Towards an Assessment of Perceived COPD Exacerbation Triggers: Initial Development and Validation of a Questionnaire Measure

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Summary at a Glance

The present study sought to develop a psychometrically valid measure of perceived triggers of exacerbations in COPD patients, the COPD Exacerbation Trigger Inventory (CETI). Patients' trigger classes and their controllability are associated with clinical outcomes and their assessment may prove useful in research and clinical settings with COPD patients, aiding exacerbation prevention and disease management.

ABSTRACT

Background and objective Prevention of exacerbations in chronic obstructive pulmonary disease (COPD) is important to slow overall declines in functioning and improve quality of life. The present study sought to develop a psychometrically valid measure of perceived triggers of exacerbations in COPD patients, the COPD Exacerbation Trigger Inventory (CETI).

Methods. Participants (N=192) were recruited through local clinics and online to complete surveys of the CETI, demographic information, disease specific information, and the COPD Assessment Test (CAT). The CETI included a free response section on patients' individual top triggers, combined with ratings of their controllability.

Results. Exploratory principle component analyses identified a stable 5-factor structure (33 items), from which trigger subscales for weather/climate, air pollution/irritants, exercise, infection/illness, and psychological factors were formed (internal consistency Cronbach's α=.90-.94). Trigger factors were associated with COPD functional status, exacerbation frequency, and healthcare utilization. Participants found personal triggers related to dust, air pollution, smoking, and physical activity to be the most easily controlled, whereas those related to psychological factors, climate, infection, respiratory symptoms, and sleep, to be more difficult to control. Greater perceived controllability of triggers was associated with lower CAT scores, indicating better health status and less impact of the disease on functioning.

Conclusion. The CETI is a psychometrically valid measure of perceived exacerbation triggers in patients with COPD. Perceived triggers are associated with clinical outcomes. Assessment of trigger classes and their controllability may prove useful in both research and clinical settings with COPD patients and to further our knowledge in prevention and disease management.

Keywords: chronic obstructive pulmonary disease (COPD); exacerbation triggers; functional status; illness perception; health status; questionnaire measure

Short title: COPD Exacerbation Triggers

INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a progressive respiratory disease and one of the leading causes of mortality[1]. Exacerbations, or significant worsening of respiratory symptoms beyond normal fluctuations, negatively impact patients' quality of life[2] and can be precipitated by a number of factors[3,4]. Exacerbations accelerate decline in lung function and are associated with high socioeconomic costs and increased risk of mortality[1,3,4].

The most commonly recognized exacerbation triggers are bacterial and viral infections[1,3,4]. Outdoor air pollution is another well-established factor linked to exacerbations, hospital admissions, or emergency treatments in COPD[5,6]. Tobacco smoking and second-hand smoke are also associated with exacerbations[7,8]. In addition, seasonal factors, such as winter weather, have been linked to exacerbations[9].

Links between psychological distress and exacerbations have also been documented[10-12]. Patients with COPD have higher levels of anxious and depressed mood[12-14] which are likely to have complex and bidirectional associations with exacerbations. The experience of strong negative emotions can be associated with elevated inflammation that promotes exacerbations[11,12]. Additionally, exacerbations are stressful experiences that might place patients at a higher risk for the development of anxiety and depression. This highlights the need for more research to elucidate the complex relationship between psychological distress and COPD exacerbations.

Whereas measures of exacerbation triggers exist for other airway diseases such as asthma[15,16], a psychometrically valid measure does not currently exist for assessing patients' perceptions of triggers factors in COPD. Although progress has been made in the treatment of

exacerbations and long-term management of the disease, efforts in assessment and prevention of exposure to exacerbation triggers from the patients' perspective have been lacking[3]. A measure of perceived COPD exacerbation triggers may help to reach this goal.

The present study sought to develop a psychometrically valid measure of perceived COPD exacerbation triggers and study its association with clinical outcomes. Because research indicates that exacerbations can be triggered by various factors and that illness perceptions are related to health status and health care use [17], we hypothesized that a reliable measure of perceived COPD exacerbation triggers would be predictive of such clinical endpoints.

METHODS

Participants

Patients were recruited through respiratory specialty clinics and online postings to self-help groups to complete either questionnaires online or as paper-and-pencil surveys, which were returned in prepaid envelopes. Inclusion criteria were a physician diagnosis of COPD, age ≥40 years, and the ability to read and understand English. Hospitalized participants were excluded to avoid confounds by treatment-induced symptomatology and experiences. The study was approved by the Baylor Research Institute IRB (No.012-085) and written informed consent was obtained from all participants, who received a compensation of $10.

Measures

The initial research version of the COPD Exacerbation Trigger Inventory (CETI)

consisted of a 53-item list of potential exacerbation triggers, including infections, illness, physical activities, moods, weather, allergens, irritants, and behaviors. In lieu of patient focus groups, the list was developed by consulting the COPD literature and COPD care professionals. An initial pool of items was then reviewed by a pulmonomolgist and a pulmonary rehabilitation specialist. Participants rated on 5-point scale (0-4: never, rarely, sometimes, most of the time, always) how often each trigger was involved in their exacerbations. In an additional section of the questionnaire, participants reported up to six most personally relevant triggers in a free-reponse format and rated their controllability (0-4: not at all, slightly, moderately, very much, completely).

Participants also reported demographics and aspects of their medical history, including diagnosis of COPD, oxygen use, history or current presence of other comorbid conditions, smoking history, medications, exacerbations in the past year, times in the past year an exacerbation caused them to seek medical attention, times in the past year an exacerbation lead to a change in their medication, and how often they received emergency treatment for COPD over their lifetime. COPD severity was calculated using the GOLD ABCD classification which is based on CAT total score and number of exacerbations in the past year.[1]

We used the 8-item COPD Assessment Test (CAT) (α=.88) (r_{tt}= 0.8), which is designed for use by health professionals and measures perceived health status in COPD[18], with higher scores indicating lower health status and more functional limitations. Use of the CAT to examine health status in patients with COPD is widespread in the literature and provides important information regarding changes in functional status. In the present study, it is used as an important clinical outcome that may be impacted by exacerbations and thus, by exposure to exacerbation

triggers. The CAT is also used in combination with either spirometric measurement or exacerbation risk to provide a comprehensive assessment of patients in clinical practice[1].

Procedure

Participants completed the questionnaire packet that included the initial 53-item research version of the CETI. Those completing the paper version were provided written information regarding study participation and confidentiality and asked to return the completed packet anonymously in a sealed envelope. Participants completing the survey online were provided a link to the Qualtrics-based survey, which provided informed consent information online at the beginning of the survey.

Statistical analysis

Principle component analysis with Varimax rotation was used to explore the structure of the CETI. With scree plots suggesting five factors, we explored five to seven factors, guided by primary factor loadings, communalities, and plausible interpretation on the basis of prior literature and clinical knowledge about triggers (airway infections, weather/climate, air pollution, psychological factors)[1-13]. Internal consistencies of the subscales were estimated using item-intercorrelations, item-total correlations, and Cronbach's α. Hierarchical linear regression analyses were used to examine prediction of COPD outcomes by trigger subscales over and above demographics, COPD severity (oxygen use or GOLD classification), COPD duration, and comorbidities. Additional regression models studied whether comorbidities predicted CETI subscales and trigger controllability. For further Methods and Results details see Supplementary Appendix S1 and S2)

RESULTS

Sample Characteristics

A total of 192 participants completed the study. A majority of the sample reported history or current presence of at least one comorbidity (Table 1).

Participants from local clinics were older, t= 3.67, p<.001; had a later COPD onset, t=4.49, p<.001; experienced fewer exacerbations in the past year, t=-2.23, p=.027; had better overall functioning based on their CAT scores, t=-2.38, p=.019; and had a greater likelihood of having comorbid conditions, χ^2= 29.12, p<.001.

Factor structure for initial CETI version

Kaiser-Meyer-Olkin measure of sampling adequacy was excellent at .92, and Bartlett's test of sphericity was significant (χ^2 (1378) = 6900.06, p < .001). An initial orthogonal five-factor solution explaining 62.8% of the variance was found to be the most plausible. This factor structure was consistently found in subsamples with and without comorbidities, as well as those recruited only through respiratory clinics (n=154). Breathing-related items (coughing, laughing, etc.) formed another potential factor, but the six-factor solution proved more unstable and item loadings were less consistent and therefore this content area was eliminated. Dust-related items were also eliminated because loadings were distributed across multiple factors. Overall, 20 items were eliminated based on content overlap with other items or unstable or low factor loadings (<.5).

The final 5-factor, 33-item solution accounted for 69.7% of the variance. All items had primary loadings over 0.5 and were grouped into the following factors: Psychology (15.9%),

Weather/Climate (13.1%), Air Pollution/Irritants (14.6%), Physical Activity (13.9%), and Illness/Infection (12.3%; see Supplementary Table S1 for factor loadings and communalities).

Psychometric Properties of CETI Subscales

Five subscales of 6-7 items each were formed with a total of 33 items (Supplementary Appendix S4, 5). Psychological items had the lowest subscale means and physical activity items had the highest (Table 2). Overall, the five subscales exhibited excellent internal consistency (α= 0.91-0.94.) and were significantly correlated with one another (Table 3).

Free Reports of Exacerbation Triggers

Free reports of triggers were grouped into categories (Fig. 1). Physical activity-related triggers were the most common, followed by climate, infection, and air pollution. Psychology-related triggers were reported by approximately 16% of participants. Rare responses that did not group into a specific category (e.g. alcohol, bending, ice cold fluids, crowds) were labeled "Other."

Observed trigger controllability ratings for each category ranged from 1.3 (1="slightly") to 2.4 (2="moderately") (Fig. 2). Participants found triggers related to dust, smoking, and air pollution to be the easiest to control, whereas those triggers related to sleep, respiratory, and infection were the least controllable (no significance testing was performed because of the limited overlap of self-reported trigger categories). Internal consistency for three to six trigger control ratings (which entailed varying numbers of the sample because not all participants reported a maximum of six triggers) were satisfactory with Cronbach's alpha ranging between α=0.78-0.86 and mean inter-item correlations between r_{ii}=0.50–0.55.

Associations of CETI subscales with Comorbidities

Examining the associations of individual comorbidities with CETI subscales, only an effect for the psychological subscale was found, with lung cancer and heart disease related to higher scores.

Associations between CETI and Clinical Outcomes

CETI trigger subscales accounted for substantial variance in CAT scores (18.2%), exacerbation frequency (13.0%), and medical treatment seeking for exacerbations (12.4% & 8.9%), beyond that accounted for by covariates (Table 4). Greater weather/climate-related triggers uniquely contributed to the prediction of higher CAT scores, greater psychological triggers to the prediction of more exacerbations, and greater infection triggers to the prediction of greater number of lifetime emergency visits and more medical treatment seeking.

Association between Trigger Controllability and Quality of Life

Controlling for oxygen use, trigger controllability accounted for 4.0% of the variance in the CAT scores beyond covariates, $F(1,105)=6.24$, $p=.014$, for R^2 change (total $R^2=0.32$, $F(8,105)=6.28$, $p<.001$). Greater controllability was associated with better health status, $t=-2.50$, $\beta=-0.21$, $p=.014$. Other associations between trigger controllability and clinical outcomes were nonsignificant.

[for further details on Results and Discussion, see APPENDIX S2 and S3].

DISCUSSION

Despite evidence for various triggers of COPD exacerbations, a standardized method for assessing patients' perceived triggers was missing. The present study developed a structured inventory for capturing perceived frequency and controllability of COPD exacerbation triggers. The 33-item, five-factor structure of the CETI was consistent across participants with and without comorbid conditions, and demonstrated excellent internal consistency.

Participants rated physical activity-related triggers the highest. Many COPD patients, particularly those with comorbidities, do not regularly engage in physical activity[19-21], although exercise is often endorsed as a potential treatment modality[22,23]. Perception of exercise as an exacerbation trigger may further hinder participation of patients in exercise or rehabilitation[24].

Infection-related triggers were also common, which reflects their recognition as risk factors for exacerbations[1,3,4]. Subscale scores were largely consistent with patients' free responses, in which physical activity, climate, infection, and air pollution were mentioned most frequently. A subset of participants (16.1%) reported psychological triggers among their top six, which is consistent with literature linking psychological distress to exacerbations[10-13,25-26].

The CETI subscales were significant and differential predictors of health status, exacerbation frequency, and healthcare use, suggesting their importance for specific COPD outcomes. Individuals who reported more weather/climate-related triggers tended to have poorer health status. Climate changes can have significant consequences for respiratory disease through a variety of pathways[27]. The current sample was collected from a humid, subtropical area of the southern continental US, which is often affected by extreme heat and air pollution during the summer months. Individuals in more extreme climates may experience more weather-related

triggers, feel more limited by their disease, and have less control over these triggers, as suggested by control ratings for that trigger domain.

The present study demonstrates the importance of psychological triggers in COPD management, particularly as such triggers may directly relate to exacerbation frequency. These findings are in line with other research that examines relations between anxiety (i.e. panic), depressed mood, and COPD[28]. Several mechanisms might explain this link.

Research has found that COPD patients with panic show heightened sensitivity to inspiratory loads, associating fear with the perception of symptoms, and possibly, exacerbations[26]. Studies of asthma have shown asssociations of stress and negative affect with bronchoconstriction and airway inflammation[29]. Physiological reactivity to distress may result in increased inflammation and therefore susceptibility to exacerbations. COPD patients also experience negative mood during exacerbations, including depression, lack of energy, anxiety, resignation, and anger [30], and could thus attribute exacerbations to psychological factors[31].

The finding that trigger perceptions predict medical treatment seeking may be clinically useful. Because many patients with COPD fail to seek medical treatment for an exacerbation[3], knowledge of exacerbation triggers may provide early warning signs[30]. Overall, the association between the CETI subscales and clinical outcomes confirms the importance of illness perception in COPD outcomes[17,31] and provides preliminary evidence for the clinical utility of this new measure.

The present study has several strengths, including recruitment of a community sample that ranged widely in comorbidities, medical treatment, and indicators of impairment. The stability of the CETI factor structure across subsamples speaks to its diverse applicability. Comorbidities are common in COPD[32], with possible shared molecular mechanisms[33], and the

fact that the factor structure was stable across patients with and without comorbidities strengthens internal validity. However, a number of limitations of our study are also worth noting. First, our work intended to inform only about patients' perceptions, not about actual exacerbation triggers. Identifying patients' observations, notions, and lay theories about triggers is an important starting point in the dialogue with the physician and in subsequent COPD self-management. Future research with systematic challenges and natural observations of both perceived and physical triggers is needed to enhance our understanding of their relationship. Second, COPD diagnosis of a small portion of the sample that was obtained online was self-reported without additional confirmation by a physician. However, given that COPD is typically under-diagnosed[1,34,35] and lack of knowledge by many patients about medical terminology[30] it seems unlikely that these participants were overreporting COPD. Finally, COPD patients may be less familiar with the "exacerbation" terminology, thus confusing exacerbations with stronger symptoms at least partially[28,37]. However, care was taken in the instructions to explain the term exacerbation in detail. Also, "control" of a trigger may have variable meanings for patients, such as limiting exposure to the trigger or overall avoidance of it. Future research may need to implement structured interview formats to ensure proper understanding of these terms.

Despite these limitations, the present study is a first step towards systematic assessment of patients' exacerbation triggers perceptions and demonstrates the importance of major trigger domains for COPD outcomes. Additional validation of the CETI would be necessary. Clinical utility of the CETI may involve facilitating interactions between physicians and patients or development of interventions that specifically target management of triggers. Research may also use the CETI to further explore the psychophysiological mechanisms of COPD, such as the link

between mood and disease processes, or how illness perceptions influence clinical outcomes in this population.

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Disclosure statement

This study was previously presented in 2015 at Annual meetings of the International Society for Advancement of Respiratory Psychophysiology and the Society of Behavioral Medicine.

REFERENCES

[1] GOLD. Global Strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease. Executive summary, updated 2018. Available from http://goldcopd.org/wp-content/uploads/2017/11/GOLD-2018-v6.0-FINAL-revised-20-Nov_WMS.pdf.

[2] Barnes N, Calverley PM, Kaplan A, Rabe KF. Chronic obstructive pulmonary disease and exacerbations: patient insights from the global Hidden Depths of COPD survey. BMC Pulm Med 2013;13:54.

[3] Wedzicha JA, Seemungal TA. COPD exacerbations: defining their cause and prevention. Lancet 2007;370:786-96.

[4] White AJ, Gompertz S, Stockley RA. Chronic obstructive pulmonary disease 6:the aetiology of exacerbations of chronic obstructive pulmonary disease. Thorax 2003;58:73-80.

[5] Laumbach RJ, Kipen HM. Respiratory health effects of air pollution: update on biomass smoke and traffic pollution. J Allergy Clin Immunol 2012;129:3-11.

[6] Ko FW, Hui DS. Air pollution and chronic obstructive pulmonary disease. Respirology 2012;17:395-401.

[7] Eisner MD, Iribarren C, Yelin EH, Sidney S, Katz PP, Sanchez G, Blanc PD. The impact of SHS exposure on health status and exacerbations among patients with COPD. Int J Chron Obstruct Pulmon Dis 2009;4:169-7

[8] Zuo L, He F, Sergakis GG, Koozehchian MS, Stimpfl JN, Rong Y, Diaz PT, Best TM. Interrelated role of cigarette smoking, oxidative stress, and immune response in COPD and corresponding treatments. Am J Physiol Lung Cell Mol Physiol 2014;307:L205-18.

[9] Donaldson GC, Seemungal TA, Patel IS, Lloyd-Owen SJ, Wilkinson TM, Wedzicha JA. Longitudinal changes in the nature, severity, and frequency of COPD exacerbations. Eur Respir J 2003;22:931-6.

[10] Atlantis E, Fahey P, Cochrane B, Smith S. Bidirectional associations between clinically relevant depression or anxiety and COPD: a systematic review and meta-analysis. Chest 2013;144:766-77.

[11] Laurin C, Moullec G, Bacon S, Lavoie K. Impact of anxiety and depression on chronic obstructive pulmonary disease exacerbation risk. Am J Respir Crit Care Med 2012;185:918-23.

[12] Xu W, Collet JP, Shapiro S, Lin Y, Yang T, Platt RW, Wang C, Bourbeau J. Independent effect of depression and anxiety on chronic obstructive pulmonary disease exacerbations and hospitalizations. Am J Respir Crit Care Med 2008;178:913-20.

[13] Maurer J, Rebbapragada V, Borson S, Goldstein R, Kunik ME, Yohannes AM, Hanania NA; ACCP Workshop Panel on Anxiety and Depression in COPD. Anxiety and depression in COPD: current understanding, unanswered questions, and research needs. Chest 2008;134(4 Suppl):43S-56S.

[14] Panagioti M, Scott C, Blakemore A, Coventry PA.Overview of the prevalence, impact, and management of depression and anxiety in chronic obstructive pulmonary disease. Int J Chron Obstruct Pulmon Dis 2014;9:1289-306.

[15] Ritz T, Kullowatz A, Kanniess F, Dahme B, Magnussen H. Perceived triggers of asthma: evaluation of a German version of the Asthma Trigger Inventory. Respir Med 2008;102:390-8.

[16] Ritz T, Steptoe A, Bobb C, Harris AH, Edwards M. The asthma trigger inventory: validation of a questionnaire for perceived triggers of asthma. Psychosom Med. 2006;68:956-65.

[17] Kaptein AA, Scharloo M, Fischer MJ, Snoei L, Cameron LD, Sont JK, Rabe KF, Weinman J. Illness perceptions and COPD: an emerging field for COPD patient management. J Asthma 2008;45:625-9.

[18] Jones P, Harding G, Berry P, et al. . Development and first validation of the COPD Assessment Test. Eur Respir J 2009;34: 648-54.

[19] Bourbeau J. Activities of life: the COPD patient. COPD 2009;6:192-200.

[20] Fastenau A, van Schayck OC, Gosselink R, Aretz KC, Muris JW. Discrepancy between functional exercise capacity and daily physical activity: a cross-sectional study in patients with mild to moderate COPD. Prim Care Respir J. 2013 Dec;22(4):425-30.

[21] McNamara RJ, McKeough ZJ, McKenzie DK, Alison JA. Physical comorbidities affect physical activity in chronic obstructive pulmonary disease: a prospective cohort study. Respirology 2014;19:866-72.

[22] Watz H, Waschki B, Meyer T, Magnussen H. Physical activity in patients with COPD. Eur Respir J 2009;33:262-72.

[23] Pitta F, Troosters T, Probst VS, Spruit MA, Decramer M, Gosselink R. Physical activity and hospitalization for exacerbation of COPD. Chest 2006;129:536-44.

[24] von Leupoldt A, Janssens T. Could targeting disease specific fear and anxiety improve COPD outcomes? Exp Rev Respir Med 2016;10:835-837.

[25] Livermore, N., Sharpe, L., McKenzie, D. Prevention of panic attacks and panic disorder in COPD. Eur Respir J 2010; 35: 557-563.

[26] Livermore, N., Butler,J., Sharpe, L., McBain, R., Gandevia, S., McKenzie, D. Panic attacks and perception of inspiratory resistive loads in chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2008; 178: 7-12.

[27] De Sario M, Katsouyanni K, Michelozzi P. Climate change, extreme weather events, air pollution, and respiratory health in Europe. Eur Respir J 2013;42:826-43.

[28] Jennings JH, DiGiovine B, Obeid D, Frank C. The association between depressive symptoms and acute exacerbations of COPD. Lung 2009;187:128-35.

[29] Ritz T, Meuret AE, Trueba AF, Fritzsche A, von Leupoldt A. Psychosocial factors and behavioral medicine interventions in asthma. J Consult Clin Psychol 2013;81:231-50.

[30] Kessler R, Ståhl E, Vogelmeier C, Haughney J, Trudeau E, Löfdahl CG, Partridge MR. Patient understanding, detection, and experience of COPD exacerbations: an observational, interview-based study. Chest 2006;130:133-42.

[31] Howard C, Hallas CN, Wray J, Carby M. The relationship between illness perceptions and panic in chronic obstructive pulmonary disease. Behav Res Ther 2009;47:71-6.

[32] Cavaillès A, Brinchault-Rabin G, Dixmier A, Goupil F, Gut-Gobert C, Marchand-Adam S, Meurice JC, Morel H, Person-Tacnet C, Leroyer C, Diot P. Comorbidities of COPD. Eur Respir Rev 2013;22:454-75.

[33] Faner R, Gutiérrez-Sacristán A, Castro-Acosta A, Grosdidier S, Gan W, Sánchez-Mayor M, Lopez-Campos JL, Pozo-Rodriguez F, Sanz F, Mannino D, Furlong LI, Agusti A. Molecular and clinical diseasome of comorbidities in exacerbated COPD patients. Eur Respir J 2015;46:1001-10.

[34] Buist AS, McBurnie MA, Vollmer WM, Gillespie S, Burney P, Mannino DM, Menezes AM, Sullivan SD, Lee TA, Weiss KB, Jensen RL, Marks GB, Gulsvik A, Nizankowska-

Mogilnicka E; BOLD Collaborative Research Group. International variation in the prevalence of COPD (the BOLD Study): a population-based prevalence study. Lancet 2007;370:741-50.

[35] Halbert RJ, Isonaka S, George D, Iqbal A. Interpreting COPD prevalence estimates: what is the true burden of disease? Chest 2003;123:1684-92.

[36] Eisner MD, Omachi TA, Katz PP, Yelin EH, Iribarren C, Blanc PD. Measurement of COPD severity using a survey-based score: validation in a clinically and physiologically characterized cohort. Chest 2010;137:846-51.

[37] Lopez-Campos JL, Calero C, Lopez-Ramirez C. Exacerbations or complications? Redefining the concepts in COPD. Int J Clin Pract 2014;68:1048.

Table 1

Sample Demographics and Disease Characteristics

Characteristic	Data
Sex, Male/Female, n (%)	101 (52.6) / 88 (45.8)
Mean Age, yrs, mean ± SD	69.5 ± 10.0
Marital Status, n (%)	
Single	40 (20.8)
Married)	102 (53.1)
Other (widowed, divorced, living with partner, separated)	47 (24.5)
Primary Race, White/Non-white, n (%)	170 (88.5) / 20 (10.4)
Occupational Status, Working/ Not working, n (%)	21 (10.9) / 131 (68.2)
Education, College/ No College, n (%)	124 (64.6) / 62 (32.3)
Current Smokers, n (%)	21 (10.9)
Patients with one or more comorbidities, n (%)	115 (59.9)
Asthma	62 (32.3)
Lung Cancer	16 (8.3)
Heart Failure	20 (10.4)
Other heart disease	7 (3.6)
Other respiratory disease	21 (10.9)
Mean age of COPD onset, yrs, mean ± SD	60.2 ± 12.0
Mean exacerbations per patient in past 12 mo., mean ± SD	1.7 ± 1.2
Currently on oxygen therapy, n (%)	85 (44.3)
GOLD Classification, n (%)	
A (Low risk, Less symptoms)	19 (9.9)
B (Low risk, More symptoms)	59 (30.7)
C (High risk, Less symptoms)	5 (2.6)
D (High risk, More symptoms)	82 (42.7)
Medications, n (%)	
Bronchodilator Only	7 (3.6)
Maintenance Medication	155 (80.7)
CAT Score, mean ± SD	19.2 ± 8.1

Table 2

Item Characteristics and Internal Consistency of the COPD Exacerbation Trigger Inventory Subscales

Trigger Subscale	Number of Items	$M_i \pm SD_i$	r_{ii} (mean)	r_{it} (range)	α
Psychological	7	1.05 ± 0.18	0.71	0.72 – 0.90	0.94
Weather/Climate	7	1.53 ± 0.28	0.59	0.50 – 0.82	0.91
Air pollution/Irritants	7	1.53 ± 0.11	0.62	0.66 – 0.81	0.92
Exercise	6	2.30 ± 0.31	0.70	0.74 – 0.85	0.93
Infection	6	1.83 ± 0.37	0.61	0.64 – 0.80	0.90

M_i = Item Mean; SD_i = Item standard deviation; r_{ii} = item intercorrelation; r_{it} = item-total correlation; α = Cronbach alpha

Table 3

Correlations Between Subscales of the COPD Exacerbation Trigger Inventory

	1	2	3	4	5
1. Psychological	-	0.64	0.52	0.50	0.47
2. Weather/Climate		-	0.63	0.57	0.57
3. Irritants			-	0.50	0.52
4. Exercise				-	0.49
5. Infection					-

*All correlations Spearman's Rho, two-tailed, $p<.01$

Table 4

Explained Variance in COPD Severity, Exacerbations, and Healthcare Use by CETI Subscales [a]

Criterion	R^2 Change[c]	p	CETI Subscale Predictors[d]	t[d]	p	R^2 for Total Model
CAT Total Score[a] (N=132)	0.182	0.000	Weather/Climate	2.25	0.027	0.472
			Infection	1.71	0.091	
# Exacerbations per year[a] (N=128)	0.130	0.001	Psychological	2.29	0.024	0.361
# Lifetime ER visits[a] (N= 124)	0.041	0.235	Infection	1.84	0.068	0.342
# Lifetime ER visits[b] (N = 119)	0.039	0.306	Infection	2.02	0.046	0.322
Medical Treatment Seeking[a] (N=121)	0.124	0.001	None			0.391
Medical Treatment Seeking[b] (N= 117)	0.089	0.008	Infection	2.09	0.039	0.441

[a] Results from hierarchical multiple linear regressions analysis after controlling for age, gender, race, and education in Step 1, COPD duration and oxygen use in Step 2, and comorbidity status in Step 3.

[b] Results from hierarchical multiple linear regressions analysis after controlling for age, gender, race, and education in Step 1, COPD duration and GOLD classification (based on CAT score and exacerbation frequency) in Step 2, and comorbidity status in Step 3.

[c] Change in R^2 for the five CETI trigger subscales entered in Step 4.

[d] Only CETI scales that contributed to prediction significantly ($p < .05$) or marginally ($p < .10$) over and above the other predictors are reported.

[e] t-test results for beta weights; positive values indicate a positive association between CETI subscale (as predictors) and dependent variable.

Figure legends

Figure 1- *Frequency of Self-Report COPD Exacerbation Triggers by Category*

Figure 2- *Average Trigger Control Ratings by Category*