

that of Dr Toon and colleagues, show a much higher isolation rate of organisms (table). We found no difference in contraceptive habit or smoking habit between these groups. We agree that many of these isolates can be regarded as normal lower genital tract commensals, but we regard *Chlamydia trachomatis*, *Trichomonas vaginalis*, and gardnerella as pathogenic organisms.

Recent data from Dundee¹ and Gateshead² indicate that many women with mildly atypical findings on cervical cytology have cervical intraepithelial neoplasia requiring treatment. Nevertheless, colposcopic facilities are not infinite and it should be remembered that some women with inflammatory smears will have a microbiological infection, which may respond to simple antimicrobial therapy without the need for colposcopic examination.

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1 Walker EM, Dodgson J, Duncan ID. Does mild atypia on a cervical smear warrant further investigation? *Lancet* 1986; ii:672-3.

2 Souttar WP, Wisdom S, Brough AK, Monaghan JM. Should patients with mild atypia in a cervical smear be referred for colposcopy? *Br J Obstet Gynaecol* 1986;93:70-4.

Childhood respiratory infection and adult chronic bronchitis in England and Wales

SIR,—Professor D J P Barker and Dr C Osmond (15 November, p 1271) used the method of Osmond and Gardner¹ to analyse deaths from lung cancer and chronic bronchitis in England and Wales during 1941-80. For both diseases they found a similar period of birth (cohort) pattern with a peak around 1900-5 for men and 1925 for women: they attributed this to the different smoking habits of successive generations. They found little evidence of a period of death effect for lung cancer but a sharp decline in both sexes for chronic bronchitis, which they attributed partly to the advent of antibiotics and partly to the decline in infant bronchitis and pneumonia in the early decades of this century.

These conclusions must be treated with extreme caution. One reason is that the estimated period of birth patterns are impossible to reconcile properly with data on smoking habits, particularly for women. Consider the smoking habits of two cohorts of women, born in 1905 and 1945, with a similar estimated period of birth value. As shown in the table, the cumulative cigarette consumption of these cohorts at any given age is vastly different. While epidemiological studies can be interpreted in terms of some benefit from tar reduction² it is difficult to see how this could explain this large anomaly. Consideration of the detailed data suggests that the declining cohort trends in risk of lung cancer and chronic bronchitis, seen in both sexes, must, at least in part, have been due to a decline in the effect of some risk factor other than smoking, quite possibly air pollution.

Cumulative cigarette consumption (thousands per adult) in two cohorts of women

Age:	20	30	40	60	80
Born 1905	0.2	2	12	43	63
Born 1954	8	36	65		

Estimated from Lee³ with annual supplements.

A second problem lies in relating the declining period of death effect for chronic bronchitis to childhood respiratory infection. If childhood infection does have an important role it should emerge from the Osmond and Gardner analysis as a birth cohort effect, not as a period of death effect.

Arguably, the Osmond and Gardner technique is useful for discriminating those diseases which are mainly characterised by birth cohort changes and those which are mainly characterised by period of death effects. Where both effects emerge from the analysis, as for chronic bronchitis, it is important, firstly, to test whether the model fits the data and, secondly, to remind readers that there are a range of other (very different) period and cohort variables that fit the data as well. Barker and Osmond do neither and overinterpret the results of these analyses.

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1 Osmond C, Gardner MJ. Age, period and cohort models applied to cancer mortality rates. *Statistics in Medicine* 1982;1:245-59.

2 Lee PN, Garfinkel L. Mortality and type of cigarette smoked. *J Epidemiol Community Health* 1981;35:16-22.

3 Lee PN, ed. *Statistics of Smoking in the United Kingdom*. 7th ed. London: Tobacco Research Council Research, 1976.

SIR,—We disagree with Mr Lee but are grateful for the opportunity to expand on a necessarily abbreviated section of our paper.

Following suggestions made by Breslow¹ and by Day and Charnay² we derived a curve which described the cohort (generation) effects of both lung cancer and chronic bronchitis (figure). For lung cancer this one curve was an adequate description of the time trends. For chronic bronchitis, however, it was necessary to add a further curve which we show as a steeply falling period of death effect (figure).

This analysis helped to determine the choice of identification^{2,3} because a submodel was adequate for lung cancer. We returned to a standard presentation of these results in the paper for simplicity. The identifications were chosen to correspond to the earlier analysis by equating gradients. Goodness of fit statistics and parameter estimates are available on request.

Mr Lee chose inappropriate cohorts for comparison. The figure and those in our paper suggest that lung cancer mortality rates for women are 25% greater in the 1945 cohort than in that of 1905. Disease rates in the two cohorts cannot be directly related because there were no overlapping ages

during the period for which data are available (1941-80). There are a number of reasons why we do not think that Mr Lee's cumulative cigarette consumption measure is helpful. For example, it fails to take account of changes in tar consumption and in the use of filters, and it is based on an overly simple model for carcinogenesis. The average number of cigarettes smoked annually by the 1905 cohort of women when aged 35-39 (that is, during 1941-5) was 1360.⁴ The corresponding figure for the 1945 cohort (that is, during 1982) is 2630.⁵ When tar corrected this reduces to 1230. This illustrates one weakness of Mr Lee's analysis.

Turning to bronchitis, our analysis showed that its trend could be decomposed into a lung cancer component, which we related to smoking, and a component that decreased regularly within each age group, which we related to childhood respiratory infection. Such a decreasing trend may be represented equally well by a cohort or period of death curve.³ Thus we find no contradiction in the appearance of a childhood related trend component in the period curve.

For these and other reasons given in our paper we restate our conclusion that the "decline in infant bronchitis and pneumonia in the early decades of this century may have determined the subsequent fall in age specific mortality from bronchitis in adults."

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1 Breslow NE. Extra-Poisson variation in log-linear models. *Appl Stats* 1984;33:38-44.

2 Day NE, Charnay B. Time trends, cohort effects and ageing as influence on cancer incidence. In: Magnus K, ed. *Trends in cancer incidence, causes and practical implications*. New York: Hemisphere Publishing Corporation, 1982.

3 Osmond C, Gardner MJ. Age, period and cohort models applied to cancer mortality rates. *Statistics in Medicine* 1982;1:245-59.

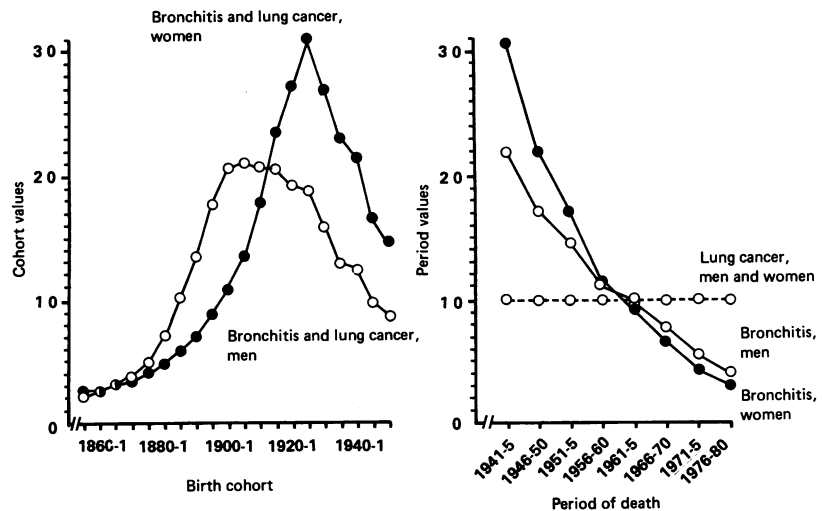
4 Lee PN, ed. *Statistics of smoking in the United Kingdom*. 7th ed. London: Tobacco Research Council, 1976.

5 Office of Population Censuses and Surveys. *General household survey 1982*. London: HMSO, 1984.

Dignity in hospital

SIR,—As a lay reader (albeit a GP's wife) I was particularly interested in the section on "dignity" in Dr Roger Homan's article (29 November, p 1417) as I have just been considering these points after a visit as an outpatient to a general hospital's x ray department.

Dr Homan states that inpatients are conditioned



Mortality trends from bronchitis and lung cancer, men and women, England and Wales 1941-80.