Some of the diseases associated with C pneumoniae

	Coronary artery disease	*Asthma*	*Rheumatoid arthritis*	*Sarcoidosis*	*Alzheimer dementia*
Main age(s) of onset	Rises steeply from 35 years onwards	Early (childhood) and late onset types recognised	30–40 years	20–30 years	Above 50 years
UK prevalence	3–4% (40–49 years) 6–7% (60–64 years)	5%; 15% in second decade	1–2%; 3% in women over 65 years	19/100 000	Exponetial increase with age
Male:female prevalence	5.5:1 (35–44 years). Ratio reduces with increasing age	Equal	1:3	Men<women	Men<women
Course	Variable but increased risk of mortality	Childhood asthma can improve in teens but frequently returns. Adult asthma can improve with age	Variable. Mortality not increased	Variable	Progressive
Geography/race	Common in countries where fat consumption is high	Uncommon in Far East and developing countries	Global although it can be uncommon in black Africans	All racial groups but 10 times more common in Afro-Caribbeans than whites	
Change in prevalence in recent times	Reducing in most developed countries	Increasing in industrialised countries	A "modern" disease? Little archaeological evidence before 15th century	No change	

Authors' response

Wong and Ward concur with the major points we made in highlighting the efforts that are required to unravel the role of *Chlamydia pneumoniae* in atherosclerosis. Their comments, however, go beyond this and focus on a possible association of *C pneumoniae* with some other diseases. Despite the occasional voice of dissent, there is now overwhelming evidence of an association between *C pneumoniae* and atherosclerosis. What this means is quite unclear and is, of course, the overriding question that needs to be resolved. Many would think an association of *C pneumoniae* with asthma[1] and to a lesser extent with sarcoidosis[1] is a reasonable proposition. However, as the microorganism is to be found in peripheral blood mononuclear cells of a substantial proportion of individuals with and without cardiovascular disease,[2] it is possible for it to lodge just about anywhere in the body and all sorts of relations with disease might be imagined, many perhaps turning out to be spurious. Indeed, is it reasonable to list rheumatoid arthritis and Alzheimer dementia in the same context as atherosclerosis? We think not. There is some evidence, which certainly needs to be substantiated, for an association between *C pneumoniae* and the HLA-B27 positive spondyloarthropathy subgroup of juvenile chronic arthritis,[3] but we are aware of only a single report of *C pneumoniae* in one adult with rheumatoid arthritis.[4] Furthermore, while there is a report of this microorganism being found in brain tissue of patients with Alzheimer disease,[5] is a single, unconfirmed report sufficient to talk in terms of an association that means anything? While it is feasible, it would, nevertheless, be startling if future research showed that this single microorganism was responsible for so many diverse diseases.

DAVID TAYLOR-ROBINSON
BRENDA J THOMAS
Genitourinary Medicine Unit, Division of Medicine (A), Imperial College School of Medicine, St Mary's Hospital, London W2 1NY, UK

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PCR to detect *M tuberculosis*

Although aspirates from solitary pulmonary nodules were not included among specimens used to assess the rapid polymerase chain reaction (PCR) technique,[1] this technique shows promise in improving the diagnostic rate for *M tuberculosis* in solitary pulmonary nodules of < 4 cm diameter.[2] Seven of eight asirates from patients with other evidence of tuberculosis (including five with bacteriological proof, and three with significant response to a therapeutic trial of chemotherapy) tested positive by PCR, and one of three not validated by either technique also tested positive by PCR.[2] The sensitivity of PCR for aspirates from solitary pulmonary nodules is therefore impressively high, with virtually no false positives. The clinical decision process for malignant solitary pulmonary nodules which at present generates a positive predictive value of the order of a mere 50%, thereby yielding an unacceptably high resection rate for benign (including tuberculous) lesions,[3] would be greatly enhanced by a more enthusiastic use of the PCR technique.

O M P JOLOBE
Department of Medicine for the Elderly, Tameside General Hospital, Fountain St, Ashton under Lyne OL6 9RW, UK

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Notice

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Further details from: Dr Jana Hercogova, Charles University 2nd Medical School, Department of Dermatology, V úvalu 84, 150 18 Prague 5, Czech Republic; tel +4202 2443 8700; fax +4202 2443 8720; email: jana.hercogova@lfmotol.cuni.cz

Corrections

In the leader by Dobbins, Kite and Wilcox in the March issue (vol 52, page 169) there is an uncorrected error in the section headed "Acridine orange staining of catheter blood." Line 4 of this section should read: "The technique requires as little as 50 µl of blood..."

In the leader by McMullin in the April issue (vol 52, page 247), a single sentence has been separated unintentionally into two. In the left hand column, three lines from the bottom of the page, the sentence as corrected should read: "As the gene is on the X chromosome and the other X chromosome is "lyonised" in females, leading to inactivation of the lyonised gene, damage to a single gene results in abnormal GPI anchor expression."