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Airflow obstruction and metabolic syndrome: the Guangzhou Biobank Cohort Study

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ABSTRACT: There is some evidence that chronic obstructive pulmonary disease (COPD) and metabolic syndrome may be related, perhaps through systemic inflammation, which is common to both. However, the association between the two conditions has not yet been clearly shown.

The present study involved 7,358 adults aged ⩾50 yrs from a population-based survey who underwent spirometry, a structured interview and measurement of fasting metabolic marker levels.

Airflow obstruction (forced expiratory volume in 1 s/forced vital capacity ratio of less than the lower limit of normal) was present in 6.7%, and the International Diabetes Federation metabolic syndrome criteria were met by 20.0%. The risk of metabolic syndrome was higher in those with airflow obstruction than in those without (odds ratio (OR) 1.47; 95% confidence interval (CI) 1.12–1.92), after controlling for potential confounders. Of the five components of metabolic syndrome, only central obesity was significantly associated with airflow obstruction (OR 1.43; 95% CI 1.09–1.88) after adjusting for body mass index. A similar association was observed in both never and current smokers.

In this Chinese sample, airflow obstruction was associated with metabolic syndrome, and, in particular, its central obesity component. This may help explain the increased risk of cardiovascular diseases in COPD, and so could guide future clinical practice.

KEYWORDS: Central obesity, China, chronic obstructive pulmonary disease, comorbidity, general population

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The term chronic systemic inflammatory syndrome has been proposed to take account of the inflammatory nature common to chronic obstructive pulmonary disease (COPD) and its comorbid conditions [1, 2]. One of these possible comorbid conditions is metabolic syndrome, an aggregate of interrelated cardiometabolic risk factors, comprising glucose intolerance, abdominal obesity, dyslipidaemia and hypertension, which are associated with an increased risk of cardiovascular disease and type 2 diabetes [3, 4].

This proposition was based on the increasing body of evidence that systemic inflammation is related to the pathogenesis of both COPD [5] and metabolic syndrome [3]. This was further supported by the finding that metabolic syndrome was more common among people with advanced COPD, compared with age- and sex-matched controls [6]. However, an association between airflow obstruction, a hallmark of COPD, and metabolic syndrome was not observed in three recent cross-sectional studies, two in population samples from Taiwan [7] and Japan [8], and one in healthy attendees of a day care centre and a home for the aged in Italy [9].

The current controversy limits an understanding of the joint aetiology and prognosis of COPD and metabolic syndrome. The aim of the present study was to examine the relationship between the two conditions, also considering each of the components of metabolic syndrome, in a large population-based sample in China.

METHODS

Participants

The Guangzhou Biobank Cohort Study, a collaboration between the Guangzhou Number 12 People's Hospital (Guangzhou, China) and the Universities of Birmingham (Birmingham, UK) and Hong Kong (Hong Kong), has been described in detail previously [10]. Participants were recruited from the Guangzhou Health and Happiness Association for Respectable Elders

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(GHHARE), a community social and welfare association unofficially aligned with the municipal government, membership of which is open to older persons for a nominal monthly fee of 4 yuan (0.5 US dollars). Approximately 7% of permanent Guangzhou residents aged ⩾50 yrs are members of GHHARE, of whom 11% (~10,000 participants) were randomly selected to take part in each of the two time periods (2003–2004 and 2005–2006). Participants were included if they were capable of consenting, ambulatory and not receiving treatment modalities for life-threatening conditions. Of those eligible, 90% of males and 99% of females participated. Ethical approval was received from the Medical Ethics Committee of the Guangzhou Medical Association (Guangzhou, China). Written informed consent was obtained from all participants.

Measurements

Each enrolled participant underwent a physical examination and a detailed medical interview [10]. Relevant to this study, blood pressure was recorded as the mean of the last two of three measurements taken using a digital sphygmomanometer (Omron 705CP; Omron, Kyoto, Japan). Waist circumference was measured horizontally around the narrowest part of the torso, between the lowest rib and the iliac crest. Levels of fasting glucose, triglyceride and high-density lipoprotein (HDL)–cholesterol were determined using an automated clinical chemical analyser (Shimadzu CL-8000; Shimadzu, Kyoto, Japan). Body mass index (BMI), calculated from weight divided by height squared, was grouped in accordance with World Health Organization recommendations for Asian populations [11].

A detailed smoking history was obtained and used to define individuals as never-, former or current smokers. Cumulative cigarette consumption in pack-years was calculated to quantify the exposure, where 1 pack-yr is equivalent to smoking a mean of 20 cigarettes·day^{-1} for 1 yr. The level of physical activity (inactive, minimally active and active) was quantified using the short version of the International Physical Activity Questionnaire [12]. Educational level (primary or below, junior middle and senior middle or above) was used as proxy for socioeconomic status.

Lung function status

Details of spirometry have been described elsewhere [13]. At least three manoeuvres were performed without use of bronchodilator, and the best measure of forced expiratory volume in 1 s (FEV_1), forced vital capacity (FVC) and FEV_1/FVC was recorded. The quality of the results was assured by their first passing a numerical check algorithm, and then by visual inspection of the flow–volume and volume–time loops. The equations derived from a Chinese population [14] were used to determine the predicted values and lower limit of normal (LLN) for lung function parameters.

Airflow obstruction was defined by an FEV_1/FVC of <LLN in those who did not report physician-diagnosed asthma and bronchiectasis. The severity of the obstruction was categorised based on percentage predicted FEV_1, modelling the staging of COPD as recommended by the Global Initiative for Chronic Obstructive Lung Disease (GOLD) [15]. Mild, moderate and severe obstruction were defined as FEV_1 of >80, 50–80 and <50% pred, respectively. Since there is evidence that restrictive lung function abnormality is also associated with metabolic syndrome [7–9], participants with restrictive abnormality, defined as an FEV_1/FVC of ⩾LLN and FVC of <LLN [16], were removed from the main analyses so that the comparison was between those with airflow obstruction and those with normal lung function. However, the relationship between restrictive abnormality and metabolic syndrome was also explored briefly.

Metabolic syndrome

Metabolic syndrome was classified according to the International Diabetes Federation (IDF) [4], which requires the presence of central obesity (defined as a waist circumference of ⩾90 cm in males and ⩾80 cm in females) plus any two of the four following criteria: 1) raised blood pressure (⩾130/85 mmHg); 2) raised fasting glucose levels (⩾5.6 mM); 3) raised triglyceride levels (⩾1.7 mM); and 4) reduced HDL–cholesterol levels: (<1.03 mM in males or <1.29 mM in females). Receiving specific treatment for a criterion was regarded as fulfilling the criterion.

Statistical analysis

Differences in characteristics by lung function status were evaluated using the Chi-squared test. ANCOVA was used to obtain age-adjusted means of metabolic marker levels. Glucose and triglyceride levels were logarithmically transformed and their geometric means are presented. Logistic regression models were constructed in order to assess the relationship between airflow obstruction and metabolic syndrome or its components. Three models are presented: model 1 was adjusted for age, sex, education, smoking and physical activity; model 2 was additionally adjusted for BMI, and model 3, which was used specifically in the assessment of the components of metabolic syndrome, also included the other four components. It was also examined whether any relationships were modified by sex, using interaction terms in the models and repeating all analyses in males and females separately. The robustness of the findings was tested against a different definition of airflow obstruction, with an FEV_1/FVC of <0.70 (compatible with the GOLD definition [15] and consistent with previous studies). All analyses were performed using Stata version 10.1 (StataCorp, College Station, TX, USA).

RESULTS

Of the 20,431 participants, 8,701 had available information and valid spirometric data. The age, sex and metabolic profile of these participants was similar to that of those without valid data. Among them, 1,343 (15.4%) were excluded because they reported having physician-diagnosed asthma or bronchiectasis, or displayed a restrictive abnormality on spirometry. The remaining 7,358 participants comprised 2,008 males (27.3%) and 5,350 females (72.7%), with an overall mean±SD age of 61.6±6.7 yrs. The prevalence of airflow obstruction was similar in males (6.4%) and females (6.9%). Compared with the participants with normal lung function, those with airflow obstruction were older, less highly educated and had had greater smoking exposure. The BMI was also lower in this group, with a significantly higher proportion (9.9%) being in the underweight category (<18.5 kg·m^{-2}) compared to those with normal lung function (3.9%). This trend was consistent in both sexes (table 1). There were no differences in blood pressure

and fasting glucose and HDL–cholesterol levels across lung function status. However, in males but not in females, those with airflow obstruction showed a significantly smaller waist circumference (79.3 *versus* 81.9 cm; p=0.001) and lower triglyceride level (1.23 *versus* 1.38 mM; p=0.021). In both sexes, the proportions of participants receiving pharmacological treatment for hypertension, diabetes and dyslipidaemia were similar in those with normal and reduced lung function (table 1).

The prevalence of metabolic syndrome in this sample was 20.0% (95% confidence interval (CI) 19.1–20.9%). Although its prevalence was higher in females (22.9%) than in males (12.4%), there was no evidence from the heterogeneity of effect across strata that the association of lung function status and metabolic syndrome varied with sex (data not shown). Results are therefore presented for males and females together. Table 2 shows the relationship between lung function status and metabolic syndrome and its components. Although metabolic syndrome (22.6 *versus* 19.8%), central obesity (34.1 *versus* 33.1%) and raised blood pressure (56.7 *versus* 53.4%) were more common in individuals with airflow obstruction than in those with normal lung function, the opposite was seen for raised fasting glucose level (34.3 *versus* 36.9%), raised triglyceride level (29.6 *versus* 33.4%) and reduced HDL–cholesterol level (15.9 *versus* 16.6%). Nevertheless, none of these differences were significant in univariate analyses. Adjusting for age, sex, education, smoking and physical activity (model 1) did not alter the lack of association, although the risk estimates changed slightly. However, metabolic syndrome was significantly associated with the presence of airflow obstruction when BMI was additionally adjusted for (model 2), with an odds ratio (OR) of 1.47 (95% CI 1.12–1.92). Among the five components of metabolic syndrome examined, only central obesity was significantly associated with airflow obstruction (OR 1.43; 95% CI 1.09–1.88).

TABLE 1 Characteristics and age-adjusted metabolic profile of 7,358 Chinese adults aged ⩾50 yrs by sex and lung function status (Guangzhou Biobank Cohort Study, 2003–2006)

	Males			Females		
	Normal lung function	Airflow obstruction	p-value	Normal lung function	Airflow obstruction	p-value
Subjects	1880 (93.6)	128 (6.4)		4982 (93.1)	368 (6.9)	
Age yrs	63.5±6.2	67.1±5.6	<0.001	60.7±6.7	62.7±6.7	<0.001
Education						
Primary or below	502 (26.7)	53 (41.4)		2367 (47.5)	215 (58.4)	
Junior middle	555 (29.5)	33 (25.8)		1293 (26.0)	82 (22.3)	
Senior middle or above	823 (43.8)	42 (32.8)	0.001	1322 (26.5)	71 (19.3)	<0.001
Smoking history						
Never	551 (29.3)	18 (14.1)		4723 (94.8)	324 (88.0)	
Former	690 (36.7)	60 (46.9)		144 (2.9)	23 (6.3)	
Current	639 (34.0)	50 (39.1)	0.001	115 (2.3)	21 (5.7)	<0.001
Smoking exposure pack-yrs	18.8±24.0	30.4±26.5	<0.001	0.6±4.4	2.1±9.1	<0.001
Physical activity						
Inactive	169 (9.0)	7 (5.5)		480 (9.6)	24 (6.5)	
Minimally active	954 (50.7)	60 (46.9)		2430 (48.8)	180 (48.9)	
Active#	757 (40.3)	61 (47.7)	0.16	2072 (41.6)	164 (44.6)	0.12
Body mass index						
<18.5 kg·m^{-2} underweight	80 (4.3)	16 (12.5)		188 (3.8)	33 (9.0)	
18.5–22.9 kg·m^{-2} normal	711 (37.8)	65 (50.8)		1870 (37.5)	146 (39.7)	
23.0–24.9 kg·m^{-2}	521 (27.7)	27 (21.1)		1255 (25.2)	87 (23.6)	
⩾25.0 kg·m^{-2} overweight	568 (30.2)	20 (15.6)	<0.001	1669 (33.5)	102 (27.7)	<0.001
Waist circumference cm	81.9 (81.4–82.2)	79.3 (77.8–80.8)	0.001	77.5 (77.2–77.7)	76.7 (75.9–77.6)	0.09
Systolic blood pressure mmHg	132.9 (131.9–133.8)	134.8 (131.1–138.5)	0.32	129.1 (128.5–129.7)	128.8 (126.6–131.0)	0.80
Diastolic blood pressure mmHg	76.3 (75.8–76.8)	76.2 (74.2–78.2)	0.92	72.6 (72.3–72.9)	71.9 (70.8–73.0)	0.23
Fasting glucose mM	5.56 (5.51–5.61)	5.39 (5.19–5.59)	0.12	5.52 (5.49–5.55)	5.44 (5.32–5.56)	0.22
Triglyceride mM	1.38 (1.35–1.41)	1.23 (1.12–1.35)	0.02	1.44 (1.42–1.46)	1.43 (1.35–1.51)	0.84
HDL–cholesterol mM	1.54 (1.52–1.55)	1.58 (1.51–1.64)	0.28	1.74 (1.73–1.75)	1.76 (1.72–1.80)	0.44
Hypertension treatment	503 (26.8)	37 (28.9)	0.60	1354 (27.2)	98 (26.6)	0.82
Diabetes treatment	138 (7.3)	8 (6.3)	0.65	383 (7.7)	29 (7.9)	0.89
Dyslipidaemia treatment	328 (6.6)	28 (7.6)	0.45	119 (6.3)	6 (4.7)	0.46

Data are presented as mean±SD, mean (95% confidence interval) or n (%); geometric means are given for fasting glucose and triglyceride levels. HDL: high-density lipoprotein. #: vigorous activity on ⩾3 days·week^{-1} achieving ⩾1,500 metabolic-equivalent-min·week^{-1} or activity on 7 days·week^{-1} achieving ⩾3,000 metabolic-equivalent-min·week^{-1}.

TABLE 2 Relationship between airflow obstruction and metabolic syndrome or its components (Guangzhou Biobank Cohort Study, 2003–2006)

	Metabolic syndrome	Central obesity[#]	Raised blood pressure[¶]	Raised fasting glucose[+]	Raised triglyceride[§]	Reduced HDL–cholesterol[f]
Normal lung function[##] n (%)	1361 (19.8)	2268 (33.1)	3664 (53.4)	2531 (36.9)	2292 (33.4)	1139 (16.6)
Airflow obstruction n (%)	112 (22.6)	169 (34.1)	281 (56.7)	170 (34.3)	147 (29.6)	79 (15.9)
Crude OR (95% CI)	1.18 (0.95–1.47)	1.05 (0.86–1.27)	1.14 (0.95–1.37)	0.89 (0.74–1.08)	0.84 (0.69–1.02)	0.95 (0.74–1.22)
Adjusted OR (95% CI)[¶¶]						
Model 1	1.01 (0.81–1.27)	0.89 (0.73–1.09)	0.98 (0.81–1.18)	0.80 (0.66–0.97)	0.82 (0.67–1.00)	0.95 (0.74–1.22)
Model 2	1.47 (1.12–1.92)	1.43 (1.09–1.87)	1.10 (0.91–1.34)	0.87 (0.71–1.06)	0.94 (0.76–1.16)	1.03 (0.80–1.33)
Model 3	–	1.43 (1.09–1.88)	1.10 (0.90–1.34)	0.83 (0.68–1.02)	0.91 (0.74–1.13)	1.03 (0.79–1.34)

HDL: high-density lipoprotein; OR: odds ratio; CI: confidence interval. [#]: waist circumference of ⩾90 cm in males or ⩾80 cm in females; [¶]: ⩾130/85 mmHg or receiving treatment for hypertension; [+]: ⩾5.6 mM or a previous diagnosis of type 2 diabetes; [§]: ⩾1.7 mM; [f]: <1.03 mM in males, <1.29 mM in females or receiving treatment for dyslipidaemia; [##]: referent category; [¶¶]: model 1 was adjusted for age, sex, education, smoking and physical activity, model 2 was additionally adjusted for body mass index, and model 3 included the other four components of metabolic syndrome, as appropriate.

There was no evidence that the relation between airflow obstruction and metabolic syndrome varied with smoking. The presence of airflow obstruction was associated with a higher prevalence of metabolic syndrome in lifelong never-smokers (adjusted OR 1.47; 95% CI 1.09–2.00) and current smokers (adjusted OR 3.46; 1.28–9.36).

The association between airflow obstruction and metabolic syndrome remained when those with self-reported physician-diagnosed asthma or bronchiectasis were included (adjusted OR 1.39; 95% CI 1.08–1.80). Replacing the definition of airflow obstruction with an FEV1/FVC of <0.70 did not alter the pattern of the relationships, although the magnitude of the OR estimate was slightly lower for metabolic syndrome (adjusted OR 1.34; 1.04–1.71) and essentially the same for central obesity (adjusted OR 1.41; 1.10–1.81).

Table 3 shows the relationship between airflow obstruction and metabolic syndrome or its central obesity component, according to the severity of obstruction. There was a significant linear trend of increasing OR for metabolic syndrome with decreasing percentage predicted FEV1 before and after adjustment for confounders, although the association was significant only in those showing severe obstruction (FEV1 <50% pred; adjusted OR 2.34; 95% CI 1.39–3.95). A similar trend was also observed in the association between airflow obstruction and central obesity.

Although the main aim of the present study was to examine the potential relationship between airflow obstruction and metabolic syndrome, the association of the latter with restrictive abnormality was also examined. The prevalence of restrictive abnormality was 13.8% (n=1,178). The OR for metabolic syndrome among this group, compared to those with normal lung function, was 1.87 (95% CI 1.59–2.20), for central obesity was 1.67 (1.40–1.99) and for hypertension was 1.19 (1.03–1.36). There were no significant associations between restrictive abnormality and the other components of metabolic syndrome.

DISCUSSION

The results of the current analysis suggest that a higher prevalence of metabolic syndrome, and, essentially, its central obesity component, is associated with airflow obstruction once BMI has been adjusted for in a sample of older adults in Guangzhou. To the best of our knowledge, the present study sample represents one of the largest studies addressing this relationship.

The finding of an association between airflow obstruction and metabolic syndrome is consistent with the results of a small case–control study [6]. The magnitude of excess risk was lower in the present study, but the previous study was based on COPD patients attending cardiopulmonary rehabilitation, where participants had more severe airflow obstruction, and the results reported were not adjusted for potential confounders.

The present results appear to contradict three recent reports, which suggested that restrictive abnormality but not airflow obstruction was associated with metabolic syndrome [7–9]. Closer scrutiny, however, shows that the population sample in the study of LIN *et al.* [7] was younger, with a mean age of 37 yrs, whereas chronic airflow obstruction is relatively uncommon before the fifth decade of life [17]. It is possible that the prevalence of obstructive lung disease was low in their sample, resulting in insufficient power for detection of any association. Although the univariate and multivariate analyses presented by FIMOGNARI *et al.* [9] suggested that obstructive abnormality was unlikely to be related to metabolic syndrome, confounders such as smoking and physical activity were not controlled for. Despite having a large sample size (n=2,396), with a similar prevalence of obstruction (7.6%) to that in the present study, NAKAJIMA *et al.* [8] detected no association between obstructive abnormality and metabolic syndrome. When fitting the present data in a similar model to that described by NAKAJIMA *et al.* [8], which included sex, age, height and smoking status (yes/no) but not BMI, the OR for metabolic syndrome was 1.05 (95% CI 0.84–1.31), which is more comparable to their finding (OR 0.88; 95% CI 0.57–1.37). It may be that adjustment for BMI might have altered their results. However, in agreement with these studies, an association between restrictive abnormality and metabolic syndrome was also observed. The adjusted OR (1.87) was similar to that reported in the Japanese study, using similar LLN criteria and

TABLE 3 Relationship between airflow obstruction and metabolic syndrome or its central obesity component by severity of airflow obstruction (Guangzhou Biobank Cohort Study, 2003–2006)

	Normal lung function	Airflow obstruction			
		Mild[#]	Moderate[¶]	Severe[+]	p-value[§]
Subjects n	6862	127	250	119	
Metabolic syndrome					
Subjects n (%)	1361 (19.8)	24 (18.9)	55 (22.0)	33 (27.7)	
Crude OR (95% CI)	1.00	0.94 (0.60–1.47)	1.14 (0.84–1.55)	1.55 (1.03–2.33)	0.05
Adjusted OR (95% CI)[f]	1.00	1.23 (0.72–2.10)	1.30 (0.90–1.87)	2.34 (1.39–3.95)	0.001
Central obesity					
Subjects n (%)	2268 (33.1)	35 (27.6)	90 (36.0)	44 (37.0)	
Crude OR (95% CI)	1.00	0.77 (0.52–1.14)	1.14 (0.88–1.48)	1.19 (0.82–1.73)	0.32
Adjusted OR (95% CI)[f]	1.00	0.94 (0.55–1.61)	1.50 (1.04–2.16)	2.03 (1.18–3.48)	0.002

OR: odds ratio; CI: confidence interval. [#]: forced expiratory volume in 1 s (FEV_1) $\geqslant$80% of the predicted value; [¶]: FEV_1 $\geqslant$50 and $<$80% pred; [+]: FEV_1 $<$50% pred; [§]: for linear trend; [f]: adjusted for age, sex, education, smoking, physical activity and body mass index.

IDF definition (1.75) [8], but less comparable with the Taiwanese (1.33) [7] and the Italian study (3.23) [9], where different definitions of restrictive abnormality (based on a fixed ratio) and metabolic syndrome were employed.

The currently available evidence has suggested that obesity (BMI of $\geqslant 30$ kg·m^{-2}) is more commonly found in patients with COPD than in the general population [18]. In the present analysis, it was found that, among the components of metabolic syndrome, central obesity was the single main factor linked with airflow obstruction. This resonates with reports of previous studies in various settings that suggested an inverse association between lung function parameters (FEV_1 and FVC) and central obesity (as measured by waist circumference or waist/hip ratio) [19–25]. However, the relationship between FEV_1/FVC ratio (as a continuous variable) and central obesity is unclear, with some [20–22], but not other [23–25], studies reporting such an association. Until recently, no previous study had demonstrated a link between the presence of airflow obstruction and central obesity. Similar to the present findings, a recent cross-sectional study from France also demonstrated that the central obesity component of metabolic syndrome was significantly associated with both obstructive and restrictive lung function impairment [26]. It is important to note that the finding that central obesity is more common in those who showed airflow obstruction than in those with normal lung function initially appears to be contradictory to the observation of a lower mean waist circumference in the former group. However, it is explained by the fact that underweight (BMI of $<$18.5 kg·m^{-2}) was more prevalent among those with airflow obstruction than those without. Leaving aside the underweight group, for any given BMI, central obesity (waist circumference of [23–25] 90 cm in males or $\geqslant$80 cm in females) was more common in those with airflow obstruction than in those with normal lung function (data not shown).

Conversely, no associations were found between airflow obstruction and other components of metabolic syndrome. Similar results were reported in the case–control study of MARQUIS *et al.* [6], except that they also found that high blood pressure was more common in COPD than in controls. The present results are in contrast with the findings of a recent analysis of the combined data from two large cohort studies (n=20,296), which showed that COPD was associated with a higher risk of diabetes (OR 1.4) and hypertension (OR 1.4). The risks were higher in those with more advanced (GOLD stage 3 and 4) COPD (OR 1.5 and 1.6, respectively) [27].

Smoking, a major risk factor for COPD, has been attributed as being one of the main causes of increased systemic inflammation [5], possibly explaining any link between COPD and other chronic conditions, such as metabolic syndrome. Nevertheless, the present results show that the association between metabolic syndrome and airflow obstruction is also found in never-smokers. It is possible that risk factors for airflow obstruction, such as passive smoking [13], may be driving the inflammatory response. In the present never-smoking subgroup, airflow obstruction was associated with metabolic syndrome in those who had been exposed to passive smoking or occupational dust but not in those without such exposures (data not shown). The inflammatory response in metabolic syndrome is particularly related to central obesity, as demonstrated by elevated levels of C-reactive protein in those with a greater waist circumference [28]. Nevertheless, it remains to be clarified how systemic inflammation associated with COPD and that associated with central obesity (and hence metabolic syndrome) might interact.

There are several potential limitations that deserve mention. Although recruited from an association whose membership is open to anyone for a low fee that is affordable by most, the present participants are unlikely to be totally representative of the older population in China. Nevertheless, bias would only be introduced if those who exhibited a specific relation between lung function status and metabolic profile, *e.g.* those who had normal lung function and metabolic syndrome, had been systematically missed, which is unlikely. In addition, the prevalences of airflow obstruction [29] and relevant cardiovascular morbid conditions, such as hypertension [30] and diabetes [31], were similar to those in recent surveys using

national samples of Chinese subjects, suggesting that it is less likely that the association has been overestimated. Although it could be inappropriate to extrapolate the present findings to other populations, especially those in the developed world, given the large proportion of never-smokers in the present sample, the findings are relevant and important to China and other developing countries, where the majority of females do not smoke, although the burden of COPD in the female population is growing. As in other large-scale prevalence surveys [29, 32], airflow obstruction was defined based on spirometric measurement. However, the use of the LLN criteria reduced the risk of overdiagnosis in the older sample [33], and a comprehensive and stringent quality check ensured that only those data which were valid and reliable were included. Although the possibility of having misclassified some asthmatic subjects as having chronic airflow obstruction could not be excluded since post-bronchodilator spirometry was not performed, the prevalence of self-reported physician-diagnosed asthma (1.7%) was comparable to that reported in a population survey (1.8% in those aged >45 yrs) [34]. Dyspnoea, which is a feature of chronic lung disease, could limit the level of physical activity and potentially confound the association between central obesity and airflow obstruction [35]. However, inclusion of dyspnoea (based on self-report using the UK Medical Research Council respiratory questionnaire) in the regression models did not alter the magnitude of the ORs (1.49 (95% CI 1.13–1.95) for metabolic syndrome and 1.41 [1.07–1.85] for central obesity). We acknowledge that, with a cross-sectional design, temporal relationship and thus causality cannot be established. Nevertheless, our message that metabolic syndrome is associated with airflow obstruction has important clinical implications, which might raise the attention of clinicians to the coexistence of the two conditions, even in the absence of smoking. Future research using a prospective study design is warranted in order to confirm the direction of the observed association and explain the underlying mechanism.

In summary, the present study suggests that the presence of airflow obstruction is related to metabolic syndrome, the risk escalating with increasing severity of obstruction. This association is limited to the central obesity component of metabolic syndrome. This finding may partly explain the elevated risk of cardiovascular disease among those with COPD. As a result of increased life expectancy, the concomitant occurrence of two or more chronic conditions (multimorbidity) is becoming increasingly common [36]. At present, most chronic conditions are managed according to clinical guidelines that were designed to cater for the specific needs of individual diseases, with little reference to multimorbidity [1, 2]. The present findings provide evidence suggesting that future guidelines should evolve and develop a holistic approach to the treatment of chronic diseases in order to meet the challenge of an ageing society.

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STATEMENT OF INTEREST

None declared.

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