Wood smoke COPD: a new description of a COPD phenotype?

To the Editor:

We read with interest the article by Camp *et al.* [1] published in the *European Respiratory Journal*. In this study, based on both qualitative and quantitative computed tomography (CT) scan measures, the authors established that women exposed to biomass smoke had less emphysema than women exposed to tobacco smoke with similar obstruction observed by spirometry [1]. In addition, they described the presence of bronchiectasis in 14% of the biomass-exposed women compared to 0% of the tobacco-exposed women, without differences between the groups in the quantitative measures of air trapping or airway thickness on the inspiratory scans [1]. Finally, the authors concluded that this is the first study showing differences in chronic obstructive pulmonary disease (COPD) phenotypes in living women with biomass- *versus* tobacco-smoke exposure [1]. However, there have been previous studies investigating the differences in clinical presentation, pulmonary function tests and CT scan findings between COPD related to wood smoke and tobacco smoke [2–5]. In 2008, a review by Torres-Duque *et al.* [6] of evidence on biomass fuels and respiratory diseases presented the existing information about the similarities and differences between COPD related to cigarette smoking and biomass smoke.

A recent study from Colombia [3] compared the CT scans and functional findings in severe COPD related to wood smoke and tobacco smoke. The airflow obstruction observed by spirometry, the hyperinflation in lung volumes and the increase of the airway resistance were similar in both groups. Based on the CT scans, we demonstrated that, unlike women with smoking-related COPD, those with wood smoke-related COPD and severe obstruction did not have emphysema, but did have significant airway involvement that manifested as peribronchial thickening (75% *versus* 10%, p=0,008) and bronchial dilation (67% *versus* 10%, p=0,024). In addition, they had tree-in-bud pattern (25%) and subsegmental atelectasis (33%), neither of which were found in those with tobacco smoke-related COPD. In concordance with the CT findings, women with wood smoke-related COPD had less decrease in the diffusing capacity for carbon monoxide (*D*LCO) and the *D*LCO/alveolar volume ratio than women with smoking related-COPD and a similar grade of obstruction [3]. This functional finding has been reported in patients with obstruction without emphysema and is probably due to severe bronchial obstruction and incomplete mixing of inspired gas during the determination of single breath *D*LCO. With these results, we postulated that the airflow obstruction in wood smoke COPD is mainly caused by severe airway involvement rather than by a loss of elastic recoil due to emphysema. Similar to our results, a recent study from Brazil [4] showed that the most common findings on CT scans in a wood smoke-exposed COPD group were bronchial wall thickening (66.7%), bronchiectasis (54.8%), mosaic perfusion pattern (45.2%), parenchymal bands, tree-in-bud pattern and laminar atelectasis ($p<0.001$ *versus* the control group for all) and, in contrast, emphysema was uncommon. The authors described a positive association between bronchial wall thickening and hour-years of wood smoke exposure [4].

In another study in women with similar age and baseline airflow obstruction, it was demonstrated that the bronchial hyperresponsiveness level was more severe in wood smoke COPD than in tobacco smoke COPD using the methacholine challenge test ($p<0.028$) [5]. With these findings, it was suggested that the observed greater airway involvement induced by the chronic exposure to wood smoke could explain the differences in bronchial hyperresponsiveness between the two groups [5].

In conclusion, the study from Camp *et al.* [1] complements the existing information about the significant differences in the clinical presentation, pulmonary function test and CT findings between wood smoke- and tobacco smoke-related COPD, reinforcing the fact that wood smoke COPD could configure a different phenotype. The information from these studies [1–5] contributes to a better understanding of COPD and suggests that pathophysiologically distinct diseases, caused by a different irritant that shares airflow obstruction, could be included within the term COPD.

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Wood smoke COPD is included in the term COPD, but different to tobacco smoke COPD: new phenotype or different disease http://ow.ly/tvfaV

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From the authors:

We greatly appreciate the letter from M. González-García and C. Torres-Duque in response to our paper comparing the different phenotypes of chronic obstructive pulmonary disease (COPD) in tobacco smoke-*versus* biomass smoke-induced COPD [1]. The information they provide from publications that we failed to cite strengthens the case for our conclusions and contributes to a better understanding of COPD sub-phenotypes.

A phenotype, according to AGUSTI [2], is the end result of the interaction between the genotype, the environment, and some degree of random variation that facilitates and/or limits these gene–environment interactions. The aim of phenotyping is to identify homogeneous groups of patients who have a different clinical course or who respond to specific therapeutic interventions. In COPD, this is an established strategy used to better understand subjects with the disease; the ''pink puffer'' and the ''blue bloater'' were the best known early phenotypes. AGUSTI [2] suggests that a clinical phenotype should predict at least one clinically relevant outcome that indicates that this would require longitudinal monitoring.

Our findings suggest that COPD associated with biomass exposure is a clinical phenotype with clear differences to COPD associated with tobacco smoking [1, 3]. The very interesting comments by M. González-García and C. Torres-Duque complement the hypothesis that COPD associated with biomass exposure is a phenotype related to airways' obstruction rather than to emphysema. However, despite the considerable cross-sectional evidence [4–7] that biomass smoke causes a different expression of COPD, there is a paucity of data on the clinical implications of this difference; for instance, is this phenotype related to a greater or lower mortality, or an accelerated or slower decline in forced expiratory volume in 1 s? As usual, much work remains to be done to discover the importance of these now well-established phenotypic differences.

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COPD associated with biomass exposure is a clinical phenotype with clear differences to COPD associated with smoking http://ow.ly/tQSJd

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