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<th>The tobacco industry and scientific publications. Challenges on grounds of self evident potential bias are not unfair.</th>
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value than measurement of the IgG anti-cardiolipin antibody and lupus anticoagulant status, both of which were negative.1 In this setting we questioned the decision to maintain the patient on lifelong warfarin, especially with the increased haemorrhagic risk of maintaining the international normalised ratio in the range 3.0-4.5 in this syndrome.3

Elizabeth M McDermott Clinical research fellow
Maddie Dudridge Senior registrar
Richard J Powell Senior lecturer
Clinical Immunology Unit, Queen’s Medical Centre, University Hospital, Nottingham NG7 2UH


Authors’ reply

EDITOR—There is no evidence to support treatment of patients with antiphospholipid antibodies who have a history of fetal loss, thrombosis, or other features of the antiphospholipid syndrome.1 Two prospective studies have addressed the treatment of antiphospholipid antibodies in pregnancy for women with or without two or more fetal losses. Both indicate that low dose aspirin and prophylactic heparin is the treatment of choice.2,3 There is no good evidence for using steroids in pregnant patients with the antiphospholipid syndrome. Indeed, the inappropriate use of steroids in pregnancy has been shown to worsen outcomes.4 We agree with Robert Llewellyn about the importance of adequate contraception and prenatal counselling in patients with systemic lupus erythematosus with or without an antiphospholipid syndrome. We provide routine contraceptive advice and counsel our obstetric colleagues, hold a joint prenatal clinic for all patients with systemic lupus erythematosus.

This patient underwent an urgent renal biopsy on referral to this centre, and we agree with Elizabeth M McDermott and colleagues about the usefulness of an early renal biopsy in the management of suspected lupus nephritis. There is no evidence that prednisolone plus intravenous pulse cyclophosphamide is superior to oral cyclophosphamide: the only controlled study showed no significant difference in renal survival.5 We believe that two to three months of daily oral cyclophosphamide at a dose of 1-5-2 mg/kg followed by daily oral azathioprine causes less gonadal toxicity and is as effective as intermittent pulse cyclophosphamide for two years. We are planning a multicentre randomised controlled study to compare the efficacy and toxicity of these regimens.

Despite having proteinuria the patient had a normal serum albumin concentration. We recognise that venous thrombosis is a complication of the nephritic syndrome. The duration of anticoagulation treatment is determined by the underlying disease and risk of recurrent thromboembolism. In a patient with a prothrombotic tendency (probable antiphospholipid syndrome) with a potentially fatal ileoceleal thrombosis (her third), five months after warfarin treatment was stopped, we believe that most doctors would accept the risk-benefit ratio of long term anticoagulation.

P Cockwell (Clinical research fellow in medicine (nephrology)) D Adju Consultant nephrologist C Gordon Senior lectures in rheumatology C O S Savage Senior lecturer in medicine (nephrology) Queen Elizabeth Hospital, Birmingham B15 2TH

3 Ral, Cohn S, Ders M, Regan L. Randomised controlled trial of aspirin and aspirin plus heparin in pregnant women with recurrent miscarriage associated with antiphospholipid antibodies (or antiphospholipid antibodies). BMJ 1997;314:557-70 (23 January)

The tobacco industry and scientific publications

Challenges on grounds of self evident potential bias are not unfair

EDITOR—Peter N Lee comments about the concern, expressed by George Davey Smith and Andrew N Phillips, that Lee’s vested interest in tobacco industry revenue to P N Lee Statistics and Computing Ltd might influence his interpretation of epidemiological evidence.6 But what is unfair about challenges on the grounds of self evident potential bias? BMJ journals now require a clear statement from authors on conflict of interest.

Nevertheless Lee has been given the privilege of reply, but he asserts only that he is widely consulted on many issues. Granted, but may we now see an audited statement on the proportion of P N Lee Ltd’s gross income from the tobacco industry during the past five years? Lee is the author of Environmental Tobacco Smoke and Mortality.7 In his conclusion to the preface of this book he states that “There is no convincing evidence that exposure to ETS [environmental tobacco smoke] results in an increased risk of death from cancer, heart disease or any other disease in non-smokers.” Would Lee now clarify in what way the tobacco industry supported the publication of his monograph and how much he received?

The problem for Lee and others who depend on revenue from the tobacco industry for a large proportion of their consultants’ income is that the industry is clearly determined to corrupt the medical and scientific literature on tobacco and health through funneling academics, conferences, publications, and delegates’ attendance at events supported by the industry in attractive venues. New initiatives include the establishment of academic posts in prestigious institutions worldwide, and especially in regions that are now prime targets for market expansion. The industry’s apparently limitless largesse is particularly noticeable in the Asia Pacific, where it is now trying to recruit health professionals as its advocates. P N Lee Ltd and others that take the industry’s commissions will have to find more novel reasons why we should not regard them as its servants and treat their outputs with circumspection.

A J Hedley Professor Department of Community Medicine, University of Hong Kong, Hong Kong

1 Lee PN. Many claims about passive smoking are simply unjustified. BMJ 1997;314:510-12 (23 January)
2 Davey Smith G, Phillips AN. Passive smoking and health: should we believe Philip Morris’s ‘experts’? BMJ 1996;313:929-33 (12 October)

Findings of scientists who were and were not funded by tobacco industry were strikingly different

EDITOR—It is hard to decide which part of Peter N Lee’s letter is the most objectionable, but it is worth commenting on three points for the sake of truth.8 Firstly, Lee whines that George Davey Smith and Andrew N Phillips mention that he receives tobacco industry funding. He implies that they insinuate that this financial support distorts his scientific veracity. But what is wrong with noting the truth about the source of his funding? I suspect that the real problem is that Lee’s long-term association with the industry, which for decades has done everything it can to obfuscate the truth, may have had its effect—perhaps subconsciously—on him.

Secondly, to support one of his arguments he cites as prime evidence a report funded by Philip Morris USA.9 He fails to mention this financial link, incestuous as it is in the context of his letter, despite his presumed search for truth. Furthermore, he does not note how the authors of that report, LeVois and Layard, obtained the data they used. For this information we need to turn to scientists who do not receive tobacco industry funding. They say it all began several years ago the tobacco industry’s lawyers obtained the American Cancer Society’s CPS [cancer prevention study] data set, ostensibly to help in preparation of the defence of the wrongfull death suit against a tobacco company. The industry’s lawyers subsequently provided this data set to two consultants, LeVois and Layard, who conducted an analysis of these data, which concluded that passive smoking did not affect the rise of heart disease.”

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