Lung function changes in patients with Chronic Obstructive Pulmonary Disease (COPD) and asthma exposed to second-hand smoke in outdoor areas

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Sheila Keogan,[1,#] Tamara Alonso,[2,3,#] Salome Sunday,[1] Olena Tigova,[3,4,5,6] Esteve Fernández,[3,4,5,6] Joan B Soriano,[2,3,*] Luke Clancy,[1,*] and the TackSHS Project Investigators (all listed in **Appendix 1**)*

[1] TobaccoFree Research Institute Ireland, Focas Research Institute, DIT, Dublin, Ireland.
[2] Hospital Universitario de la Princesa, Universidad Autónoma de Madrid, Madrid, Spain.
[3] Centro de Investigación en Red de Enfermedades Respiratorias (CIBERES), Instituto de Salud Carlos III (ISCIII), Madrid, Spain.
[4] Tobacco Control Unit, Catalan Institute of Oncology (ICO); L'Hospitalet de Llobregat (Barcelona), Catalonia.
[5] Tobacco Control Research Group, Bellvitge Biomedical Research Institute (IDIBELL), L'Hospitalet de Llobregat, Catalonia.
[6] School of Medicine and Health Sciences, Bellvitge Campus, Universitat de Barcelona, L'Hospitalet de Llobregat, Catalonia.

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Joint first authorship
* Joint senior authorship

Address for correspondence:
Dr. Joan B Soriano
Hospital Universitario de la Princesa, Diego de León 62, Neumología 6ª planta, 28030-Madrid, Spain
Email: jbsoriano2@gmail.com
Cellular: +34 618 86 77 69

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Abstract

Background: Further evidence is needed on the effects that short- and long-term exposure to secondhand smoke (SHS) have on the respiratory health of patients with lung disease. Within the TackSHS project we aimed to assess the acute respiratory effects in lung function that result from short-term SHS exposure among patients with asthma and chronic obstructive pulmonary disease (COPD).

Methods: The study design was an intervention trial with measurements before/after exposure to SHS in legal outdoor smoking areas. We studied patients with asthma or COPD from Czechia, Ireland, and Spain. Forced spirometry, peak flow and carbon monoxide (CO) measurements were performed pre- and 24 hours post- exposure.

Results: Overall, 60 patients were studied, 30 with asthma, and 30 with COPD; 35 (58.3%) were female. There were no significant differences observed in exhaled CO between pre- and 24 hours post-exposure neither in women ($p = 0.210$), nor in men ($p = 0.169$).

A statistically significant decrease in forced vital capacity (FVC) was seen, overall, in asthma participants ($p = 0.02$) and in forced expiratory volume in the first second (FEV_1), ($p = 0.02$), FVC ($p = 0.04$) and peak expiratory flow rate (PEFR) ($p = 0.04$) in female asthmatic participants. The observed decreases in respiratory measurements in COPD were not significant. There were no reported increases in symptoms, respiratory medication, or use of health services 24 hours after the exposure.

Conclusion: We conclude that acute, short-term SHS exposure had a statistically significant effect on spirometry in female asthma patients but did not significantly modify spirometric indices 24 hours later in COPD patients.

Abstract word count: 249 words

Introduction

Research produced in the past three decades has provided substantial evidence of the harm that short and long-term exposure to secondhand smoke (SHS) represents to respiratory and cardiovascular health of adults and children. [1,2,3,4,5]

While interest in SHS exposure prevalence and its effects is increasing, [6,7] important gaps in evidence remain, and should be considered when designing national and international health policies aiming to protect those with lung disease.[8] One of these gaps is a scarcity of studies objectively measuring markers of SHS in settings where smoking is not regulated, such as private places and outdoor public places.

In many European countries, the introduction of smoke-free laws has prompted the proliferation of outdoor areas where smoking is permitted, and there exist places where non-smokers are still exposed to some levels of SHS. The possible health effects of such exposures are unknown. We are not aware of any studies in patients in such smoking areas. Some effects can be expected, given the known irritant effects of SHS or some of the constituents of SHS[9], but it may be difficult to demonstrate any short-term measurable effects especially for ethical considerations unless in uncontrolled, real-life conditions.

The project "Tackling secondhand tobacco smoke and e-cigarette emissions: exposure assessment, novel interventions, impact on lung diseases and economic burden in diverse European populations" (the TackSHS project) www.tackshs.eu was developed to address these and other challenges in the research of SHS and second-hand e-cigarette aerosols (SHA) from electronic cigarettes. TackSHS aims to comprehensively elucidate the impact that SHS and SHA have on the European population and how health impacts vary according to socioeconomic and other demographic characteristics, with a particular emphasis on specific vulnerable groups.[10]

Asthma and chronic obstructive pulmonary disease (COPD) are the two most frequent chronic respiratory diseases;[11] although all respiratory patients should refrain from smoking, many are still exposed to SHS from first- to fourth-hand smoking.[12]

This paper aims to assess if the acute effects in lung function that result from short-term SHS exposure among patients with stable doctor-diagnosed asthma and COPD.

Methods

The current study, led by TobaccoFree Research Institute Ireland (TFRI) is part of the TackSHS project[10] and was conducted in Liberia (Czechia), Dublin (Ireland) and Madrid (Spain). The study design was an intervention trial with measurements before/after exposure to SHS. The study research protocol received ethical approval in each country and is registered at clinicaltrials.gov with identifier NCT03074734. All participants signed a written informed consent form.

Participants

Inclusion criteria for asthma patients were: confirmed doctor-diagnosed asthma, aged 18 years and older, fully ambulatory, and crucially only patients with a history of frequent visits to smoking outdoor areas were included. Consequently more asthmatic smokers volunteered than may be representative of many asthmatic populations. The asthma patients were known to the referring physicians and regarded as stable with a mean asthma control test score (ACT) of 21.8 (SD 3.6). Inclusion criteria for COPD patients were: confirmed doctor-diagnosed COPD patients with confirmed airflow limitation by post-bronchodilator spirometry with FEV1/FVC < 0.7, aged 50 to 70 years old, current- or ex-smokers, fully ambulatory, and again with a history of frequent visits to smoking outdoor areas, clinically stable and a mean COPD Assessment Test (CAT) score of 11.6 (SD 7.6). Exclusion criteria for both asthma and COPD patients were: under 18 years old, on oxygen therapy, never smokers in COPD patient group, undergoing treatment for acute exacerbations, pregnant women and patients not giving a history of visits to outdoor smoking areas of pubs.(Table 1).

This study involved 60 patients (30 asthma and 30 COPD patients) in Czechia (30 patients), Ireland (10 patients), and Spain (20 patients) between April 2018 and April 2019. All participants were administered a validated symptoms questionnaire. The COPD Assessment Test (CAT) for all COPD patients,[13] and the Asthma Control Test (ACT) for all asthmatic patients,[14] pre- and post-peak flow, spirometry, and CO measurements were carried out by the study researchers in each country. They were advised to take their usual

medication and dosage and to carry their inhalers with them and to note any need for extra medication.

Intervention

An outdoor smoking area was defined in Irish law as: 'a place or premises, or part of a place or premises that, is fully uncovered by any roof, fixed or mobile, or a place or premises that is covered by a roof, so long as not more than 50% of the perimeter (outside) is covered by a wall, windows, gate or similar'.

The intervention was exposure to SHS in outside smoking areas. Study subjects were asked to spend at least 15 minutes in the outdoor smoking area, with a preferable time of 30-60 mins. They recorded time of entry to the outside area and duration of exposure. They also noted the characteristics of the area including the number of walls and the type of roof or canopy and the number of smokers present in the area.

Outcome measures included pulmonary function changes in terms of forced spirometry and peak flow measurement pre- and post-exposure to SHS within 24 hours. Spirometry guidelines from the American Thoracic Society (ATS)/European Respiratory Society (ERS) Task Force were used[15], which suggests that three acceptable manoeuvres should be achieved. The best two measurements must fulfil the reproducibility criteria. For FEV_1 and FVC, the best two values should be within 5% or 150 mL of each other, whichever is greater. If FVC is <1.0 L, then the values should be within 100 ml. All spirometers used had valid calibration certificates. These measurements are the standard ones done in each of the three venues and the patients and researchers are familiar with the equipment and manoeuvres and serial stored data were available for comparison.

The patients completed a questionnaire pre-exposure to SHS, which included items on respiratory medication, symptoms, and previous visits to emergency room or hospital admissions. Any significant variation from this was noted at the post exposure consultation but none were reported.

Statistical analysis

Data is presented as mean and standard deviation (SD) for continuous variables, or percentage for qualitative variables, as appropriate, and their 95% confidence interval (CI). Differences within groups were compared with Chi^2 tests for categorical variables and Student t test or Wilcoxon test for continuous variables. A *p* value lower than 0.05 was considered statistically significant. Both STATA and SPSS were used in the analyses.

Results

Overall, 61 patients were recruited; three had to have their recording repeated due to equipment failure and one Irish COPD patient was not repeated. Therefore, 60 patients were finally studied (**Figure 1**), with a 98.4% response rate; 30 with asthma, and 30 with COPD, of whom 35 (58.3%) were female. Those with asthma were 19 (63.3%) female with a mean (SD) age of 46.9 (18.7) years, with 26.7% current smokers; while those with COPD were 16 (53.3%) female with age of 63.3 (10.2) years, with 70.0% current smokers (**Table 1**). Seven (23.3%) patients with asthma and 10 (33.3%) patients with COPD were living with a smoker. Most COPD patients had symptomatic, active disease as per their CAT 11.6 (7.5) score, while most asthma patients had their asthma controlled as per their ACT 21.8 (3.6). When in the outside areas, most participants (83.3%) were not exposed to aerosols of electronic cigarettes (data not shown in the tables).

There were no significant differences in severity of disease by sex in asthma (p=0.813) or in COPD (p=0.770) scores (**Table 2**).

Pulmonary function

There was a small but statistically significant reduction in FVC, in asthmatic patients (p=0.02) (**Table 3**). In female asthmatic patients, however, there were statistically significant changes in (FVC) p=0.04, (FEV1) p=0.02 and (PEFR) p=0.04 but not in males.

There were no statistically significant changes in pulmonary function tests in COPD in either sex but respiratory parameters were numerically lower post exposure **(Table 3)**

Exhaled breath Carbon Monoxide parts per million (CO ppm):

The CO in smokers was 10.21 (SD 10.03) ppm, and 1.47 (SD 1.22) ppm, in non-smokers pre-exposure and did not change significantly post-exposure (p=0.08) and (p=0.83), respectively.

Discussion

The TackSHS project involves a comprehensive and innovative approach to enhance scientific understanding of SHS in chronic lung respiratory diseases. At the societal level, we aim to raise awareness of the risks from SHS and the health burden that SHS exposure presents. Our study explored the perceived effects on breathing in patients with asthma and COPD and measured changes in spirometry and peak flow in these respiratory patients after exposure to SHS in outside areas.

Our main findings were that short-term SHS exposure did not result in any significant change in symptoms or noticeable change in medication usage, in 24 hours monitoring among patients with asthma and COPD, in contrast with prior results obtained[16].

There were significant decreases in pulmonary function in asthma patients, particularly in females. These changes were of less than 100 ml, which were not noticed as changes in symptoms by the patients. In the COPD patients there was a reduction in FEV1, FVC and PEFR but it was not statistically significant.

These small changes in both diseases are of particular interest because while not of immediate clinical significance they are enough to raise the possibility of small losses of lung function which may become cumulative with continuing, or even if intermittent, exposure. This was a once off short exposure and it can be envisaged that repeated and or prolonged exposure to SHS might produce cumulative damage in these and other lung function measurements. Previous studies have shown that airway oxidative stress remains increased after the exposure to SHS, representing a possible mechanism for the persistence of the deleterious effects of SHS after the end of the exposure[17].

Patients with asthma, not unexpectedly, were more sensitive to the effects of exposure but likewise may be more reversible. The long-term consequences may be more serious for COPD patients where changes may be more permanent and progressive[18]. Serial self-monitoring may be possible for such patients and be more definite but even with this short intervention it would appear reasonable to caution patients with these diseases to avoid such exposure.

Some of the strengths of this research include the real-world conditions of SHS exposure[19], and an identical protocol applied in the three countries with quite different climates and smoking behaviours. However, a number of limitations must be considered. Changes in forced spirometry were performed the next day, so acute, immediate effects of SHS exposure in FEV_1, FVC or in PEFR could not be assessed. Nor were serial

measurements made to determine the time course of the response and whether there was any continuing loss which may contribute to a gradual decline in lung function; this may be the case particularly in COPD patients. It was not considered reasonable or feasible to undertake forced spirometry in outdoor areas in the frame of this study. We initially considered to have a more extensive battery of lung tests, up to pletismography. However, for practicality reasons, measuring mid-expiratory flows, inspiratory capacity, and other lung parameters, required further standardisation of spirometers and staff, that added time and complexity to our simple study design. But, they all should be considered areas for future research. With 60 respiratory patients, we must admit our sample size is relatively modest, given a reduced budget and limited availability of monitoring devices in each country, which were sterilised, reset and reused. No a priori formal sample size was estimated, and we think inappropriate to calculate any posterior power calculation, as there are many outputs and secondary analyses of interest. However, our findings should be considered as proof of concept of larger, more ambitious studies on the unaccounted tobacco-related hazards suffered by our asthma and COPD patients.

New research should explore the long-term effects of exposure to SHS in outside areas in the respiratory health of asthma and COPD patients. Continuous monitoring of lung function (with home, portable spirometers[20]) and of respiratory symptoms and other biosensors. This could be coupled with more accurate and continuous environmental assessment of particles and nicotine in the air. These studies, however, will deal with the challenge to differentiate exposure to SHS from tobacco, environmental pollution and also exposure to SHA. Hence, the effects of SHA in patients with respiratory diseases such as asthma and COPD needs further research. In our study, most patients (83.3%) were not exposed to electronic cigarettes precluding an assessment.

We conclude that acute, short-term SHS exposure among patients with asthma and COPD modifies spirometric indices and is a hazard for patients with these diseases, but did not result in reporting of increased symptoms, use of health services or require medical intervention.

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Authors contribution LC, SK and EF had the original idea for the study. SK trained all the researchers in the use of the monitoring devices, collected the data in Ireland and was responsible for data management for all three sites. Dr Milada Šípková collected the data in Czechia and PP performed the fieldwork in Spain.

MP-S and SSC carried out the statistical analysis with the supervision of TA and JBS and LC; TA and JBS wrote the first draft of the article in collaboration with LC, SK and EF; all the authors contributed to manuscript preparation and approved its final version prior to submission.

LC and JBS are the guarantors.

Competing Interests the authors declare no competing interests.

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Appendix 1

***The TackSHS Project Investigators:**

Catalan Institute of Oncology (ICO); Bellvitge Biomedical Research Institute (IDIBELL), Spain: Esteve Fernández, Yolanda Castellano, Marcela Fu, Montse Ballbè, Beladenta Amalia, Olena Tigova

Public Health Agency of Barcelona (ASPB), Spain: Maria José López, Xavier Continente, Teresa Arechavala, Elisabet Henderson

Istituto di Ricerche Farmacologiche Mario Negri IRCCS (IRFMN), Italy: Silvano Gallus, Alessandra Lugo, Xiaoqiu Liu, Cristina Bosetti, Elisa Borroni, Enrico Davoli; **Istituto DOXA, Worldwide Independent Network/Gallup International Association, Italy:** Paolo Colombo

University of Stirling (UNISTIR), the UK: Sean Semple, Rachel O'Donnell, Ruaraidh Dobson

TobaccoFree Research Institute Ireland (TFRI), Ireland: Luke Clancy, Sheila Keogan, Hannah Byrne, Shasha Li

Hellenic Cancer Society - George D. Behrakis Research Lab (HCS), Greece: Panagiotis Behrakis, Anna Tzortzi, Constantine Vardavas, Vergina Konstantina Vyzikidou, Gerasimos Bakelas, George Mattiampa

Fondazione IRCCS Istituto Nazionale dei Tumori (INT), Italy: Roberto Boffi, Ario Ruprecht, Cinzia De Marco, Alessandro Borgini, Chiara Veronese, Martina Bertoldi, Andrea Tittarelli

Istituto per lo Studio, la Prevenzione, e la Rete Oncologica (ISPRO), Italy: Giuseppe Gorini, Giulia Carreras, Barbara Cortini, Simona Verdi, Alessio Lachi, Elisabetta Chellini

Polytechnic University of Cartagena (UPCT), Spain: Ángel López Nicolás, Marta Trapero-Bertran, Daniel Celdrán Guerrero

European Network on Smoking and Tobacco Prevention (ENSP), Belgium: Cornel Radu-Loghin, Dominick Nguyen, Polina Starchenko

Instituto de Investigación Sanitaria del Hospital Universitario La Princesa (IP), Spain: Joan B Soriano, Julio Ancochea, Tamara Alonso, María Teresa Pastor, Marta Erro, Ana Roca, Patricia Pérez, Elena García Castillo

Table 1. Demographic and clinical characteristics of patients

	Asthma n=30	COPD n=30	All n=60
Age (years)	46.9 (18.7)	63.3 (10.2)	55.1(17.1)
Weight (Kg)	75.6 (18.1)	80.3(16.5)	78 (17.3)
Sex			
Male	11 (36.7%)	14 (46.7%)	25 (41.7%)
Female	19 (63.3%)	16 (53.3%)	35 (58.3%)
Smoking status			
Current-smoker	8 (26.7%)	21 (70%)	29 (48.3%)
Ex-smoker	6 (20.0%)	9 (30.0%)	15 (25.0%)
Never-smoker	16 (53.3%)	0 (0%)	16 (26.7%)
Lives with a smoker			
Yes	7 (23.3%)	10 (33.3%)	17 (28.3%)
No	23 (76.7%)	20 (66.7%)	43 (71.7%)
ACT score	21.8 (3.6)	-	-
CAT score	-	11.6 (7.5)	-
Number of smokers present in the outdoor smoking area during patient visit			
1-5	22 (73.3%)	22 (73.3%)	44 (73.3%)
More than 5	8 (26.7%)	8 (26.7%)	16 (26.7%)
Any unscheduled doctor visit			
Yes	0 (0%)	0 (0%)	0 (0%)
No	30 (100%)	30 (100%)	60 (100%)

Table 1 footnote: Mean (SD)/n (%); COPD: Chronic Obstructive Pulmonary Disease; ACT: Asthma Control Test, with range from 5 to 25; CAT: COPD Assessment Test, with range from 0 to 40

Figure 1. STROBE flowchart of participation

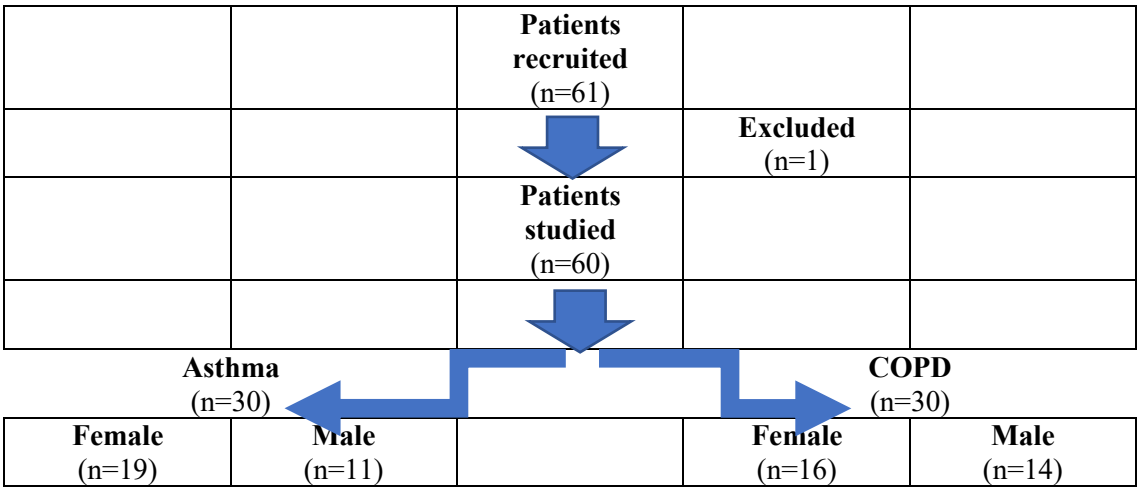

Table 2. Symptom questionnaires by diagnosis and sex

Asthma patients			
	Female n=19	**Male n=11**	**p - value**
ACT Mean (SD) range	21.7 (3.5) [14 – 25]	22.0 (4.0) [11 - 23]	0.813
COPD patients			
	Female n=16	**Male n=14**	**p - value**
CAT Mean (SD) range	11.2 (5.8) [2 - 23]	12.1 (9.2) [3 - 37]	0.770

Table 2 footnote: ACT: Asthma Control Test, with range from 5 to 25; CAT: COPD Assessment Test, with range from 0 to 40

Table 3. Comparison of Forced Expiratory Volume (FEV_1), Forced Vital Capacity (FVC) and Peak Expiratory Flow Rate (PEFR) before and after SHS Exposure by Gender and Disease

FEV_1(litres in 1st second)					
Asthma					
	Baseline FEV_1 Mean (SD)	Post Exposure FEV_1. Mean (SD)	Mean difference (95% CI)	t (df)*	P-value
All Asthma (n=30)	3.01 (1.05)	2.97 (1.10)	0.03 (-0.04, 0.11)	0.99 (29)	0.33
Male (n=11)	3.72 (1.12)	3.78 (1.19)	-0.06 (-0.23, -0.09)	-0.86 (10)	0.41
Female (n=19)	2.59 (0.76)	2.50 (0.74)	0.09 (0.01, 0.17)	2.50 (180	0.02
COPD					
	Baseline FEV_1 Mean (SD)	Post Exposure FEV_1. Mean (SD)	Mean difference (95% CI)	t (df)*	P-value
All COPD (n=30)	1.80 (0.56)	1.77 (0.54)	0.03 (-0.03, 0.09)	1.00 (29)	0.33
Male (n=14)	1.98 (0.53)	1.97 (1.72)	0.01 (-0.10, 0.13)	0.25 (14)	0.81
Female (n=16)	1.61 (0.55)	1.56 (0.56)	0.05 (-0.02, 0.12)	1.54 (14)	0.15
FVC (litres)					
Asthma					

	Baseline FVC Mean (SD) lts	Post Exposure FVC. Mean (SD) lts	Mean difference (95% CI)	t (df)*	P-value
All Asthma (n=30)	3.97 (1.33)	3.89 (1.37)	0.07 (0.00, 0.15)	2.30 (29)	0.02
Male (n=11)	5.10 (1.29)	5.05 (1.37)	0.19 (-0.07, 0.18)	0.91 (10)	0.38
Female (n=19)	3.32 (0.84)	3.22 (0.19)	0.10 (0.00-0.90)	2.15 (18)	0.04
COPD					
	Baseline FVC Mean (SD) lts	Post Exposure FVC. Mean (SD) lts	Mean difference (95% CI)	t (df)*	P-value
All COPD (n= 30)	2.90 (0.81)	2.87 (0.77)	0.03 (-0.07, 0.12)	0.62 (29)	0.54
Male (n=14)	3.26 (0.79)	3.27 (0.67)	-0.00 (-0.16, 0.16)	-0.01 (14)	0.99
Female (n=16)	2.55 (0.69)	2.49 (0.22)	0.06 (-0.06, 0.18)	1.03 (14)	0.32
PEFR (litres per min)					
Asthma					
	Baseline PEFR Mean (SD) l/min	Post Exposure PEFR. Mean (SD) l/min	Mean difference (95% CI)	t (df)*	P-value
All Asthma (n=30)	434 (211)	426 (223)	8.59 (-13.84, 31.03)	0.78 (29)	0.43
Male (n=11)	568 (199)	584 (240)	-16.82 (-66.76-33.13)	-0.75 (10)	0.5
Female (n=19)	357 (181)	334 (155)	23.31 (1.09, 45.52)	2.20 (18)	0.04
COPD					
	Baseline PEFR Mean (SD)l/min	Post Exposure PEFR. Mean (SD)l/min	Mean difference (95% CI)	t (df)*	P-value
All COPD (n=30)	343 (183)	328 (151)	14.27 (-16.83, 45.37)	0.94 (27)	0.35
Male (n=14)	377 (212)	345 (174)	31.77 (-21.60, 85.14)	1.30 (12)	0.22
Female (n=16)	312 (154)	313 (132)	-0.89 (-40.78, 38.99)	-0.04 (14)	0.96

Table 3 footnote: COPD: Chronic Obstructive Pulmonary Disease; * Student´s t-test (degrees of freedom); 95% CI: 95% Confidence Interval