

- ⁴ Greenberg ER, Vessey M, McPherson K, *et al*. Cigarette smoking and cancer of the uterine cervix. *Br J Cancer* 1985; **51**: 139–41.
- ⁵ Howe GR, Westhoff C, Vessey M, *et al*. Effects of age, cigarette smoking and other factors on fertility: findings in a large prospective study. *Br Med J* 1985; **290**: 1697–1700.
- ⁶ Baron JA. Smoking and estrogen-related disease. *Am J Epidemiol* 1984; **119**: 9–22.

SIR—Mant *et al* have expressed concern that the use of hospital controls, “inexact age matching and the loss of subjects from socially deprived areas”, may be responsible for our finding of a negative association between current cigarette smoking and the risk of benign breast disease.¹ Since our results were not altered when age was included as a continuous variable in logistic regression models, we do not understand how differences in the age distribution of the cases and controls could have influenced our results. We are similarly unclear how the loss of subjects from areas designated by the local police department as unsafe for home interviews could have affected our results since there is no reason to believe that the cases who were excluded would be more likely to be current smokers than the controls who were excluded. Furthermore, this exclusion resulted in the loss of less than 2% of the cases and controls.

In our paper, we did express a similar concern to that of Mant *et al* regarding the use of hospital controls. However, we attempted to minimise potential selection biases by examining the influence of a large number of potential confounders, including indices of medical care utilisation and the practice of breast self-examination, but the odds ratios were not materially altered in these analyses. In addition, we reported that the inverse association with current smoking was evident for both inpatients and outpatients as well as when the cases were compared to a subgroup of controls with acute surgical conditions.

Since we found no evidence that former smokers were at a reduced risk of benign breast disease, we believe that it is important to ascertain smoking status at the time of diagnosis. At least in the United States, the proportion of ex-smokers among women aged 20 years and older has virtually doubled between 1965 and 1980.² The proportion of ex-smokers (13%) among the cases in the data presented from the Oxford-FPA study is considerably lower than that obtained among our cases (23%). Since cigarette smoking has been found to alter the risk of several oestrogen-related disorders³ and since benign breast disease is believed to be hormonally related,⁴ the inverse association with current cigarette smoking that we observed appears to be biologically plausible.

However, in light of the difficulty of choosing an appropriate control group for studying benign breast diseases, we welcome further data to substantiate or refute our findings.

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References

- ¹ Berkowitz GS, Canny PF, LiVolsi VA, *et al*. Cigarette smoking and benign breast disease. *J Epidemiol Community Health* 1985; **39**: 308–13.
- ² US Dept HHS/Public Health Service. Health, United States, 1982. Washington, DC: Govt Printing Office, Publ No (PHS) 83–1232, 1982; 83.
- ³ Baron JA. Smoking and estrogen-related disease. *Am J Epidemiol* 1984; **119**: 9–22.
- ⁴ Sitruk-Ware R, Sterkers N, Mauvais-Jarvis P. Benign breast disease. I. Hormonal investigation. *Obstet Gynecol* 1979; **53**: 457–60.

Low tar means less tar

SIR—Dr M A H Russell *et al* (1986; **40**:80–3) conclude, from the measurement of plasma nicotine, plasma cotinine, and COHb%, that despite “compensation”

Letters

smokers of low tar cigarettes have an estimated "intake" of tar some 25% lower than that of smokers of higher yielding brands.

They emphasise, as has been previously reported,¹ that when making interproduct comparisons and extrapolating from one smoke marker to another, it is important to take into account differing ratios of these markers (as measured under standard conditions) for the products being compared.

In a very different study² of demographically representative samples of regular middle tar and regular low tar smokers, I found that estimates of mouth tar intake (derived from 24 hour butt nicotine analyses) indicated a 32% lower intake for the low tar smokers. The results also showed a similar reduction in tar intake (30%) for the middle tar smokers when they were asked to smoke a low tar cigarette. The indirect methodology employed in that report has now been validated through a detailed experimental study (RGR, in preparation).

Russell's study examined the tar intake of heavy smokers (around 30 cigarettes per day) and he was cautious in extrapolating his findings to lighter smokers. However, the low tar smokers in my study² consumed on average 17.5 cigarettes a day, which indicates that consumption is not a critical factor in determining smoking patterns.

Russell's results are thus supported in studies of (a) widely differing design and methodology, (b) both representative and non-representative populations of smokers, and (c) both habitual low tar versus habitual middle tar smoking and acute switching between middle tar and low tar smoking.

The fact that these studies indicate comparable results supports the validity of the overall conclusion that low tar means less tar.

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References

- ¹ Rawbone RG. Low tar smoking versus middle tar smoking. *Br Med J* 1980; (26 July): 309.
- ² Rawbone RG. Switching to low tar cigarettes: are the tar league tables revelant? *Thorax* 1984; **39**: 657-62

Occupation and testicular cancer

SIR—In their paper, McDowall and Balarajan (*Journal of Epidemiology and Community Health*. 1986; **40**, 26-9) were trying to determine whether an observed association between occupation and testicular cancer could highlight aspects of modern

living that may contribute to an increased incidence of the disease. Deriving their data from mortality statistics (ICD:9 186), they were unable to differentiate between the two main histological categories of testicular cancer: seminoma and teratoma. The very different pathologies of seminoma and teratoma and the differences in their epidemiological characteristics probably suggest different aetiological factors. (Seminomas occur more frequently than teratomas, their incidence peaks earlier, and a larger percentage occur in social classes I and II¹).

Although the authors did mention the possible confounding influence of survival time, this was in relation to testicular cancer *en masse*, and they did not indicate what type of cancer patient was surviving. The treatment of seminomas has remained fairly constant over their study period (1971-80), orchidectomy and/or orchidectomy with radiotherapy constituting standard procedure. It is likely therefore that the type of seminoma patient dying has also remained constant. The same is not true, however, for teratoma patients. In 1975, marked progress in cytotoxic chemotherapy, particularly in the use of cisplatin in the treatment of germ cell tumours, increased the percentage of those surviving from malignant teratoma (and seminoma with extralymphatic metastatic disease) from 8% to 90%.² Thus, in the study period the composition of patients will have altered appreciably.

A scrutiny of the death certificates to identify the occupations separately for patients with teratomas and seminomas would be an important next step.

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References

- ¹ Waterhouse JAH. Epidemiology of testicular tumours in management of testicular tumours. 1985; Supp 6, 78; 3-7.
- ² Oliver RID. Rare cancers and specialist centres. *Br Med J* 1986; **292**: 641-2.

Testis cancer

McDowall and Balarajan¹ describe an increasing incidence of testis cancer in young men in England and Wales; they later mention an increased mortality. Their premise for concluding an increased incidence is based on the assumption of rising mortality rates as previously reported.² This parallel between increasing mortality and incidence may have been true when the original paper by Davies was published, but the fact is that mortality from testis cancer in England and Wales is now decreasing. In a paper in this Journal, we