established the temporality of the disease events, which was otherwise lacking in the study. The overall burden of disease (number, duration and severity of TB relapse) and respiratory morbidity in childhood [2] are other potential confounding factors that were not considered in the study.

Radiologically, healed pulmonary TB can present with cicatrization, fibro-cavitary disease, end-stage lung destruction, pulmonary calcification, bronchiectasis, trachea-bronchial stenosis *etc.* [3]. With such diverse and mixed presentations, combined obstructive and restrictive defects seem highly probable, but were not observed in the study.

In general, asymptomatic lung function defects in patients with past TB do not require any treatment. The study would have been clinically more relevant if the authors had given the percentage of previously treated TB patients with symptomatic lung function defects, particularly airway obstruction, who were likely to utilise medical resources in future.

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Healed pulmonary TB often presents with residual lung function defects which may require further treatment <http://ow.ly/TXfLm>

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From the authors:

We are grateful to Aggarwal and colleagues for their interest in our paper [1]. They are correct to point out the limitations of cross-sectional studies, particularly when they rely on recalled information, and we discussed these at some length in the paper. However, evidence needs to be evaluated in the round. Not all error is biased; the outcomes (lung function) that we used were measured objectively and, as the results were not shared with the participants at the time, are unlikely to have influenced their answers to questions about a past history of tuberculosis. We agree, of course, that objective measures of tuberculosis would have been preferable, but it seems more likely that these would have reduced random error and so strengthened the current findings. We hope to add these to future investigations in the Burden of Obstructive Lung Disease (BOLD) cohort.

We also discussed at some length the problem of inferring causality from cross-sectional studies when the order of events is unclear. In this case, reverse causation (low lung function causing tuberculosis) seems unlikely, but finding that the tuberculosis preceded the lung function decline would have only slightly reduced, and not excluded, the possibility of both being explained by a third, unmeasured, variable. The argument against this rests largely on the strength of the association and the failure to account for it by adjustment for other known factors.

Having raised a concern over recall errors, Aggarwal and colleagues suggest we should have used time to “lung defects” after the occurrence of tuberculosis and “total burden” of disease as exposure variables. In the BOLD study, we do not have this information but it would likely be even less reliable than the information on history of tuberculosis itself.

We did have information on childhood hospitalisation for respiratory disease. We did not report this in the paper, largely because of the strong possibility of recall bias in this variable among those with symptomatic disease. However, adjusting for this variable does not alter the association between lung

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function and a self-reported history of tuberculosis (airflow obstruction: adjusted odds ratio (aOR) 2.46 versus 2.51; spirometric restriction: aOR 2.11 versus 2.13).

We did indeed find, as Aggarwal and colleagues expected, that some participants had both obstructive and restrictive defects. In a sensitivity analysis, we excluded 482 participants who had both a forced expiratory volume in 1 s/forced vital capacity (FVC) ratio below the lower limit of normal (LLN) and FVC<LLN, and showed that the associations were still present between tuberculosis and both obstruction and restriction. This demonstrates that there are independent associations between tuberculosis and these two outcomes.

Finally, Aggarwal and colleagues showed interest in knowing the proportion of participants with a history of tuberculosis and airflow obstruction who had symptoms. Of those with a history of tuberculosis and airflow obstruction, 54% had wheeze and 33% had modified Medical Research Council grade ≥ 2 dyspnoea.

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A history of tuberculosis is an important risk factor for obstructive disease and low lung function <http://ow.ly/TXvgO>

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Number needed to treat: enigmatic results for exacerbations in COPD

To the Editor:

SUISSA [1] provides the event-based number needed to treat (NNT) for a reduction in exacerbations with fluticasone/salmeterol compared with placebo for the TORCH (Towards a Revolution in COPD Health) trial by period of follow-up (0–1 years after treatment, 1–2 years after treatment and 2–3 years after treatment) [2]. These NNT data are misleading as they fail to recognise that in chronic obstructive pulmonary disease (COPD) trials, patients who exacerbate are more likely to withdraw from the trial than patients with no exacerbations. KEENE *et al*. [3] show that for the TRISTAN (TRial of Inhaled STeroids AND long-acting β_2 agonists) trial, the exacerbation rate is more than three per year among placebo patients withdrawing prior to 1 year compared with an exacerbation rate of one per year for patients completing a year of placebo treatment. In the TORCH trial, 25% of the placebo group withdrew compared with 15% of the fluticasone/salmeterol arm during the first year of follow-up; therefore, patients entering the second year of the trial on placebo and on fluticasone/salmeterol were no longer directly comparable. This is not accounted for in the calculation of exacerbation rates or NNT during years 2 and 3, as presented in table 2 of the study by SUISSA [1].