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Causal mechanisms of a scapular stabilization intervention for patients with subacromial pain syndrome: a secondary analysis of a randomized controlled trial

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Abstract

Background: Causal mediation analysis is one way to bridge this gap by exploring the causal pathways of a given intervention. The aim of this study was to assess whether scapular motion, position, and periscapular muscle strength are mediators for pain and shoulder disability outcomes following a scapular stabilization intervention for patients with subacromial pain syndrome.

Methods: Sixty patients were randomized into two groups: scapular stabilization or periscapular strengthening exercises. The intervention consisted of three sessions per week for 8 weeks. The primary outcome measures were pain and disability and the following outcome measures were considered as potential mediators: scapular motion, scapular position, periscapular muscle strength, age, duration of symptoms, and side of the complaint. A model-based inference approach with bootstrap simulations was used to estimate the average causal mediation effect, average direct effect, and the average total effect from the data of a randomized clinical trial that evaluated the effect of adding scapular stabilization exercises to a scapulothoracic strengthening program in people with subacromial pain syndrome.

Results: The results demonstrated that none of the putative mediators were influenced by the intervention. However, muscle strength of serratus anterior, upper, middle, and lower trapezius muscles was associated with shoulder disability.

Conclusion: Scapular kinematic and periscapular muscle strength did not mediate the effect of scapular stabilization exercises on shoulder pain or disability scores in subjects with subacromial pain syndrome. Muscle strength of serratus anterior, upper, middle and lower trapezius were associated with shoulder disability scores at 8-weeks follow-up.

Keywords: Shoulder pain, Mediation analysis, Prognosis analysis, Rehabilitation

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What's already known about this topic?

- Causal mediation analysis provides information about the mechanisms though which an intervention may or may not work.

- To date, there are no mediation studies investigating the causal pathway though which scapular-focused interventions may work in people with shoulder pain.

What does this study add?

- Scapular kinematics and periscapular muscle strength does not seem to be mediators of effect of a scapular-focused intervention.

- Future studies should investigate the role of already known biopsychossocial variables as putative mediators of effect of exercise-based interventions for people with shoulder pain.

Background

Subacromial pain syndrome (SAPS) is the most prevalent disorder in the shoulder region, accounting for up to 50–70% of all shoulder complaints in primary care [1]. Exercises are frequently indicated as the first line of treatment for improving pain and disability in people with SAPS, and usually include a combination of scapular stabilization exercises, rotator cuff strengthening and stretching [2, 3]. However, the causal mechanisms associated with exercise-based interventions for shoulder pain complaints are not fully understood.

The scientific literature suggests that an adequate scapular motion may be crucial to shoulder function [4, 5]. Subjects with SAPS present less scapular upward rotation and increased scapular internal rotation [6, 7] and anterior tilt [8] than asymptomatic subjects. Recently, the use of better statistical tools for biomechanical data analysis (i.e. Principal Component Analysis) showed differences in scapular anterior/posterior tilt and forward/backward translation in subjects with SAPS [9]. Despite the lack of high-quality evidence to support a causal association between these biomechanical changes with pain and disability in people with SAPS, it is believed that scapular motion impairments contribute to the development and maintenance of shoulder pain [4], as highlighted by a consensus statement from 2013 [10] and by the Movement System Diagnosis framework [10, 11]. Therefore, interventions for subjects with shoulder pain commonly focus on improving scapular movement [10, 11]. However, although there is some emerging evidence to support scapular-focused exercises for reducing shoulder pain and disability in the short-term [12–14], most of the available studies have a high risk of bias. This is why all available systematic reviews recommended that more trials with better methodologies and larger sample sizes were needed to provide more quality of evidence about the efficacy of scapular stabilization exercises in people with SAPS [15, 16].

Considering the limitations of those studies, we conducted a randomized controlled trial (RCT) to evaluate the incremental effect of scapular stabilization exercises to a scapulothoracic strengthening program in people with SAPS [17]. The results of this study showed that no effects were found when added a scapular stabilization exercise to a scapulothoracic strengthening program for patients with shoulder pain on pain and shoulder disability. These results bring into question the role of scapula on symptoms improvement in patients with SAPS.

Traditional RCTs analyses do not provide insight into the mechanisms through which an intervention works, given that its focus is to identify between-group differences in clinical outcomes [18, 19]. Causal mediation analysis (CMA) is one way to bridge this gap by exploring the causal pathways of a given intervention [18]. Therefore, the main goal of this study was to verify why the intervention did not worked by analyzing the effect of the scapular stabilization exercises on scapular motion, position and muscle strength, as well as to verify the effect of these intermediate variables on the outcomes using CMA.

Methods
Participants
The randomized controlled trial involved 60 individuals with SAPS, recruited from March 2016 to June 2017. Patients were referred by an orthopedic physician to physiotherapy due to shoulder pain. We included patients with positive results for 3 out of 5 SAPS tests: Neer, Hawkins-kennedy, painful arc, pain or weakness resistant to external rotation and Jobe [20]. The exclusion criteria were: history of shoulder trauma or surgery; total rotator cuff or biceps brachii tendon rupture (imaging exam or self-report); sports activities involving the upper limbs; individuals with neurological disorders and alterations in cognitive function (e.g., stroke, epilepsy, multiple sclerosis, Parkinson's disease, and peripheral neuropathy); shoulder pain for primary involvement in the cervical or thoracic region; systemic disease involving the joints (e.g., rheumatoid arthritis); carpal tunnel syndrome; and underwent physiotherapeutic treatment of the shoulder in the last 6 months [17]. A detailed description of the trial was presented by Hotta et al. [17] (clinicaltrials.gov: NCT02695524). A brief overview of the trial is presented below.

Randomization and interventions
Participants included in the study were randomized into two groups: periscapular strengthening and scapular

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stabilization exercises. Interventions were carried out for 8 weeks, three times a week, on non-consecutive days. Each session lasted 50 min and individuals were treated separately. Participants assigned to the periscapular strengthening group (PSG) performed only six exercises of periscapular strengthening (upper trapezius, middle trapezius, lower trapezius, and serratus anterior). Participants allocated to the scapular stabilization group (SSG) performed the same six periscapular strengthening exercises applied to PSG, and six scapular stabilization exercises, emphasizing retraction and depression of the scapula, were added to this group [17].

Assessment time points
Patients characteristics, outcome measures, primary and alternative mediators, and potential confounders were measured at baseline prior to randomization. The putative mediators were measured after 4 weeks of the beginning of the treatment. Outcomes were measured right after the end of the treatment (i.e. 8 weeks).

Primary outcome measures
Shoulder disability was assessed by The Shoulder Pain and Disability Index (SPADI). The SPADI is valid and reliable (Cronbach Alpha = 0.89) for the assessment of individuals with shoulder disorders (Martins et al., 2010). The score of the questionnaire ranges from 0 to 100 points, and higher scores indicate higher disability [21]. The minimal clinically important difference (MCID) considered for the questionnaire was 10 points [22].

Pain intensity was assessed by the 0–10 numerical pain rating scale [23, 24]. Changes of 15 to 20% from baseline values were considered clinically relevant [25].

Putative mediators
The primary mediators were scapular motion and position, measured through a digital inclinometer (Lafayette®, Lafayette Instrument Company, Ind., USA) and expressed in degrees. We measured scapular upward/downward rotations and anterior/posterior tilt at rest (scapular position), 90° and 180° of arm elevation (scapular motion). Scapular upward/downward rotations and anterior/posterior tilt measurements presented intra-rater reliability ranging from good to excellent, with standard error of measurement ranging 2 to 2.8 degrees [26, 27] and criterion validity ranging from good to excellent [26, 28]. Other mediators included muscle strength of serratus anterior, upper, middle, and lower trapezius [29]. The evaluation of the isometric strength was performed using a portable dynamometer that has excellent reliability [26, 27] (Lafayette®, Lafayette Instrument Company, Ind., USA).

Potential confounders
We assumed that both the intervention-mediator and intervention-outcome paths were not confounded due to randomization. Given the mediator cannot be randomized, we assumed that the mediator-outcome path might be confounded by pain duration, age, sex and baseline measures of mediators and outcomes. Therefore, we controlled the analyses in the outcome regression models for pain duration, age, sex and baseline measures of mediators and outcomes. Besides that, the outcome regression models for pain and disability, with scapular position and motion as mediators, were also controlled for muscle strength and all outcome models were controlled for the dominant side of complaint. The hypothesized causal pathways are presented in Figs. 1 and 2.

Sample size
The sample size for the main trial was estimated to identify clinically meaningful between-group differences in shoulder function, considering a minimal clinically important difference of 10 points in the global SPADI score, with alpha set at 0.05, power 80%, and a 20% sample loss. The minimum sample size for the original trial was 30 on each intervention arm. The trial had no dropouts, with 60 participants (30 each group) completing the study.

We conducted a post hoc power calculation as suggest by the literature [30, 31] using the estimator for joint indirect effect developed by Vittinghoff and Neilands [32]. We performed two sample size estimations: (1) one assuming a large treatment-mediator and mediator-outcome effect (r = 0.65); (2) another assuming a moderate treatment-mediator and mediator-outcome effect (r = 0.3). The remaining variables were kept the same for both analysis, and were as follows: absence of exposure-mediator confounding (i.e. error term correlation coefficient = 0.0), given the design of the study (i.e. clinical trial); a moderate confounding for the mediator-outcome (r = 0.3) as suggested by Vittinghoff and Neilands [32]; with power set at 0.8. The coefficients were standardized.
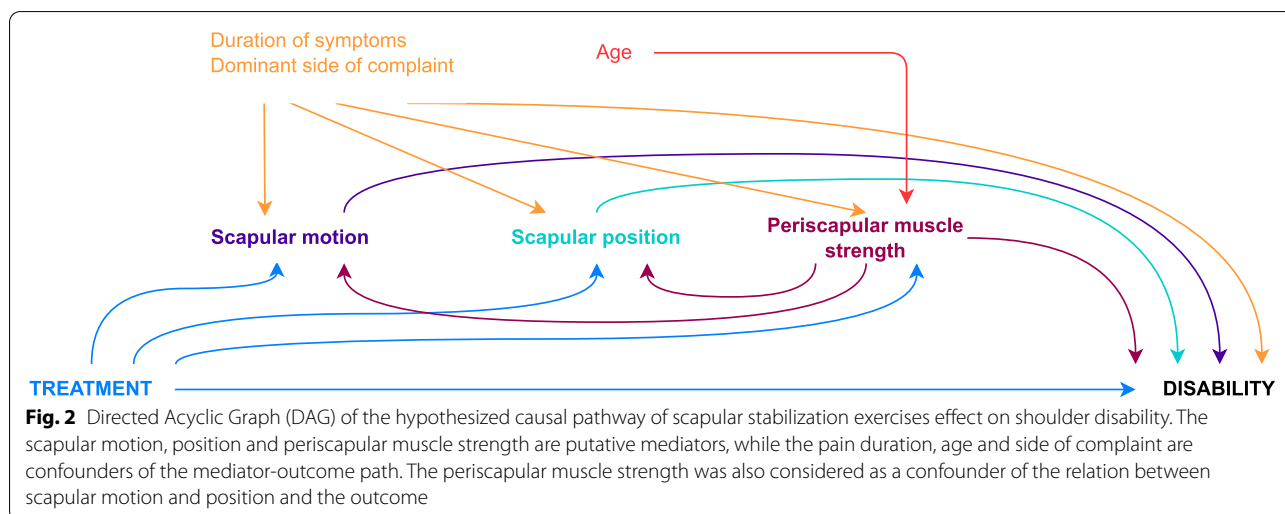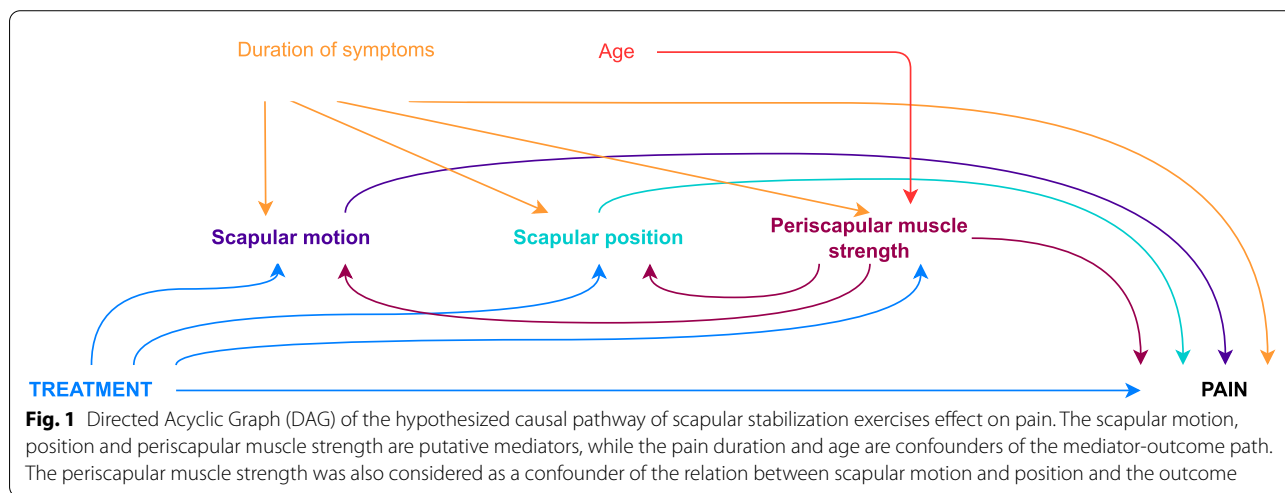
Data analysis
Analyses were performed in R (The R Foundation for Statistical Computing). The causal mediation analysis was performed using the *mediation* package [33]. A model-based inference approach was used to estimate the average causal mediation effect (ACME), average direct effect (ADE) and the average total effect (Fig. 3) [33, 34]. Two regression models were created: the mediator model and the outcome model. As there was no total effect, we decided to conduct several univariate mediation models to verify where the causal pathway break down. The mediator model was constructed with treatment

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allocation as the independent variable and the putative mediator as the dependent variable. The outcome model was constructed with the treatment allocation and the putative mediator as independent variables and the outcome as independent variable. The outcome models were adjusted for potential confounders. Continuous mediators and outcomes that were normally distributed were modelled using linear models (*lm*). However, if the data was skewed or the assumptions of the linear model were violated, mediators and outcomes were modelled using robust linear models[22] or generalized linear models (*glm*) with respective family and link function [35].

The *mediate* function was used to estimate the value of the mediator and outcome. The simulated potential values of the mediator and outcome was used to compute the ACME, ADE and average total effect. We used 1000 bootstrap simulations to generate 95% confidence intervals (95% CI) if linear assumptions of mediator and/ or outcome models were not violated. Non-parametric bootstrap simulations were used if the linear assumptions of the mediator and/or outcome models were violated.

We performed sensitivity analysis for unmeasured confounding to assess how a hypothetical level of unmeasured confounding would impact on ACME. We used the *medsens* function to explore the level of confounding due to unknown confounders from the mediator ant outcome models. The level of confounding [ρ (rho)] is represented by the correlation between the error terms from the mediator and outcome models. The level of confounding ranged from −1 to 1. A ρ = 0 suggests no correlation between error terms and can be interpreted as the absence of unmeasured confounding [34].

Fig. 1 Directed Acyclic Graph (DAG) of the hypothesized causal pathway of scapular stabilization exercises effect on pain. The scapular motion, position and periscapular muscle strength are putative mediators, while the pain duration and age are confounders of the mediator-outcome path. The periscapular muscle strength was also considered as a confounder of the relation between scapular motion and position and the outcome

Fig. 2 Directed Acyclic Graph (DAG) of the hypothesized causal pathway of scapular stabilization exercises effect on shoulder disability. The scapular motion, position and periscapular muscle strength are putative mediators, while the pain duration, age and side of complaint are confounders of the mediator-outcome path. The periscapular muscle strength was also considered as a confounder of the relation between scapular motion and position and the outcome

Results

The intervention had no additional effect on pain intensity and shoulder disability. The causal mediation analysis demonstrated that the intervention did not influence the primary mediators (ie. scapular motion and position) or the alternative mediators (ie. serratus anterior, upper, middle, and lower trapezius strength). The alternative mediators serratus anterior, upper, middle and lower trapezius strength were associated with shoulder disability.

The full description of the causal mediation analysis is described in Table 1.

The sensitivity analysis showed that our estimated causal mediation effects were stable across all possible levels of residual confounding for the pain models. The disability models were relatively unstable for some levels of residual confounding. The sensitivity analysis plots for each model are reported in Figures S1 and S2 in the Supplementary file.

The analyses of post hoc power calculation suggested that assuming large treatment-mediator and mediator-outcome effects, a minimum of 67 participants were required. Assuming moderate treatment-mediator and mediator-outcome effects, a minimum of 342 participants were required. Those analyses showed that our study is underpowered for detecting a large and moderate mediating effect. Considering that, the coefficients presented in Table 1 were standardized.
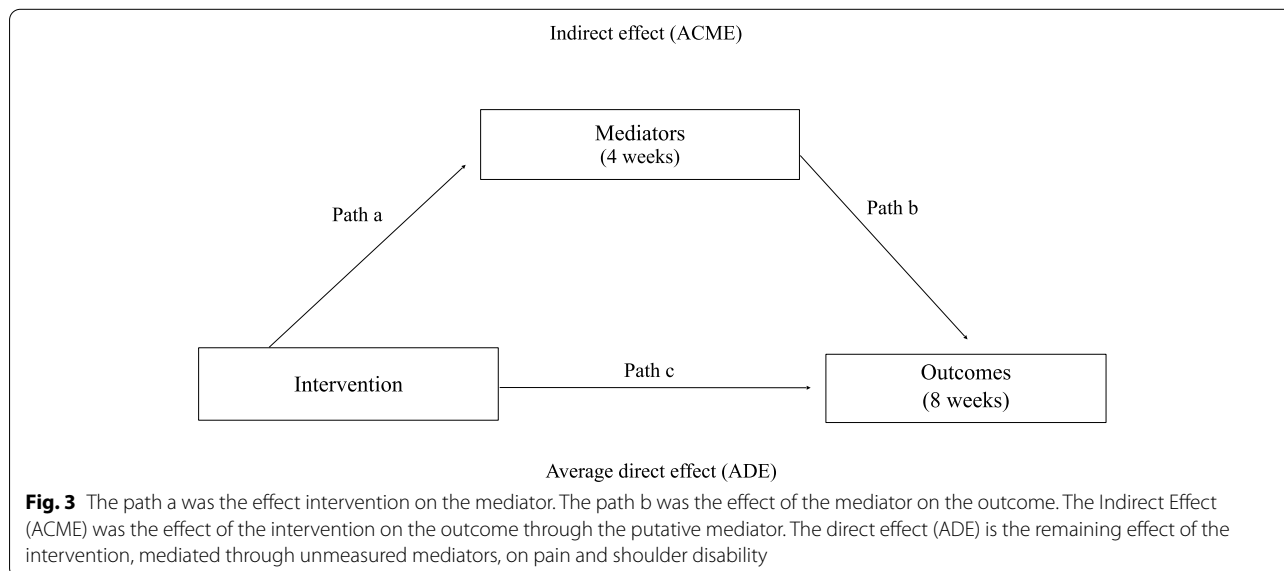
Discussion

The main purpose of this secondary analysis was to assess the mechanism of action of a scapular intervention program for patients with subacromial pain syndrome, considering scapular motion, position and periscapular muscle strength as possible mediators in people with SAPS. The results of the causal mediation analysis demonstrated that scapular motion, position and periscapular muscle strength were not influenced by the intervention (i.e., path a), while muscle strength of SA, UT, MT and LT was associated with shoulder disability (i.e., path b).

Our findings challenge the assumption that scapular-focused interventions may lead to better clinical outcomes by altering scapular movement pattern, position and muscle strength [13, 25, 36]. This assumption is partially supported by previous clinical trials that reported conflicting evidence that interventions targeting scapular movement may lead to better clinical outcomes [13, 14]. However, these previous clinical trials have high risk of bias due to sample size, lack of allocation concealment, heterogeneity in term of outcome measurement and lack of intention-to-treat analysis [13, 14]. Our findings suggest the path between intervention and mediators (i.e., scapular movement pattern, position, or muscle strength) were not significant. The causal paths between muscle strength and disability scores were unstable and may be biased by unmeasured mediator-outcome confounding.

Due to the multifactorial nature of SAPS and the uncertain relationship between pain and shoulder disability with movement and posture, some methodology for measuring scapular motion and position have been studied in the literature [5]. A recent cross-section study reported a fair association between improvement in shoulder function and improvement in scapular dyskinesis, and no association between pain and scapular dyskinesis [37]. Therefore, future studies should explore the causal effects between improvement in shoulder function and scapular dyskinesis, since we do not evaluate the presence of this condition in our participants.

Fig. 3 The path a was the effect intervention on the mediator. The path b was the effect of the mediator on the outcome. The Indirect Effect (ACME) was the effect of the intervention on the outcome through the putative mediator. The direct effect (ADE) is the remaining effect of the intervention, mediated through unmeasured mediators, on pain and shoulder disability

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The use of RCT design allows controlling for intervention-outcome confounding, but not for mediator-outcome confounding. The results of the Hotta et al. [17] clinical trial demonstrated that there were no differences between the group that received additional scapular stabilization exercises with a focus on scapular retraction and scapular depression movements when compared to the muscle strengthening control group. Therefore, despite the effect of the intervention on the mediators was not significant, the causal mechanism of scapular orientation and muscle strength with pain and disability remains uncertain.

Some authors have questioned the theoretical concept of scapular stabilization and the function of a "stable

Table 1 Effect decomposition for each single-mediator model

Analysis	Intervention-mediator effect (Path a)	Mediator-outcome effect (Path b)	ATE	ADE	ACME	Proportion Mediated (%)
Pain						
Scapular Position						
SUR 0°	0.01 (−1.9 to 2.9)	0.23 (0.02 to 0.45)	−0.34 (−1.89 to 1.24)	−0.36 (−2 to 1.29)	0.02 (−0.32 to 0.35)	−0.05 (−1.89 to 2)
SAT 0°	0.07 (−2.4 to 2.6)	0.01 (−0.14 to 0.18)	−0.31 (−1.85 to 1.44)	−0.32 (−1.9 to 1.4)	0.006 (−0.26 to 0.37)	−0.01 (−1.23 to 1.96)
Scapular Motion						
SUR 90°	0.5 (−3.2 to 3.3)	−0.06 (−0.18 to 0.05)	−0.01 (−0.3 to 0.12)	−0.24 (−1.82 to 1.48)	−0.26 (−1.84 to 1.49)	0.06 (−1.02 to 1.52)
SUR 180°	−0.04 (−4.8 to 4.7)	−0.19 (−0.28 to 0.1)	−0.63 (−2.4 to 1.9)	−0.7 (−2.26 to 1.18)	0.05 (−0.32 to 0.30)	−0.07 (−0.07 to 2.32)
SAT 90°	−0.16 (−2.51 to 2.47)	−0.13 (−0.3 to 0.03)	−0.25 (−1.97 to 1.43)	−0.26 (−2 to 1.93)	0.01 (−0.2 to 0.33)	0.004 (−1.5 to 2)
SAT 180°	0.09 (−4.2 to 4.4)	0.44 (−0.35 to 0.53)	−0.23 (−2 to 1.47)	−0.46 (−2 to 1)	0.23 (−0.17 to 1)	−0.99 (−4.3 to 3.91)
Muscle Strength						
SA	0.12 (−1.5 to 1.6)	−0.51 (−0.73 to 0.02)	−0.3 (−1.8 to 1)	−0.08 (−1.57 to 1.24)	−0.3 (−1.4 to 1.4)	0.3 (−3 to 4)
UT	0.09 (−1.5 to 1.7)	−0.31 (−0.55 to 0.06)	−0.25 (−0.2 to 0.2)	−0.19 (−0.3 to 0.7)	−0.9 (−0.1 to 0.09)	0.10 (−0.41 to 0.9)
MT	−0.03 (−0.68 to 0.7)	−0.19 (−0.28 to 0.1)	−0.05 (−1.65 to 1.4)	−0.6(−1.8 to 1.1)	0.36 (−0.2 to 1.2)	−0.10 (−9.68 to 6.8)
LT	−0.6 (−0.69 to 0.8)	−0.19 (−0.28 to 0.1)	−0.27 (−1.79 to 1.2)	−0.3 (−1.89 to 0.89)	0.2 (−0.27 to 0.71)	−0.63 (−3.88 to 4.59)
Shoulder Disability						
Scapular Position						
SUR 0°	0.01 (−1.9 to 2)	−0.04 (−1.6 to 1.5)	−0.3 (−2.2 to 3.3)	−0.42 (−2.27 to 1.32)	0.06 (−0.35 to 0.64)	0.03 (−1.33 to 1.19)
SAT 0°	0.07 (−2.4 to 2.6)	0.19 (−1 to 1)	5 (−6 to 16.2)	3 (−6.16 to 14.9)	0.8 (−1.6 to 5.3)	0.04 (−1.55 to 1.97)
Scapular Motion						
SUR 90°	0.49 (−3 to 3.38)	0.19 (−0.73 to 1.13)	6 (−6 to 10)	5 (−6 to 14.36)	0.6 (−3.4 to 4.59)	0.01 (−1.6 to 2.41)
SUR 180°	−0.04 (−4.8 to 4.7)	−0.14 (−0.84 to 0.55)	4.6 (−7 to 14)	4 (−8 to 13)	0.6 (−1.88 to 5.3)	0.05 (−1.55 to 1.97)
SAT 90°	−0.01 (−2.55 to 2.24)	0.31 (−0.17 to 2.47)	7 (−5.5 to 16)	6.9 (−5.5 to 16)	0.1 (−3.3 to 2.73)	0.02 (−1.97 to 2)
SAT 180°	0.09 (−4.2 to 4.4)	0.25 (−0.44 to 0.96)	6 (−5 to 16.3)	4 (−6 to 14)	1.08 (−5.82 to 6.44)	0.07 (−1.2 to 2)
Muscle Strength						
SA	0.12 (−1.56 to 1.8)	−0.64 (−2.2 to −0.97)***	1 (−9.44 to 13)	0.15 (−4 to 17)	−0.05 (−2 to 1.67)	−0.05 (−1 to 1.2)
UT	0.09 (−1.5 to 1.6)	−0.44 (−5 to −1.3)**	2.86 (−8 to 14)	5.1 (−5.15 to 17.3)	−2.3 (−7 to 2)	−0.8 (−9.78 to 12)
MT	−0.03 (−0.68 to 0.75)	−0.42 (−4.9 to −0.2)*	0.25 (−5.79 to 6)	1 (−10 to 12)	−0.8 (−4 to 2)	−3.2 (−3.3 to 3)
LT	−0.06 (−0.69 to 0.8)	−0.3 (−4 to −1)**	−0.2 (−5.79 to 6)	1 (−10.8 to 13)	−1.1 (−5 to 1)	0.5 (−3.3 to 3)

ATE Average total effect, *ADE* Average direct effect, *ACME* Average causal mediation effect, *SUR* Scapular upward rotation, *SAT* Scapular anterior tilt, *SA* Serratus anterior, *UTS* Upper trapezius, *MT* Middle trapezius, *LT* Lower trapezius

Standardized coefficients with their 95% confidence intervals unless otherwise stated

*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$

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base" for upper limb movement [38]. The "scapular stability" paradigm is considered flawed due to limitations with regards to the definition of concept of joint stability and the knowledge that shoulder movement is complex and may be better represented through a dynamic systems perspective [38, 39]. Considering that there is a greater number of muscles than degrees of freedom that involves the shoulder girdle and upper limb joints, to complete a task, from the point of view of dynamic systems theory, the central nervous system could use a variety of segmental coordination patterns, within an optimal range of variability [38, 39]. Thus, the prescription of exercises aimed to stabilize the scapula and influence its movement pattern, in patients with scapular dyskinesis, for instance, are being made in a completely arbitrary way, since the role of scapular motion and position on shoulder pain and disability is still not fully understood.

The motor control intervention proposed in our clinical trial [17] was based on scapular retraction and depression movements, one possible explanation for the absence of intervention-mediators effect upon scapular position is that this intervention did not offered adequate motor stimulus to induce changes in motor behavior. Experimental paradigms have evaluated movement variability, systematic movement error and motor action planning and defined that kinematic accuracy and learning have limited generalization and are encoded in extrinsic coordinates [40]. Internal learning models transform biomechanical properties (i.e. strain, stiffness) in motion and the ability to anticipate dynamic effects, such as torque [40]. Current literature suggests that motor learning and the consolidation of kinematics and dynamics occur independently [40] and that the generalization of motor learning is influenced not only by prediction errors, but also by the history of implicitly remembered contexts in which the training occurred [41]. Those principles from dynamic systems suggest that isolated scapular stabilization exercises are unlikely to change scapular movement pattern. A better approach would be to improve motor behavior by exposing patients to functional movements of the upper limb [38].

Limitations

This study had some limitations. Our main goal was to better understand the causal pathways through which a scapular stabilization program may have affected pain and disability scores. Therefore, one challenge of our study was to measure scapular movement and this is a common issue in the field. We measured two scapular rotations (upward-downward rotation, and anterior-posterior tilt) at different glenohumeral positions. Given the challenges in measuring scapular movement, we had to conduct several univariate causal mediation analyses

when assessing whether scapular movement mediated the effects of the treatment. The post hoc power calculation showed that we are underpowered for the mediation analysis, considering a large treatment effect. That increases the odds of Type 2 error. Nevertheless, when a causal question is important, it is preferable to have multiple studies with imprecise estimates than having no study at all [30]. Considering the limitations, this study can be a guide for the development of future studies with power in this area.

Conclusion

Scapular motion, position and periscapular muscle strength did not mediate the effect of scapular stabilization exercises on shoulder pain and disability in subjects with subacromial pain syndrome. Muscle strength of serratus anterior, upper, middle, and lower trapezius mediated changes in shoulder disability, but these estimates were unstable due to possible unmeasured mediator-outcome confounding.

Abbreviations
ACME: Average causal mediation effect; ADE: Average direct effect; CMA: Causal mediation analysis; DAG: Directed Acyclic Graph ; LT: Lower trapezius; MT: Middle trapezius; PSG: Periscapular group; RCT: Randomized controlled trial; SAPS: Subacromial pain syndrome; SSG: Scapular Stabilization group; SPADI: Shoulder pain and disability index; SA: Serratus anterior; UT: Upper trapezius.

Supplementary Information
The online version contains supplementary material available at https://doi.org/10.1186/s40945-022-00138-1.

Additional file 1: Figure S1. Sensitivity analysis plots for each single mediator model for pain. The correlation between the error terms in the mediator and outcome regression models (ρ) is plotted against the average causal mediation effect (ACME). The estimated ACME (assuming sequential ignorability) is the dashed line and the 95% confidence intervals are represented by the shaded regions. **Figure S2.** Sensitivity analysis plots for each single mediator model for shoulder function. The correlation between the error terms in the mediator and outcome regression models (ρ) is plotted against the average causal mediation effect (ACME). The estimated ACME (assuming sequential ignorability) is the dashed line and the 95% confidence intervals are represented by the shaded regions.

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Availability of data and materials
The datasets generated during and/or analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
Participation was voluntary, and all participants gave written informed consent after receiving oral and written information. Furthermore, the participants could withdraw without any consequences. The study was approved by the local ethic committee at University of São Paulo (CAAE:52563216.0.0000.5414). This study did require public trials registry. The clinical trial data were prospectively registered at ClinicalTrials.gov (NCT02695524).

Consent for publication
Not applicable.

Competing interests
The authors (GHH, RKA, DCR, KJM, ASO) declare that they have no competing interests.

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