Pergamon

European Journal of Cancer Vol. 32A, No. 5, pp. 836–841, 1996

Printed in Great Britain
0959-8049/96 $15.00+0.00

0959-8049(95)00650-8

Original Paper

Risk Factors for Invasive Cervix Cancer in Young Women

J. Cuzick,[1] P. Sasieni[1] and A. Singer[2]

[1]Department of Mathematics, Statistics and Epidemiology, Imperial Cancer Research Fund, London WC2A 3PX, U.K.; and [2]Department of Obstetrics and Gynaecology, Whittington Hospital, London, U.K.

The aim of the study was to evaluate risk factors for invasive cervix cancer in young British women and to look for factors which might explain its increased incidence in younger ages. A case-control study involving 121 women with invasive cervix cancer diagnosed before the age of 40 years, and 241 matched controls was undertaken. Questions were asked about demography, sexual behaviour, reproductive and contraceptive history, smoking, diet and hygiene. Age at first intercourse and lifetime number of sexual partners were found to be the most important factors, whereas parity, educational attainment and social class were not related to risk. Smoking was only weakly related to risk ($P = 0.01$) and this effect disappeared after adjustment for sexual behaviour variables. A weak protective effective of increased fruit consumption was observed ($P = 0.03$), but again this became non-significant after adjustment for sexual behaviour. Only factors related to sexual behaviour were found to be important in this study and other cofactors often associated with invasive cancer in older women were not found to be involved. It is possible that genetic factors influencing viral persistence, and specific more virulent strains of human papillomavirus may be more important in early onset cases. Copyright © 1996 Elsevier Science Ltd

Key words: invasive cervical cancer, young women, case-control study
Eur J Cancer, Vol. 32A, No. 5, pp. 836–841, 1996

INTRODUCTION

ALTHOUGH STILL the commonest form of cancer in the developing world (and second ranking in women world-wide) [1], the incidence of, and mortality from, cervix cancer are generally declining in most developed countries [2, 3]. The most notable exception to this trend is the increase seen in young women in the U.K. over the last 30 years [4]. More recently, smaller increases have been observed in young women in Scandinavia, Eastern Europe, Australia and New Zealand [2, 3]. Furthermore, the decline seen in the United States at all ages may now have reversed in the youngest ages [5]. The increase seen in young British women appears to have stabilised in the past few years [6].

In an effort to determine the reasons for the British increase, we have conducted a case-control study of invasive cancer in women diagnosed under the age of 40 years in Greater London. The main factors studied related to sexual behaviour, reproductive history, methods of contraception and cigarette consumption. These are all well established factors in invasive disease [7], and have also been found to be related to disease in a study of pre-invasive neoplasms (CIN) in young women within the geographic area studied here [8]. We have also attempted to study dietary factors in a subset of these women, particularly the consumption of fresh fruits and vegetables, for which a protective effect has been suggested both directly [9–15] and indirectly from serum measurements of vitamin E, β-carotene and folate [8, 12, 15–18]. Material was not collected to study the role of the human papillomavirus (HPV).

Correspondence to J. Cuzick.
Revised 21 Nov. 1995; accepted 24 Nov. 1995.

PATIENTS AND METHODS

Cases were selected from women with histological confirmed squamous or adenosquamous carcinoma as a first primary, diagnosed before or at the age of 40 years, in a number of North London hospitals during the period July 1985–1991. Fourteen per cent of the cases had micro-invasive disease, 62% were stage Ib and 24% were stage II or worse. (The hospital notes for 13 of the cases were insufficient to determine the stage.)

An attempt was made to interview every eligible case from the participating hospitals, but complete coverage of a defined geographical area was not obtained because some hospitals chose not to participate. In addition, some patients had died or were too ill to be interviewed when identified. All cases

were interviewed within 2 years of diagnosis and most (65%) within 6 months. For each case, we attempted to find two controls from the same GP practice and within 2 years of the case's age (58% within 1 year, 95% within 2 years, all within 4 years). To do this, we selected up to six potential controls. These controls were ordered and the first two were approached. If either could not be contacted or refused, the next potential control was contacted until either two controls had been selected or all six potential controls had been exhausted. In some instances, GPs declined to participate in the study or, very rarely, no controls could be found among the six potential controls. In these situations, controls were selected from another GP practice with similar socio-economic characteristics. Details of control selection are shown in Table 1. Frequently, it was only necessary to write to two women in order to get two controls in suburban areas, but the response rate was not nearly as good for women with an inner city address. In many of these instances, the potential controls must have moved without informing their GPs of their change of address.

Cases were interviewed mostly in hospital. Controls were interviewed mostly at their GPs' surgery, although some were interviewed at home. Interviews took between 30 and 60 min and contained questions about demography, smoking, reproductive and contraceptive history, diet and hygiene. Blood samples for determination of micronutrients were not taken because of the difficulty in interpretation of the results in patients with invasive cancer, many of whom were receiving either cytotoxic agent or radiotherapy.

The study was analysed by conditional logistic regression, which is appropriate for matched study designs. Trend tests were based on the likelihood ratio statistic. In some cases, a second trend test was performed which allowed a test of dose–response only within the group of users. *P* values were based on a χ^2 approximation to these statistics and are two-sided in the sense that they would reject the null hypothesis under either an increasing or decreasing trend. When studying age at first intercourse, the most relevant reference group was the group with ages less than 16 years, which had the highest risk. For comparability with other variables, we report relative protection (the inverse of relative risk) for this group.

Continuous variables were grouped into intervals and coded as integers accordingly. Where sensible, the intervals were defined as in [8]. As a result of the close matching, it was not necessary to adjust for age. In multivariate analyses, adjustment was made for other confounding variables. It proved particularly important to adjust for age at first intercourse and number of sexual partners when considering odds ratios associated with use of contraceptives and smoking.

RESULTS

A total of 121 cases and 241 controls were recruited into the study. Only 4 patients refused to join but several others were too ill to join the study or could not be contacted. The source of controls for these cases is shown in Table 1. Their average age was 33.6 years (cases) and 33.5 years (controls) (NS). Twenty-four per cent of both cases and controls were under 30 years, and 48% were 35 years or older. Educational level attained was not different between cases and controls. Although women with 'A' level qualifications but no degree had the lowest odds ratios (Table 2), there was no indication that women with a degree were at reduced risk.

Factors related to sexual behaviour proved to be the most important for predicting risk (Tables 3 and 4). Both number of partners and age at first intercourse (AFI) were highly significant and remained so after adjustment for the other variables, although in both cases the χ^2 values were substantially reduced by adjustment for the other variable. The strength of each variable was similar. Number of partners did not show a strong trend above three partners. The number of partners in the last 2 years was only a weak predictor of risk (odds ratio (OR) for two or more partners 1.5, 95% CI = 0.76–2.8, data not shown), suggesting that partners at an early age or at least some years before the development of cancer are more relevant to the development of invasive cervical cancer than those in the years immediately prior to diagnosis. Women with a history of genital warts were at increased risk (OR = 3.4, $P = 0.002$), and this remained strong after adjustment for other sexual variables (OR = 2.49, $P = 0.03$) (Table 5).

Reproductive factors were weaker predictors of risk than sexual behaviour. Nulliparous women were at slightly higher risk than parous women ($P = 0.3$), but no further effects of high parity were seen. Among parous women, a late age at first birth was protective (Table 6), although this was not significant after adjustment for sexual behaviour. Curiously, the protective effect of late age at menarche became more significant after adjustment for sexual behaviour (Table 7, $P = 0.02$).

No clear risk associated with use of oral contraceptives was apparent (Table 8). Overall users had a non-significantly lower risk than never users. Women who used oral contraceptives for between 2 and 8 years had a risk that was less than half that of non-users, but among users a weak increasing trend with duration of use was found, so that women who used oral contraceptives for more than 8 years had about the same risk as never users (Table 8).

Use of the diaphragm or condoms was found to be protective and this was particularly apparent when use was long

Table 1. Source of controls

Source and number of controls/case	Number of cases	Average number of women approached per control
Two controls: both from case GP	97	1.8
Two controls: one from case GP, one from another GP	8	2.5
Two controls: both from another GP	13	2.2
Only one control: from case GP	2	4.0
Three controls: all from case GP	1	1.0
Total	121	1.9

Table 2. Education and social class

	Per cent of cases	Per cent of controls	Odds ratio (95% CI)
Highest educational qualification			
None	37	28	1‡
CSE	13	15	0.66 (0.31–1.42)
'O' levels	27	28	0.70 (0.38–1.30)
'A' levels	10	15	0.56 (0.25–1.22)
Degree	13	14	0.73 (0.34–1.57)
Trend			$\chi^2 = 1.69$, $P = 0.2$
Social class†			
I	9	12	1‡
II	31	34	1.2 (0.5–2.7)
III NM	12	15	0.8 (0.3–2.2)
III M	36	24	1.9 (0.8–4.6)
IV	9	12	0.8 (0.3–2.1)
V	3	3	1.0 (0.2–4.9)
Trend			$\chi^2 = 0.40$, $P = 0.7$

†Social class calculated from father's occupation, where available, otherwise the social class of the woman or her partner, whichever was higher, was used. ‡Reference category.

Table 3. Relative protection against cervical cancer according to age at first sexual intercourse (AFI)

Age (years)	Per cent of cases	Per cent of controls	1/odds ratio (95% CI)	Adjusted† 1/odds ratio (95% CI)
≤16	43	26	1‡	1‡
17–18	44	36	1.34 (0.80–2.2)	1.22 (0.70–2.1)
19	7	12	2.65* (1.09–6.4)	1.92 (0.76–4.9)
≥20	6	26	6.59*** (2.8–15.5)	4.51** (1.78–11.4)
Trend			$\chi^2_1 = 26.67$, $P < 0.001$	$\chi^2_1 = 10.7$, $P = 0.001$

†Adjusted for number of sexual partners, using four indicator variables (for 2, 3–5, 6–19 and ≥20 partners).
‡Reference category.
*$0.01 < P \leq 0.05$.
**$0.001 < P \leq 0.01$.
***$P \leq 0.001$.

Table 4. Risk of disease according to number of sexual partners

No. of sexual partners	Per cent of cases	Per cent of controls	Odds ratio (95% CI)	Adjusted† odds ratio (95% CI)
1	7	29	1‡	1‡
2	6	13	2.39 (0.74–7.73)	1.84 (0.56–6.07)
3–5	35	23	8.2*** (3.15–21.32)	6.18***(2.33–16.36)
6–19	42	29	6.62***(2.68–16.35)	4.29** (1.7–10.9)
≥20	10	6	8.17***(2.61–25.62)	4.32* (1.28–14.53)
Trend			$\chi^2_1 = 25.69$, $P < 0.001$	$\chi^2_1 = 9.87$, $P = 0.002$

†Adjusted for age at first sexual intercourse.
‡Reference category.
*$0.01 < P \leq 0.05$.
**$0.001 < P \leq 0.01$.
***$P \leq 0.001$.

term. Women who used barrier methods for more than 8 years had 15% of the risk of women who never used them. Use of the IUD did not affect risk in any meaningful way.

Smoking did not appear to be a strong predictor of risk in this study (Table 9). Current smokers were at twice the risk of never smokers, but the difference disappeared after adjustment for sexual behaviour. No trend with total number of pack-years was observed, although the rates were generally in excess of unity in the higher categories.

Dietary data measuring intake of fresh fruit and vegetables

Table 5. Per cent of women with a history of genital warts prior to diagnosis

Genital warts	Per cent of cases	Per cent of controls	Odds ratio (95% CI)	Adjusted† odds ratio (95% CI)
No	86	96	1‡	1‡
Yes	14	4	3.4** (1.56–7.43)	2.49* (1.07–5.80)

*$0.01 < P \leq 0.05$. **$0.001 < P \leq 0.01$. †Adjusted for number of partners and AFI. ‡Reference category.

Table 6. Risk of disease according to parity and age at first birth

	Per cent of cases	Per cent of controls	Odds ratio (95% CI)	Adjusted† odds ratio (95% CI)
Parity				
0	28	24	1‡	1‡
1	20	22	0.75 (0.38–1.47)	0.68 (0.32–1.44)
2	32	36	0.75 (0.42–1.36)	0.77 (0.37–1.57)
≥3	20	18	0.90 (0.44–1.81)	0.86 (0.35–2.09)
Trend			$\chi^2_1 = 0.17$, $P = 0.68$	$\chi^2_1 = 0.11$, $P = 0.74$
Age at first birth (years)				
Nulliparous	28	24	1‡	1‡
≤19	25	12	1.72 (0.85–3.49)	1.23 (0.50–3.02)
20–24	24	26	0.81 (0.43–1.53)	0.86 (0.39–1.87)
25–29	16	27	0.50 (0.25–1.01)	0.67 (0.31–1.46)
≥30	7	12	0.55 (0.22–1.34)	0.55 (0.21–1.48)
Trend§			$\chi^2_1 = 10.91$, $P = 0.001$	$\chi^2_1 = 2.97$, $P = 0.09$

†See Table 5. ‡Reference category. §For age at first birth after adjusting for nulliparity.

Table 7. Risk of disease according to age at menarche

	Per cent of cases	Per cent of controls	Odds ratio (95% CI)	Adjusted† odds ratio (95% CI)
Age at menarche (years)				
≤12	45	38	1‡	1‡
13–14	42	47	0.70 (0.43–1.15)	0.57 (0.32–1.04)
≥15	13	15	0.68 (0.34–1.37)	0.39 (0.17–0.89)
Trend			$\chi^2_1 = 1.89$, $P = 0.17$	$\chi^2_1 = 5.68$, $P = 0.02$

†See Table 5. ‡Reference category.

were available for 101 matched sets. Fresh fruit was the only variable which showed a significant difference between cases and controls and this disappeared after adjustment for sexual behaviour (Table 10). Blood samples were not obtained, so serum levels of micronutrients could not be assessed.

DISCUSSION

Although our method of case ascertainment was not population based, we have recruited 88% of eligible cases in the participating hospitals. We feel this sample is likely to be representative of all cases in this area. Controls were selected from the same GP practices as cases and were closely matched for age. On average, 1.9 controls were contacted for each control interviewed, which is very satisfactory for such a study in a metropolitan area. Many of the lost potential controls were not at the address recorded by the GP (especially in inner city areas) and failure to reach potential controls is probably the major factor for non-participation. Only very few women refused to participate in this study.

Education and social class (based on father's occupation) were not risk factors in this study. This is in contrast to the strong effect of education found in our study of CIN in this population [8] and many other studies of invasive cancers in older women [19, 20]. Although our cases were matched to controls from the same practice, considerable variability in social class and educational attainment existed in this population, suggesting that this may be less important in young cases with invasive cancer. The reasons for this are not clear, but may indicate that cancers are more related to genetic factors or aspects of sexual behaviour which do not vary across social classes. However, it could also be a cohort effect, but this can only be resolved by studies of these women in future years.

As in most studies of cervix cancer at older ages, age at first intercourse and number of sexual partners were the major factors in determining risk. They exhibited some degree of independence, but χ^2 values were more than halved when each was adjusted for the other making it impossible to determine which of these factors is most important. The fact that the number of partners in the last 2 years was not a factor

Table 8. Risk according to duration of contraception by oral contraceptives, barrier contraception and IUD

	Per cent of cases	Per cent of controls	Odds ratio (95% CI)	Adjusted† odds ratio (95% CI)
Months of oral contraceptive use				
0	11	8	1‡	1‡
1–23	12	15	0.61 (0.23–1.63)	0.72 (0.23–2.22)
24–47	9	15	0.45 (0.17–1.20)	0.31*(0.10–0.96)
48–95	21	34	0.43 (0.18–1.02)	0.35*(0.13–0.98)
96–143	31	20	1.19 (0.50–2.84)	0.87 (0.31–2.41)
≥144	17	8	1.67 (0.62–4.51)	1.08 (0.35–3.34)
Trend (1)			$\chi_1^2 = 3.95$, $P = 0.049$	$\chi_1^2 = 0.56$, $P = 0.45$
Trend (2)§ (users only)			$\chi_1^2 = 9.38$, $P = 0.002$	$\chi_1^2 = 3.49$, $P = 0.06$
Months of barrier contraception				
0	47	32	1‡	1‡
1–23	21	28	0.53* (0.29–0.97)	0.55 (0.27–1.12)
24–47	16	15	0.70 (0.36–1.35)	0.75 (0.36–1.56)
48–95	12	11	0.72 (0.35–1.48)	0.44 (0.19–1.04)
≥96	4	14	0.18**(0.06–0.54)	0.14**(0.04–0.47)
Trend (1)			$\chi_1^2 = 8.42$, $P = 0.005$	$\chi_1^2 = 9.70$, $P = 0.002$
Trend§ (users only)			$\chi_1^2 = 1.21$, $P = 0.27$	$\chi_1^2 = 3.13$, $P = 0.077$
IUD duration				
0	74	70	1‡	1‡
1–23	9	11	0.77 (0.36–1.66)	0.78 (0.31–1.95)
24–47	7	4	1.54 (0.58–4.05)	1.95 (0.65–5.83)
≥48	10	15	0.58 (0.28–1.21)	0.52 (0.22–1.21)
Trend (1)			$\chi_1^2 = 1.17$, $P = 0.28$	$\chi_1^2 = 0.99$, $P = 0.32$

*$0.01 < P \leq 0.05$. **$0.001 < P \leq 0.01$. †See Table 5. ‡Reference category. §Trend tests adjusted for no use of particular method of contraception.

Table 9. Risk according to smoking status and total consumption of cigarettes

	Per cent of cases	Per cent of controls	Odds ratio (95% CI)	Adjusted† odds ratio (95% CI)
Smoking status				
Never	28	42	1‡	1‡
Ex	31	27	1.72 (0.98–3.04)	1.30 (0.67–2.55)
Current	41	31	1.97*(1.16–3.34)	1.23 (0.66–2.29)
Trend			$\chi_1^2 = 6.52$, $P = 0.01$	$\chi_1^2 = 0.34$, $P = 0.56$
Pack-years				
0	33	47	1‡	1‡
0.1–5	19	16	1.65 (0.86–3.17)	1.29 (0.64–2.61)
5.1–10	19	13	2.37*(1.16–4.84)	1.82 (0.81–4.08)
10.1–20	20	19	1.50 (0.78–2.88)	0.89 (0.42–1.90)
≥20	9	5	3.02*(1.18–7.75)	1.72 (0.62–4.74)
Trend (1)			$\chi_1^2 = 5.88$, $P = 0.015$	$\chi_1^2 = 0.37$, $P = 0.54$
Trend (2) (with intercept)			$\chi_1^2 = 0.30$, $P = 0.58$	$\chi_1^2 = 0.02$, $P = 0.90$

†See Table 5. ‡Reference category.

suggests that, even in young women, the majority of invasive cervical cancers require at least 2 years to develop. Unlike some studies in less developed countries [20–23], we did not see a strong effect of parity or age at first birth. In fact, nulliparous women were at marginally higher risk than parous women. This may reflect behavioural changes brought about by widespread use of oral contraceptives at young ages, so that highly sexually active women do not become pregnant. Total duration of oral contraceptives use was not related to risk, but use of a barrier method of contraception was seen to be protective.

The importance of sexual behaviour variables and the striking protection offered by long-term use of barrier methods strongly implicate a sexually transmitted factor as the main determinant of invasive cancer in young women. This factor has been shown to be the human papilloma virus (HPV) in studies of older women and also for CIN 3 in younger women. This is also likely to be the case here, but we were unable to collect appropriate material in both cases and controls to assess this directly.

Smoking was not a strong factor in our study. This is surprising in view of its consistent association with invasive disease in older women and its strong association with CIN 3 in a comparison study in the same geographical area. This may be a chance observation since the confidence intervals still include typical values found in other studies. However,

Table 10. Risk of disease according to consumption of fruit, leafy vegetables and other vegetables

	Per cent of cases	Per cent of controls	Odds ratio (95% CI)	Adjusted† odds ratio (95% CI)
Fruit (pieces per week)	(n = 101)	(n = 201)		
0	15	14	1‡	1‡
1–6	58	45	1.49 (0.74–3.03)	2.18 (0.92–5.17)
7	19	22	0.90 (0.39–2.10)	1.43 (0.50–4.07)
⩾8	8	19	0.44 (0.16–1.19)	0.67 (0.19–2.35)
Trend			$\chi^2_1 = 4.45$, $P = 0.03$	$\chi^2_1 = 0.97$, $P = 0.33$
Leafy vegetables (per week)	(n = 95)	(n = 192)		
⩽2	21	20	1‡	1‡
3–6	51	39	1.21 (0.59–2.49)	1.39 (0.58–3.32)
⩾7	28	41	0.65 (0.30–1.39)	0.59 (0.24–1.48)
Trend			$\chi^2_1 = 2.18$, $P = 0.14$	$\chi^2_1 = 2.55$, $P = 0.11$
Other vegetables (per week)	(n = 93)	(n = 193)		
⩽2	26	19	1‡	1‡
3–6	52	53	0.74 (0.39–1.42)	0.67 (0.31–1.43)
⩾7	22	28	0.64 (0.27–1.48)	0.67 (0.23–1.98)
Trend			$\chi^2_1 = 1.16$, $P = 0.28$	$\chi^2_1 = 0.73$, $P = 0.39$

†See Table 5. ‡Reference category.

La Vecchia and associates [24] found duration of smoking to be the most important factor in their study, and it could be that the latency related to this factor is too long for it to be a factor in young women, and that some other more rapid mechanism is involved here.

We found some weak evidence for a protective effect of fresh fruit, but this disappeared after adjustment for sexual behaviour. More work is needed to clarify the role of diet in cervix cancer. Future studies should also explore the role of HPV in early onset cervix cancer and examine genetic factors.

1. Parkin DM, Pisani P, Ferlay J. Estimates of the worldwide incidence of eighteen major cancers in 1985. *Int J Cancer* 1993, **54**, 594–606.
2. Cuzick J, Boyle P. Trends in cervix cancer mortality. *Cancer Surv* 1988, **7**, 417–439.
3. Beral V, Hermon C, Munoz N. Cervical cancer. *Cancer Surv* 1994, **19/20**, 265–285.
4. Cook GA, Draper GJ. Trends in cervical cancer and carcinoma *in situ* in Great Britain. *Br J Cancer* 1984, **50**, 367–375.
5. Miller BA, Ries LAG, Hankey BF, *et al.* (editors). *SEER Cancer Statistics Review*: 1973–1990. NIH Publ. No. 93-2789, 1993. National Cancer Institute, Bethesda, Maryland, U.S.A., 1993.
6. Sasieni P. Trends in cervical cancer mortality. *Lancet* 1991, **338**, 818–819.
7. Muñoz N, Bosch FH. Epidemiology of cervical cancer. In Muñoz N, Bosch FH, Jensen OM, eds. *Human Papillomavirus and Cervical Cancer*. Lyon, IARC Scientific Publications, 1989, 9–39.
8. Cuzick J, Singer A, DeStavola BL, *et al.* Case-control study of risk factors for cervical intraepithelial neoplasia in young women. *Eur J Cancer* 1990, **26**, 684–690.
9. Romney SL, Palan PR, Duttagupta C, *et al.* Retinoids and the prevention of cervical dysplasia. *Am J Obstet Gynecol* 1981, **141**, 890–894.
10. Marshall JR, Graham S, Byers T, *et al.* Diet and smoking in the epidemiology of cancer of the cervix. *J Natl Cancer Inst* 1983, **70**, 847–851.
11. La Vecchia C, Franceschi S, Decarli A, *et al.* Dietary vitamin A and the risk of invasive cervical cancer. *Int J Cancer* 1984, **34**, 319–322.
12. Brock KE, Berry G, Brinton La, *et al.* Nutrients in diet and plasma and risk of *in situ* cervical cancer. *J Natl Cancer Inst* 1988, **80**, 580–585.
13. Verreault R, Chu J, Mandelseon M, *et al.* A case-control study of diet and invasive cervical cancer. *Int J Cancer* 1989, **43**, 1050–1054.
14. Ziegler RG, Brinton LA, Hamman RF, *et al.* Diet and the risk of invasive cervical cancer among white women in the United States. *Am J Epidemiol* 1990, **132**, 432–445.
15. Van Eenwyk J, Davis FG, Bowen PE. Dietary and serum carotenoids and cervical intraepithelial neoplasia. *Int J Cancer* 1991, **48**, 34–38.
16. Harris RWC, Brinton LA, Cowdell RH, *et al.* Characteristics of women with dysplasia or carcinoma *in situ* of the cervix uteri. *Br J Cancer* 1986, **42**, 359–369.
17. Palan PR, Romney SL, Mikhail M, *et al.* Decreased plasma B-carotene levels in women with uterine cervical dysplasia and cancer. *J Natl Cancer Inst* 1988, **80**, 454–455.
18. Van Eenwyck J, Davis FG, Colman N. Folate, Vitamin C, and cervical intraepithelial neoplasia. *Cancer Epidemiol, Biomarkers Prev* 1992, **1**, 119–124.
19. Stocks P. Cancer of the uterine cervix and social conditions. *Br J Cancer* 1955, **IX**, 487–494.
20. Parrazini F, La Vecchia C, Negri E, *et al.* Reproductive factors and the risk of invasive and intraepithelial cervical neoplasia. *Br J Cancer* 1989, **59**, 805–809.
21. Jussawalla DJ, Deshpande VA, Standfast SJ. Assessment of risk patterns in cancer of the cervix. A comparison between Greater Bombay and western countries. *Int J Cancer* 1971, **7**, 259–268.
22. Brinton LA, Reeves WC, Brenes MM, *et al.* Parity as a risk factor for cervical cancer. *Am J Epidemiol* 1989, **130**, 486–496.
23. Williams MA, Kenya PR, Mati JKG, *et al.* Risk factors for invasive cervical cancer in Kenyan women. *Int J Epidemiol* 1994, **23**, 906–912.
24. La Vecchia C, Franceschi S, Decarli A, *et al.* Sexual factors, venereal diseases and the risk of intraepithelial and invasive cervical neopasia. *Cancer* 1986, **58**, 935–941.

Acknowledgements—We thank our interviewers Judith Young, SRN, and Jane Webster, SRN, for their outstanding job in locating and interviewing participants, the many consultants and GPs who co-operated with the study, and especially the women themselves who gave their time and provided personal details in such a helpful spirit.