



Published in final edited form as:

Ann Behav Med. 2012 August ; 44(1): 52–65. doi:10.1007/s12160-012-9348-7.

Behavioral Medicine Approaches to Chronic Obstructive Pulmonary Disease

Andreas von Leupoldt, Ph.D.,

Department of Psychology, University of Hamburg, Von-Melle-Park 5, 20146 Hamburg, Germany.
Department of Systems Neuroscience, University Medical Center Hamburg-Eppendorf, Hamburg, Germany

Anja Fritzsche, Ph.D.,

Department of Psychology, University of Hamburg, Von-Melle-Park 5, 20146 Hamburg, Germany

Ana F. Trueba, M.A.,

Department of Psychology, Southern Methodist University, Dallas, TX, USA

Alicia E. Meuret, Ph.D., and

Department of Psychology, Southern Methodist University, Dallas, TX, USA

Thomas Ritz, Ph.D.

Department of Psychology, Southern Methodist University, Dallas, TX, USA

Andreas von Leupoldt: andreas.vonleupoldt@uni-hamburg.de

Abstract

Background—Chronic obstructive pulmonary disease (COPD) is a prevalent respiratory disease and associated with considerable individual and socioeconomic burden. Recent research started examining the role of psychosocial factors for course and management of the disease.

Purpose—This review provides an overview on recent findings on psychosocial factors and behavioral medicine approaches in COPD.

Results—Research has identified several important psychosocial factors and effective behavioral medicine interventions in COPD. However, there is considerable need for future research in this field.

Conclusions—Although beneficial effects of some behavioral medicine interventions have been demonstrated in COPD, future research efforts are necessary to study the effects of distinct components of these interventions, to thoroughly examine promising but yet not sufficiently proven interventions, and to develop new creative interventions.

Keywords

Behavioral medicine; Chronic obstructive pulmonary disease; COPD; Psychosocial; Treatment

Introduction

Chronic obstructive pulmonary disease (COPD) is a progressive chronic respiratory disease and associated with considerable individual as well as social and economic burden [1].

Already a leading cause of morbidity and mortality worldwide, the prevalence of COPD is projected to dramatically increase in the upcoming decades implying significant consequences for public health care systems [1, 2]. In addition to the prominent physiological symptoms, psychosocial aspects play an important role in the course and management of the disease. However, compared to other chronic diseases such as asthma, behavioral medicine approaches in COPD have not received the same level of systematic research activity or public interest [3]. Therefore, the present review provides an introduction to the disease characteristics and current medical treatment approaches, followed by an overview on important psychosocial influences as well as behavioral medicine diagnostic and treatment approaches to COPD. Moreover, needs for future research in this area will be highlighted.

Definition, Epidemiology, and Pathophysiology of COPD

COPD is a chronic respiratory disease with some significant extrapulmonary (systemic) effects that may contribute to the severity in individual patients [1]. It is characterized by progressive airflow limitation which, in contrast to asthma, is not fully reversible and associated with abnormal inflammatory responses of the lung to noxious particles or gases [1]. The chronic airflow limitation is caused by a mixture of small airway disease (subtype obstructive bronchiolitis) and parenchymal destruction (subtype emphysema) with varying contributions of these factors between patients. The chronic inflammation leads to structural changes and narrowing of the small airways [1, 4, 5]. The cardinal symptom of COPD is dyspnea (breathlessness), the experience of uncomfortable breathing, which constitutes a frightening experience for many patients [1, 6]. At early stages of COPD, dyspnea usually develops during physical activities and exercise, whereas at later stages of the disease, it is already present at rest and is caused by hyperinflation of the lungs (i.e., an increase of end-expiratory lung volume due to airflow limitation), weakened respiratory muscles, or insufficiencies in gas exchange. The perception of dyspnea involves sensory and emotional aspects [7–10], which are presumably controlled by distinct brain areas [11–16], but respective neuroimaging data are currently not available in patients with COPD. In particular, the emotional aspects of perceived dyspnea seem to be closely linked to psychosocial factors and disease-related behavior in the everyday life of patients [8, 17–19]. Other major symptoms of COPD are cough and augmented sputum production. Prominent extrapulmonary symptoms or comorbidities include skeletal muscle wasting, nutritional abnormalities, systemic inflammation, cardiovascular disease, osteoporosis, lung cancer, depression, sleep disorders, and diabetes, which can potentiate the morbidity of COPD [1, 20].

Although the natural history and the course of the disease vary between patients, the most commonly encountered risk factor for developing COPD is cigarette smoking [1], with up to 90% of all deaths from COPD being attributable to smoking [21]. Further risk factors include inhalational exposures to occupational dusts, chemicals, and in many countries air pollution due to burning wood and biomass fuels. Additionally, other influences such as infections in early life, genetic predispositions, and pre-existing asthma might be contributory in some individuals [1]. In general, COPD is a progressive disease, especially if the exposure of patients to noxious agents continues.

The international Burden of Obstructive Lung Disease study has demonstrated a prevalence of stage II or more severe COPD of 10% across 12 sites on different continents. However, prevalence rates showed considerable variations between countries with the lowest rates (9%) observed in Canada, Germany, and Australia and the highest rates observed in South Africa and the Philippines (22% and 19%, respectively) [22]. Older age was consistently related to an increased risk of developing COPD, with the highest rates being found in those

over 60 years of age [2, 22, 23]. It is assumed that in many cases, COPD is not detected due to insufficient diagnostics or because milder symptoms in the first stage of the disease remain often unrecognized by patients [4]. The prevalence and burden of COPD are projected to dramatically increase in the coming decades, which is partly related to changes in the age structure of the global population, with more individuals reaching an older age at which COPD usually develops [1, 2]. For example, the Global Burden of Disease study concludes that COPD worldwide will increase its ranking from 2002 to 2030 for disease-related deaths from rank 5 to rank 4 and for disability-adjusted life years lost from rank 11 to rank 7 [24]. The economic burden of the disease is substantial. For the USA, the estimated costs for health care expenditures and lost productivity due to COPD in the year 2010 were nearly \$50 billion USD [25].

According to well-established guidelines, a clinical diagnosis of COPD should be considered in any individual over age 40 who demonstrates dyspnea (usually progressive and worse with exercise), chronic cough or sputum production, and/or a history of exposure to risk factors for the disease such as tobacco smoke [1]. The diagnosis should be confirmed by a spirometric lung function test. The presence of a postbronchodilator ratio of the forced expiratory volume in 1 s (i.e., the amount of air that can be exhaled during a specific forceful exhalation maneuver)/forced vital capacity (FEV_1/FVC) < 0.70 and FEV_1 < 80% of the normative values for a given age and gender confirms the presence of a not fully reversible airflow limitation and helps differentiating COPD from asthma. The assessment of the severity level of COPD is based on the severity of lung function impairment, the patient's level of symptoms, and the presence of complications which are grouped to four severity levels as suggested by Global Initiative for Chronic Obstructive Lung Disease (GOLD; see Table 1), but also by other guidelines (e.g., [4, 26]).

COPD exacerbations have been characterized in various ways in the past [27]. Most recent guidelines [1] suggest that an exacerbation should be defined as: "... an event in the natural course of the disease characterized by a change in the patient's baseline dyspnea, cough and/or sputum that is beyond normal day-to-day variations, is acute in onset, and may warrant a change in regular medication..." (p. 64). Exacerbations can be caused by bacterial or viral infections and environmental pollutants, but in 1/3 of severe exacerbations underlying causes have not been identified [1]. Because exacerbations are associated with stronger inflammatory responses, reduced quality of life and health status as well as a poor prognosis of the disease, the prevention, early detection, and immediate treatment of exacerbations is a major goal in the management of COPD.

Under certain circumstances (e.g., advanced stages of the disease, family history of COPD), arterial blood gas measurements, chest radiography, exercise tests, respiratory muscle function tests, auscultations, or alpha-1 antitrypsin deficiency screenings might provide important additional diagnostic information. Because COPD is a progressive disease, symptoms and lung function should continuously be monitored in order to determine possible modifications in the therapy. Due to their negative interactions with the course of disease, comorbidities should be identified and managed [1].

Medical Treatment of COPD

Pharmacologic therapy in patients with COPD is the first choice of treatment used to prevent and control respiratory symptoms, to reduce frequency and severity of exacerbations, as well as to improve health status and exercise tolerance. Although most medications do not reverse the long-term decline of lung function, limited data suggest that the rate of this decline might be slowed with some medications [28, 29]. Current guidelines [1, 4, 26, 30] recommend short-acting or long-acting bronchodilator treatments as the mainstay of

pharmacological therapy, which decrease airway smooth muscle tone, thus improving expiratory flow rates, reducing hyperinflation, and resulting in reduced dyspnea. These treatments are preferably administered by inhalation and attention to effective drug delivery and inhaler technique is essential. For symptomatic patients with severe forms of COPD or for specific subgroups, additional treatments can be indicated, e.g., with glucocorticosteroids or antibiotics. Optimal pharmacotherapy of COPD should be individualized based on the patient's level of disease severity as assessed by spirometric lung function tests, symptoms and disability, frequency of acute exacerbations, and occurrence of side effects which necessitates continuous monitoring. In some very severely affected patients intermittent or long-term oxygen therapy, ventilator support or surgical procedures (e.g., lung volume reduction, lung transplantation) might become necessary [1, 4, 26, 30]. Severe exacerbations can require emergency department treatments or hospitalizations. Because comorbid symptoms are frequent in COPD, they often require additional medical treatments.

Psychosocial Influences on COPD

Psychosocial factors play an important role in COPD. Due to the chronic and progressive character of the disease, patients are not only physically limited, but often show great reductions in their psychological and social functioning which can feedback on the course of disease as well as on the social environment [1, 31]. However, research efforts on the specific mechanisms linking psychosocial factors and COPD have so far remained very limited, and only the past few years have seen an acceleration [3]. In general, both direct physiological (e.g., increased inflammatory processes due to high individual or social stress levels) and indirect behavioral pathways (e.g., poor disease management due to comorbid psychological symptoms) might link psychosocial aspects with the course of COPD, as has been demonstrated for other respiratory diseases such as asthma [32–35].

Comorbidity of COPD with Psychological Disorders

In patients with COPD, comorbid psychological symptoms are highly prevalent, in particular depression and anxiety. Recent studies reported prevalence rates ranging from 8% to 80% for symptoms of depression and from 6% to 74% for symptoms of anxiety [31, 36]. This large variance is presumably related to the large differences in measurement instruments, diagnostic criteria, study designs, and illness severity. In a meta-analysis of 13 studies by Yohannes et al. [37], the pooled analysis revealed prevalence rates for depression and anxiety of 40% and 36%, respectively, which might be considered as more realistic benchmark figures. Previous studies demonstrated that comorbid depression and anxiety in COPD patients is related to a worse course of the disease including increased mortality, more frequent exacerbations, persistent smoking, longer and more frequent hospitalizations, increased symptom burden, worsened physical and social functioning, decreased quality of life, reduced activity levels, and less favorable outcome of pulmonary rehabilitation (PR) [36, 38–46]. Importantly, psychological comorbidities often remain undetected and untreated in patients with COPD [47]. A previous study demonstrated that in less than 44% of COPD patients, clinically relevant anxiety and/or depression was correctly diagnosed and that only 31% of these patients received any treatment for these psychological comorbidities [48]. Therefore, the detection and treatment of comorbid psychological symptoms in patients with COPD remains a major clinical target.

At present, the exact causes for the high prevalence of psychological symptoms in patients with COPD are widely unknown. A population-based longitudinal study suggested that pre-existing symptoms of anxiety and depression were associated with the new onset of dyspnea, whereas pre-existing dyspnea was only weakly associated with later symptoms of anxiety and depression [49]. However, this sample only included patients with asthma as well as individuals without respiratory symptoms, thus preventing specific conclusions for

patients with COPD. Other authors emphasized the role of disease severity and/or repeated experiences with frightening episodes of dyspnea as potential causes for the later development of psychological comorbidities (in particular anxiety), especially for patients who tend to misinterpret or catastrophize bodily sensations [39, 40, 50]. It might further be speculated that a common factor is underlying both COPD and psychopathology, e.g., systemic inflammatory processes or genetic influences [51]. Once comorbid psychopathology and COPD are present, both direct physiological and indirect behavioral pathways might link these comorbidities with a worse course of disease. Whereas studies for the direct physiological pathway are scarce in COPD patients, several studies demonstrated that psychopathology in COPD is related to worse exercise performance [39, 40, 44, 52]. This might be indicative of a behavioral pathway such that anxious avoidance or depression related motivational difficulties result in avoidance of activities that could lead to dyspnea, a sedentary lifestyle, and a further decrease in patients' health status. Another behavioral pathway of comorbid psychological symptoms is their negative impact on adherence to prescribed treatments, in particular smoking cessation or medication adherence, which is generally regarded as poor in COPD patients [53]. Overall, future studies are needed to specify the exact direct and indirect mechanisms that link psychopathology and worse course of COPD.

Social Aspects in COPD

A growing body of studies suggests that supportive social relationships might be related to a more favorable course of disease in patients with COPD [54]. In this regard, patients living with a partner or perceiving high satisfaction with available social support showed less hospital readmission rates, dyspnea, or depressive symptoms as well as improved exercise tolerance and survival rates compared to patients living alone or perceiving low satisfaction with social support [55, 56]. However, other studies failed to observe such associations or found patients living alone to show even greater improvements in quality of life after pulmonary rehabilitation [57, 58]. The specific mechanisms that might underlie possible associations between stronger social support and better COPD disease status remain to be investigated. It has been speculated that social support might be effective in absorbing distress caused by the deleterious consequences of the illness, in enhancing perceived self-efficacy, and/or in promoting positive adaptive health behaviors including treatment adherence [53, 55, 59]. Recent findings further suggest that also social comparison processes, i.e., the degree to which patients perceive themselves as similar to other patients with more or less favorable states of disease, can impact outcomes of pulmonary rehabilitation [60]. Importantly, some studies have demonstrated that not only patients with COPD but also their partners show lower quality of life and stronger symptoms of anxiety and depression which, in turn, can interact with the patients' level of physical symptoms [61, 62]. These data indicate that partners of COPD patients can be substantially affected by the disease of their ill partners and suggest a need for interventions that include partners [3]. Overall, research on social factors in COPD is still in its infancy, and larger, controlled studies are necessary to increase our understanding on specific mechanisms, qualities, and effects of these interactions.

Diagnostic Assessment of COPD in Behavioral Medicine

A detailed diagnostic assessment of patients with COPD is not limited to physiological and functional aspects, but should also explore the impact of the disease on feelings of depression and anxiety, activity limitations, missed work, the economic situation, family routines, as well as smoking status and motivation to quit smoking [1, 63]. In addition, the overlap of symptoms of depression and anxiety with symptoms of COPD makes a sound diagnostic procedure essential. In the daily clinical routine, a first and quick screening can already be performed with simple verbal questions asking patients for their current

experience with respective symptoms, such as the standardized anxiety and depression screening questions from the Patient Health Questionnaire [64] or the Hospital Anxiety and Depression Scale [65], which avoids overlap with COPD-typical symptoms of fatigue, sleep disturbance, or dyspnea. If relevant psychological symptoms become obvious, patients should be referred to a specialized health care professional trained in clinical psychological diagnostics and treatments. For an overview of further questionnaire instruments for the assessment of psychological, but also disease-specific symptoms, experience of the disease, quality of life, or relevant behaviors, the reader is referred to previous reviews [31, 66, 67] and the website of the American Thoracic Society's Behavioral Science Assembly (<http://qol.thoracic.org/sections/instruments/ko/index.html>). Because patients' perception of symptoms, activity limitations, and health-related quality of life are often not well correlated with pulmonary function measurements, GOLD-defined disease stages, or physician's perceptions, the use of such validated self-report outcome measurements of health status (e.g., health-related quality of life, functional and emotional status) is now recognized as being key in capturing the patient's experience. Using these measures health care professionals can determine what is really important to the individual patient, highlight differences between patients, and optimize treatment efforts [66]. It must be noted, however, that several questionnaire measures commonly used in patients with COPD have so far not been validated for this specific population (in particular respective cutoff values), which warrants future research efforts.

Due to the progressive character of COPD, a close monitoring of physiological, functional, psychological, and social aspects has been recommended in recent guidelines [1]. This should also include the assessment of adherence to prescribed treatments such as medication intake, which is usually poor in COPD and related to significant health and economic burden [68, 69]. Medication adherence can be measured by biochemical evaluation of drug levels, electronic devices assessing the use of pills, inhalers or canisters, diaries as well as pharmacy records filling prescriptions. However, these techniques are rarely used in routine praxis due to high costs, administrative complexity, and/or problems with reliability [53]. Simple questions to the patients are perhaps the easiest approach to assess adherence, but the typical limitations of this approach (retrospective biases or inaccuracies in the form of overreporting of medication use) can also be expected in COPD [53, 68].

Behavioral Medicine Treatment

An effective management and treatment of COPD should be aimed at several goals: relief of symptoms, prevention of disease progression, improvement of exercise tolerance, improvement of health status and quality of life, prevention and treatment of complications and exacerbations, as well as reduction of mortality, which should be achieved with a minimum of treatment side effects [1]. Due to its somatic origin and potentially severe consequences, patients with COPD have to be diagnosed and medically treated by a physician, preferably with specific pulmonary training. However, many aspects of the disease require a more comprehensive treatment approach according to the biopsychosocial model of diseases to reduce the impact of the disease on quality of life and health status. This requires a multidisciplinary approach combining various forms of treatment, which have to be tailored to individual patient's needs and should continuously be monitored [1, 4]. Ideally, COPD care should be delivered by a multidisciplinary team including physicians, nurses, physiotherapists, exercise physiologists, kinesiologists, occupational therapists, dietician, social worker, mental health trained worker, behavior nurse therapist, clinical psychologist, or liaison psychiatrist [4, 26, 30]. Behavioral medicine offers a variety of relevant techniques and interventions that can be effective in alleviating the burden of COPD by initiating important behavioral change in patients. Most of the techniques reviewed below are part of comprehensive treatment programs such as pulmonary

rehabilitation or are used in some other form of combination, which makes it difficult to study their distinct individual effects.

Pulmonary Rehabilitation

The joint guidelines of the American Thoracic Society and the European Respiratory Society define pulmonary rehabilitation as "...evidence-based, multidisciplinary, and comprehensive intervention for patients with chronic respiratory diseases who are symptomatic and often have decreased daily life activities." [70]. The main goals of PR are the reduction of symptoms, the improvement of quality of life, and the increase in physical and emotional participation in everyday activities. A large body of evidence has demonstrated the beneficial effects of PR in several outcome domains for patients with COPD (Table 2). Therefore, treatment guidelines and systematic reviews strongly recommend PR for all disease stages including older patients, current smokers, and patients after exacerbations [1, 70–73]. Components of PR vary between programs and depend on individual needs of the patients. Common elements of comprehensive PR besides monitoring and optimization of pharmacological treatments are exercise training, patient education/behavioral training, nutrition counseling, psychosocial support, smoking cessation support, breathing therapy, and respiratory muscle training. PR can be performed in inpatient and outpatient settings. The optimal duration of PR remains to be established, but some evidence suggests that longer compared to shorter programs are more effective [74].

Exercise Training

Exercise training is the cornerstone of pulmonary rehabilitation and the best available means of improving skeletal muscle function in COPD. It is particularly indicated for those patients who have decreased exercise tolerance, exertional dyspnea or fatigue, and/or impairment of activities of daily living [70]. Improvements in skeletal muscle function after exercise training enhance exercise capacity despite the absence of improvements in lung function. Furthermore, the improved oxidative capacity and efficiency of the skeletal muscles lead to less alveolar ventilation for a given work rate resulting in reductions of dynamic hyperinflation and exertional dyspnea [70, 72]. Patients should perform a minimum of 20 sessions of exercise at a frequency of at least three times per week, and regular supervision of exercise sessions is necessary to achieve optimal physiologic benefits. Exercise should be adapted to individual patients' characteristics and limitations. Symptoms such as dyspnea and objective markers such as heart rate at the gas exchange threshold, blood pressure, or power output can be used to target training intensity [70].

Training components include lower extremity exercise (e.g., stationary cycle ergometer, treadmill, walking) as well as upper extremity exercise (e.g., arm cycle ergometer, free weights, elastic bands). Although both low-intensity and high-intensity exercise training produce clinical benefits in patients with COPD, training at higher exercise intensity seems to be associated with greater physiologic benefits than lower intensity training [70, 72]. A combination of endurance and strength training is probably the best strategy to treat peripheral muscle dysfunction because it leads to combined improvements in muscle strength and whole body endurance. In patients with airflow limitation, optimal bronchodilator therapy should be given prior to exercise training to reduce dyspnea and improve exercise tolerance [75]. Symptoms of depression and anxiety can limit the benefits of exercise training due to poor motivation and/or fear of symptoms such as exertional dyspnea and, thus, should be targeted before and during trainings [18, 39, 40, 44].

Physical inactivity has been demonstrated to be the strongest predictor of all-cause mortality in COPD [76]. Moreover, benefits achieved during PR wane after completion of PR, but if exercise training is continued at home, the health status of patients remains above levels

before PR [1, 77]. Therefore, a transfer of exercise motivation into everyday life is essential and underlines the need for behavioral changes that have to be initiated and supported during PR. If PR is not available, patients should be encouraged to lead an active life and to undertake a home-based exercise program to prevent progressive skeletal muscle deconditioning associated with inactivity [26]. Although home-based programs might be less individualized and certainly lack the daily physical presence of PR experts, some promising effects have been reported including improvements in dyspnea, quality of life, and exercise capacity [78–80].

Patient and Self-Management Education

Patient education plays an important role in the treatment of COPD and is a core component of pulmonary rehabilitation, although outcomes are often difficult to measure. The character of education is changing from traditional didactic lectures to more interactive self-management education, which not only conveys pure information but also includes the teaching of self-management skills emphasizing illness control through modifications of patients' health behavior [70]. Therefore, behavioral approaches such as cognitive behavioral therapy (CBT) are ideally suited to establish effective self-management skills. Self-management education is recommended in most treatment guidelines for COPD; however, topics suggested in specific programs vary. Typical components include the pathophysiology of lung disease, proper use of medication, benefits of exercise and physical activities, irritant avoidance/smoking cessation, prevention and early treatment of exacerbations, coping with COPD, end-of-life planning, control of anxiety and stress, breathing strategies, and bronchial hygiene techniques [1, 26, 70]. In particular, changes in self-efficacy due to self-management education have been suggested as being a key factor for improving clinical outcomes including treatment adherence [54, 70, 81]. Addressing patients' illness perceptions or beliefs about the disease and its treatment is also viewed as important [53, 82].

Recent systematic reviews have demonstrated that self-management education can reduce hospitalizations and improve quality of life, dyspnea, and exercise capacity in patients with COPD [83, 84]. However, more and larger randomized controlled trials with long-term follow-ups are needed to develop clear recommendations regarding the form and specific contents of self-management education programs in COPD. An interesting alternative to conventional programs might be internet-based programs, in which patients can participate from home. Pilot studies have demonstrated promising results of an internet-based dyspnea self-management program for COPD patients, including improvements in dyspnea, self-reported exercise time, physical functioning, and self-efficacy for managing dyspnea [85, 86].

Smoking Cessation

Tobacco smoke is the main risk factor for the development and poor prognosis of COPD, nonetheless up to 43% of patients with moderate to severe COPD continue to smoke [87], rendering smoking a major target for behavioral interventions. Indeed, international guidelines consistently recommend smoking cessation as the single most effective means of preventing COPD and of slowing the progress of the disease [1, 4, 26, 63, 88]. Findings from The Lung Health Study have shown that smoking cessation improves pulmonary function, dyspnea, and chronic cough, as well as reduces exacerbations, slows the rate of lung function loss, and lowers mortality in patients with COPD [89–91].

Several interventions are available to motivate and support individuals to quit smoking, often as an integral part of PR treatment programs. These include low intensity interventions such as brief advice by physicians or nurses, tailored self-help materials, and proactive

telephone counseling [63]. However, more intensive interventions are more effective than less intensive interventions. Evidence-based medicine reviews suggest that particularly counseling and group counseling are effective in helping individuals stop smoking [63, 92, 93]. Recent treatment guidelines and meta-analyses recommend the combination of psychosocial interventions and pharmacotherapy (e.g., nicotine replacement therapy, varenicline, bupropion) to support smoking cessation in patients with COPD [1, 4, 26, 63, 88, 94]. Typical success rates after 1 year from smoking cessation studies in COPD patients show considerable variations ranging from about 5% up to 35% in highly intensive programs and are usually lower compared with smokers without COPD [63]. These poor rates underline the urgent need for improved interventions aimed at supporting smoking cessation in COPD.

Respiratory Muscle Training

Functional inspiratory muscle strength and inspiratory muscle endurance are often compromised in patients with COPD. This results in respiratory muscle weakness which contributes to hypercapnia (i.e., increased levels of blood carbon dioxide), dyspnea, and reduced exercise performance [70]. Inspiratory muscle training is aimed at increasing inspiratory muscle strength, usually by having patients breathe through defined resistances (i.e., inspiratory resistive training, threshold loading). A recent meta-analysis has demonstrated that this training improves inspiratory muscle strength and endurance, functional exercise capacity, dyspnea, and quality of life in patients with COPD [95]. Specifically, strength training of the respiratory muscles was shown to be effective, whereas endurance training was less effective. In particular for COPD patients with inspiratory muscle weakness, the addition of inspiratory muscle training to a general exercise training program seems to improve exercise performance and is recommended in some guidelines as adjunctive therapy in PR [1, 70, 95, 96]. Similar to exercise training programs, training effects wear off in the long-term, and therefore, patients require special encouragement to continue with the training [95].

Breathing Training

Breathing training is recommended as complimentary intervention in the management of COPD in several reviews and guidelines [70, 97–99]. It includes a number of controlled-breathing exercises aimed at improving different pathophysiological aspects of lung diseases, such as reducing dynamic hyperinflation of the rib cage and improving regional ventilation, gas exchange, respiratory muscle function, and thoracoabdominal coordination [70, 98]. Prominent examples are pursed-lip-breathing, active expiration, and body positioning. Pursed-lip breathing can improve hyperinflation and gas exchange by prolonged expiration through half-opened lips and has been effective in reducing dyspnea in some patients with COPD [98, 100, 101]. Active compared to passive expiration involves contractions of abdominal muscles and might improve diaphragm-functioning and ventilation in COPD, but its significance for relieving dyspnea remains unclear and even dysfunctional effects such as increased rib cage hyperinflation have been reported [98, 102]. Body positioning techniques, especially forward leaning, improve diaphragmatic functioning and chest wall movement and reduce accessory muscle recruitment and dyspnea in patients with COPD [70, 98]. Overall, breathing training should be individualized and require careful patient selection, adequate instructions, and control of the techniques and their effects [70, 98].

Nutrition Counseling

In patients with COPD, nutritional status has an important impact on symptoms, disability, and prognosis and both overweight and underweight can be problematic. In particular, malnutrition has been observed in many patients (i.e., reduction of body mass index, BMI or

fat free mass), which is a prognostic risk factor for increased morbidity and mortality in COPD [103, 104]. The reasons for malnutrition in COPD are multifactorial and can include effects of increased work of breathing due to abnormal respiratory mechanics, effects of systemic inflammation, decreased dietary intake, dental problems, and dyspnea during or after eating [105]. It is therefore recommended in some guidelines to identify these patients by measuring BMI or fat free mass, to identify and to correct the causes for malnutrition [1]. This might include training of adequate eating behavior (e.g., higher caloric intake; small, frequent meals), correction of poor dentition, treatment of relevant comorbidities, and nutritional supplementation. Although active nutritional supplementation in undernourished patients with COPD can lead to weight gain, improved respiratory muscle function, and exercise performance, there is currently not enough evidence for its use as sufficient single intervention, but rather in combination with exercise training or within comprehensive PR programs [1, 30, 70, 105]. In addition, the long-term effects and optimal types of nutritional supplementation as well as long-term effects of respective pharmacological interventions (e.g., anabolic steroids) are not fully understood.

Cognitive Behavioral Therapy for Comorbid Anxiety and Depression

The high prevalence of psychological symptoms in patients with COPD necessitates adequate treatment. The few available studies on psychotropic medications (e.g., antidepressants, anxiolytics) have provided only weak evidence to support routine application in COPD patients and have shown considerable side effects [99, 106]. Greater compliance was observed in studies using CBT. Typical components include behavioral activation, enhancement of competency through skill-building exercises, cognitive restructuring to establish a more adaptive cognitive style, as well as psycho-education targeting automatic thoughts and their influence on feelings [107–110]. Furthermore, problem-solving techniques, sleep-management skills, thought stopping, self-instructional training, as well as non-specific relaxation or stress management techniques are often added, which should be adapted to the specific needs of these patients [111, 112]. With specific relevance to COPD, CBT addresses cognitive distortions about physical limitations, the emotional effects of such distortions, the vicious cycle of dyspnea–anxiety–activity avoidance–more dyspnea, and exposure to feared situations such as exercise. Usually, group sessions have been implemented in previous studies, which ranged from a single session up to 12-week programs and sometimes included additional supportive telephone contacts [50, 106].

Although CBT is effective for treating depression and anxiety in individuals without respiratory diseases [113–115] and has been recommended in some guidelines for the modification of health behavior in patients with COPD by improving self-management skills [1], findings in COPD patients are still limited. Recent reviews reported some positive effects of CBT in terms of reducing symptoms of depression or anxiety, but also in other outcome domains such as improvements in walking distance, quality of life, and hospital admissions in patients with COPD, especially in more recent studies [50, 106, 116–118]. However, more randomized controlled studies are necessary to draw definite conclusions on which specific CBT components are effective for which specific subpopulation of COPD patients as well as the ideal length of these interventions. These studies should also examine the additional benefits that specific CBT techniques might have for supporting smoking cessation or its effects on other outcomes such as perceived dyspnea or exercise tolerance.

Relaxation Techniques

Although significant effects on lung function due to relaxation cannot be expected, some guidelines list relaxation techniques such as muscle relaxation, imagery, and yoga as complementary interventions to reduce symptoms of anxiety, stress, or feelings of dyspnea

in patients with COPD [4, 70, 72]. The rationale of relaxation techniques is a reduction of physiological arousal, which results in parallel decreases of mental stress and ventilatory demand. However, acute effects of relaxation can also include bronchoconstriction [119, 120]. Although beneficial effects of relaxation on symptoms of anxiety, depression, or stress have been demonstrated in individuals without lung disease (e.g., [121, 122], there is at present insufficient evidence to suggest relaxation as effective routine intervention in patients with COPD. This is related to the small number of available studies which are characterized by heterogeneous methodological quality and partly conflicting results [97, 99]. However, pilot studies have suggested some beneficial effects of muscle relaxation and yoga on anxiety, dyspnea, and exercise performance [107, 123]. Again, future randomized controlled studies with adequate test power will be necessary for definite conclusion on whether these positive effects are clinically meaningful, long-lasting, and observable in which subtypes of patients.

Biofeedback Training

The limited number of currently available studies prevents a recommendation of biofeedback as routine intervention for COPD. However, a few smaller studies suggested some potential of biofeedback of abdominal muscles, arterial oxygen saturation, or ventilation patterns, respectively, when used to support breathing training in patients with COPD [124–126]. In addition, heart rate variability biofeedback combined with pulse oximetry biofeedback during walking exercise showed some improvements in exercise capacity, quality of life, self-efficacy, and dyspnea in one uncontrolled pilot study [127]. A potential mechanism for the observed improvements with heart rate variability biofeedback might be reduced phase differences between respiration and heart rate due to slowed breathing rates, which are assumed to improve gas exchange in the lungs [128, 129]. A promising variant of ventilation feedback training, which is aimed at prolonging exhalations by visual feedback of inspiratory and expiratory durations, could be its combination with cycle or treadmill exercise trainings in COPD patients. Collins et al. [130, 131] demonstrated that the combination of ventilation feedback plus exercise training increased exercise duration and expiratory time and reduced exercise-induced hyperinflation more when compared to exercise training without ventilation feedback or ventilation feedback alone. Overall, well-powered future randomized controlled studies are needed to further elaborate the beneficial effects of biofeedback techniques in COPD.

Distraction Techniques

Unconditioned patients with COPD often experience aversive dyspnea when starting exercise training after years of sedentary life-style. Attentional distraction has been suggested as helpful in reducing the perceived level of exertional dyspnea [132, 133], thereby increasing motivation for and adherence to training sessions. Attentional distraction is the re-direction of the focus of the conscious attentional state away from one sensation (e.g., dyspnea) toward another more pleasant sensation (e.g., music), which reduces cognitive processing resources for the former sensation. This rationale has been confirmed in research studying respiratory-related evoked potentials in the electroencephalogram, which showed reduced neural processing of respiratory sensations during attentional distraction compared to recruitment of attention conditions [134, 135]. However, research in COPD patients has produced conflicting results, with some studies showing reductions of exertional dyspnea and/or increases in exercise performance during attentional distraction by music [19, 136, 137], whereas other studies failed to observe beneficial effects on dyspnea [138–140]. Due to this inconclusive evidence, recent systematic reviews and guidelines do not support the routine use of attentional distraction for COPD [97, 99].

Alternative Techniques

Despite optimal pharmacological therapy, psychosocial support, and pulmonary rehabilitation, many patients with COPD continue experiencing symptoms such as dyspnea, which underlines the need for additional strategies to alleviate symptoms and to improve patients' quality of life. In this regard, recent pilot studies have investigated positive mood, humor, and laughter as well as singing as alternative techniques. Laughter is assumed to change the breathing pattern in addition to its positive effects on mood. A small uncontrolled study demonstrated that a single group intervention with the performance of a professional clown increased cheerfulness in patients with COPD [141]. Most interestingly, induced moderate laughter and smiling were associated with short-term reductions in hyperinflation, whereas intensive laughter was associated with an increase in hyperinflation. Another small study demonstrated that COPD patients' overall sense of humor was correlated with fewer symptoms of depression and anxiety and enhanced quality of life but suggested laughter induced by a humorous film clip to be associated with increased hyperinflation [142]. Moreover, short-lasting positive, compared to negative, mood states induced by watching affective picture series reduced dyspnea in patients with COPD during cycleergometer exercise without effects on cardiopulmonary measures [18].

Learning to sing is assumed to improve control of breathing and body posture. Two small controlled studies investigated the effects of weekly group singing classes in patients with COPD and reported improvements in quality of life, anxiety levels, and overall well-being [143, 144]. The majority of patients described singing classes as a positive experience associated with an improved sense of achievement and self-efficacy. However, whereas some small effects on ventilatory parameters were found in one study [143], no effects on ventilatory or exercise parameters were observed in the second study [144], which makes it difficult to estimate whether physiological mechanisms have contributed to these improvements. Again, future and well-controlled large studies are needed to investigate the effectiveness and underlying mechanisms of these, but also of new alternative techniques aimed at relieving the symptom burden of patients with COPD.

Conclusion

COPD is a highly prevalent, severe, and progressive respiratory disease with a major negative impact both on the patients' personal and socioeconomic level. In addition to progress in elucidating pathophysiological mechanisms and medical treatment options, recent research has demonstrated the important role of psychosocial factors in the course and management of the disease. Particularly, associations of psychological comorbidities, such as anxiety and depression, with worse disease status and treatment outcomes are now well established, although improving detection rates for these comorbidities in clinical diagnostic routine remains a major target. There is a considerable need for further research on the direct and indirect mechanisms of psychosocial influences on the pathophysiology of COPD. Progress in behavioral medicine approaches to COPD has been made in areas of pulmonary rehabilitation, exercise training, respiratory muscle training, and self-management education, although future studies are still needed on the effectiveness of distinct components of these interventions. Moreover, additional research on the optimal transfer of beneficial treatment effects in clinical settings into long-lasting improvements in the daily life of patients is necessary. In this context, home-based or internet-based interventions might be valuable new forms of intervention but require further evaluations. Areas in need of further research activity are the evaluation of CBT for patients with comorbid psychological disorders, breathing training, relaxation training, biofeedback training, social factors, and some aspects of nutrition, as well as more creative interventions such as distraction, humor, or singing. Given the important role of smoking for the development and progress of COPD, more research is needed to improve the success rates of interventions

aimed at smoking cessation. A significant increase in research efforts in these areas will be necessary to further advance behavioral medicine treatment options for one of the leading chronic disease conditions of the twenty-first century.

Acknowledgments

The work on the present manuscript was supported by a stipend (Heisenberg-Stipendium, LE 1843/9-2) from the German Research Society (Deutsche Forschungsgemeinschaft) to A.v.L. and partially by a grant of the National Institutes of Health (NIH R01 HL-089761) to T.R. and A.E.M.

References

1. Global Initiative for Chronic Obstructive Lung Disease (GOLD). Global Strategy for Diagnosis, Management, and Prevention of COPD. 2010. Available from: <http://www.goldcopd.com>
2. Gershon AS, Warner L, Cascagnette P, Victor JC, To T. Lifetime risk of developing chronic obstructive pulmonary disease: A longitudinal population study. *Lancet*. 2011 Sep 10; 378(9795): 991–6. [PubMed: 21907862]
3. Kaptein AA, Scharloo M, Fischer MJ, Snoei L, Hughes BM, Weinman J, et al. 50 years of psychological research on patients with COPD—road to ruin or highway to heaven? *Respir Med*. 2009 Jan; 103(1):3–11. [PubMed: 18930645]
4. National Clinical Guideline Centre. Chronic obstructive pulmonary disease: Management of chronic obstructive pulmonary disease in adults in primary and secondary care. 2010. Available from: <http://www.nice.org.uk/CG101>
5. O'Donnell DE, Banzett RB, Carrieri-Kohlman V, Casaburi R, Davenport PW, Gandevia SC, et al. Pathophysiology of dyspnea in chronic obstructive pulmonary disease: A roundtable. *Proc Am Thorac Soc*. 2007 Mai;4(2):145–68. [PubMed: 17494725]
6. American Thoracic Society. Dyspnea Mechanisms, assessment, and management: A consensus statement. *Am J Respir Crit Care Med*. 1999 Jan; 159(1):321–40. [PubMed: 9872857]
7. Carrieri-Kohlman V, Gormley JM, Eiser S, Demir-Deviren S, Nguyen H, Paul SM, et al. Dyspnea and the affective response during exercise training in obstructive pulmonary disease. *Nurs Res*. 2001 Juni;50(3):136–46. [PubMed: 11393635]
8. Lansing RW, Gracely RH, Banzett RB. The multiple dimensions of dyspnea: Review and hypotheses. *Respir Physiol Neurobiol*. 2009 Mai;167(1):53–60. [PubMed: 18706531]
9. Meek PM, Lareau SC, Hu J. Are self-reports of breathing effort and breathing distress stable and valid measures among persons with asthma, persons with COPD, and healthy persons? *Heart Lung*. 2003 Okt;32(5):335–46. [PubMed: 14528191]
10. von Leupoldt A, Dahme B. Differentiation between the sensory and affective dimension of dyspnea during resistive load breathing in normal subjects. *Chest*. 2005 Nov; 128(5):3345–9. [PubMed: 16304282]
11. Davenport PW, Vovk A. Cortical and subcortical central neural pathways in respiratory sensations. *Respir Physiol Neurobiol*. 2009 Mai;167(1):72–86. [PubMed: 18977463]
12. Evans KC. Cortico-limbic circuitry and the airways: Insights from functional neuroimaging of respiratory afferents and efferents. *Biol Psychol*. 2010 Apr; 84(1):13–25. [PubMed: 20211221]
13. von Leupoldt A, Dahme B. Cortical substrates for the perception of dyspnea. *Chest*. 2005 Juli; 128(1):345–54. [PubMed: 16002956]
14. von Leupoldt A, Sommer T, Kegat S, Baumann HJ, Klose H, Dahme B, et al. The unpleasantness of perceived dyspnea is processed in the anterior insula and amygdala. *Am J Respir Crit Care Med*. 2008 Mai;177(9):1026–32. [PubMed: 18263796]
15. von Leupoldt A, Sommer T, Kegat S, Eippert F, Baumann HJ, Klose H, et al. Down-regulation of insular cortex responses to dyspnea and pain in asthma. *Am J Respir Crit Care Med*. 2009 Aug 1; 180(3):232–8. [PubMed: 19483110]
16. Schön D, Rosenkranz M, Regelsberger J, Dahme B, Büchel C, von Leupoldt A. Reduced perception of dyspnea and pain after right insular cortex lesions. *Am J Respir Crit Care Med*. 2008 Dez;178(11):1173–9. [PubMed: 18776150]

17. Peiffer C. Dyspnea and emotion: What can we learn from functional brain imaging? *Am. J Respir Crit Care Med.* 2008 Mai;177(9):937–9.
18. von Leupoldt A, Taube K, Henkhus M, Dahme B, Magnussen H. The impact of affective states on the perception of dyspnea in patients with chronic obstructive pulmonary disease. *Biol Psychol.* 2010 Apr; 84(1):129–34. [PubMed: 21768011]
19. von Leupoldt A, Taube K, Schubert-Heukeshoven S, Magnussen H, Dahme B. Distractive auditory stimuli reduce the unpleasantness of dyspnea during exercise in patients with COPD. *Chest.* 2007 Nov; 132(5):1506–12. [PubMed: 17890458]
20. Barnes PJ, Celli BR. Systemic manifestations and comorbidities of COPD. *Eur Respir J.* 2009 Mai; 33(5):1165–85. [PubMed: 19407051]
21. Chen JC, Mannino DM. Worldwide epidemiology of chronic obstructive pulmonary disease. *Curr Opin Pulm Med.* 1999 März;5(2):93–9. [PubMed: 10813258]
22. Buist AS, McBurnie MA, Vollmer WM, Gillespie S, Burney P, Mannino DM, et al. International variation in the prevalence of COPD (the BOLD Study): A population-based prevalence study. *Lancet.* 2007 Sep 1; 370(9589):741–50. [PubMed: 17765523]
23. Menezes AMB, Perez-Padilla R, Jardim JRB, Muiño A, Lopez MV, Valdivia G, et al. Chronic obstructive pulmonary disease in five Latin American cities (the PLATINO study): A prevalence study. *Lancet.* 2005 Nov 26; 366(9500):1875–81. [PubMed: 16310554]
24. Mathers CD, Loncar D. Projections of global mortality and burden of disease from 2002 to 2030. *PLoS Med.* 2006 Nov.3(11):e442. [PubMed: 17132052]
25. National Heart, Lung, and Blood Institute. Morbidity and mortality chartbook on cardiovascular, lung and blood diseases. 2009. Available from: <http://www.nhlbi.nih.gov/resources/docs/cht-book.htm>
26. O'Donnell DE, Hernandez P, Kaplan A, Aaron S, Bourbeau J, Marciniuk D, et al. Canadian Thoracic Society recommendations for management of chronic obstructive pulmonary disease—2008 update—highlights for primary care. *Can Respir J.* 2008 Feb; 15(Suppl A):1A–8A.
27. Wedzicha JA, Seemungal TAR. COPD exacerbations: Defining their cause and prevention. *Lancet.* 2007 Sep 1; 370(9589):786–96. [PubMed: 17765528]
28. Celli BR, Thomas NE, Anderson JA, Ferguson GT, Jenkins CR, Jones PW, et al. Effect of pharmacotherapy on rate of decline of lung function in chronic obstructive pulmonary disease: Results from the TORCH study. *Am J Respir Crit Care Med.* 2008 Aug 15; 178(4):332–8. [PubMed: 18511702]
29. Decramer M, Celli B, Kesten S, Lystig T, Mehra S, Tashkin DP. Effect of tiotropium on outcomes in patients with moderate chronic obstructive pulmonary disease (UPLIFT): A prespecified subgroup analysis of a randomised controlled trial. *Lancet.* 2009 Okt;374(9696):1171–8. [PubMed: 19716598]
30. Vogelmeier C, Buhl R, Criée CP, Gillissen A, Kardos P, Köhler D, et al. Guidelines for the diagnosis and therapy of COPD issued by Deutsche Atemwegsliga and Deutsche Gesellschaft für Pneumologie und Beatmungsmedizin. *Pneumologie.* 2007 Mai;61(5):e1–40. [PubMed: 17436190]
31. Maurer J, Rebbapragada V, Borson S, Goldstein R, Kunik ME, Yohannes AM, et al. Anxiety and depression in COPD: Current understanding, unanswered questions, and research needs. *Chest.* 2008 Okt;134(4 Suppl):43S–56S. [PubMed: 18842932]
32. Kullowatz A, Kanniss F, Dahme B, Magnussen H, Ritz T. Association of depression and anxiety with health care use and quality of life in asthma patients. *Respir Med.* 2007 März;101(3):638–44. [PubMed: 16891108]
33. Sandberg S, Järvenpää S, Penttinen A, Paton JY, McCann DC. Asthma exacerbations in children immediately following stressful life events: a Cox's hierarchical regression. *Thorax.* 2004 Dez; 59(12):1046–51. [PubMed: 15563703]
34. Wright RJ. Perinatal stress and early life programming of lung structure and function. *Biol Psychol.* 2010 Apr; 84(1):46–56. [PubMed: 20080145]
35. Kullowatz A, Rosenfield D, Dahme B, Magnussen H, Kanniss F, Ritz T. Stress effects on lung function in asthma are mediated by changes in airway inflammation. *Psychosom Med.* 2008 Mai; 70(4):468–75. [PubMed: 18480192]

36. Yohannes AM, Willgoss TG, Baldwin RC, Connolly MJ. Depression and anxiety in chronic heart failure and chronic obstructive pulmonary disease: Prevalence, relevance, clinical implications and management principles. *Int J Geriatr Psychiatry*. 2010 Dez;25(12):1209–21. [PubMed: 20033905]
37. Yohannes AM, Baldwin RC, Connolly MJ. Mood disorders in elderly patients with chronic obstructive pulmonary disease. *Reviews in Clinical Gerontology*. 2000; 10(02):193–202.
38. de Voogd JN, Wempe JB, Koëter GH, Postema K, van Sonderen E, Ranchor AV, et al. Depressive symptoms as predictors of mortality in patients with COPD. *Chest*. 2009 März;135(3):619–25. [PubMed: 19029432]
39. Eisner MD, Blanc PD, Yelin EH, Katz PP, Sanchez G, Iribarren C, et al. Influence of anxiety on health outcomes in COPD. *Thorax*. 2010 März;65(3):229–34. [PubMed: 20335292]
40. Giardino ND, Curtis JL, Andrei A-C, Fan VS, Benditt JO, Lyubkin M, et al. Anxiety is associated with diminished exercise performance and quality of life in severe emphysema: A cross-sectional study. *Respir Res*. 2010; 11:29. [PubMed: 20214820]
41. Laurin C, Labrecque M, Dupuis G, Bacon SL, Cartier A, Lavoie KL. Chronic obstructive pulmonary disease patients with psychiatric disorders are at greater risk of exacerbations. *Psychosom Med*. 2009 Juli;71(6):667–74. [PubMed: 19561164]
42. Ng T-P, Niti M, Tan W-C, Cao Z, Ong K-C, Eng P. Depressive symptoms and chronic obstructive pulmonary disease: Effect on mortality, hospital readmission, symptom burden, functional status, and quality of life. *Arch Intern Med*. 2007 Jan 8; 167(1):60–7. [PubMed: 17210879]
43. Vögele C, von Leupoldt A. Mental disorders in chronic obstructive pulmonary disease (COPD). *Respir Med*. 2008 Mai;102(5):764–73. [PubMed: 18222685]
44. von Leupoldt A, Taube K, Lehmann K, Fritzsche A, Magnussen H. The impact of anxiety and depression on outcomes of pulmonary rehabilitation in patients with COPD. *Chest*. 2011 Sep; 140(3):730–6. [PubMed: 21454397]
45. Xu W, Collet J-P, Shapiro S, Lin Y, Yang T, Platt RW, et al. Independent effect of depression and anxiety on chronic obstructive pulmonary disease exacerbations and hospitalizations. *Am J Respir Crit Care Med*. 2008 Nov 1; 178(9):913–20. [PubMed: 18755925]
46. Quint JK, Baghai-Ravary R, Donaldson GC, Wedzicha JA. Relationship between depression and exacerbations in COPD. *Eur Respir J*. 2008 Juli;32(1):53–60. [PubMed: 18321938]
47. Kim HF, Kunik ME, Molinari VA, Hillman SL, Lalani S, Orengo CA, et al. Functional impairment in COPD patients: The impact of anxiety and depression. *Psychosomatics*. 2000 Dez;41(6):465–71. [PubMed: 11110109]
48. Kunik ME, Roundy K, Veazey C, Soucek J, Richardson P, Wray NP, et al. Surprisingly high prevalence of anxiety and depression in chronic breathing disorders. *Chest*. 2005 Apr; 127(4):1205–11. [PubMed: 15821196]
49. Neuman A, Gunnbjörnsdóttir M, Tunsäter A, Nyström L, Franklin KA, Norrman E, et al. Dyspnea in relation to symptoms of anxiety and depression: A prospective population study. *Respir Med*. 2006 Okt;100(10):1843–9. [PubMed: 16516455]
50. Livermore N, Sharpe L, McKenzie D. Panic attacks and panic disorder in chronic obstructive pulmonary disease: A cognitive behavioral perspective. *Respir Med*. 2010 Sep; 104(9):1246–53. [PubMed: 20457513]
51. Al-shair K, Kolsum U, Dockry R, Morris J, Singh D, Vestbo J. Biomarkers of systemic inflammation and depression and fatigue in moderate clinically stable COPD. *Respir Res*. 2011; 12:3. [PubMed: 21208443]
52. Spruit MA, Watkins ML, Edwards LD, Vestbo J, Calverley PMA, Pinto-Plata V, et al. Determinants of poor 6-min walking distance in patients with COPD: The ECLIPSE cohort. *Respir Med*. 2010 Juni;104(6):849–57. [PubMed: 20471236]
53. Bourbeau J, Bartlett SJ. Patient adherence in COPD. *Thorax*. 2008 Sep; 63(9):831–8. [PubMed: 18728206]
54. Kaplan, RM.; Ries, AL. Chronic obstructive pulmonary disease: Behavioural assessment and treatment. In: Kaptein, AA.; Creer, TL., editors. *Respiratory Disorders and Behavioral Medicine*. London: Martin Dunitz; 2002. p. 85-116.

55. Grodner S, Prewitt LM, Jaworsk BA, Myers R, Kaplan RM, Ries AL. The impact of social support in pulmonary rehabilitation of patients with chronic obstructive pulmonary disease. *Annals of Behavioral Medicine*. 1996 Sep;18:139–45.
56. Wong AWM, Gan WQ, Burns J, Sin DD, van Eeden SF. Acute exacerbation of chronic obstructive pulmonary disease: Influence of social factors in determining length of hospital stay and readmission rates. *Can Respir J*. 2008 Okt;15(7):361–4. [PubMed: 18949105]
57. Haave E, Hyland M. Different short-term and longitudinal results on perceived health status for asthma and COPD patients after pulmonary rehabilitation. Patients living alone have the largest improvements in perceived quality of life. *Chron Respir Dis*. 2008; 5(2):69–73. [PubMed: 18539719]
58. Trappenburg JC, Troosters T, Spruit MA, Vandebrouck N, Decramer M, Gosselink R. Psychosocial conditions do not affect short-term outcome of multidisciplinary rehabilitation in chronic obstructive pulmonary disease. *Arch Phys Med Rehabil*. 2005 Sep; 86(9):1788–92. [PubMed: 16181944]
59. Kara Ka ikçi M, Alberto J. Family support, perceived self-efficacy and self-care behaviour of Turkish patients with chronic obstructive pulmonary disease. *J Clin Nurs*. 2007 Aug; 16(8):1468–78. [PubMed: 17655535]
60. Petersen S, Taube K, Lehmann K, Van den Bergh O, von Leupoldt A. Social comparison and anxious mood in pulmonary rehabilitation: The role of cognitive focus. *Brit J Health Psychol*. 2011;110.1111/j.2044-8287.2011.02048.x
61. Köhl K, Schürmann W, Rief W. Mental disorders and quality of life in COPD patients and their spouses. *Int J Chron Obstruct Pulmon Dis*. 2008; 3(4):727–36. [PubMed: 19281087]
62. Pinto RA, Holanda MA, Medeiros MMC, Mota RMS, Pereira EDB. Assessment of the burden of caregiving for patients with chronic obstructive pulmonary disease. *Respir Med*. 2007 Nov; 101(11):2402–8. [PubMed: 17624751]
63. Tønnesen P, Carrozzi L, Fagerström KO, Gratiou C, Jimenez-Ruiz C, Nardini S, et al. Smoking cessation in patients with respiratory diseases: A high priority, integral component of therapy. *Eur Respir J*. 2007 Feb; 29(2):390–417. [PubMed: 17264326]
64. Kroenke K, Spitzer RL, Williams JB. The PHQ-9: Validity of a brief depression severity measure. *J Gen Intern Med*. 2001 Sep; 16(9):606–13. [PubMed: 11556941]
65. Zigmond AS, Snaith RP. The hospital anxiety and depression scale. *Acta Psychiatr Scand*. 1983 Juni;67(6):361–70. [PubMed: 6880820]
66. Jones PW, Price D, van der Molen T. Role of clinical questionnaires in optimizing everyday care of chronic obstructive pulmonary disease. *Int J Chron Obstruct Pulmon Dis*. 2011; 6:289–96. [PubMed: 21697993]
67. ZuWallack RL, Haggerty MC, Jones P. Clinically meaningful outcomes in patients with chronic obstructive pulmonary disease. *Am J Med*. 2004 Dez;117(Suppl 12A):49S–59S. [PubMed: 15693643]
68. Lareau SC, Yawn BP. Improving adherence with inhaler therapy in COPD. *Int J Chron Obstruct Pulmon Dis*. 2010; 5:401–6. [PubMed: 21191434]
69. Vestbo J, Anderson JA, Calverley PMA, Celli B, Ferguson GT, Jenkins C, et al. Adherence to inhaled therapy, mortality and hospital admission in COPD. *Thorax*. 2009 Nov; 64(11):939–43. [PubMed: 19703830]
70. Nici L, Donner C, Wouters E, Zuwallack R, Ambrosino N, Bourbeau J, et al. American Thoracic Society/European Respiratory Society statement on pulmonary rehabilitation. *Am J Respir Crit Care Med*. 2006 Juni;173(12):1390–413. [PubMed: 16760357]
71. Lacasse Y, Goldstein R, Lasserson TJ, Martin S. Pulmonary rehabilitation for chronic obstructive pulmonary disease. *Cochrane Database Syst Rev*. 2006; (4):CD003793. [PubMed: 17054186]
72. Ries AL, Bauldoff GS, Carlin BW, Casaburi R, Emery CF, Mahler DA, et al. Pulmonary rehabilitation: Joint ACCP/ AACVPR evidence-based clinical practice guidelines. *Chest*. 2007 Mai;131(5 Suppl):4S–42S. [PubMed: 17494825]
73. Puhan MA, Gimeno-Santos E, Scharplatz M, Troosters T, Walters EH, Steurer J. Pulmonary rehabilitation following exacerbations of chronic obstructive pulmonary disease. *Cochrane Database Syst Rev*. 2011; (10):CD005305. [PubMed: 21975749]

74. Beauchamp MK, Janaudis-Ferreira T, Goldstein RS, Brooks D. Optimal duration of pulmonary rehabilitation for individuals with chronic obstructive pulmonary disease—a systematic review. *Chron Respir Dis*. 2011; 8(2):129–40. [PubMed: 21596893]
75. Casaburi R, Kukafka D, Cooper CB, Witek TJ Jr, Kesten S. Improvement in exercise tolerance with the combination of tiotropium and pulmonary rehabilitation in patients with COPD. *Chest*. 2005 März;127(3):809–17. [PubMed: 15764761]
76. Waschki B, Kirsten A, Holz O, Müller K-C, Meyer T, Watz H, et al. Physical activity is the strongest predictor of all-cause mortality in patients with COPD: A prospective cohort study. *Chest*. 2011 Aug; 140(2):331–42. [PubMed: 21273294]
77. Griffiths TL, Burr ML, Campbell IA, Lewis-Jenkins V, Mullins J, Shiels K, et al. Results at 1 year of outpatient multidisciplinary pulmonary rehabilitation: A randomised controlled trial. *Lancet*. 2000 Jan 29; 355(9201):362–8. [PubMed: 10665556]
78. Behnke M, Jörres RA, Kirsten D, Magnussen H. Clinical benefits of a combined hospital and home-based exercise programme over 18 months in patients with severe COPD. *Monaldi Arch Chest Dis*. 2003 März;59(1):44–51. [PubMed: 14533282]
79. du Moulin M, Taube K, Wegscheider K, Behnke M, van den Bussche H. Home-based exercise training as maintenance after outpatient pulmonary rehabilitation. *Respiration*. 2009; 77(2):139–45. [PubMed: 18667807]
80. Maltais F, Bourbeau J, Shapiro S, Lacasse Y, Perrault H, Baltzan M, et al. Effects of home-based pulmonary rehabilitation in patients with chronic obstructive pulmonary disease: A randomized trial. *Ann Intern Med*. 2008 Dez;149(12):869–78. [PubMed: 19075206]
81. Harris M, Smith BJ, Veale A. Patient education programs—can they improve outcomes in COPD? *Int J Chron Obstruct Pulmon Dis*. 2008; 3(1):109–12. [PubMed: 18488433]
82. Kaptein AA, Scharloo M, Fischer MJ, Snoei L, Cameron LD, Sont JK, et al. Illness perceptions and COPD: An emerging field for COPD patient management. *J Asthma*. 2008 Okt;45(8):625–9. [PubMed: 18951252]
83. Effing T, Monnikhof EM, van der Valk PDLPM, van der Palen J, van Herwaarden CLA, Partidge MR, et al. Self-management education for patients with chronic obstructive pulmonary disease. *Cochrane Database Syst Rev*. 2007; (4):CD002990. [PubMed: 17943778]
84. Peytremann-Bridevaux I, Staeger P, Bridevaux P-O, Ghali WA, Burnand B. Effectiveness of chronic obstructive pulmonary disease-management programs: Systematic review and meta-analysis. *Am J Med*. 2008 Mai;121(5):433–43.e4. [PubMed: 18456040]
85. Nguyen HQ, Carrieri-Kohlman V, Rankin SH, Slaughter R, Stulbarg MS. Is Internet-based support for dyspnea self-management in patients with chronic obstructive pulmonary disease possible? Results of a pilot study. *Heart Lung*. 2005 Feb; 34(1):51–62. [PubMed: 15647734]
86. Nguyen HQ, Donesky-Cuenco D, Wolpin S, Reinke LF, Benditt JO, Paul SM, et al. Randomized controlled trial of an internet-based versus face-to-face dyspnea self-management program for patients with chronic obstructive pulmonary disease: Pilot study. *J Med Internet Res*. 2008; 10(2):e9. [PubMed: 18417444]
87. Calverley PMA, Anderson JA, Celli B, Ferguson GT, Jenkins C, Jones PW, et al. Salmeterol and fluticasone propionate and survival in chronic obstructive pulmonary disease. *N Engl J Med*. 2007 Feb 22; 356(8):775–89. [PubMed: 17314337]
88. Andreas S, Hering T, Mühlig S, Nowak D, Raupach T, Worth H. Smoking cessation in chronic obstructive pulmonary disease: An effective medical intervention. *Dtsch Arztebl Int*. 2009 Apr; 106(16):276–82. [PubMed: 19547629]
89. Anthonisen NR, Connett JE, Kiley JP, Altose MD, Bailey WC, Buist AS, et al. Effects of smoking intervention and the use of an inhaled anticholinergic bronchodilator on the rate of decline of FEV1. The Lung Health Study. *JAMA*. 1994 Nov 16; 272(19):1497–505. [PubMed: 7966841]
90. Kanner RE, Connett JE, Williams DE, Buist AS. Effects of randomized assignment to a smoking cessation intervention and changes in smoking habits on respiratory symptoms in smokers with early chronic obstructive pulmonary disease: The Lung Health Study. *Am J Med*. 1999 Apr; 106(4):410–6. [PubMed: 10225243]

91. Scanlon PD, Connett JE, Waller LA, Altose MD, Bailey WC, Buist AS. Smoking cessation and lung function in mild-to-moderate chronic obstructive pulmonary disease. The Lung Health Study. *Am J Respir Crit Care Med.* 2000 Feb; 161(2 Pt 1):381–90. [PubMed: 10673175]
92. Rigotti NA, Munafo MR, Stead LF. Interventions for smoking cessation in hospitalised patients. *Cochrane Database Syst Rev.* 2007; (3):CD001837. [PubMed: 17636688]
93. Stead LF, Lancaster T. Group behaviour therapy programmes for smoking cessation. *Cochrane Database Syst Rev.* 2005; (2):CD001007. [PubMed: 15846610]
94. van der Meer RM, Wagena EJ, Ostelo RWJG, Jacobs JE, van Schayck CP. Smoking cessation for chronic obstructive pulmonary disease. *Cochrane Database Syst Rev.* 2003; (2):CD002999. [PubMed: 12804448]
95. Gosselink R, De Vos J, van den Heuvel SP, Segers J, Decramer M, Kwakkel G. Impact of inspiratory muscle training in patients with COPD: What is the evidence? *Eur Respir J.* 2011 Feb; 37(2):416–25. [PubMed: 21282809]
96. 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 Sep; 20(3):570–6. [PubMed: 12358330]
97. Bausewein C, Booth S, Gysels M, Higginson I. Non-pharmacological interventions for breathlessness in advanced stages of malignant and non-malignant diseases. *Cochrane Database Syst Rev.* 2008; (2):CD005623. [PubMed: 18425927]
98. Gosselink R. Controlled breathing and dyspnea in patients with chronic obstructive pulmonary disease (COPD). *J Rehabil Res Dev.* 2003 Okt;40(5 Suppl 2):25–33. [PubMed: 15074451]
99. Marciniuk D, Goodridge D, Hernandez P, Rocker G, Balter M, Bailey P, et al. Managing dyspnea in patients with advanced chronic obstructive pulmonary disease: A Canadian Thoracic Society clinical practice guideline. *Can Respir J.* 2011 Apr; 18(2):69–78. [PubMed: 21499589]
100. Bianchi R, Gigliotti F, Romagnoli I, Lanini B, Castellani C, Grazzini M, et al. Chest wall kinematics and breathlessness during pursed-lip breathing in patients with COPD. *Chest.* 2004 Feb; 125(2):459–65. [PubMed: 14769725]
101. Nield MA, Soo Hoo GW, Roper JM, Santiago S. Efficacy of pursed-lips breathing: A breathing pattern retraining strategy for dyspnea reduction. *J Cardiopulm Rehabil Prev.* 2007 Aug; 27(4): 237–44. [PubMed: 17667021]
102. Gigliotti F, Romagnoli I, Scano G. Breathing retraining and exercise conditioning in patients with chronic obstructive pulmonary disease (COPD): A physiological approach. *Respir Med.* 2003 März;97(3):197–204. [PubMed: 12645825]
103. Gray-Donald K, Gibbons L, Shapiro SH, Macklem PT, Martin JG. Nutritional status and mortality in chronic obstructive pulmonary disease. *Am J Respir Crit Care Med.* 1996 März; 153(3):961–6. [PubMed: 8630580]
104. Wilson DO, Rogers RM, Wright EC, Anthonisen NR. Body weight in chronic obstructive pulmonary disease. The National Institutes of Health Intermittent Positive-Pressure Breathing Trial. *Am Rev Respir Dis.* 1989 Juni;139(6):1435–8. [PubMed: 2658702]
105. King DA, Cordova F, Scharf SM. Nutritional aspects of chronic obstructive pulmonary disease. *Proc Am Thorac Soc.* 2008 Mai;5(4):519–23. [PubMed: 18453365]
106. Fritzsche A, Clamor A, von Leupoldt A. Effects of medical and psychological treatment of depression in patients with COPD—a review. *Respir Med.* 2011 Okt;105(10):1422–33. [PubMed: 21680167]
107. Brenes GA. Anxiety and chronic obstructive pulmonary disease: Prevalence, impact, and treatment. *Psychosom Med.* 2003 Dez;65(6):963–70. [PubMed: 14645773]
108. Butler AC, Beck A. Cognitive therapy for depression. *The Clinical Psychologist.* 1995; 48(3):3–5.
109. Ingram, RE. *The International Encyclopedia of Depression.* New York: Springer; 2009.
110. Emery CF, Schein RL, Hauck ER, MacIntyre NR. Psychological and cognitive outcomes of a randomized trial of exercise among patients with chronic obstructive pulmonary disease. *Health Psychol.* 1998 Mai;17(3):232–40. [PubMed: 9619472]
111. Kunik ME, Braun U, Stanley MA, Wristers K, Molinari V, Stoebner D, et al. One session cognitive behavioural therapy for elderly patients with chronic obstructive pulmonary disease. *Psychol Med.* 2001 Mai;31(4):717–23. [PubMed: 11352373]

112. Kunik ME, Veazey C, Cully JA, Soucek J, Graham DP, Hopko D, et al. COPD education and cognitive behavioral therapy group treatment for clinically significant symptoms of depression and anxiety in COPD patients: A randomized controlled trial. *Psychol Med.* 2008 März;38(3): 385–96. [PubMed: 17922939]
113. Butler AC, Chapman JE, Forman EM, Beck AT. The empirical status of cognitive-behavioral therapy: A review of meta-analyses. *Clin Psychol Rev.* 2006 Jan; 26(1):17–31. [PubMed: 16199119]
114. Mitte K. A meta-analysis of the efficacy of psycho- and pharmacotherapy in panic disorder with and without agoraphobia. *J Affect Disord.* 2005 Sep; 88(1):27–45. [PubMed: 16005982]
115. Stewart RE, Chambless DL. Cognitive-behavioral therapy for adult anxiety disorders in clinical practice: A meta-analysis of effectiveness studies. *J Consult Clin Psychol.* 2009 Aug; 77(4):595–606. [PubMed: 19634954]
116. Baraniak A, Sheffield D. The efficacy of psychologically based interventions to improve anxiety, depression and quality of life in COPD: A systematic review and meta-analysis. *Patient Educ Couns.* 2011 Apr; 83(1):29–36. [PubMed: 20447795]
117. Hynninen MJ, Bjerke N, Pallesen S, Bakke PS, Nordhus IH. A randomized controlled trial of cognitive behavioral therapy for anxiety and depression in COPD. *Respir Med.* 2010 Juli;104(7): 986–94. [PubMed: 20346640]
118. Livermore N, Sharpe L, McKenzie D. Prevention of panic attacks and panic disorder in COPD. *Eur Respir J.* 2010 März;35(3):557–63. [PubMed: 19741029]
119. Lehrer PM, Hochron SM, Mayne TM, Isenberg S, Lasoski AM, Carlson V, et al. Relationship between changes in EMG and respiratory sinus arrhythmia in a study of relaxation therapy for asthma. *Appl Psychophysiol Biofeedback.* 1997 Sep; 22(3):183–91. [PubMed: 9428968]
120. Ritz T. Relaxation therapy in adult asthma. Is there new evidence for its effectiveness? *Behav Modif.* 2001 Sep; 25(4):640–66. [PubMed: 11530720]
121. Jorm AF, Morgan AJ, Hetrick SE. Relaxation for depression. *Cochrane Database Syst Rev.* 2008; (4):CD007142. [PubMed: 18843744]
122. Lehrer, PM.; Woolfolk, RL.; Sime, WE. *Principles and Practice of Stress Management.* 3. New York: Guilford; 2007.
123. Donesky-Cuenco D, Nguyen HQ, Paul S, Carrieri-Kohlman V. Yoga therapy decreases dyspnea-related distress and improves functional performance in people with chronic obstructive pulmonary disease: A pilot study. *J Altern Complement Med.* 2009 März;15(3):225–34. [PubMed: 19249998]
124. Estève F, Blanc-Gras N, Gallego J, Benchetrit G. The effects of breathing pattern training on ventilatory function in patients with COPD. *Biofeedback Self Regul.* 1996 Dez;21(4):311–21. [PubMed: 9031710]
125. Reybrouck T, Wertelaers A, Bertrand P, Demedts M. Myo-feedback training of the respiratory muscles in patients with chronic obstructive pulmonary disease. *J Cardiopulm Rehabil.* 1987; 7:18–22.
126. Tjep BL, Burns M, Kao D, Madison R, Herrera J. Pursed lips breathing training using ear oximetry. *Chest.* 1986 Aug; 90(2):218–21. [PubMed: 3731893]
127. Giardino ND, Chan L, Borson S. Combined heart rate variability and pulse oximetry biofeedback for chronic obstructive pulmonary disease: Preliminary findings. *Appl Psychophysiol Biofeedback.* 2004 Juni;29(2):121–33. [PubMed: 15208975]
128. Vaschillo E, Vaschillo B, Lehrer P. Heartbeat synchronizes with respiratory rhythm only under specific circumstances. *Chest.* 2004 Okt;126(4):1385–6. [PubMed: 15486413]
129. Yasuma F, Hayano J-I. Respiratory sinus arrhythmia: Why does the heartbeat synchronize with respiratory rhythm? *Chest.* 2004 Feb; 125(2):683–90. [PubMed: 14769752]
130. Collins EG, Fehr L, Bammert C, O’Connell S, Laghi F, Hanson K, et al. Effect of ventilation-feedback training on endurance and perceived breathlessness during constant work-rate leg-cycle exercise in patients with COPD. *J Rehabil Res Dev.* 2003 Okt;40(5 Suppl 2):35–44. [PubMed: 15074452]

131. Collins EG, Langbein WE, Fehr L, O'Connell S, Jelinek C, Hagarty E, et al. Can ventilation-feedback training augment exercise tolerance in patients with chronic obstructive pulmonary disease? *Am J Respir Crit Care Med.* 2008 Apr 15; 177(8):844–52. [PubMed: 18202351]
132. von Leupoldt A, Dahme B. Psychological aspects in the perception of dyspnea in obstructive pulmonary diseases. *Respir Med.* 2007 März;101(3):411–22. [PubMed: 16899357]
133. von Leupoldt A, Seemann N, Gugleva T, Dahme B. Attentional distraction reduces the affective but not the sensory dimension of perceived dyspnea. *Respir Med.* 2007 Apr; 101(4):839–44. [PubMed: 16971103]
134. Davenport PW, Chan P-YS, Zhang W, Chou Y-L. Detection threshold for inspiratory resistive loads and respiratory-related evoked potentials. *J Appl Physiol.* 2007 Jan; 102(1):276–85. [PubMed: 17008431]
135. Webster KE, Colrain IM. The respiratory-related evoked potential: Effects of attention and occlusion duration. *Psychophysiology.* 2000 Mai;37(3):310–8. [PubMed: 10860409]
136. Bauldoff GS, Hoffman LA, Zullo TG, Sciarba FC. Exercise maintenance following pulmonary rehabilitation: Effect of distractive stimuli. *Chest.* 2002 Sep; 122(3):948–54. [PubMed: 12226037]
137. Thornby MA, Haas F, Axen K. Effect of distractive auditory stimuli on exercise tolerance in patients with COPD. *Chest.* 1995 Mai;107(5):1213–7. [PubMed: 7750308]
138. Bauldoff GS, Rittinger M, Nelson T, Doehrel J, Diaz PT. Feasibility of distractive auditory stimuli on upper extremity training in persons with chronic obstructive pulmonary disease. *J Cardiopulm Rehabil.* 2005 Feb; 25(1):50–5. [PubMed: 15714113]
139. Brooks D, Sidani S, Graydon J, McBride S, Hall L, Weinacht K. Evaluating the effects of music on dyspnea during exercise in individuals with chronic obstructive pulmonary disease: A pilot study. *Rehabil Nurs.* 2003 Dez;28(6):192–6. [PubMed: 14649167]
140. Pfister T, Berrol C, Caplan C. Effects of music on exercise and perceived symptoms in patients with chronic obstructive pulmonary disease. *J Cardiopulm Rehabil.* 1998 Juni;18(3):228–32. [PubMed: 9632325]
141. Brutsche MH, Grossman P, Müller RE, Wiegand J, Pello, Baty F, et al. Impact of laughter on air trapping in severe chronic obstructive lung disease. *Int J Chron Obstruct Pulmon Dis.* 2008; 3(1): 185–92. [PubMed: 18488442]
142. Lebowitz KR, Suh S, Diaz PT, Emery CF. Effects of humor and laughter on psychological functioning, quality of life, health status, and pulmonary functioning among patients with chronic obstructive pulmonary disease: A preliminary investigation. *Heart Lung.* 2011 Aug; 40(4):310–9. [PubMed: 21724041]
143. Bonilha AG, Onofre F, Vieira ML, Prado MYA, Martinez JAB. Effects of singing classes on pulmonary function and quality of life of COPD patients. *Int J Chron Obstruct Pulmon Dis.* 2009; 4:1–8. [PubMed: 19436683]
144. Lord VM, Cave P, Hume VJ, Flude EJ, Evans A, Kelly JL, et al. Singing teaching as a therapy for chronic respiratory disease—a randomised controlled trial and qualitative evaluation. *BMC Pulm Med.* 2010; 10:41. [PubMed: 20682030]

Table 1GOLD classification of COPD severity based on post-bronchodilator FEV₁ with associated symptoms [1]

COPD stage	Spirometry	Common symptoms
Stage I: mild	FEV ₁ ≥80% predicted FEV ₁ /FVC<0.7	Chronic cough or sputum production may be present, but not always
Stage II: moderate	50% ≤FEV ₁ <80% predicted FEV ₁ /FVC<0.7	Dyspnea during exertion, chronic cough or sputum production may be present,
Stage III: severe	30% ≤FEV ₁ <50% predicted FEV ₁ /FVC<0.7	Greater dyspnea, reduced exercise capacity, fatigue, repeated exacerbations which impact quality of life
Stage IV: very severe	FEV ₁ <30% predicted FEV ₁ /FVC<0.7	Increasing symptoms, respiratory failure or cardiac comorbidities possible, greater reductions in quality of life, exacerbations may be life-threatening

COPD chronic obstructive pulmonary disease, *FEV₁* forced expiratory volume in 1 s, *FVC* forced vital capacity, *GOLD* Global Initiative for Chronic Obstructive Lung Disease

Table 2

Evidenced based benefits of pulmonary rehabilitation in patients with COPD (modified after GOLD [1])

Benefits of pulmonary rehabilitation
Improves exercise capacity
Reduces the perceived intensity of breathlessness
Improves health-related quality of life
Reduces the number of hospitalizations and days in the hospital
Reduces milder forms of anxiety and depression associated with COPD
Improves muscle functioning
Benefits extend beyond the immediate period of training
Improves survival

COPD chronic obstructive pulmonary disease, *GOLD* Global Initiative for Chronic Obstructive Lung Disease