain inventory review. Occup Med (Chic Ill). 2016;66:496–7.
53. Boonstra AM, et al. Cut-off points for mild, moderate, and severe pain on the numeric rating scale for pain in patients with chronic musculoskeletal

pain: variability and influence of sex and catastrophizing. Front Psychol. 2016;7:1466.
54. Van der Ploeg HM. Validity of the Zelf-Beoordelings-Vragenlijst (A Dutch version of the Spielberger State-Trait Anxiety Inventory). Ned. Tijdschr. Psychol. 1980.
55. Roelofs J, et al. Norms for the Beck Depression Inventory (BDI-II) in a large Dutch community sample. J Psychopathol Behav Assess. 2013;35:93–8.
56. Pescatello LS. ACSM's guidelines for exercise testing and prescription. Lippincott Williams & Wilkins; 2014.
57. Janssens L, et al. Inspiratory muscle training affects proprioceptive use and low back pain. Med Sci Sports Exerc. 2015;47:12–9.
58. Myers J, et al. Exercise capacity and mortality among men referred for exercise testing. N Engl J Med. 2002;346:793–801.
59. Iwakura M, et al. Estimation of minimal clinically important difference for quadriceps and inspiratory muscle strength in older outpatients with chronic obstructive pulmonary disease: a prospective cohort study. Phys Ther Res. 2021;24:35–42.
60. Mcgough JJ, Faraone SV. Estimating the size of treatment effects: Moving Beyond P Values. (2009).
61. Bursac Z, Gauss CH, Williams DK, Hosmer DW. Purposeful selection of variables in logistic regression. Source Code Biol Med. 2008;3:17.
62. Austin PC, Steyerberg EW. The number of subjects per variable required in linear regression analyses. J Clin Epidemiol. 2015;68:627–36.
63. Ren Z, Guo J, He Y, Luo Y Wu, H. Effects of Inspiratory Muscle Training on Respiratory Muscle Strength, Lactate Accumulation and Exercise Tolerance in Amateur Runners: A Randomized Controlled Trial. . 15, 705 (2025).
64. Archiza B, et al. Effects of inspiratory muscle training in professional women football players: a randomized sham-controlled trial. J Sports Sci. 2018;36:771–80.
65. Finta R, Nagy E, Bender T. The effect of diaphragm training on lumbar stabilizer muscles: a new concept for improving segmental stability in the case of low back pain. J Pain Res. 2018;28:3031–45.
66. Polkey MI, Moxham J. Improvement in volitional tests of muscle function alone may not be adequate evidence that inspiratory muscle training is effective. 23:5–6 Preprint at (2003).
67. Paz AL, Barral-Fernandez M, Van Baelen, L, Holmstock L, Langer, D. Learning effect of respiratory muscle endurance tests in healthy adults. Preprint at (2024).
68. Schaun GZ, Pinto SS, Brasil B, Nunes GN, Alberton CL. Neuromuscular adaptations to sixteen weeks of whole-body high-intensity interval training compared to ergometer-based interval and continuous training. J Sports Sci. 2019;37:1561–9.
69. Lötters F, Van Tol B, Kwakkel G, Gosselink R. Effects of controlled inspiratory muscle training in patients with COPD: a meta-analysis. Eur Respir J. 2002;20:570–7.
70. McConnell AK, Caine MP, Sharpe GR. Inspiratory muscle fatigue following running to volitional fatigue: the influence of baseline strength. Int J Sports Med. 1997;18:169–73.
71. Archiza B, et al. Temporal characteristics of exercise-induced diaphragmatic fatigue. J Appl Physiol. 2018;124:906–14.
72. Smith JR, et al. Influence of exercise intensity on respiratory muscle fatigue and brachial artery blood flow during cycling exercise. Eur J Appl Physiol. 2014;114:1767–77.
73. Westbrook J, MacBean V. The influence of affective state on respiratory muscle activity. Clin Physiol Funct Imaging. 2019;39:291–5.
74. Esser RW, et al. Brain activation during perception and anticipation of dyspnea in chronic obstructive pulmonary disease. Front Physiol. 2017;8:617.
75. Currow DC, et al. Breathlessness, anxiety, depression, and function–the BAD-F study: a cross-sectional and population prevalence study in adults. J Pain Symptom Manage. 2020;59:197-205. e2.
76. Romer LM, Polkey MI. Exercise-induced respiratory muscle fatigue: implications for performance. J Appl Physiol. 2008;104:879–88.
77. Mølmen KS, Almquist NW, Skattebo Ø. Effects of exercise training on mitochondrial and capillary growth in human skeletal muscle: a systematic review and meta-regression. Sports Med. 2025;55:115–44.
78. Thum JS, Parsons G, Whittle T, Astorino TA. High-intensity interval training elicits higher enjoyment than moderate intensity continuous exercise. PLoS ONE. 2017;12:e0166299.
79. Verges S, Boutellier U, Spengler CM. Effect of respiratory muscle endurance training on respiratory sensations, respiratory control and exercise performance: a 15-year experience. Respir Physiol Neurobiol. 2008;161:16–22.
80. Ando R, et al. Effect of inspiratory resistive training on diaphragm shear modulus and accessory inspiratory muscle activation. Appl Physiol Nutr Metab. 2020;45:851–6.
81. de Voogd JN, Sanderman R, Postema K, van Sonderen E, Wempe JB. Relationship between anxiety and dyspnea on exertion in patients with chronic obstructive pulmonary disease. Anxiety Stress Coping. 2011;24:439–49.
82. Brooks C, Kennedy S, Marshall PWM. Specific trunk and general exercise elicit similar changes in anticipatory postural adjustments in patients with chronic low back pain: a randomized controlled trial. Preprint at (2012).
83. Van Wesemael S. et al. The effect of different types of breathing interventions on spinal pain. (2025).

Publisher's Note