Introduction

According to the Global Initiative for Chronic Obstructive Lung Disease (GOLD), chronic obstructive pulmonary disease (COPD) is a common, preventable, and treatable disease characterized by persistent respiratory symptoms and airflow limitation due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases [1]. It is currently the fourth leading cause of death and the fifth leading cause of disability globally [2]. Prior studies in hospitals and clinics in Trinidad have reported a COPD prevalence of approximately 21% [3,4]. A similar prevalence has been reported for type 2 diabetes mellitus (DM) in this population [5]. The interaction between these two quite common conditions in Trinidad and Tobago, to the best of our knowledge, has not been previously studied.

Although persistent airflow limitation is the defining feature of COPD, it is a complex, heterogeneous, and multi-morbid condition [6], with a typical COPD patient likely to report having four or more comorbidities [7]. In the National Health and Nutrition Examination Survey 2001-2008 [8], DM was identified as the third most influential comorbidity on self-rated health among COPD patients. Furthermore, comorbid diabetes has been found to increase the risk of hospitalization and mortality in patients with COPD in the United States and Australia [9].

European and North American studies have found that DM is more common in COPD patients [10,11] with an odds ratio of up to 2.0 for diabetes in COPD [10]. It is possible that DM may develop among patients with COPD as a result of chronic inflammation, hypoxia, or concomitant corticosteroid treatment or that they may both share a common antecedent in the form of cigarette smoking.

This study aimed to define the prevalence of diabetes in a cohort of Trinidian subjects with COPD and investigate whether it impacted outcome measures related to COPD, including lung function, exacerbations, quality of life and depression scales, and mortality.
MATERIALS AND METHODS

Study Design and Sample

This was a cross-sectional, follow-up study, and so it utilized an antecedent cohort of patients (n=105) [12,13], but due to attrition, only 58 were recruited of which 2 were excluded (each having a total hemoglobin of <7 g/dL). Thus, 49 new participants were sought via convenience sampling. All of our final 105 participants originated from the chest clinics in the three major general hospitals in Trinidad (Port-of-Spain, Mount Hope, and San Fernando).

Authors declare that the research was conducted according to the principles of the World Medical Association Declaration of Helsinki “Ethical Principles for Medical Research Involving Human Subjects” (amended in October 2013). Approvals were obtained from the ethics committee of the Faculty of Medical Sciences, University of the West Indies, St. Augustine. Written informed consent was obtained from all patients who participated in this study.

COPD Diagnosis and Spirometry

All participants had a clinical diagnosis of COPD, which was confirmed using spirometry, and were aged ≥40 years. Patients with a post-bronchodilator ratio of the forced expiratory volume in 1 second (FEV₁) to the forced vital capacity (FVC) of ≥0.70 [1], contraindications to spirometry [14], and patients who had known severe anemias, acute blood losses, recent transfusions, or pregnancy [15] were excluded from the study. Also, patients whose total hemoglobin was <7 g/dL were excluded as it would have rendered subsequent testing of the glycated hemoglobin level to be inaccurate.

Demographic and anthropometric data including height and weight were measured and recorded. Patients with a body mass index (BMI) <21 kg/m² were considered as cachectic [16]. Post-bronchodilator spirometry was performed using a hand-held turbine spirometer (Micro Spirometer Cat No: MS01 Micro Medical Limited, Rochester, Kent, United Kingdom) after administration of 300 μg of aerosolized salbutamol [14]. This was not technically possible in two patients who were unable to complete the FVC maneuver. Predicted FEV₁ and FVC were calculated using the Global Lung Initiative 2012 Excel sheet calculator [17]. The percentage of predicted FEV₁ values were subsequently used to classify the severity of airflow limitation in our patients according to the GOLD stages [1].

Diabetes Diagnosis

The glycated hemoglobin (HbA1c) level was tested on a finger-prick capillary blood sample using a digital communication analyzer (DCA) Vantage Analyzer (Siemens Healthcare Diagnostics Ltd., Frimley, Camberley, United Kingdom). Patients were classed as having DM if they gave a positive medical history and/or were on treatment for such, or if independently their HbA1c was ≥6.5% in keeping with the American Diabetes Association criteria [15]. According to the same criteria, individuals with glucose levels above normal but not yet in the diabetic range are recognized as having pre-diabetes and being at increased risk for developing DM [15]. Patients with an HbA1c ranging between 5.7% and 6.4% were classed as having pre-diabetes and those with an HbA1c of <5.7% as normal [15]. All individuals who had an HbA1c of ≥5.7% and were not known to be diabetic or on treatment for diabetes were given a referral letter to an appropriate diabetic service.

Questionnaires

Patients were classified as smokers if they smoked a minimum of 100 cigarettes in their lifetime and otherwise as never smokers [18]. Patients were asked about the number of exacerbations in the past year requiring hospitalizations and their inhaled, oral, and intravenous corticosteroid usage in the past 3 to 4 months. Patients who used inhaled corticosteroids were found to have used a combination of long-acting beta₂-agonist and corticosteroid inhalers (e.g., formoterol/budesonide or salmeterol/fluticasone propionate inhalers) alone or together with additional corticosteroid inhalers (e.g., fluticasone propionate or beclomethasone dipropionate inhalers). Two questionnaires on the quality of life were administered: St. George’s Respiratory Questionnaire (SGRQ) [19] and COPD Assessment Test (CAT) [20] as well as two on depression: Center for Epidemiologic Studies Depression Scale (CES-D) [21] and Center for Epidemiologic Studies Depression Scale-Revised (CESD-R) [22] after the necessary permissions were obtained. For all questionnaires, increasing scores indicated worsening parameters, and for this analysis, clinically significant depression was defined as a score of ≥16 on the CES-D and CESD-R scales [23, 24]. Of the total studied population (n=105), we were able to collect 1-year mortality data on 89 participants.

Statistical Analysis

Statistical analyses were performed using the IBM Statistical Package for the Social Sciences Statistics Version 20 (IBM SPSS Statistics Corp.; Armonk, NY, USA). Variables were expressed as mean (standard deviation, SD), median (interquartile range, IQR), or number (n, %) as appropriate. The chi-squared or Kruskal-Wallis tests were used to test the differences in discrete variables. Whereas for continuous variables, the t-test or Mann-Whitney U test were used. Spearman’s (Spearman’s rho, rs) or Pearson’s (Pearson’s r) correlations were employed in comparisons of variables as appropriate. Associations were considered significant at the 5% level.

RESULTS

From our study population of 105 COPD patients, 83.8% were male, and the mean (SD) age was 67.4 (11.0) years (Table 1). Patients were primarily of Indo-Trinidadian (52.4%) and Afro-Trinidadian (38.1%) ethnicities, and the median (IQR) BMI was 25.4 (22.1, 28.7) kg/m². A significant number of the participants never smoked (22.3%). The majority of patients were of the GOLD Stage 2 COPD severity (50.0%) with 80.8% being GOLD Stage 2 or greater.

Figure 1 shows that of 105 patients, 42 had diabetes, of which up to 38% were newly diagnosed, having an HbA1c in the diabetic range. A further 40.0% of the total cohort was pre-diabetic, and 20.0% was normal. As shown in Table 1, subjects with DM had a significantly greater median (IQR) BMI (27.3 (24.1, 30.4)) than non-diabetics (24.2 (21.2, 27.2)) (p=0.001). HbA1c levels increased with body weight...
Table 1. Demographic and anthropometric data and outcome measures for all subjects and for non-diabetics and diabetics individually

<table>
<thead>
<tr>
<th>Variables</th>
<th>Total (n=105)</th>
<th>Non-diabetes (n=63)</th>
<th>Diabetes (n=42)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean (SD) age, years</td>
<td>67.4 (11.0)</td>
<td>66.5 (12.3)</td>
<td>68.7 (8.7)</td>
<td>0.314b</td>
</tr>
<tr>
<td>Male sex (n (%))</td>
<td>88 (83.8)</td>
<td>51 (81.0)</td>
<td>37 (88.1)</td>
<td>0.330f</td>
</tr>
<tr>
<td>Ethnicity (n (%))</td>
<td>0.213f</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Afro-Trinidadian</td>
<td>40 (38.1)</td>
<td>29 (46.0)</td>
<td>11 (26.2)</td>
<td></td>
</tr>
<tr>
<td>Indo-Trinidadian</td>
<td>55 (52.4)</td>
<td>26 (41.3)</td>
<td>29 (69.0)</td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>3 (2.9)</td>
<td>2 (3.2)</td>
<td>1 (2.4)</td>
<td></td>
</tr>
<tr>
<td>Mixed</td>
<td>7 (6.7)</td>
<td>6 (9.5)</td>
<td>1 (2.4)</td>
<td></td>
</tr>
<tr>
<td>Mean (SD) height, cm</td>
<td>164.3 (8.2)</td>
<td>165.0 (8.5)</td>
<td>163.3 (7.6)</td>
<td>0.296h</td>
</tr>
<tr>
<td>Median (IQR) weight, kg</td>
<td>69.4 (60.0,77.9)</td>
<td>66.0 (58.5,75.3)</td>
<td>72.2 (64.0,83.8)</td>
<td>0.016f</td>
</tr>
<tr>
<td>Median (IQR) BMI, kg/m²</td>
<td>25.4 (22.1,28.7)</td>
<td>24.2 (21.2,27.2)</td>
<td>27.3 (24.1,30.4)</td>
<td>0.001i</td>
</tr>
<tr>
<td>Cachexia (BMI &lt;21 kg/m²) (n (%))</td>
<td>15 (14.7)</td>
<td>13 (21.0)</td>
<td>2 (5.0)</td>
<td>0.026f</td>
</tr>
<tr>
<td>Lung function parameters</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FEV₁, L (mean (SD))</td>
<td>1.47 (0.61)</td>
<td>1.50 (0.63)</td>
<td>1.44 (0.60)</td>
<td>0.654b</td>
</tr>
<tr>
<td>FEV₁ % predicted (mean (SD))</td>
<td>59.8 (22.0)</td>
<td>60.3 (22.7)</td>
<td>59.0 (21.1)</td>
<td>0.766b</td>
</tr>
<tr>
<td>FVC, L (mean (SD))</td>
<td>2.59 (0.87)</td>
<td>2.65 (0.85)</td>
<td>2.50 (0.92)</td>
<td>0.421b</td>
</tr>
<tr>
<td>FVC % predicted (mean (SD))</td>
<td>81.7 (23.4)</td>
<td>83.3 (22.5)</td>
<td>79.2 (24.7)</td>
<td>0.385b</td>
</tr>
<tr>
<td>FEV₁/FVC, % (median (IQR))</td>
<td>59.9 (47.6,66.3)</td>
<td>59.8 (45.7,66.3)</td>
<td>59.9 (51.1,66.1)</td>
<td>0.661f</td>
</tr>
<tr>
<td>PEFR, L/min (median (IQR))</td>
<td>209 (134,294)</td>
<td>215 (131,289)</td>
<td>208 (141,303)</td>
<td>0.725f</td>
</tr>
<tr>
<td>GOLD stages (n (%))</td>
<td></td>
<td></td>
<td></td>
<td>0.770f</td>
</tr>
<tr>
<td>Stage 1 (FEV₁ ≥80% predicted)</td>
<td>20 (19.2)</td>
<td>11 (17.7)</td>
<td>9 (21.4)</td>
<td></td>
</tr>
<tr>
<td>Stage 2 (50% ≥80% predicted)</td>
<td>52 (50.0)</td>
<td>32 (51.6)</td>
<td>20 (47.6)</td>
<td></td>
</tr>
<tr>
<td>Stage 3 (30% ≥50% predicted)</td>
<td>18 (17.3)</td>
<td>10 (16.1)</td>
<td>8 (19.0)</td>
<td></td>
</tr>
<tr>
<td>Stage 4 (FEV₁ &lt;30% predicted)</td>
<td>14 (13.5)</td>
<td>9 (14.5)</td>
<td>5 (11.9)</td>
<td></td>
</tr>
<tr>
<td>HbA1c, % (median (IQR))</td>
<td>6.1 (5.7,6.7)</td>
<td>5.8 (5.5,6.0)</td>
<td>6.8 (6.5,7.6)</td>
<td>&lt;0.001i</td>
</tr>
<tr>
<td>At least one exacerbation in past year (n (%))</td>
<td>33 (31.4)</td>
<td>19 (30.2)</td>
<td>14 (33.3)</td>
<td>0.731f</td>
</tr>
<tr>
<td>Smoking history (n (%))</td>
<td></td>
<td></td>
<td></td>
<td>0.985f</td>
</tr>
<tr>
<td>Never smoked</td>
<td>23 (22.3)</td>
<td>13 (21.0)</td>
<td>10 (24.4)</td>
<td></td>
</tr>
<tr>
<td>Quit &gt;1 year ago</td>
<td>62 (60.2)</td>
<td>39 (62.9)</td>
<td>23 (56.1)</td>
<td></td>
</tr>
<tr>
<td>Current smoker or quit ≤1 year ago</td>
<td>18 (17.5)</td>
<td>10 (16.1)</td>
<td>8 (19.5)</td>
<td></td>
</tr>
<tr>
<td>Smoking pack-years (median (IQR))</td>
<td>37.5 (10.0,62.5)</td>
<td>35.5 (12.5,60.0)</td>
<td>40.0 (4.3,69.5)</td>
<td>0.658f</td>
</tr>
<tr>
<td>Corticosteroid use (n (%))</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inhaled</td>
<td>73 (70.2)</td>
<td>42 (66.7)</td>
<td>31 (75.6)</td>
<td>0.330f</td>
</tr>
<tr>
<td>Oral</td>
<td>10 (9.6)</td>
<td>5 (7.9)</td>
<td>5 (12.2)</td>
<td>0.472f</td>
</tr>
<tr>
<td>Intravenous</td>
<td>2 (1.9)</td>
<td>0 (0.0)</td>
<td>2 (4.9)</td>
<td>0.077f</td>
</tr>
<tr>
<td>SGRQ scores (mean (SD))</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Symptoms</td>
<td>44.42 (22.67)</td>
<td>43.09 (23.40)</td>
<td>46.42 (21.64)</td>
<td>0.464b</td>
</tr>
<tr>
<td>Activity</td>
<td>60.81 (28.35)</td>
<td>59.68 (29.60)</td>
<td>62.52 (26.62)</td>
<td>0.617b</td>
</tr>
<tr>
<td>Impacts</td>
<td>34.40 (20.88)</td>
<td>33.61 (21.66)</td>
<td>35.37 (19.84)</td>
<td>0.640b</td>
</tr>
<tr>
<td>Total</td>
<td>44.09 (21.10)</td>
<td>43.12 (22.14)</td>
<td>45.55 (19.62)</td>
<td>0.566b</td>
</tr>
<tr>
<td>CAT score (mean (SD))</td>
<td>16.0 (8.8)</td>
<td>15.5 (9.4)</td>
<td>16.7 (7.8)</td>
<td>0.499b</td>
</tr>
<tr>
<td>CES-D score (median (IQR))</td>
<td>7.5 (2.0,16.0)</td>
<td>6.5 (2.0,16.3)</td>
<td>8.0 (3.0,16.5)</td>
<td>0.426f</td>
</tr>
<tr>
<td>CESD-R score (median (IQR))</td>
<td>7.0 (3.0,19.0)</td>
<td>7.0 (2.0,20.0)</td>
<td>9.0 (4.0,18.3)</td>
<td>0.393f</td>
</tr>
<tr>
<td>Clinically significant CES-D score (i.e., ≥16) (n (%))</td>
<td>27 (26.0)</td>
<td>16 (25.8)</td>
<td>11 (26.2)</td>
<td>0.965f</td>
</tr>
<tr>
<td>Clinically significant CESD-R score (i.e., ≥16) (n (%))</td>
<td>33 (31.4)</td>
<td>20 (31.7)</td>
<td>13 (31.0)</td>
<td>0.932f</td>
</tr>
<tr>
<td>1-year mortality (n (%))</td>
<td>6 (6.9)</td>
<td>1 (1.9)</td>
<td>5 (14.3)</td>
<td>0.026f</td>
</tr>
</tbody>
</table>

*comparing non-diabetes and diabetes; **based on patient’s perception; †calculated using the Global Lungs Initiative 2012 Excel sheet calculator; ‡one pack-year is defined as smoking 20 cigarettes daily for 1 year; *in past 3 to 4 months; ψChi-squared test; χKruskal-Wallis test; t-test; Mann-Whitney U test; BMI: body mass index; CAT: COPD assessment test; SD: standard deviation; CES-D: center for epidemiologic studies depression scale; CESD-R: center for epidemiologic studies depression scale-revised; SGRQ: St. George’s Respiratory Questionnaire

Non-diabetes includes normal and pre-diabetes, and diabetes includes a prior medical history and newly diagnosed diabetes
Lung Function
cantly (p=0.026) greater than among non-diabetics (n=1).

There were no significant differences observed between diab-
etics and non-diabetics in lung function parameters, GOLD
stages, history of COPD exacerbations, smoking history and
pack-years of smoking, corticosteroid use, and CAT, SGRQ,
CES-D, and CESD-R scores. However, the number of deaths
after 1 year among those with diabetes (n=5) was signifi-
cant (p=0.026). Cachexia was noted more frequently among non-diabetics (p=0.026).
The use of intravenous corticosteroids in the past 3 to 4
months was associated with an increased HbA1c (r=0.199,
p=0.043). Among the patients who used conventional long-
acting beta2-agonist and corticosteroid combination inhalers
(n=73), those who used additional corticosteroid inhalers
(n=4) had increased HbA1c levels (r=0.261, p=0.007), and
diabetes was more prevalent among them (r=0.248,
p=0.011). A negative association was found between the
FVC percentage predicted and HbA1c (Pearson’s r=-0.210,
p=0.011). A negative association was found between the
FVC percentage predicted and HbA1c (Pearson’s r=-0.210,
p=0.011).

FEV1, FVC, and the peak expiratory flow rate decreased with
Lung Function

Exacerbations
The occurrence of at least one exacerbation in the past year
was associated with a history of oral corticosteroid use
(r=0.198, p=0.044) along with increased dosages of inhaled
corticosteroids per day (r=0.208, p=0.035). There were also
significant associations between patients with at least one
exacerbation in the past year and CAT (r=0.350, p<0.001),
SGRQ symptoms (r=0.435, p<0.001), SGRQ activity
(r=0.201, p=0.040), SGRQ impacts (r=0.353, p<0.001),
SGRQ total (r=0.338, p<0.001), CES-D (r=-0.232, p=0.018),
and CESD-R (r=0.256, p=0.008) scores.

Quality of Life and Depression
All SGRQ and CAT scores were positively associated with
inhaled corticosteroid use (0.198≤r≤0.329, p≤0.044) and
the dosage (0.188≤r≤0.236, p<0.05). All SGRQ and CAT
scores were positively (p<0.001 for all) related to CES-D
(0.554≤r≤0.705 for SGRQ, r=0.664 for CAT) and CESD-R
(0.629≤r≤0.792 for SGRQ, r=0.719 for CAT) scores. As
expected, CAT scores correlated well (p<0.001) to the SGRQ
total (r=0.820) and component scores. Notably, CES-D and
CESD-R scores correlated well together (r=0.925, p<0.001).
From Table 1, it can be seen that 26.0% and 31.4% of the
cohort of patients had clinically significant depression scores
as assessed by the CES-D and CESD-R, respectively.

DISCUSSION
The prevalence of diabetes and pre-diabetes in our COPD
cohort (n=105) was 40% each, leaving only 20% with normal
glucose tolerance as assessed by HbA1c. We found HbA1c to be associated with the use of intravenous cortico-
steroids, increased BMI, and a decreased FVC percentage
predicted. Patients with at least one COPD exacerbation in
the past year or those with reduced lung function were more
likely to have a worse quality of life and higher depression
scores. Overall, 31.4% of our cohort had clinically signifi-
cant depression scores. Also, after a 1-year follow-up, diabet-
etics in our cohort had a significantly greater mortality than
non-diabetics.

Comparison with Existing Literature
The high prevalence of 40% diabetes among COPD patients
in our study differs markedly from the 12.7% reported by
dealt with participants from the general community, whereas
our study looked at patients in hospital clinics. Also, they
included patients with respiratory symptoms, but without
spirometric abnormality. In fact, only 12.8% of patients in
that study were GOLD Stage 2 or higher, compared to 80.8%
in our COPD cohort. In another US study, in which over half
of the study population had what was defined as moderate to
high complexity disease (complexity of the illness being used
as a proxy for COPD GOLD stage severity), diabetes preva-
ence was found to be comparatively higher at 21.9%–28.8% (com-
mercial and Medicare dataset populations respectively)
[10]. In a UK study, Feary et al. [11] analyzed primary care
records and found that 12.2% of patients with a diagnosis of
COPD also had a diagnosis of DM or were on treatment for
such. They also found that COPD patients have a two-fold
higher risk of having new-onset DM. This is consistent with
our finding of 40% DM among our COPD cohort when com-
pared with that of 20.5% reported previously in the general
population of Trinidad and Tobago aged 16-64 years [5].

Additionally, the high prevalence of DM could be attributed
to the high mean (SD) age of our cohorts, 67.4 (11.0) years.
Hennis et al. [25] observed a higher incidence of DM with
increasing age, while the prevalence of DM in citizens of
Trinidad and Tobago aged 55-64 years was 27.6%, greater
than the mean percentage for the entire Trinidadian popula-
tion mentioned above [5]. This is possibly due to declining
glucose tolerance caused by peripheral insulin resistance
[26] along with an increase in visceral fat [27], and it is vis-
ceral fat that may be the likely cause of the relatively higher
BMI among our diabetics. Interestingly, we found a signifi-

Figure 1. Graph showing the prevalence of diabetes in COPD patients
HbA1c: glycated haemoglobin; COPD: chronic obstructive pulmonary disease
cantly greater number of cachectic patients among non-diabetics than diabetics. Still, diabetics and pre-diabetics, both of which are closely associated with obesity, comprised a large proportion of our COPD cohort, and this may explain the relatively lower prevalence of cachexia of 14.7% compared with 20%-40% reported in the existing literature [16].

In our study, we found that all patients who used inhaled corticosteroids within the past 3 to 4 months exhibited abnormal HbA1c levels in the diabetic range, that is >6.5%. Whether this association is cause or effect could not be established in this cross-sectional study. Rogliani et al. [28] recently found an association between the use of inhaled corticosteroids and the presence of diabetes in COPD, but that the use of a combination corticosteroid and beta-agonist inhaler reduced this. In our study, increased HbA1c levels and diabetes prevalence were seen among patients who used additional inhaled corticosteroids along with the combination long-acting beta-agonist and steroid inhalers.

There was a negative correlation between the HbA1c and FVC percentage predicted, with diabetics having a mean (SD) FVC percentage predicted of 79.2% (24.7%). This suggests a mild restrictive defect being associated with DM and increased HbA1c, consistent with the previous literature [29]. This restrictive disposition may be because of our diabetics’ higher BMI, but low-grade systemic inflammation or hyperglycemia-induced diabetic microangiopathy may also be involved [29].

We found that patients with at least one COPD exacerbation in the past year or those with reduced lung function were more likely to have a worse quality of life and higher depression scores. These results are consistent with the literature [23,30,31], and in fact, a more recent study by Yohannes et al. [24] found that patients with depression experienced more exacerbations. Depression is well known to be more prevalent in COPD, and the prevalence of depression in our COPD cohort was found to be 31.4%, compared with 14% in a community sample from the Trinidadian population [32]. Furthermore, previous studies have found similar prevalences of 21.6%-38% of depression in COPD patients [23,24]. Also, depression has been found to be associated with increased 3-year but not 1-year mortality [33].

In keeping with the findings of prior studies that comorbid DM is associated with a higher risk of death [9] the 1-year mortality among diabetics in this study was significantly higher (p=0.026) than among non-diabetics.

Strengths and Limitations
This study is as far as we know one of the first in the West Indies, a region with a high background prevalence of type 2 diabetes, to objectively evaluate the prevalence of diabetes in a COPD population and to demonstrate not only the doubled risk of diabetes, but also a heightened risk of mortality. Interestingly, up to 38% of our diabetics were diagnosed incidentally, and among those who were non-diabetic, 67% had pre-diabetes. Previous studies on COPD and DM have not reported prevalence rates for pre-diabetes as here reported. Although an American Diabetes Association expert panel report highlighted the higher rates of progression of pre-diabetic individuals (up to 70%) toward diabetes [33], many studies on COPD comorbidities did not focus on its prevalence.

To the best of our knowledge, this study is also one of the first in the West Indies to utilize the CESD-R and furthermore, confirm its strong correlation (p<0.001) to its predecessor, the CES-D.

In this study, a point-of-care HbA1c assay (Siemens DCA Vantage Analyzer) was used, although we note that the American Diabetes Association does not recommend its use for diagnosis [15]. Leca et al. [34] found that point-of-care assays under-evaluated HbA1c levels as compared to the central laboratory, and as such, we may have underestimated the prevalence of DM. Also, we note that of all the point-of-care HbA1c assays available, the Siemens DCA Vantage Analyzer has the best sensitivity and specificity for diagnosis [35]. Lastly, the Global Lung Initiative equations may not have adequately covered Trinidadian ethnic groups [17]. Finally, the presence of multiple comorbidities was not studied, and they might have influenced some of the functional and mortality outcomes in our study population.

In conclusion, we observed a 40% prevalence of diabetes in this COPD cohort. From our findings, whereas the co-existence of DM does not appear to affect the functional measures of COPD significantly including FEV1, exacerbation frequency, quality of life, and depression, there is a notable increase in 1-year mortality. It is hoped that this study highlights the necessity of screening for diabetes in COPD patients as 38% of our diabetic patients were newly diagnosed, and its presence impacts on 1-year mortality. We recommend further studies to confirm these findings and also to explore the influence of DM on COPD prognosis and mortality.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Faculty of Medical Sciences, The University of the West Indies, St Augustine Campus and the North-West and North-Central Regional Health Authorities, Trinidad and Tobago.

Informed Consent: Written informed consent was obtained from all patients who participated in this study.

Peer-review: Externally peer-reviewed.


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