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We may return unduly long letters to the author for shortening so that we can offer readers as wide a selection as possible. We receive so many letters each week that we have to omit some of them. Letters must be signed personally by all their authors. We cannot acknowledge their receipt unless a stamped addressed envelope or an international reply coupon is enclosed.

Correspondents should present their references in the Vancouver style (see examples in these columns). In particular, the names and initials of all authors must be given unless there are more than six, when only the first three should be given, followed by et al; and the first and last page numbers of articles and chapters should be included. Titles of papers are not, however, included in the correspondence section.

Passive smoking and lung cancer

SIR,—Dr Takeshi Hirayama has shown that passive smoking increases one's risk of lung cancer (17 January, p 183). He found this risk to be dose related, and to be about a third to a half that of active smoking. If that is correct then the effect of smoking will be compounded from three sources: one's spouse, one's workmates, and oneself.

Smokers tend to marry each other.¹ The left-hand side of the table below gives smoking habits of a cohort of 222 couples married in Edinburgh in 1972. If we now add one-third of the spouse's intake for each person, the figures change to those shown on the right-

hand side. The number of non-smoking women falls from 150 (67%) to 94 (42%), and the number of heavily smoking women (20 or more a day) rises from 29 (13%) to 37 (17%).

This ignores whether smoking is together at home or separately at work. But smoking is linked to social class, where again like marries like.¹² If one smokes at work, one is likely to work among smokers; and one's spouse and his or her workmates are also likelier to be smokers. Away from work all will tend to congregate in smoking areas of cinemas, trains, etc.

Moreover, if passive smoking carries such a

high risk as Dr Hirayama suggests, then a large element of the risk of active smoking must come from rebreathing one's own fumes in a smoky room. Perhaps, therefore, the mortality from lung cancer could be substantially reduced simply by more powerful ventilation or by smoking out of doors.

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 Sutton GC. Ann Hum Biol 1980;7:449-56.
 Coleman DA. In: Chester R, Peel J, eds. Equalities and inequalities of family life. London: Academic Press, 1977: ch 2.

Number of couples in Edinburgh cohort of 222 with various levels of smoking by husband and wife

| Α | ctive smoking | : | | | |
|-----|--------------------|----------|--------|--------|-----|
| | No of | | Women | | |
| Men | cigarettes/ day | 0 | 1-9 | 10-19 | ≥20 |
| Z | 0 | 88 | 5 | 7 | 7 |
| | 1-9 10-19 | 19 11 | 2 7 | 1 5 | 1 |
| | 20+ | 32 | 6 | 10 | 17 |

| | No of | | Women | | |
|-----|--------------------|----|----------|-------|---------|
| Men | cigarettes/ day | 0 | 1-9 | 10-19 | ≥20 |
| Ž | 0 | 88 | 4 | 0 | 0 |
| | 1-9 10-19 | 6 | 15 | 7 | 7 |
| | 20 + | ŏ | 14 23 | 21 | 3 27 |

SIR,—Dr Takeshi Hirayama's study (17 January, p 183) made extremely interesting reading.

Following the work of Brunnemann¹ and others on the chemistry of tobacco smoke, some observers, including the BMJ^2 and myself,^{3 4} have emphasised the importance of sidestream smoke as a source of environmental carcinogens, and have proposed that passive

smoking could be a hitherto unrecognised cause of cancer in non-smokers. Others have belittled this notion, and even the World Health Organisation concluded recently that passive smoking "is probably free of risk in the sense of producing serious disease.'

With the finding of Miller⁶ that wives of smoking husbands die, on average, four years earlier than wives of non-smokers, the likelihood of a carcinogenic effect of passive smoking became even stronger, and various other suggestive findings have also been reported.7 8 Now, with the major contribution from Dr Hirayama, the evidence becomes less circumstantial, and it seems that a carcinogenic effect can at last be attributed to passive smoking with some certainty.

What degree of exposure is necessary to produce an effect? This question requires urgent attention. It seems likely that the presence or absence of a smoking husband provides only a crude measure of the degree to which a wife passively smokes. In many cases passive smoking outside the home could be more important, particularly with wives who work, or spend much of their time in places of entertainment; and whether there was exposure in childhood (or even in utero9) could also be important. Thus it is possible that some of the control women actually received greater exposure than members of the experimental group. Certainly it seems unlikely that the control group represented a truly "basal" lung cancer rate. As Dr Hirayama notes, in the rural setting passive smoking outside the home is likely to be less than in urban surroundings, and in fact in the younger couples working agriculturally the lung cancer rate in the women with non-smoking husbands was half that of their urban counterparts. Other workers have also found that the lung cancer rate is lower in rural non-smokers than in urban non-smokers.10 From Dr Hirayama's work, though, it would seem that this advantage of rural living can be completely negated, and actually reversed, by living with a smoker, suggesting that passive smoking is a more important component of the so-called "urban factor" in the causation of lung cancer than general atmospheric pollution, at least in non-smokers.

It is to be hoped that further studies will become available that confirm (or refute) Dr Hirayama's findings, and which take into account some of the other sources of exposure noted, so that the risk to non-smokers can be delineated and quantified with increased precision. In the light of Dr Hirayama's study, and other recent demonstrations of the harmfulness of passive smoking (not to mention aesthetic considerations), it will be remarkable if society continues to sanction such a ubiquitous and damaging form of pollution just so that a habituated minority can gratify themselves with a psychostimulant drug whenever they please. It is to be hoped that every effort will now be made by the authorities to minimise the pestilential and incredibly selfish practice of smoking in public. The smoker's right to smoke if he chooses is not denied; but he has no right to make that choice for other people-and that is exactly what he does when he smokes in public, for those around him become involuntary smokers.

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Brunnemann KD, Adams JD, Ho DPS, Hoffman D. In Proceedings of the fourth joint conference on the sensing of environmental pollutants, New Orleans, 1977. American Chemical Society, 1978:876-80.
 Anonymous. Br Med J 1978;ii:453-4.
 Stock SL. New Sci 1980;2 October:10-3.
 Stock SL. Lancet 1980;ii:1082.
 WHO Expert Committee on Smoking Control. Controlling the smoking epidemic. Technical Report Series 636. Geneva: World Health Organisation, 1979:28.
 Miller GH. Toward of Parachina 1979 44.5.5

1979:28.

Miller GH. Journal of Breathing 1978;41:5-9.
Hinds MW, Kolonel LN. Lancet 1980;ii:703.
Lyon JL, Klauber MR, Gardner JW, Smart CR.
N Engl J Med 1976;294:129-33.
Everson RB. Lancet 1980;ii:123-7.
Buell P, Dunn JE. Arch Environ Health 1967;15: 291-7.

Cancer of the cervix and screening

SIR.—The letter from Dr A M Adelstein and others (14 February, p 564) is timely and of importance. I would like to reinforce the conclusions made that the women most at risk from developing cancer of the cervix stay away from screening programmes.

Figures obtained from the Information Services Division of the Scottish Common Services Agency linking Registrar General figures for deaths with the names of those who died enabled us to check the smear records of these women. In the years 1973 to 1978 in the Grampian Region there were 115 deaths from cancer of the cervix. Nine of these women had had a previous smear, but 106 had not. Thus over 90% of the women in the Grampian Region who died from cancer of the cervix had never had a smear.

Surely if screening was mandatory at time of pregnancy the increase in deaths of the young women could be prevented. Perhaps maternity grants should not be paid until the woman can show she has had a satisfactory smear, or the doctor should not be paid his fee until he has taken a satisfactory smear.

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Age of mothers with breast cancer and sex of their children

SIR,—Recently Drs H Olsson and L Brandt (18 October, p 1029) reported a relationship between the age at diagnosis in women with breast cancer and the sex of their offspring. These authors found that women who had given birth to two or more boys were diagnosed with their breast cancer at an average age of 49 years, whereas those who had given birth to only girls had an average age of diagnosis of 61 years. This difference seemed

so marked that we decided to examine it in our own data.

Information has been collected on the sex of the first four children in two studies carried out at the A Maxwell Evans Clinic.12 The data from these two studies were combined, yielding information on 1022 newly diagnosed women with primary breast cancer who had had four or fewer children and for whom the sex of each child was known. Table I shows mean ages at diagnosis by number and sex of children. There is clearly no regular trend in age with the number of boys born for any given total number of children (t=0.13. p = 0.9 for linear contrast); however, the mean age at diagnosis decreases with an increasing total number of children, irrespective of their sex. Study of menopausal and postmenopausal women separately did not show any association with the sex of the children, but showed that the decreasing mean age at diagnosis with increasing parity was restricted to postmenopausal patients.

Thus we cannot reproduce the results of Olsson and Brandt from our series, which is much larger than theirs. Nor are we convinced that our findings of a decreasing age at diagnosis with increasing parity is meaningful. To see if this trend was consistent, we compared out study series with two other groups, all patients seen at our centre in 1976 and in 1977, who were not included in the earlier series (table II). Although there is a tendency for age at diagnosis to be associated with parity, it is irregular. Data based on patients seen at a major clinic are difficult to interpret in this context as the patients seen are selected; for example, the mean age of all patients seen in 1977 was 57.5 years, compared with 60 for all patients registered with breast cancer in British Columbia.

We believe that the findings of Drs Olsson and Brandt may, despite their "statistical significance," represent a chance finding in their small data set. Certainly the association they have reported does not appear in patients in British Columbia.

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¹ Elwood JM, Godolphin W. Br J Cancer (in press).
² Elwood JM, Coldman AJ. Cancer (in press).

Prevention of doxorubicin-induced alonecia

SIR,—The paper by Justine E Anderson and others (7 February, p 423) on the prevention,

TABLE I-Mean age at diagnosis of breast cancer by number and sex of children

| No of children | 0 | 1 | l | | 2 | | | 3 | | | | | 4 | | |
|--|-------------|------------|------------|------------|-------------|------------|------------|------------|------------|------------|------------|------------|------------|------------|------|
| No of boys | 0 | 0 | 1 | 0 | 1 | 2 | 0 | 1 | 2 | 3 | 0 | 1 | 2 | 3 | 4 |
| Mean age at diagnosis No of patients | 56·3 284 | 57·9 93 | 56·3 71 | 56·2 65 | 55·5 134 | 53·7 83 | 54·1 26 | 51·7 71 | 52·7 75 | 56·0 26 | 51·9 11 | 52·9 21 | 50·6 30 | 51·3 28 | 51·5 |

TABLE II—Mean age at diagnosis of breast cancer patients for study group, and patients seen at clinic in years 1976 and 1977

| | Study group | | 1976 | patients | 1977 patients | | |
|---|-------------|-----------|----------|-----------|---------------|-----------|--|
| | Mean age | Sample No | Mean age | Sample No | Mean age | Sample No | |
| 0 | 56⋅3 | 284 | 58.3 | 137 | 57.4 | 132 | |
| 1 | 57.2 | 164 | 58.4 | 101 | 62.9 | 81 | |
| 2 | 55.1 | 282 | 55.5 | 193 | 56.6 | 174 | |
| 3 | 53.0 | 198 | 55-5 | 112 | 57·1 | 104 | |
| 4 | 51.5 | 94 | 57.8 | 71 | 53.4 | 57 | |