These tests in screening 3, 4 (as opposed to contact with a known index case) in patients receiving haemodialysis, which is important for the management of patients with CKD, particularly before transplantation. 5 Both publications favoured the IGRA tests over the TST in this patient group, but also identified limitations with these tests. There is also one large multicentre study in immunocompromised patients currently underway across Europe, and this includes groups of patients with CKD as well as those with solid organ transplants (Tuberculosis Network European Clinical Trials Group). It is hoped that this study will report next year and will give us definitive data on the relative merits of each of the IGRA tests as well as the TST in this complex group of patients.

Heather Milburn, 1 Neil Ashman, 2 Peter Davies, 3 on behalf of the BTS Guidelines Group for TB in Renal Patients
1Department of Respiratory Medicine, Guy’s and St Thomas’ NHS Foundation Trust, London, UK; 2Department of Respiratory Medicine, Guy’s Hospital, London SE1 9RT, UK; 3Department of Respiratory Medicine, Liverpool Hospitals Trust, Liverpool, UK
Correspondence to Dr Heather Milburn, Chest Clinic, Guy’s Hospital, London SE1 9RT, UK; heather.milburn@gstt.nhs.uk
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Eosinophils best marker of steroid response
There are important aspects of the study design that cast doubt on the claim of Cowan et al that ‘modified responses’ to corticosteroids occur in patients with non-eosinophilic asthma. 5

First, the population recruited was more likely to include patients who experienced loss of control of their asthma after steroid withdrawal than those who remained stable or improved. This increases the potential for regression to the mean as well as identifying a particularly steroid-responsive population. Secondly, it is not possible to make any firm claims about the efficacy of inhaled corticosteroids in either population as the intervention was not placebo controlled. In the only placebo-controlled trial, Berry et al showed no evidence of a response to inhaled corticosteroids in patients with non-eosinophilic asthma.

A more reasonable interpretation of the authors’ findings is that there is a much greater response to re-introduction of inhaled corticosteroids in patients classified as eosinophilic compared with non-eosinophilic. This reinforces the view that the presence of sputum eosinophilia is a strong predictor of steroid responsiveness. The apparent relationship between the fraction of exhaled nitric oxide (FeNO) and improvement in airway responsiveness after re-introduction of inhaled steroids in the non-eosinophilic patients is interesting. One possible explanation is that an increased FeNO is an early marker of returning eosinophilic airway inflammation. The concept that non-eosinophilic asthma can be subclassified into a group that is non-eosinophilic as a result of treatment and a group where eosinophilic inflammation is not a component of the disease is supported by a recent study investigating the presence of eosinophilic proteins in airway macrophages. 6

Neil Martin, 1 Chris E Brightling, 1 Ian D Povard 2
1Institute for Lung Health, Glenfield Hospital, Leicester, UK; 2Glenfield Hospital, Leicester, UK
Correspondence to Neil Martin, Institute for Lung Health, Glenfield Hospital, Groby Road, Leicester LE3 9Qp, UK; nmartin@doctors.org.uk
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Authors’ response
We are grateful to Dr Martin et al for their comments, and accept that our study had...