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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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Exercise and cardiovascular benefit in subjects with COPD: the need for randomised trials

To the Editor:

We read with interest the work on arterial stiffness in patients with chronic obstructive pulmonary disease (COPD) and the potential role of pulmonary rehabilitation by VANFLETEREN *et al.* [1] in the *European Respiratory Journal*. Arterial stiffness, as assessed by aortic pulse wave velocity (PWV), is an independent predictor of cardiovascular outcome and may improve risk stratification in selected patient groups [2]. Furthermore, there is now firm evidence that aortic stiffness is increased in patients with COPD [3, 4], even in those without coexistent diabetes mellitus or overt cardiovascular disease. Increases in arterial stiffness have a number of deleterious nonatherosclerotic consequences including renal, cardiac and further vascular damage.

In addition to age, aortic PWV is affected by structural and functional components as well as the distending pressure: mean arterial blood pressure (MAP). There is accumulating evidence of a role for chronic inflammation [5] in both the functional and structural elements, and interventional anti-inflammatory trials have suggested improved aortic stiffness in other inflammatory conditions. In patients with COPD, several studies have shown associations of aortic stiffness with systemic inflammatory mediators [3, 6] and one determined increased vascular wall inflammation using positron emission tomography/computed tomography fluorodeoxyglucose imaging [7]. Anti-inflammatories may be one way to reduce aortic stiffness in COPD but others also need to be explored.

Numerous therapeutic strategies have been employed to attenuate the increased aortic PWV across many conditions, including pharmacological, nutritional and lifestyle modification, such as optimising exercise. Indeed, interpretation of many trials has proved problematical as many of the interventions, including exercise, have produced significant reductions in MAP [8].

It is against this background that two studies in subjects with COPD have shown significant reductions in aortic PWV with exercise and/or pulmonary rehabilitation [9, 10]. As expected, there were significant reductions in MAP that would largely account for the more modest reduction in aortic stiffness that, in its own right, would confer beneficial cardiological status to subjects. It is therefore very unexpected that the recent study by VANFLETEREN *et al.* [1] failed to produce any decrease in MAP despite a major aerobic component to the training. This may be attributed to the ability of the participants to maintain sufficient exercise intensity or confounders such as medications or timing of assessments. Furthermore, failure to