combination screening with TST or IGRA should be considered. There remains a need for a robust national screening strategy that promotes the detection of latent as well as active tuberculosis.

David Thomas, Miles Jarvis, Alan Williams

Royal Bournemouth and Christchurch Hospitals NHS foundation Trust, Department of Thoracic Medicine, Royal Bournemouth Hospital, Bournemouth, UK

Correspondence to David Thomas, Royal Bournemouth and Christchurch Hospitals NHS foundation Trust, Department of Thoracic Medicine, Royal Bournemouth Hospital, Castle Lane East, Bournemouth BH7 7DW, UK; david.thomas@rbch.nhs.uk

Competing interests None.

Provenance and peer review Not commissioned; not externally peer reviewed.

Accepted 2 December 2010
Published Online First 16 February 2011

Thorax 2011;**66**:1010–1011.
doi:10.1136/thx.2010.152868

REFERENCES

1. **Moore-Gillon J,** Davies PDO, Ormarod LP. Rethinking TB screening: politics, practicalities and the press. *Thorax* 2010;**65**:663–5.
2. **Health Protection Agency**. *Tuberculosis in the UK: annual report on tuberculosis surveillance in the UK 2008*. London: Health Protection Agency Centre for Infections, 2008.
3. **Turner J.** *Reporting of new entrant chest x-rays. Internal audit*. Dorset: Department of Thoracic Medicine Royal Bournemouth Hospital, 2006.
4. **NICE**. *Tuberculosis: clinical diagnosis and management of tuberculosis, and measures for its prevention and control*. London: NIHCE, 2006.
5. **Thomas D,** Jarvis M, Williams A. Do the NICE TB screening guidelines for new entrants under-diagnose cases of latent TB infection? *Thorax* 2010;**65**(Suppl IV): A100.

Authors' response

We welcome the correspondence[1 2] relating to our recent editorial[3] and thank the authors for their interest.

Mr Thomas from the Royal Bournemouth Hospital presents two interesting local audits. The first highlights the possibility of wasted resources in screening individuals identified by the Port Health Control Unit as having an abnormal chest x-ray. The second demonstrates the potential shortcoming in the NICE guidelines, which can lead to an underidentification of latent tuberculosis (TB) infection. We share Mr Thomas's concerns, particularly on this latter point.

Dr Pareek and colleagues from Imperial College and the Health Protection Agency show that the NICE guidance is often not followed and that it is TB services which serve the areas with the highest TB burden which appear least likely to undertake screening for latent TB infection. This latter finding is, we would argue, unsurprising when resources are as scarce as they are in TB services. Those in high burden areas will be concentrating on 'fire fighting'—treating those cases of actual disease which actually do arise, with little or no time left for detecting latent infection and preventing the emergence of new active cases. This is likely to be a funding issue. Even in 2001, only one in six of high TB burden districts met minimum staffing recommendations.[4] Despite the DH funding template produced in 2007, this is widely ignored by primary care trusts as 'not applying to them', continuing the under-resourcing of TB services in many high (and some low) burden districts.

These authors also highlight the deviation from the NICE guidance in the latent TB infection screening methods employed, and we agree that there is a need for effective national coordination. They agree with our arguments regarding the need to change policy and we too feel that there is a need for an expanded evidence base to determine the most effective structure for the future. We also agree on the need for health economic analysis in these cost-constrained times. We understand that this will be provided in the updated economic appraisal of this area, which should be part of the revised NICE TB guidelines in January 2011, and believe it will be fully supportive of our case.

John C Moore-Gillon,[1] Peter D O Davies,[2] L Peter Ormerod[3]

[1]Department of Respiratory Medicine, St Bartholomew's and Royal London Hospitals, London, UK; [2]Department of Respiratory Medicine, Cardiothoracic Centre, Liverpool, UK; [3]Department of Respiratory Medicine, Royal Blackburn Hospital, Lancashire, UK

Correspondence to John C Moore-Gillon, Barts and the London NHS Trust, St Bartholomew's Hospital, London EC1A 7BE, UK; john.mooregillon@btinternet.com

Competing interests None.

Provenance and peer review Not commissioned; not externally peer reviewed.

Accepted 2 December 2010
Published Online First 16 February 2011

Thorax 2011;**66**:1011.
doi:10.1136/thx.2010.156968

REFERENCES

1. **Thomas D,** Jarvis M, Williams A. Rethinking TB screening: politics, practicalities and the press. *Thorax* 2011;**66**:1010–11.
2. **Pareek M,** Abubakar I, White PJ, *et al*. UK immigrant screening is inversely related to regional tuberculosis burden. *Thorax* 2011;**66**:1010.
3. **Moore-Gillon J,** Davies PD, Ormerod LP. Rethinking TB screening: politics, practicalities and the press. *Thorax* 2010;**65**:663–5.
4. **Ormerod LP.** Serial surveys of tuberculosis nurse and support staff in England and Wales in 1998 and 2001. Commun Dis Public Health. 2002;**5**:336–7

Sarcoidosis is a Th1/Th17 multisystem disorder: wider implications

Facco *et al* demonstrated elevated levels of T helper 17 (Th17) cells in the peripheral blood and in the bronchoalveolar lavage of patients with active sarcoidosis; increased expression of interleukin 17 (IL-17) and *IL-23R* in lung and lymph node specimens was also noted.[1] These results suggest a role for the IL-23/Th17 inflammatory axis in the pathogenesis of sarcoidosis.

Crohn's disease (CD) is a severe inflammatory bowel disease (IBD). Many components of the IL-23 pathway (*IL23R*, *IL12B*, *STAT3*, *JAK2*, *TYK2*) are true IBD susceptibility genes, suggesting a crucial role for this pathway in maintaining intestinal immune homeostasis.[2]

We recently reported the development of multisystem sarcoidosis in two CD patients who had received maintenance therapy with natalizumab, a selective adhesion molecule inhibitor that prevents lymphocyte migration to the gut.[3] We hypothesised that natalizumab may have contributed to the development of the disease by allowing dysregulated lymphocyte trafficking to the respiratory mucosa and other extraintestinal mucosal surfaces in genetically predisposed individuals.

Complex disease genetics has been revolutionised by the advent of genome-wide association (GWA) studies. Shared susceptibility genes between IBD and other immune mediated/inflammatory disorders (ankylosing spondylitis, psoriasis, systemic lupus erythematosus, rheumatoid arthritis, asthma, atopic dermatitis, coeliac disease, multiple sclerosis, type 1 and type 2 diabetes mellitus, mycobacterial disease) have already emerged, paralleling the reported epidemiological evidence.[4] A combined analysis of a limited (100 kb) GWA study in CD and sarcoidosis identified a common susceptibility locus on 10p12.2 (but not at GWA levels of significance).[5]

We feel that this recent publication provides further intriguing evidence of common immunopathogenic pathways between CD and sarcoidosis. Ongoing research into common pathways and susceptibility regions between these two granulomatous conditions is essential, with further exploration of the IL-23/Th17 axis looking increasingly like a good starting point.

Acknowledgements Professor Jack Satsangi for his helpful input.

C A Parisinos

Gastrointestinal Unit, Western General Hospital, Edinburgh, UK

Correspondence to Constantinos A Parisinos, Gastrointestinal Unit, Molecular Medicine Centre, University of Edinburgh, Western General Hospital, Crewe Road, Edinburgh EH4 2XU, UK; c.a.parisinos@doctors.org.uk

Competing interests None.

Provenance and peer review Not commissioned; internally peer reviewed.

Accepted 26 April 2011
Published Online First 8 June 2011

Thorax 2011;**66**:1011–1012.
doi:10.1136/thoraxjnl-2011-200290