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CORRESPONDENCE

Authors' response

We thank Dr Connell and colleagues for their interesting letter in response to the 2010 British Thoracic Society guidelines for the management of tuberculosis infection and disease in patients with chronic kidney disease (CKD),[1] and for demonstrating their recent experience with both commercially available interferon-γ release assays (IGRA) and the Mantoux tuberculin skin test (TST) in a group of patients with CKD who had been exposed to tuberculosis. This is a welcome addition to the literature which currently remains sparse in this patient group, particularly in the UK.

We note the disappointingly poor completion of the TST (in only 48%) and subsequent reduction in positive TST responses. We can only assume that the patients, who were initially inpatients at the time of contact, subsequently dispersed to be managed in satellite clinics. In the past we have managed this problem by teaching patients and their carers to read the TST and have followed this up with a telephone call 48 h after administration of the Mantoux test. While not ideal, this has worked well for similar patients who live a considerable distance from a centre (H Milburn, unpublished data 2009).

It is interesting that Connell and colleagues did not find any association of any of the three tests with length of exposure to the index case, as suggested in other studies for the IGRA tests but not the TST.[2] It is possible that larger numbers would be needed to demonstrate such an association. This study also described the performance of the three tests in a contact tracing situation, so the numbers tested have depended on the numbers thought to have had significant contact with a particular index case.

We are only aware of two published studies on the relative use of all three of 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.[1] 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.

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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.[1]

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*[2] 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 (FE_{NO}) 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 FE_{NO} 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.[3]

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Authors' response

We are grateful to Dr Martin *et al* for their comments, and accept that our study had