REVIEW

Evidence for cognitive–behavioral strategies improving dyspnea and related distress in COPD

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Background: Dyspnea is a complex, prevalent, and distressing symptom of chronic obstructive pulmonary disease (COPD) associated with decreased quality of life, significant disability, and increased mortality. It is a major reason for referral to pulmonary rehabilitation.

Methods: We reviewed 23 COPD studies to examine the evidence for the effectiveness of cognitive–behavioral strategies for relieving dyspnea in COPD.

Results: Preliminary evidence from randomized controlled trials exists to support cognitive–behavioral strategies, used with or without exercise, for relieving sensory and affective components of dyspnea in COPD. Small to moderate treatment effects for relieving dyspnea were noted for psychotherapy (effect size [ES] = 0.08–0.25 for intensity; 0.26–0.65 for mastery) and distractive auditory stimuli (ES = 0.08–0.33 for intensity; 0.09 to −0.61 for functional burden). Small to large dyspnea improvements resulted from yoga (ES = 0.2–1.21 for intensity; 0.67 for distress; 0.07 for mastery; and −8.37 for functional burden); dyspnea self-management education with exercise (ES = −0.14 to −1.15 for intensity; −0.62 to −0.69 for distress; 1.04 for mastery; 0.14–0.35 for self-efficacy); and slow-breathing exercises (ES = −0.34 to −0.83 for intensity; −0.61 to −0.80 for distress; and 0.62 for self-efficacy). Cognitive–behavioral interventions may relieve dyspnea in COPD by (1) decreasing sympathetic nerve activity, dynamic hyperinflation, and comorbid anxiety, and (2) promoting arterial oxygen saturation, myelinated vagus nerve activity, a greater exercise training effect, and neuroplasticity.

Conclusion: While evidence is increasing, additional randomized controlled trials are needed to evaluate the effectiveness of psychosocial and self-management interventions in relieving dyspnea, in order to make them more available to patients and to endorse them in official COPD, dyspnea, and pulmonary rehabilitation practice guidelines. By relieving dyspnea and related anxiety, such interventions may promote adherence to exercise programs and adaptive lifestyle change.

Keywords: chronic obstructive pulmonary disease, dyspnea, anxiety, slow breathing, distractive auditory stimuli, self-management

Introduction

Alleviating dyspnea is a primary goal of pulmonary rehabilitation (PR) and self-management (SM) interventions in chronic obstructive pulmonary disease (COPD).[1] Dyspnea is a "subjective experience of breathing discomfort."[2] It is perceived via two main afferent neural pathways: (1) chemoreceptors and vagal C fibers from the lung parenchyma and airways, and (2) chest wall mechanoreceptors and vagal stretch receptors, which are subsequently processed in the insular cortex and corticolimbic system.[3] The insula appears to have a primary role in the perception of dyspnea based on its interoception function. The insula works closely with the amygdala and anterior

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cingulated cortex to modulate dyspnea-related attention and affect based on arousal and valence.[4]

There can be remarkable variation between self-reported dyspnea and the degree of pulmonary obstruction.[5,6] Dyspnea includes both conscious awareness of physical sensation intensity and affective judgment and response.[7,8] The American Thoracic Society's (ATS) 2012 practice guidelines[2] divide dyspnea into three domains: sensory perceptual experience, affective distress, and symptom impact or burden. Dyspnea can be strongly influenced by negative affectivity, anxiety, and distress.[5,8–12] Anxiety may contribute to hyper-perception of dyspnea or an exaggerated or distorted perception relative to the severity of pulmonary dysfunction.[7,13] A distressing emotional response to dyspnea may lead to a heightened attention to breathing, inducing further anxiety and causing avoidance behavioral responses.[7] Dyspnea has been shown to be a better predictor of mortality than pulmonary function (forced expiratory volume in 1 second [FEV_1]).[14]

Patients with COPD have been shown to experience significantly increased comorbid psychological distress and anxiety compared with the general population[15–19] and to have higher sympathetic nerve activity and parasympathetic dysfunction.[20,21] It is estimated that the prevalence of anxiety is between 10% and 19% in stable COPD and between 9% and 58% in patients with unstable COPD resulting from a recent exacerbation.[16]

There is strong evidence supporting the effectiveness of PR that consists of at least 4 weeks of exercising training in improving dyspnea in adults with COPD.[22] PR practice guidelines recommend a program of exercise training (ET) for the muscles of ambulation as a mandatory component of PR for patients with COPD.[22] To date, there is little understanding of how pulmonary rehabilitation works to relieve dyspnea in patients with COPD and how much improvements relate to a conditioning effect, desensitization to the sensory experience, decreased sympathetic activity, and improved self-efficacy.[2,23]

While, based on strong evidence, ET is considered an essential component of PR, PR is not just limited to ET,[23] and also includes collaborative psychosocial, behavioral, and SM interventions integrated into long-term COPD disease management.[22,24] The evidence supporting these adjunctive therapies is weaker due to inconsistency in the research findings and variability in the quality of the studies.[22] The effectiveness of non-exercise and non-pharmacological COPD treatment components in alleviating dyspnea in COPD is not well known. More focused research is needed to determine whether adjunctive therapies to exercise are effective in relieving dyspnea in PR. The best ways to maintain PR gains post-discharge have also not been well established.

Because dyspnea is a symptom, it can only be measured by patient self-report. Measuring the affective component of dyspnea, in addition to the sensory component (intensity), is important to more comprehensively evaluate the outcomes of PR and cognitive–behavioral interventions (see Table 1 for a summary of dyspnea assessments, their minimal clinical important difference (MCID) if known, and reference citations).

Table 1 Dyspnea assessments used in review studies

Instrument name	Constructs	MCID
Borg Breathlessness Scale (Borg)[64]	Dyspnea intensity	1.0 unit
Borg Dyspnea Distress Scale (Borg[DD])[25,65]	Dyspnea distress	1.0 unit
Breathlessness Beliefs Questionnaire[66]	Dyspnea-related anxiety: somatic and activity avoidance beliefs	Not known
Chronic Respiratory Disease Questionnaire (CRQ)[67–69]	Dyspnea intensity with activities (dyspnea scale, CRQ-D); dyspnea-related anxiety (mastery scale, CRQ-M)	0.5 per item
COPD Self-Efficacy Scale (CSES)[70]	Dyspnea self-efficacy	Not known
Dyspnea Management Questionnaire (DMQ)[39,71,72]	Dyspnea intensity with activities; dyspnea-related anxiety; activity avoidance; self-efficacy	Not known
Interpretation of Breathing Problems Questionnaire (IBPQ)[73]	Dyspnea-related anxiety; catastrophic thoughts	Not known
Modified Medical Research Council Dyspnea Scale (mMRC)[1]	Dyspnea impact or burden	Not known
Self-Efficacy for Managing Shortness of Breath (SEMSOB)[74]	Dyspnea self-efficacy	Not known
St George's Respiratory Questionnaire (SGRQ)[2,75]	Dyspnea impact or burden (activity scale: SGRQ-A)	4 units
Transition Dyspnea Index (TDI)[76]	Dyspnea impact or burden	1.0 unit
University of California, San Diego Shortness of Breath Questionnaire (SOBQ)[64,77]	Dyspnea intensity with activities	5 units
Visual Analog Scale – Unpleasantness (VAS-U)[64,62]	Dyspnea affect	10–20 units
Visual Analog Scale – Dyspnea Distress (VAS-DD), Dyspnea Anxiety (VAS-DA), shortness of breath (VAS-SOB)[54,64]	Dyspnea distress; dyspnea-related anxiety; dyspnea intensity	10–20 units

Abbreviations: MCID, minimal clinical important difference; COPD, chronic obstructive pulmonary disease; D, dyspnea scale; M, mastery scale; A, activity scale.

This review was conducted to critique the evidence for current adjunctive psychosocial therapies used both during and after PR to specifically manage and relieve the sensory and affective components of dyspnea in patients with COPD. For this review, we focused on cognitive–behavioral and SM therapies that could complement ET and pharmacologic interventions in PR. Only COPD randomized controlled trials (RCTs) that included at least one outcome measure of dyspnea were included in this review. The review focused on studies published between 1996 and 2013. The studies were analyzed and grouped into the following categories: meditation, cognitive–behavioral therapy (psychotherapy), breathing exercises, dyspnea SM education, and distractive auditory stimuli (DAS). Given the wide scope of this review, a systematic review was not conducted.

Meditation

Six mind–body meditation studies met our inclusion criteria, with sample sizes ranging between 10 and 158, and COPD severity levels varying considerably between the groups from mild to very severe (see Table 2). Two studies showed trends in effectively reducing dyspnea distress (the Borg and the Chronic Respiratory Disease Questionnaire – Modified [CRQ-M]) post-intervention following Iyengar yoga and Tai Chi interventions compared with usual care control groups ($P = 0.07–0.08$).[25,26] An additional study found within-group effects for reducing functional burden of dyspnea (St George's Respiratory Questionnaire – Activity scale [SGRQ-A]) following a 3-month yoga breathing intervention but not in the control group ($P < 0.01$).[27] Only one Tai Chi study conducted a follow up evaluation post-intervention at 6 months and found no significant differences for dyspnea when combining Tai Chi with PR.[28] However, the mean change scores at 6 months for the group receiving Health Qigong Tai Chi plus PR for CRQ dyspnea and CRQ mastery exceeded the MCID scores, while the mean change scores within the comparison PR group did not.[28]

In contrast, Mularski et al[29] identified no differences between or within groups for dyspnea following an 8-week group "Mindfulness-Based Breathing Therapy" (MBBT) intervention when compared with a support group. Of note, this study suffered from an especially high drop-out rate of 43% after randomization. Also, an RCT by Chan et al[30] of a 3-month Tai Chi Qigong (TCQ) intervention found no effects of TCQ or breathing exercises for dyspnea compared with a control group as measured by the Borg scale.

Summary

Yoga is a mind–body intervention that comprises a wide variety of approaches, including meditation (dhyana), breathing exercises (pranayamas), optimal coping principles (swadhyaya), exercise (asanas), nourishing diet, and positive thinking.[27,31] Its ability to improve symptoms such as dyspnea has only been recently studied.[31] While the evidence supporting mind–body interventions for improving dyspnea is very preliminary, these interventions appear to be promising for improving the debilitating effects of dyspnea in COPD, especially dyspnea distress or anxiety and dyspnea impact. The ATS practice guidelines for dyspnea (2012)[2] concluded that there were currently insufficient data to recommend mindfulness treatment or yoga for relieving dyspnea.

Cognitive–behavioral therapy

Cognitive–behavioral therapy (CBT) is a "structured, psychological intervention in which the patient works collaboratively with the therapist to identify the types and effects of thoughts, beliefs, attributions, and interpretations on current symptoms, feeling states and/or problem areas."[32] We identified two CBT psychotherapy RCTs that met our inclusion criteria (see Table 3).

Livermore et al[33] demonstrated that dyspnea anxiety (measured by the Interpretation of Breathing Problems Questionnaire [IBPQ]) was reduced after a CBT intervention at 6, 12, and 18 month follow up evaluations, but not post-intervention at 4 weeks in adults with mild to severe COPD.

However, Kunik et al[34] found that CBT was no more effective than COPD education on CRQ-dyspnea and CRQ-mastery scores at the end of an 8-week treatment and at a 12-month follow up in male adults with moderate to very severe COPD. Both treatments were found to have small within-group effects for improving dyspnea intensity and mastery post-intervention ($P < 0.01$).

Summary

CBT is considered a primary treatment approach in national practice guidelines for relieving anxiety in diverse populations, as supported by strong evidence.[35] Clearly though, more research is needed on the effectiveness of CBT on dyspnea and dyspnea-related distress relief for COPD. CBT strategies have enormous potential for relieving the affective dimension of dyspnea.[9,15,36] The same corticolimbic areas of the brain activated during dyspnea have been found to be altered following CBT therapy in people with anxiety disorders, with increased activity found in the anterior cingulated cortex and

Table 2 Meditation studies

Study	Design/treatment	N	FEV$_1$ % predicted M ± SD	Group	Dyspnea score: short-term, M (SD)	Effect size	MCID	Dyspnea score: long-term (6 month follow up), M (SD)
Donesky-Cuenco et al[25]	RCT; 1 hour, twice weekly; 12 weeks	14	51.2 ± 10.5	E: Yoga	E: 3.8 (2.3)–3.8 (1.5) Borg[6MW]	0.2	X	
		15	44.4 ± 19	C: Usual care	C: 2.9 (1.6)–3.3 (2.5)		X	
					E: 2.6 (2.8)–1.6 (1.7) Borg[DD a]	0.67	√	
					C: 1.1 (1.1)–1.4 (1.5)		X	
					E: 14.6 (3.0)–16.8 (6.3) CRQ-D	0.07	X	
					C: 12.9 (6.1)–15.2 (6.6)		X	
					E: 22.4 (3.5)–22.3 (4.0) CRQ-M	0.07	X	
					C: 19.5 (5.0)–19.4 (5.3)		X	
Yeh et al[26]	RCT; 1 hour twice weekly; 12 weeks	5	53 ±7	E: Tai chi	E: 4.4–5.7 CRQ-D		√mod	
		5	47 ±7	C: Usual care	C: 6.0–6.4		X	
					E: 4.2–6.5 CRQ-M[a]		√large	
					C: 6.0–5.7		X	
					E: 39–27 UCSD		√	
					C: 20–22		X	
					E: 105–135 CSES			
					C: 135–137			
Chan et al[30]	RCT; TCQ: 1 hour twice weekly for 3 months. Measures: 0, 6 weeks, 3 months	60	Data not available	E: TCQ	E: 1.98 (1.21)–1.84 (1.38)[6wk]–1.86 (1.25)[3m] Borg[6MW]	−0.11	X	
		50		C$_1$: BT	C$_1$: 1.38 (1.74)–1.74 (1.37)–1.70 (1.38)	−0.1		
		48		C$_2$: Control	C$_2$: 1.51 (1.43)–2.02 (1.50)–2.06 (1.53)	0.23, 0.20	X, X	
Ng et al[28]	RCT; one 45-minute session; 4 weeks	23	37.1 ± 2.2	E: TCQ + PR	Not reported			E: 0.64 (0.26 SE) CRQ-D[6m] C: 0.26 (0.21) E: 0.64 (0.29) CRQ-M C: 0.33 (0.20)
		29	36.8 ± 2.1	C: PR alone				
Mularski et al[29]	RCT; once-weekly group MBBT, 8 weeks; support group, matched time; diary	20	Data not available	E: MBBT	E: 4.6–4.3 Borg[6MWT]		X	
		29		C: Support group	C: 4.6–4.6		X	
					E: 26.4 VAS (at rest)			
					C: 27.9			
					E: 50.7 VAS (dyspnea activity)			
					C: 49.3			
					E: 68.6–71.2 SGRQ-A		X	
					C: 69.4–69.8		X	
Katiyar and Bihari[27]	RCT; 3-month training	24	48 ± 2.4	E: Yoga breathing	E: 66 (2.1)–50 (1.7) SGRQ-A[‡]	−8.37	√	
		24	48 ± 2	C: Control	C: 63–62		X	

Notes: ‡P < 0.01 for within-group mean change; [a]P = 0.07–0.08.
Abbreviations: Borg, modified Borg scale; BT, breathing training; C, control or comparison group; CRQ-D, Chronic Respiratory Disease Questionnaire, dyspnea subscale; CRQ-M, Chronic Respiratory Disease Questionnaire, mastery scale; CSES, COPD Self-Efficacy Scale; DD, dyspnea distress; E, experimental group; FEV$_1$, forced expiratory volume in 1 second; m, months; M, mean; MBBT, mindfulness-based breathing therapy; MCID, minimal clinical important difference; mod, moderate; PR, pulmonary rehabilitation; RCT, randomized controlled trial; SD, standard deviation; SE, standard error; SGRQ-A, St George's Respiratory Questionnaire, activity scale; TCQ, Tai Chi Qigong; UCSD, University of California, San Diego, Shortness of Breath Questionnaire; VAS, visual analog scale; wk, weeks; 6MW, 6-minute walk distance (feet); √, exceeded MCID; X, did not meet MCID.

Table 3 Cognitive–behavioral therapy studies

Study	Design/intervention	N	FEV_1% predicted, M ± SD	Group	Dyspnea score; short-term, M (SD)	Effect size	MCID	Dyspnea score; long-term (6–18 month), M (SD)	Effect size
Livermore et al[33]	RCT; Indiv CBT, post-PR, 1 hour once per week, 4 weeks	21	57.0 ± 25.1	E: CBT	E: 7.0 (4.3) IBPQ^wk4			IBPQ^6m**, 12m**, 18m**	0.23
		20	51.1 ± 15.0	C: Usual care	C: 8.2 (4.8)				
Kunik et al[34]	RCT; 1 hour per week, 8 weeks; CBT delivered in groups of up to ten patients	118	45.3 ± 16.8	E: CBT	E: 2.99 (1.33)–3.36 (1.64)‡ CRQ-D	0.25	X	E: 2.99 (1.33)–3.38 (1.96)^12m CRQ-D	0.23
		120	46.8 ± 17.5	C: COPD educ	C: 3.11 (1.53)–3.51 (3.04)‡	0.17	X	C: 3.11 (1.53)–3.60 (1.91)	0.28
					E: 4.00 (1.83)–4.47 (1.82)‡ CRQ-M	0.26	X	E: 4.00 (1.83)–4.62 (1.82) CRQ-M	0.34
					C: 3.95 (1.85)–4.37 (1.82)‡	0.23	X	C: 3.95 (1.85)–4.37 (1.77)	0.23

Notes: **$P < 0.01$ favoring treatment group; ‡$P < 0.01$ for within-group mean change.
Abbreviations: C, control or comparison group; CBT, cognitive–behavioral therapy; COPD, chronic obstructive pulmonary disease; CRQ-D, Chronic Respiratory Disease Questionnaire, dyspnea scale; CRQ-M, Chronic Respiratory Disease Questionnaire, mastery scale; E, experimental group; educ, education; FEV_1, forced expiratory volume in 1 second; IBPQ, Interpretation of Breathing Problems Questionnaire; Indiv, individual; m, months; M, mean; MCID, minimal clinical important difference; PR, pulmonary rehabilitation; RCT, randomized controlled trial; SD, standard deviation; wk, weeks; √, exceeded MCID; X, did not meet MCID.

decreased activity in the amygdala.[37] Preliminary evidence suggests that anxiety plays an important role in mediating the experience of dyspnea in COPD.[15] More research is needed to better understand how anxiety and other affective factors impact dyspnea in order to improve dyspnea treatment.[15,36] More research is needed on the effectiveness of CBT for female adults with COPD, particularly since anxiety affects women more than men with COPD.[18,38] In order to measure the effectiveness of CBT on dyspnea in COPD, it may be important to measure the constructs of anxiety and dyspnea-related anxiety separately.[39]

Slow-breathing exercises

We identified seven studies that investigated the effects of slow-breathing exercises on dyspnea in COPD, with sample sizes between 30 and 53 participants (Table 4). Five studies evaluated patients with moderate to very severe COPD; one study with just severe COPD, and one with moderate to severe COPD. The effects of breathing exercises with biofeedback, pursed-lips breathing (PLB), diaphragmatic breathing (DB), and singing were studied.

Three RCTs found statistically significant between-group differences favoring slow-breathing exercise interventions. Yamaguti et al[40] (n = 30) reported that the disabling impact of dyspnea in the DB group was significantly reduced compared with a control group ($P < 0.05$) as measured by the modified Medical Research Council (mMRC) scale. Improved abdominal motion with normal breathing ($P < 0.001$) and DB ($P < 0.05$) at 4 weeks, and greater diaphragmatic mobility ($P < 0.001$), were identified in the DB group. Also, using the Borg scale, Nield et al[41] (n = 53) reported improved dyspnea at 3 months ($P < 0.05$) after a 4-week PLB with arterial oxygen saturation (SaO_2) biofeedback intervention compared with inspiratory muscle training (IMT), expiratory muscle training (EMT), and control groups. In 2007, Nield et al[42] also found decreased mean Borg dyspnea scores for patients assigned to a PLB intervention group ($P = 0.05$) at a 2 month follow up compared with comparison EMT and control groups. The dyspnea reductions were clinically significant for the PLB group at 4 weeks and 12 weeks, exceeding the MCID of five units for the University of Southern California, San Diego, Shortness Of Breath Questionnaire (UCSD SOBQ), while dyspnea worsened in the other two groups at 12 weeks.

In three additional studies, while significant differences were not found between the treatment and comparison groups, within-group differences post-intervention were clinically significant for the breathing intervention groups.[43–45]

Table 4 Breathing studies

Study	Design/intervention	N	FEV$_1$ % predicted, M ± SD	Group	Dyspnea score; short-term, M (SD)	Effect size	MCID	Dyspnea score; long-term (2–3 months), M (SD)	Effect size
Yamaguti et al[40]	RCT, pre- post-test; 3 indiv 45-minute weekly sessions, 4 weeks	15 15	43.4 42.4	E: DBT C: Control	E: 2.6–2.0* C: 2.8–2.5 mMRC	−0.41 (b/w group)			
Nield et al[41]	RCT; 0, 4 week, 3 monthly; 4-weekly training sessions	53	38 ± 14	E: PLB C$_1$: IMT C$_2$: EMT C: Control	VAS UCSD Borg			Borg$^{6MW\ 3m*}$	
Nield et al[42]	RCT; 0, 4, 12-week time points; weekly monitored breathing sessions in laboratory and daily home practice for 4 weeks	14 13 13	35 ± 8 43 ± 16 40 ± 15	E$_1$: PLB E$_2$: EMT C: Control	E$_1$: 3.9 (1.3)–3.2 (0.9) Borg E$_2$: 4.2 (0.9)–3.7 (0.7) C: 3.8 (0.7)–3.2 (0.8) E$_1$: 68 (24)–60 (16) UCSD E$_2$: 65 (19)–63 (18) C: 58 (28)–64 (20)	−0.63 −0.62 −0.80 −0.39 −0.11 0.25	X X X √ X √worse	E$_1$: 3.9 (1.3)–3 (1.0)# Borg^{2m} E$_2$: 4.2 (0.9)–3.9 (0.7) C: 3.8 (0.7)–4 (1.4) E$_1$: 68 (24)–59 (17) UCSD E$_2$: 65 (19)–68 (22) C: 58 (28)–69 (24)	−0.78 −0.37 0.18 −0.43 0.15 0.42
Collins et al[43]	RCT; 36 ET training sessions; VF with monitor display of RR and goal	17 16 16	41 ± 18 43 ± 15 46 ± 16	E: VF + PR C$_1$: PR alone C$_2$: VF alone	5.6 (4.1)–1.3 (1.4)‡ Borg^{TET} 6.2 (3.8)–2.2 (2.4)‡ 5.7 (3.0)–3.3 (2.8) E: +4 (6) CRQ-D† C$_1$: +6 (5)† C$_2$: +6 (5)‡ E: 3 (3)‡ CRQ-M C$_1$: NR/NS C$_2$: NR/NS	−1.40 −1.26 −0.83	√ √ √		
Bonilha et al[46]	RCT; 24, 1 hour weekly group training classes; 24 weeks	15 15	48.8 ± 20.8 53.4 ± 20.1	E: Singing C: Handcraft	E: 0.7 (1.2) TDI C: 0.3 (1.7)		X X		
van Gestel et al[44]	RCT; 4-week PR program; 10 × 30-minute VF sessions; PLB and DB	20 20	47.0 ± 19.9 44.3 ± 17.3	E: VF + PR C: PR	E: 0.88 (0.74) CRQ-D C: 0.57 (1.01) E: 0.62 (0.97) CRQ-M C: 0.45 (0.90) E: −0.1 (1.0) Borg$^{post-6MW}$ C: 0.3 (1.7)		√ √ √ X X X		
Scherer et al[45]	RCT; twice daily 15 minutes, 5 days/week, 8 weeks	15 15	50.2 ± 4.4 52.3 ± 3.5	E: RMT C: BT	E: 4.7 TDI C: 2.9		√ √		

Notes: *$P < 0.05$, favoring experimental/treatment group; †$P < 0.05$ for within-group mean change; ‡$P < 0.01$ for within-group mean change; ‡$P < 0.05$ for within-group mean change; #$P = 0.05$.

Abbreviations: Borg, modified Borg scale 0–10 point; BT, breathing training; C, control or comparison group; CRQ-D, Chronic Respiratory Disease Questionnaire, dyspnea scale; CRQ-M, Chronic Respiratory Disease Questionnaire, mastery scale; DB, diaphragmatic breathing; DBT, diaphragmatic breathing; E, experimental group; ET, exercise training; EMT, expiratory muscle training; FEV$_1$, forced expiratory volume in 1 second; indiv, individualized; IMT, inspiratory muscle training; m, months; min, minutes; M, mean; MCID, minimal clinical important difference; mMRC, modified Medical Research Council scale; NR, not reported; NS, not significant; PLB, pursed-lips breathing; PR, pulmonary rehabilitation; RCT, randomized controlled trial; RMT, respiratory muscle endurance training; SD, standard deviation; TDI, Transition Dyspnea Index; TET, treadmill exercise test; UCSD, University of California, San Diego Shortness of Breath Questionnaire; VAS, visual analog scale; VE, vocalization exercises; VF, ventilation feedback training; 6MW, 6-Minute Walk Test; √, exceeded MCID; X, did not meet MCID; b/w, between; RR, respiratory rate.

Collins et al[43] found no differences for dyspnea between three groups (ET plus ventilation feedback [VF], ET alone, and VF alone) as measured by the CRQ and Borg scales (n = 49). A large (−0.83 with the Borg scale) was found for the VF-alone group for dyspnea, which consisted of 36 sessions (30–35 minutes each) of training in slow breathing using a pneumotachometer interfaced to a computer with visual display and respiratory rate goal feedback, combined with low-intensity exercise. A larger dyspnea effect size (ES) was found for the ET plus VF group (ES = −1.40). Exercise-induced dynamic hyperinflation was significantly less in the ET plus VF group than in the other two groups ($P \leq 0.019$).

van Gestel et al[44] also investigated the benefits of combining breathing training with PR compared with PR alone on dyspnea in COPD (n = 40). While no between-group differences were identified for dyspnea as measured by the CRQ and Borg scales, the CRQ-M score improvement at discharge met the 0.5 unit MCID in the experimental group only.

Similarly, in the RCT by Scherer et al,[45] the disabling dyspnea impact of both the respiratory muscle training (RMT) group and a comparison breathing training group improved as measured by the Transition Dyspnea Index (TDI). Change within the groups was clinically significant, but there was no difference between the groups ($P = 0.09$).

Finally, Bonilha et al[46] compared dyspnea outcome scores following a novel singing intervention compared with a handcraft control intervention as measured by the TDI (n = 30). They found no differences between the groups for dyspnea.

Summary

Some evidence from small RCTs exists to support slow-breathing exercises (both PLB and DB). The types of breathing exercises studied and the protocols used varied widely between the COPD studies. Slow-breathing training was combined with PR ET in two studies.[43,44]

The mechanisms for improvements in dyspnea with slow-breathing exercises continue to be debated. Collins et al[43] found a decrease in dyspnea to be related to a smaller end-expiratory lung volume in the VF and ET group. PLB, which reduces respiratory rate, may also improve dyspnea by reducing sympathetic activity and enhancing baroreflex efficiency in adults with COPD. Dyspnea improvements following DB in one study were found to be aided by improved abdominal motion and greater diaphragmatic mobility with normal breathing and DB at 4 weeks compared with a control group.[40] Prolonged exhalations from breathing exercises and singing may increase the influence of the myelinated

vagus, which decreases heart rate and improves state regulation.[47,48] Both singing and PLB recruit the muscles of the face, which are linked to neural regulation of the autonomic nervous system.[47] The findings of several studies support that slow-breathing training alone (without an ET effect) can reduce dyspnea.[40,42,43] The American College of Chest Physicians (ACCP) consensus statement[49] on the management of dyspnea with advanced lung disease recommends PLB for effective dyspnea relief.

Dyspnea self-management

Education that includes SM education is considered an important component of PR.[22] SM education is distinct from traditional patient education as it focuses on (1) non-didactic education based on the patient's agenda and problems identified by the patient, (2) collaborative decision making using action plans, (3) teaching problem-solving skills, and (4) increasing both self-efficacy and knowledge to promote behavior change.[50] Self-efficacy in SM programs is enhanced through action plans, feedback of progress, modeling of behaviors, reinterpretation of symptoms, and social persuasion.[51] Practice guidelines recommend collaborative SM education as a component of PR.[22]

We included five dyspnea SM studies in our review, with samples of 39–103 participants. Most of the studies evaluated participants with moderate to very severe COPD. Two of the studies found significant differences between the groups favoring experimental groups receiving both SM education and 24 CBT-based ET sessions (Table 5).[52,53] Stulbarg et al[52] found that the dyspnea SM and exercise group (D-SM plus 24 exercise sessions [Ex$_{24}$]) had better CRQ dyspnea, CRQ Mastery and Borg scores than the comparison groups (D-SM alone and D-SM plus 4 exercise sessions [Ex$_4$]), with mostly large ESs ($P < 0.05$; ES = −0.62 to −1.15). The dyspnea SM-alone group had small effects on CRQ dyspnea and CRQ Mastery (0.31, 0.34, respectively), but these did not reach the MCID of 0.5 units. Similarly, the study by Carrieri-Kohlman et al[53] found that the experimental group that received D-SM and Ex$_{24}$ had better CRQ dyspnea intensity and mastery scores and Borg scores than did groups receiving D-SM alone and D-SM plus Ex$_4$ interventions at 2 months ($P < 0.01$). They also found a reduction in the Borg dyspnea score following an incremental treadmill test at a 12-month follow up ($P < 0.01$) favoring the D-SM plus Ex$_{24}$ group.

In the study by Carrieri-Kohlman et al,[54] the dyspnea intensity, dyspnea distress, and dyspnea-anxiety of both intervention groups (CBT-based D-SM strategies with ET and exercise-alone groups) improved. Differences between

Table 5 Dyspnea self-management studies

Study	Design/intervention	N	FEV$_1$ % predicted, M ± SD	Group	Dyspnea score; post-intervention (short-term), M (SD)	Effect size	MCID	Dyspnea score; long-term (2- to 12-month follow up), M (SD)	Effect size
Stulbarg et al[52]	RCT; D-SM: 3 hours of indiv educ and indiv HEP with pedometer and daily log measurement: 0 and 2 months	36 33 34	42.5 ± 18.4 48.5 ± 12.6 43.7 ± 9.9	C$_1$: D-SM C$_2$: D-SM + Ex$_4$ E: D-SM + Ex$_{24}$	C$_1$: 16.0 (5.6)–17.7 (5.5) CRQ-D	0.31	X	E: 4.8–5.9 (1.6)† CRQ-D^{8wk}	0.80
					C$_2$: 17.4 (4.2)–21.1 (5.3)†	0.77	√small	C: 5.1 (1.1)–5.7 (1.7)†	0.42
					E: 15.8 (4.9)–20.8 (4.5)†,**	1.06	√mod		
					C$_1$: 5.1 (1.4)–1.5 (2.5) TDI		√		
					C$_2$: 5.4 (1.4)–1.5 (2.7)		√		
					E: 4.9 (1.7)–2.0 (2.2)		√		
					C$_1$: 53.3 (17.7)–51.6 (15.6) UCSD	−0.10	X		
					C$_2$: 50.6 (18.0)–46.1 (17.5)	−0.25	X		
					E: 54.7 (15.1)–52.4 (16.7)	−0.14	X		
					C$_1$: 18.6 (5.3)–20.3 (4.7)† CRQ-M	0.34	X		
					C$_2$: 19.9 (4.6)–21.8 (4.9)‡	0.40	X		
					E: 17.9 (4.9)–22.6 (4.1)†,*	1.04	√mod		
					C$_1$: 4.7 (2.0)–4.4 (1.6) Borg^{ITT}	−0.17	X		
					C$_2$: 4.9 (2.3)–4.2 (2.4)	−0.30	X		
					E: 4.8 (1.8)–3.0 (1.3)*,**	−1.15	√		
					C$_1$: 3.8 (1.8)–3.9 (2.0) Borg^{6MW}	0.05	X		
					C$_2$: 4.0 (1.9)–3.3 (1.9)*	−0.37	X		
					E: 4.7 (2.6)–3.4 (1.4)**	−0.62	√		
Carrieri-Kohlman et al[54]	RCT; 12 Ex sessions over 4–6 weeks; 8 weeks of home walking with pedometer; 0, 4–6 wks; 8-week follow up	24 27	40 ± 11 36 ± 10	E: D-SM + Ex$_{12}$ C: Ex$_{12}$ only	E: 4.8 (1.1)–5.3 (1.5)‡ CRQ-D	0.38	√		
					C: 5.1 (1.1)–5.8 (1.6)‡	0.51	√		
					E: 142 (58)–97 (64)‡ VAS-SOB$^{TST\ isostage}$	−0.74	√		
					C: 155 (42)–78 (55)‡,*	−1.57	√		
					E: 113 (67)–72 (65)‡ VAS-DD^{TST}	−0.62	√		
					C: 133 (62)–49 (49)‡,*	−1.50	√		
					E: 97 (76)–50 (60)‡ VAS-DA^{TST}	−0.69	√		
					C: 110 (76)–42 (51)‡	−1.05	√		
					220 mm VAS		[MCID 20 mm]		
Carrieri-Kohlman et al[53]	RCT; repeated measures: 0, every 2 months for 1 year; either 4 or 24 Ex sessions; SM: four 1 hour and two ½ hr (4 month, 8 month) indiv sessions, home walking, biweekly telephone calls	36 33 34	44.8 ± 1.4 (for total)	C$_1$: D-SM C$_2$: D-SM + Ex$_4$ E: D-SM + Ex$_{24}$	C$_1$: 16 (0.8)–1.7^{Δ},† CRQ-D^{2m}		C$_1$: X	C$_1$: 16–4.5† CRQ-D^{12m}	
					C$_2$: 17.2 (0.8)–3.7†,Δ		C$_2$: √small	C$_2$: 17.2–4.2†	
					E: 15.8 (0.8)–5.0*,†,Δ		E: √mod	E: 15.8–3.2†	
					C$_1$: 18.4 (0.8)–1.8^{Δ} CRQ-M		C$_1$: X	C$_1$: 18.4–2.5† CRQ-M^{12m}	
					C$_2$: 19.9 (0.8)–1.9^{Δ}		C$_2$: X	C$_2$: 19.9–1.4	
					E: 17.9 (0.8)–4.7†,**,Δ		E: √mod	E: 17.9–1.6	
					C$_1$: 0.5 (0.1)–0^{Δ} Borg^{ITT}		X	C$_1$: 0.5 to –0.1 Borg^{ITT,12m}	
					C$_2$: 0.5 (0.1)–0^{Δ}		X	C$_2$: 0.5–0.1	
					E: 0.6 (0.1) to –0.2*,**,†,Δ		X	E: 0.6 to –0.2*,**,†	

Study	Design	n	Mean ± SD	Groups	Outcomes	Effect size	MCID
Davis et al[56]	RCT: same as for Stulbarg et al[52]. 0 and 8 weeks	36 32 34	44 ± 19 49 ± 13 44 ± 10	C₁: D-SM C₂: D-SM + Ex₄ E: D-SM + Ex₂₄	C₁: 2.97 (0.69)–2.98 (0.63) CSES C₂: 3.28 (0.70)–3.26 (0.81) E: 3.10 (0.92)–3.22 (0.85) C₁: 4.44 (2.5)–5.7 (2.2)‡ SEMSOB C₂: 5.7 (2.6)–6.5 (2.5) E: 6.1 (2.7)–7.0 (2.4)†	0.02 −0.3 0.14 0.54 0.31 0.35	
Nguyen et al[55]	RCT; repeated measures: 0, 3 month, 6 month	20 19	50.3 ± 17.6 49.0 ± 16.8	C: fD-SM E: eD-SM	C: 15.9 (5.4)–19.2 (5.8)–19.9 (6.2)‡ CRQ-D E: 18.8 (6.2)–22.3 (4.6)–21.3 (6.0)‡ C: 20.4 (5.2)–22.3 (5.8)–22.4 (5.5)‡ CRQ-M E: 21.7 (3.2)–23.6 (2.9)–23.6 (3.7)† C: 4.6 (2.4)–5.5 (3.3)–5.0 (3.6) CSES† E: 4.7 (2.3)–6.8 (2.3)–6.7 (2.6)†	0.59/0.69 0.64/0.41 0.34/0.37 0.62/0.55 0.31/0.13 0.91/0.81	√/√ √/√ X/√ X/X

Notes: *$P < 0.05$, favoring treatment group; **$P < 0.01$ favoring treatment group; †$P < 0.05$ for within-group mean change; ‡$P < 0.01$ for within-group mean change.
Abbreviations: Ex, exercise sessions; Borg, modified Borg scale; C, control or comparison group; CRQ-D, Chronic Respiratory Disease Questionnaire, dyspnea scale; CRQ-M, Chronic Respiratory Disease Questionnaire, mastery scale; CSES, COPD Self-Efficacy Scale; D-SM, dyspnea self-management; E, experimental group; eD-SM, Internet-based dyspnea self-management; educ, education; fD-SM, face-to-face dyspnea self-management; FEV, forced expiratory volume in 1 second; HEP, home exercise program; indiv, individualized; ITT, incremental treadmill test (isotime); m, months; M, mean; MCID, minimal clinical important difference; mod, moderate; RCT, randomized controlled trial; SD, standard deviation; SEMSOB, Self-Efficacy for Managing Shortness of Breath; SM, self-management; SOB, shortness of breath; TDI, Transition Dyspnea Index; TST, treadmill stress test; UCSD, University of California, San Diego Shortness of Breath Questionnaire; VAS, 100 mm visual analog scale; VAS-DA, visual analog scale – dyspnea anxiety; VAS-DD, visual analog scale – dyspnea distress; wk, weeks; 6MW, 6-Minute Walk Test; Δ, difference score; √, exceeded MCID; X, did not meet MCID.

the groups ($P < 0.05$) were found for dyspnea intensity and dyspnea distress (as measured by a visual analog scale [VAS] during a treadmill stress test), favoring the comparison group receiving exercise alone. However, at a 2-month follow up, the CBT-SM plus exercise group had a larger treatment effect (0.80) than the exercise alone group (0.42) for dyspnea intensity (CRQ), which was clinically significant. It may be that a CBT-SM intervention adjunctive to exercise improves dyspnea more in the long-term by promoting healthy lifestyle change compared with an exercise-alone intervention.

In an innovative study by Nguyen et al[55] (n = 39), a face-to-face D-SM study was compared with a web-based D-SM intervention. ET was not combined with the SM intervention. No differences were found between the groups for dyspnea based on the delivery mode ($P > 0.05$). Large within-group dyspnea self-efficacy treatment effects were found for the web-based SM intervention (0.91), with effects maintained at 6 months compared with small effects (0.31 and 0.13, respectively) for the face-to-face SM intervention.

Summary

Evidence supports a CBT-based COPD intervention consisting of 24 supervised treadmill exercise sessions (conceptualized as dyspnea exposure therapy) combined with D-SM education for decreasing dyspnea in the short- and long-term (at 12 months). Preliminary evidence from four studies also exists of clinically significant within-group effects for stand-alone D-SM programs that include an individualized home exercise program, with small to large effects for improving dyspnea.[52,53,55,56]

Web-based and telephone technology have begun to be used for delivering COPD SM interventions. Smartphones and computer technologies have enormous potential for advancing PR and D-SM programs in the future.[57] Future studies should consider these delivery methods for SM interventions.

Distractive auditory stimuli

We identified three RCTs evaluating the effectiveness of music in relieving dyspnea in adults with COPD, with samples of 24–64 participants (Table 6). Participants' COPD severity was moderate to very severe in two studies and moderate to severe in one study. Two of the three studies evaluated the effects of combining music in the form of DAS with exercise. DAS or music has been conceptualized as a CBT strategy that provides desensitization of dyspnea and anxiety during physical exertion such as exercise.[58]

Table 6 Distractive auditory stimulus (DAS/music) studies

Study	Design/intervention	N	FEV₁% predicted, M ± SD	Group	Dyspnea score; short-term, M (SD)	MCID	Effect size
Singh et al[61]	RCT; pre- post-test design; 30 minutes, twice on same day; PMR pre-recorded; inpatient intervention	32 32	49 ± 11 54 ± 11	E: Music C: PMR	E: 49.1 (17.1)^{pre1}–34.7 (14.3)^{post1} VAS* C: 46 (13)^{pre1}–38.4 (14.6)^{post1} E: 47 (16.2)^{pre2}–23.9 (10.2)^{post2} * C: 45.4 (11.3)^{pre2}–32.5 (14.4)^{post2}	√ X √ √	−0.91 −0.55 −1.71 −1.0
Bauldoff et al[59]	RCT; repeated measures: 0, 4, 8 weeks; post-PR, home walking program with DAS and electronic pedometer/log; Music: 90–110 bpm	12 12	40.4 ± 15.6 42.3 ± 10.5	E: DAS (music) C: HEP only	E: 60.2–52.2–44.2^{8wks} ** UCSD C: 45.5–47.1–53.3 E: 2.9 (1.2)–3.3 (1.2)–3.0 (1.2) Borg^{6MW} C: 3.6 (1.1)–3.2 (1.1)–3.2 (0.8) E: 63.8 (24.2)–65.9 (23.3)–67.8 (19.9) SGRQ-A C: 62.0 (19.9)–61.5 (17.8)–64.3 (16.1)	√$^{4, 8wks}$ √$^{worse, 8wks}$ X X √$^{worse, 8wks}$ X	 E: 0.33, 0.08 C: −0.36, −0.42 E: 0.09, 0.18 C: −0.03, 0.13
Bauldoff et al[60]	RCT; post-PR; UET sessions 15 minutes, 2–3 times/week for 4 weeks; portable music	10 10 10	33.8 ± 16.3 41.3 ± 17.8 46.1 ± 19.9	E₁: UET + DAS^{Mod} E₂: UET + DAS^{Slow} C: UET	E₁: 65.8 (21.8)–62.3 (18.5) UCSD^{4wks} E₂: 58.4 (22.9)–53.9 (22.3) C: 55.2 (22.8)–54.0 (16.7) E₁: 78.8 (10.5)–72.3 (16.7) SGRQ-A E₂: 77.7 (15.9)–68.3 (15.1) C: 79.4 (15.6)–76.8 (15.7)	X X X √ √ X	−0.17 −0.20 −0.06 −0.47 −0.61 −0.17

Notes: *$P < 0.05$, favoring treatment group; **$P < 0.01$ favoring treatment group.
Abbreviations: Borg, modified Borg scale; bpm, beats per minute; C, control or comparison group; DAS, distractive auditory stimuli; E, experimental group; FEV₁, forced expiratory volume in 1 second; HEP, home exercise program; M, mean; mod, moderate; MCID, minimal clinical important difference, PMR, progressive muscle relaxation; postx, post session x; PR, pulmonary rehabilitation; prex, pre session x; RCT, randomized controlled trial; SD, standard deviation; SGRQ-A, St George's Respiratory Questionnaire, activity scale; UCSD, University of California, San Diego Shortness of Breath Questionnaire; UET, upper extremity training exercises using upper body ergometry; VAS, 100 mm visual analog scale; 6MW, 6-Minute Walk Test, meters; √, exceeded MCID; X, did not meet MCID.

A difference was found for dyspnea with activities ($P < 0.01$) in the short term favoring the DAS intervention in a small RCT; the group difference exceeded 5.0 units MCID for the UCSD SOBQ.[59] The DAS group listened to self-selected upbeat music of 90–110 beats per minute during unsupervised home walking. Participants in both study groups (the DAS and Home Exercise Program [HEP] only groups) were given an unsupervised home walking program using a pedometer post-PR. DAS appeared to promote better tolerance of dyspnea and a greater training effect.

Bauldoff et al[60] evaluated the effectiveness of combining DAS with a 4-week upper extremity (UE) training program. No differences were found for dyspnea between the three groups ($P > 0.05$). The disabling impact of dyspnea with activities (as measured by the SGRQ-A subscale) showed clinically significant reductions in the DAS groups (of greater than the MCID of 4 points) but not in the control group of UE training alone. A moderate treatment effect (−0.61) was found for the slow-tempo DAS intervention (60–89 beats per minute).

Singh et al[61] studied the effects of music compared with a progressive muscle relaxation (PMR) intervention in medically stable inpatients with COPD. Both music (self-selected, non-lyrical classical instrumental, 60–80 beats per minute) and PMR were clinically effective (exceeding the MCID) in immediately reducing dyspnea as measured by the vertical VAS. The dyspnea and anxiety improvements for the music group were higher than for the PMR group ($P < 0.01$). Both interventions showed medium to large ESs on reducing dyspnea intensity.

Summary

The three RCTs provide supporting evidence for the use of music as a CBT-based distraction strategy. A theoretical rationale for DAS or music is that it occupies the attention channels that normally carry dyspneic information.[60] Music may promote desensitization to dyspnea during ET.[58] By reducing dyspnea, DAS appears to promote greater exercise tolerance, (including walking distance[59] and UE repetitions[60]) and positive affect, and to reduce anxiety.[59,61] These outcomes are important goals of PR for patients with COPD.[62,63]

Conclusion

CBT and SM interventions hold promise in effectively relieving disabling sensory and affective dyspnea in COPD. Recent research shows that such interventions can enhance COPD dyspnea outcomes when used in combination with, and adjunctive to, ET. These interventions appear to improve dyspnea and counteract related emotional stress and fight-or-flight physiological and behavioral responses. CBT interventions focused specifically on dyspnea exposure and

cognitive restructuring may be especially useful in relieving dyspnea distress and warrant further research.[15] Additional clinical trials of CBT strategies are needed to provide stronger evidence of their effectiveness in relieving dyspnea with COPD. Future studies should include validated measures of both affective and sensory dyspnea to more comprehensively measure the effectiveness of dyspnea treatments.

Disclosure

The authors report no conflicts of interest in this work.

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