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ORIGINAL RESEARCH

Increased Incidence of Chronic Obstructive Pulmonary Disease in Women Due to Long-Term Passive Smoking

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Objective: To investigate the impact of long-term passive smoking on the pathogenesis of chronic obstructive pulmonary disease (COPD) in women.

Methods: We conducted a community-based cross-sectional study involving 2,360 women aged ≥40 years in Jinan, China (October 1, 2022-April 30, 2023). Participants underwent comprehensive assessments including pulmonary function tests (spirometry), hematological analyses, and structured questionnaires evaluating COPD symptoms and passive smoking exposure. Based on exposure history, subjects were stratified into long-term passive smoking (LPS, n = 610) and non-passive smoking (NPS, n = 1,750) cohorts.

Results: Comparative analysis revealed significant pulmonary function impairment in the LPS group versus NPS controls: lower FEV1 (2.97±0.61 vs 3.25±0.37 L, $p < 0.05$), reduced FEV1% predicted (78.20±10.18 vs 81.47±14.69, $p < 0.05$), decreased FEV1/FVC ratio (83.32±11.20 vs 87.23±10.32%, $p < 0.05$). Small airway dysfunction was more pronounced in LPS participants, evidenced by: diminished MEF75% (77.58±11.95 vs 86.08±14.02 L/s, $p < 0.05$), reduced MEF50% (62.76±19.79 vs 89.36±16.78 L/s, $p < 0.05$), lower MMEF (80.87±12.80 vs 87.46±11.26 L/s, $p < 0.05$). The LPS group demonstrated: higher prevalence of preserved ratio impaired spirometry (PRISm, 5.74% vs 2.91%); increased annual exacerbation frequency ($p < 0.05$), elevated systemic inflammatory markers ($p < 0.05$), greater symptom severity ($p < 0.05$).

Conclusion: Our findings demonstrate that chronic passive smoke exposure constitutes an independent risk factor for COPD development in women, associated with higher disease prevalence, accelerated pulmonary function decline, increased exacerbation frequency and enhanced systemic inflammation.

Keywords: women, passive smoking, chronic obstructive pulmonary disease, pulmonary function, preserved ratio impaired spirometry

Introduction

Chronic obstructive pulmonary disease (COPD) is a chronic respiratory disorder characterized by persistent airflow limitation and respiratory symptoms. Although COPD is a preventable and treatable disease, it ranks as the fifth leading cause of death in China,[1] remains a major contributor to global morbidity and mortality, is now one of the top three causes of death worldwide[2,3] The pathogenesis of COPD is multifactorial, involving complex interactions between gene-environment.[4,5] Tobacco smoking and the inhalation of toxic particles and gases from household and outdoor air pollution are the main environmental exposures leading to COPD.[6,7] Among these factors, cigarette smoking is predominant, accounting for 80–90% of COPD cases in Western countries and 72% in China.[8] Earlier smoking initiation, prolonged duration, higher cumulative exposure (pack-years), deeper smoke inhalation, and accelerated annual decline in forced expiratory volume in one second (FEV1) are strongly associated with increased COPD risk and mortality in smokers

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compared to non-smokers.[9] Quit smoking is useful to improve the symptoms, respiratory function and metabolic parameters of COPD patients.[10] Notably, 25–51% of COPD patients report no history of active smoking.[7,11] The 2018 China Adult Tobacco Survey revealed that 68.1% of non-smokers are exposed to secondhand smoke (SHS), which contains numerous toxic and carcinogenic compounds.[12] SHS exposure represents a critical public health concern, with even transient contact inducing adverse effects on respiratory, cardiovascular, immune, and endocrine systems.[13,14] Epidemiological studies implicate SHS in the etiology of cardiovascular diseases,[15] childhood asthma,[16] and lung cancer,[17,18] contributing to an estimated 1% of global mortality.[19] Mechanistically, SHS shares pathophysiological pathways with active smoking, including oxidative stress and airway inflammation, thereby increasing risks of respiratory symptoms and COPD development.[20,21] While direct causal evidence linking SHS to COPD incidence remains limited, a large European prospective case-control study identified SHS as a significant risk factor for COPD.[18] Longitudinal investigations, including the Singapore Chinese Health Study and a 17-year Chinese cohort, demonstrate associations between SHS exposure and elevated COPD mortality.[22,23] However, conflicting findings from US case-control[24] and Hong Kong COPD patient studies[25] highlight ongoing controversies regarding SHS-related COPD risk in non-smokers.

Sex-specific analyses reveal notable disparities: 14.1% of males with airflow obstruction are non-smokers compared to 26.8% of females.[26] Over the past two decades, COPD prevalence has remained higher in females across most age groups.[27,28] Emerging evidence suggests that biological vulnerabilities in never-smoking women may interact with SHS exposure to amplify COPD susceptibility.[29,30] This study aims to elucidate the relationship between passive smoking and COPD risk in females, providing evidence to advocate for smoke-free public policies and reduce SHS exposure among women.

Materials and Methods

Study Population

A total of 2,360 female volunteers aged ≥40 years (mean age: 55.6 years; range: 40–86) were recruited from homogeneous communities in western Jinan between October 1, 2022, and April 30, 2023. Inclusion criteria: All subjects were free of active smoking, they were divided into long-term passive smoking group (LPS, n = 610) and non-passive smoking group (NPS, n1,750) according to whether they were exposed to long-term secondhand smoke indoors (≥0.5/d for ≥5 years). The LPS group was further categorized according to the duration of exposure: <2 0 years group; 20≤ group <30 group; ≥30 years group. Exclusion criteria: (1) active smokers; (2) unclear spousal smoking history; (3) occupational dust exposure; (4) acute respiratory infections; (5) contraindications for spirometry; (6) chronic hepatic/renal diseases, severe cardiovascular pathologies, immunologic disorders, malignancies, hematologic diseases, pregnancy/lactation, or psychocognitive impairments affecting questionnaire validity. The experiments were carry out in accordance with the Declaration of Helsinki (2013) of the World Medical Association and the protocol received ethical approval from the Institutional Review Board of Shandong Second Provincial General Hospital, with written informed consent obtained from all participants.

Study Design

Questionnaire Survey

Trained investigators administered standardized questionnaires to assess demographic characteristics (age, marital status, education level), clinical parameters (annual acute exacerbation frequency), and respiratory symptoms using the modified Medical Research Council (mMRC) dyspnea scale (range: 0–4) and the COPD Assessment Test (CAT) scale (range: 0–40).

Pulmonary Function Testing

Spirometry was performed using Jaeger Masterscreen PFSystem (CareFusion, Germany) following ATS/ERS guidelines. COPD diagnosis followed 2021 GOLD criteria.[31] All pulmonary function test values are results post-bronchodilation.

Arterial Blood Gas and Complete Blood Count

Arterial blood gas: Measured using GEM 3000 analyzer (Instrumentation Laboratory, USA) under room air conditions. Complete blood count: Analyzed via Mindray BC-6800 hematology system (Shenzhen, China).

Statistical Analysis

Data analyzed with SPSS 23.0 (IBM, USA). Categorical data are summarized as frequencies and percentages. For continuous data, normally distributed variables are summarized as mean ± SD and never-normally distributed variables as median (interquartile range [IQR]). Between-group comparisons: Independent *t*-test (normal distribution). Mann–Whitney *U*-test (non-normal distribution). Statistical significance thresholds: $p < 0.05$.

Results

Baseline Characteristics

No significant differences were observed in age, BMI, marital status, education, or alcohol consumption between groups (Table 1).

Laboratory and Pulmonary Function Parameters

The long-term passive smoking group showed significantly lower EFV1 (2.97+0.61 vs 3.25+0.37; $p < 0.05$), FEV1% predicted (78.20 ± 10.18 vs 81.47 ± 14.69; $p < 0.05$) and FEV1/FVCex% (83.32 ± 11.20 vs 87.23 ± 10.32; $p < 0.05$). Compromised small airway function parameters including MEF75% (77.58 ± 11.95 vs 86.08 ± 14.02), MEF50% (62.76 ± 19.79 vs 89.36 ± 16.78), and MMEF (80.87 ± 12.80 vs 87.46 ± 11.26) (all $p< 0.05$)).; alongside elevated WBC counts (9.33 ± 7.94 vs 7.31 ± 2.67; $p < 0.05$) and HB (137.02±23.66 vs 124.88±18.86; $p < 0.05$); Preserved ratio impaired spirometry (PRISm) cases was more frequent in the exposed group ($p < 0.05$) (Table 2).

Table 1 Baseline Characteristics

Parameter	LPS (n = 610)	NPS (n = 1,750)	*p*
Age (years)	58 (40–73)	60 (41–76)	0.07
BMI (kg/m²)	24.9 ± 4.5	24.7 ± 4.1	0.31
Married (%)	98.2	97.0	0.11
College education (%)	44.9	45.6	0.15
Alcohol consumption (%)	11.8	10.7	0.45

Abbreviations: LPS, long-term passive smoking; NPS, on-passive smoking.

Table 2 Laboratory and Pulmonary Function Parameters (Mean ± SD)

Parameter	LPS (n = 610)	NPS (n = 1,750)	*p*
PH	7.41±0.04	7.42±0.09	0.32
PO_2(mmHg)	78.36±11.21	80.76±9.3	0.07
PCO_2(mmHg)	43.91±8.56	40.67±6.11	0.12
HB (g/L)	137.02±23.66	124.88±18.86	<0.05
WBC ($10^{\wedge 9}$L)	9.33±7.94	7.31±2.67	<0.05
FEV1 (L)	2.97+0.61	3.25+0.37	<0.05
FEV1(%) Pre	78.20±10.18	81.47±14.69	<0.05
FEV1/FVCex (%)	83.32±11.20	87.23±10.32	<0.05
RV/TLC (%)	41.44±4.79	36.82±3.77	0.09
MEF75 (%)	77.58±11.95	86.08±14.02	<0.05
MEF50 (%)	62.76±19.79	89.36±16.78	<0.05
MMEF (%)	80.87±12.80	87.46±11.26	<0.05
PRISm (No.[%])	35 (5.74%)	54 (2.91%)	<0.05

Abbreviations: LPS, long-term passive smoking; NPS, on-passive smoking.

Table 3 COPD Grade According to GOLD

COPD According to GOLD	LPS		NPS	
	No.	%	No.	%
NO COPD	512	83.93	1693	96.74*
GOLD I	40	6.67	26	1.49*
GOLD II	42	6.89	24	1.37*
GOLD III	11	1.80	6	0.34*
GOLD IV	5	0.82	1	0.06*

Notes: *p < 0.01 compared with the LPS.
Abbreviations: LPS, long-term passive smoking; NPS, on-passive smoking.

Table 4 COPD Symptoms and Exacerbations (Median [IQR])

Parameter	LPS (n = 98)	NPS (n = 57)	*p*
Acute exacerbations (/yr)	2(1–3.5)	1(0–1.5)	<0.05
Cough/sputum duration (yr)	5(1–11)	2(0.5–5)	<0.05
mMRC	2(0–3)	1(0–2)	<0.05
CAT	15 (6–23)	9(3–14)	<0.05

Abbreviations: LPS, long-term passive smoking; NPS, on-passive smoking.

COPD Symptoms and Exacerbations

Compared to the NPS group, the LPS group had more severe airflow obstruction ($p < 0.01$) (Table 3). The COPD in LPS group reported more frequent acute exacerbations ($p < 0.05$), prolonged cough/sputum duration ($p < 0.05$), and higher mMRC ($p < 0.05$) and CAT scores ($p < 0.05$) (Table 4).

Discussion

COPD is a globally recognized common respiratory disorder that severely threatens human health, characterized by high disability and mortality rates, imposing substantial economic and psychological burdens on both society and families. In China, the incidence, prevalence, and disease burden of COPD significantly exceed those in Western developed countries, establishing it as a critical public health concern.[32] While smoking remains the single greatest risk factor for COPD, over half of COPD cases are estimated to be attributable to other causes,[11] with a notably higher proportion occurring among non-smokers.[26] Recent epidemiological data from China[33] reveal a 13.7% prevalence of COPD among adults aged ≥40 years, with gender-specific rates of 19.0% in males and 8.1% in females. Approximately 100 million COPD patients exist nationwide, including 31.5 million females (32%). Previous studies indicate greater secondhand smoke exposure among female non-smokers,[34–36] with higher COPD prevalence in non-smoking African American women compared to Caucasians, potentially linked to passive smoking.[16–19] Passive smoking ≥30 minutes/day can trigger early pathological changes such as oxidative stress,[37,38] so we used this as a limit for passive smoking to investigate the association between passive smoking and COPD in Chinese women.

Distinct from prior COPD-passive smoking studies,[14,15] this research employs the diagnostic gold standard post-bronchodilator pulmonary function testing[31] and specifically examines Chinese females. Furthermore, we eliminated confounding from active smoking by excluding current/former smokers. Spirometry, the gold standard for COPD diagnosis, quantifies airflow limitation severity. COPD was defined as post-bronchodilator FEV1/FVC <70%, with severity staged by FEV1% predicted. Airflow limitation leads to pulmonary hyperinflation, evidenced by elevated RV/TLC. Compared to non-passive smoking controls, the long-term passive smoking group demonstrated significantly reduced the FEV1, FEV1/FVC and FEV1% predicted, alongside increased RV/TLC, indicating substantial large airway damage from chronic passive smoke exposure. PRISm, an alternative designation for GOLD-Unclassified, identifies individuals with normal FEV1/FVC ratios but abnormal lung function (post-bronchodilator FEV1 <80% predicted),[39] distinguishing this pattern from restrictive and nonspecific abnormalities. PRISm patients carry elevated risks for

developing airflow obstruction,[39,40] with female PRISm-to-COPD progression strongly predicted by passive smoking.[41] Our study revealed higher PRISm prevalence in long-term passive smokers. Additionally, significant between-group differences emerged in small airway function indices, consistent with smoking-related COPD patterns of severe small airway obstruction and emphysema, validating small airway vulnerability in females. Both active and passive smoking thus induce concurrent large and small airway damage, leading to irreversible pulmonary impairment and progressive airflow obstruction, ultimately increasing COPD risk. The elevated COPD prevalence in female passive smokers aligns with previous findings.[29]

The mMRC scale, recommended by GOLD guidelines for dyspnea assessment,[31] correlates well with COPD symptoms, health status,[42] and mortality prediction.[43,44] For COPD patients, the CAT can predict the health status deterioration, depression, mortality and exacerbation risk (Jones et al 2009).[45,46] Our results demonstrate significantly prolonged cough/sputum duration, higher mMRC and CAT scores in passive smoking COPD patients versus controls, suggesting passive smoking exacerbates symptoms, potentially increases mortality, more severe symptoms and risk of acute exacerbation. Acute exacerbation history remains the strongest exacerbation predictor independent of GOLD staging.[47] Analysis revealed significantly more prior-year exacerbations in passive smokers, indicating passive smoke exposure as a high-risk factor for acute COPD episodes in women.

Current smokers and those suffering from other environmental exposures have increased mucous cells as well as cellular hyperplasia in small and large airways.[48,49] COPD patient might produce neutrophils nearly 25-fold greater than in healthy, so neutrophilic inflammation may contribute to emphysema in COPD during acute inflammatory. Analogous to smoking-related COPD's elevated neutrophil counts,[50–52] passive smoking females also exhibited significantly higher neutrophil levels. These pathological changes manifest as bronchial mucosal congestion/edema, abnormal smooth muscle contraction, airway narrowing, impaired gas exchange, and alveolar hyperinflation, collectively aggravating respiratory symptoms.[7]

Smoke-free laws have been adopted to protect against exposure to smoke in some public locations increasingly.[53] Many studies have found protective associations between smoke-free policies and COPD-related hospitalizations, mortality and 30-day hospital readmissions.[54–56] There are many studies especially for smoke-free policies in children,[57] but little about women.[58,59] The World Health Organization needs more research on tobacco use among women and tobacco control policies for women specific.[58,60,61] Our findings further support the need for public health interventions targeted to never-smoking women, recommending implementation of smoke-free policies in both homes and the workplace.[35,58]

In conclusion, the present study demonstrates that long-term passive smoking significantly increases the risk and severity of COPD among women. Specifically, long-term passive smoking is associated with reduced pulmonary function, exacerbated inflammation, and increased frequency of acute exacerbations. Notably, a high prevalence of preserved ratio impaired spirometry was observed in the LPS population, suggesting early airflow limitation. This finding further supports the health benefits of legislation to reduce secondhand smoke exposure among women. However, potential biases in participant selection or unaccounted confounders may affect the interpretation of the findings and conclusions, therefore, it is recommended that more in-depth validation studies to refine evidence be conducted by expanding the sample size.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Disclosure

The authors report no conflicts of interest in this work.

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