

caused by undiagnosed cardiovascular disease such as congestive heart failure.

I suspect that their slightly increased utilisation of respiratory care during the 12 months before the year 11 follow-up was due to asthma (diagnosed subsequent to their baseline visit), viral upper respiratory infections, hay fever or cardiovascular disease, and never due to a true COPD exacerbation. Their lung function simply could not have fallen enough during the 11 years (with a mean decline of 440 ml from a mean FEV₁ of 99% predicted) to have caused a COPD exacerbation. As with the 11-year follow-up of the Lung Health Study cohort of adult smokers with an FEV₁/FVC <0.70, fewer than 10% of those with a baseline FEV₁ above 80% predicted had developed a post-bronchodilator FEV₁ below 60% predicted.⁴

Smokers with normal or near-normal FEV₁ should be urged and helped to quit smoking, not given a diagnostic label of "COPD" which risks inappropriate treatments.⁵ Other causes for their chronic cough, phlegm and dyspnoea should be sought.

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Authors' reply

We would like to thank Dr Enright for his comments regarding our article.¹ He suggests that the fixed forced expiratory volume in 1 s/forced vital capacity (FEV₁/FVC) ratio as proposed by the GOLD guidelines largely misclassifies subjects in population studies. Many subjects labelled as mildly obstructive would have normal spirometry and thus had no chronic obstructive pulmonary disease (COPD) if the lower limit of normal (LLN) range for FEV₁/FVC was used instead of the

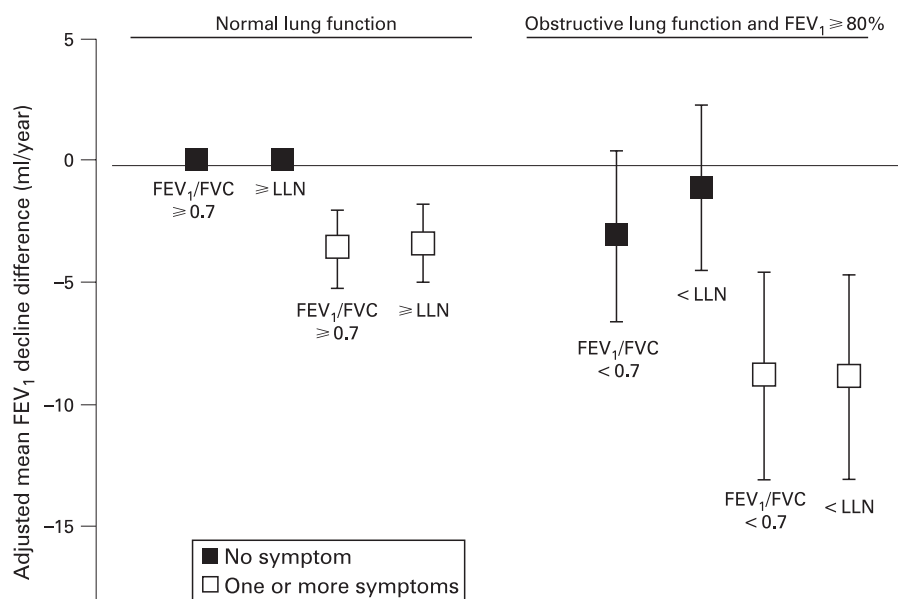


Figure 1 Difference in adjusted* decline in forced expiratory volume in 1 s (FEV₁; ml/year with 95% confidence interval) over 11 years in subjects with normal spirometry or mild obstruction, stratified at SAPALDIA 1 (1991) by modified GOLD[†] and symptom[‡] categories or lower limit of normal (LLN) of FEV₁ to forced vital capacity (FEV₁/FVC) ratio.[†] *Adjusted for age, age squared, gender, baseline FEV₁, smoking status, lifetime smoking (packs/year), baseline body mass index, weight change, education level, nationality and study area (random effect). [†]Pre-bronchodilator spirometry. [‡]One or more symptoms (report of chronic cough or phlegm or shortness of breath while walking). Normal lung function, no symptom = reference category.

fixed ratio (<0.70). This implies that the COPD definition relies only on spirometry. However, the main point of our paper is that respiratory symptoms are key features for defining COPD because their presence predicts long-term functional decline, respiratory care utilisation and quality of life in subjects with mild obstruction. In fig 1 we compare the decline in FEV₁ using the LLN and the GOLD criterion for obstruction. No relevant difference was observed between the two defining criteria. Thus, the use of LLN as a definition of early obstruction would not alter the main conclusion of our study.

The letter also points out that subjects with mild obstruction have symptoms not directly related to COPD but to other illnesses. In our cohort the prevalence of obesity or cardiovascular disease is low in symptomatic subjects with mild obstruction, and subjects with self-reported doctor-diagnosed asthma were excluded from the study at entry. Moreover, in SAPALDIA the occurrence of respiratory symptoms (as defined in our paper) was strongly associated with an accelerated decline in FEV₁ even in subjects with normal FEV₁ (≥80%) at baseline (p<0.0001). This suggests that respiratory symptoms related to COPD may occur even when the FEV₁ is superior to 80%, a finding that is consistent with other studies. For example, in the Copenhagen

City Heart Study, 35% of subjects with mild COPD reported chronic mucus hypersecretion and 22% wheeze with dyspnoea.²

In conclusion, we believe that assessment of respiratory symptoms contributes to identification of a group of patients at risk who may develop disease and poor long-term outcome, and therefore deserve to be closely followed. We fully agree with Dr Enright that smoking cessation is the primary treatment in patients with mild COPD.

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