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Reproductive variables and risk of uterine cervical cancer in Norwegian registry data

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The influence of reproductive variables on cervical cancer incidence, controlling for other sociodemographic factors, was estimated in Norwegian register and census data, using Poisson regression models. Among the 1.3 million women under observation, a total of 2,870 cases of cervical cancer were diagnosed. According to models restricted to parous women, parity level had no independent impact on cervical cancer incidence, but a clear effect of age at first birth was noted. It was most pronounced in the squamous cell carcinomas, where the incidence was reduced by 48 percent from age at first birth < 21 years to age at first birth 27+ years. Women without children had the same cervical cancer incidence as parous women with a first birth after age 24. The sociodemographic variables controlled for exerted a strong net effect on the cervical cancer incidence. Educational level was related inversely to the cancer risk. Moreover, an increased risk was seen for women who had given birth when they were still single (never married) and for those who were divorced/separated at the time of the last previous census. A fairly small excess risk was found to be associated with living in non-rural compared with rural areas. *Cancer Causes and Control* 1996, 7, 351–357

Key words: Cervical cancer, Norway, reproductive variables.

Introduction

Cervical cancer is the fifth most common neoplasm worldwide and accounts for 11.6 percent of all female malignancies.[1] Marked cross-national differences in incidence rates have been found, and the rates are usually higher in urban than in rural areas.[2] In addition, several studies have revealed that cervical cancer occurs more often among women in the lower socioeconomic groups.[3]

Historically, epidemiologic data has linked sexual behavior to cervical cancer.[4] In the mid-1800s, the disease was found more often in married than in single women. In addition, the absence of cervical cancer among nuns and those who had never been sexually active, suggested that the disease was transmitted sexually.[5] Early onset of sexual activity, multiple sex partners, 'promiscuity' of the partner, bad hygiene, and histories of sexually transmitted diseases have been reported to increase the cervical cancer risk.[3]

More recent research has provided strong evidence for a causal relationship between human papillomavirus (HPV) and cervical cancer. However, a high proportion of HPV-infected women do not develop invasive disease, which indicates that other risk factors – *e.g.*, smoking, oral contraceptive (OC) use, and other sexually transmitted diseases – may operate as cofactors.[6] In addition, case-control studies[7,8] have suggested that high intake of vitamins A, C, and E, and β-carotene is associated with a reduced risk of cervical cancer.

The role played by pregnancy is difficult to assess because of the correlations with important sexual risk factors. In some recent case-control studies,[9,10] where the confounding effects of sexual factors were controlled

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for, a relatively high cancer risk was found among multiparous women. Early age at first birth also has been reported as a risk factor,[11] possibly with an effect that differs significantly between squamous cell carcinomas and adenocarcinomas.[12]

The aim of the present study was to estimate how the total number of livebirths and mother's age at first birth influence the incidence of uterine cervical cancer, controlling for other sociodemographic factors. The analysis is based on Norwegian registry data (the Cancer Registry of Norway linked to demographic life histories derived from the Central Population Register of Norway and the population censuses). Unfortunately, no information on sexual habits was available in these data, but different sociodemographic factors were used as indicators. Nevertheless, due to the extraordinary size of the dataset and the histology-specific models, the study might give important contributions to the exploration of reproductive variables and cervical cancer risk.

Materials and methods

Materials

The study is a follow-up of all Norwegian women (1.3 million) born between 1935 and 1971. Individual reproductive histories were extracted from the Central Population Register of Norway. Nearly all livebirths up to 1991 were included. The few adopted children are linked to their social rather than their biological mothers. Individual sociodemographic characteristics from the censuses of 1960, 1970, and 1980 were included.

Since 1953, the Cancer Registry of Norway has received information on all cancer patients in the population. The reporting system is based on pathology and cytology reports, clinical records, and death certificates, and provides information about site, histologic type, and stage of disease at the time of diagnosis. Registration is based on a modified version of ICD-7.[13] The cancer data were linked to the demographic life histories by means of the personal identification number that every individual living in Norway for some period after 1960 has been assigned.

A total of 2,870 patients with cervical cancer were included in the study population. Altogether 84.1 percent (2,413) of the cases were squamous cell carcinomas and 8.7 percent (249) were adenocarcinomas.

Methods

Poisson regression models were estimated in the Epicure[14] program to assess the effects of reproductive and other factors on the cervical cancer incidence. The results were expressed as relative risks (RR), and their 95 percent confidence intervals (CI) were given. Throughout the analysis, a significance level of 0.05 was used.

The individuals contributed to the population at risk from the age of 20 years, but not before 1960. The latter restriction is introduced because only the less severe cancer cases, from which the patients survived until 1960 and thus received a personal identification number, are included in the linked data for the period 1953-60. The individuals were followed until death, emigration, development of cervical cancer, or until end of follow-up (31 December 1991).

For each month, the following variables (all of which were time-varying) were defined; time period (1960-69, 1970-79, 1980-85, and 1986-91), age, parity, age at first birth, and place of residence. Education and marital status also were included in the analysis.

For age, five-year intervals were used. This appeared to be a sufficient control for age because use of 2.5-year age intervals did not alter the effect estimates.

Parity was defined as the number of livebirths before the month in focus. The categories 0, 1, 2, 3, and 4+ livebirths were used. Age at first birth, only defined for women who were not childless that month, was entered into the model as a four-level variable (< 21, 21-23, 24-26, and 27+).

Educational level referred to was that recorded at the last previous census (0-10 years earlier), and was grouped into three categories: low (seven to nine years of schooling); medium (10-12 years of schooling); and high (13 or more years of schooling).

Marital status also referred to the last previous census. A distinction was made between married, divorced/separated, widowed, and never-married. Among the latter, a subgroup of women who had given birth to at least one child before the last census, was defined as well.

Place of residence referred to the month in focus, and only the categories 'rural' and 'non-rural' were used.

Results

Table 1 describes the RR of uterine cervical cancer (all histologic subgroups pooled), and for the two main histologic subgroups (squamous cell carcinoma and adenocarcinoma), separately. Exposures and cancer cases at parity 0 level were excluded in these calculations, *i.e.*, the follow-up was from entry into motherhood. In the squamous cell carcinomas, there was an increase in incidence from the 1960s until the 1970s. Thereafter, the incidence declined. In the adenocarcinomas, however, there was an increasing trend during the entire time-period 1960-91. The age profile differed somewhat between the two histologic types. In the squamous cell carcinomas, the incidence increased until the age group 40 to 44 years before

Table 1. Relative risk (RR) and 95% confidence interval (CI) of uterine cervical cancer in parous Norwegian women

	Total			Squamous cell carcinoma			Adenocarcinoma		
	RR	(CI)	No.[c]	RR	(CI)	No.[c]	RR	(CI)	No.[c]
Time period									
1960-69[a]	1.00	—	(111)	1.00	—	(104)	1.00	—	(7)
1970-79	1.82	(1.48-2.23)	(857)	1.78	(1.44-2.20)	(789)	1.14	(0.50-2.62)	(36)
1980-85	1.51	(1.22-1.87)	(636)	1.31	(1.05-1.64)	(513)	2.31	(1.02-5.23)	(65)
1986-91	1.48	(1.19-1.83)	(937)	1.27	(1.01-1.59)	(743)	2.52	(1.11-5.71)	(109)
Age									
20-24[a]	1.00	—	(56)	1.00	—	(52)	1.00	—	(2)
25-29	3.18	(2.37-4.27)	(317)	3.10	(2.29-4.22)	(278)	4.47	(1.02-19.54)	(20)
30-34	6.09	(4.52-8.21)	(637)	6.05	(4.43-8.27)	(552)	8.90	(2.03-39.02)	(49)
35-39	7.20	(5.29-9.80)	(652)	7.34	(5.31-10.13)	(565)	9.75	(2.17-43.83)	(52)
40-44	7.49	(5.45-10.28)	(480)	7.54	(5.40-10.53)	(402)	9.33	(2.03-42.79)	(41)
45-49	6.92	(4.95-9.67)	(252)	6.48	(4.54-9.25)	(190)	11.44	(2.45-53.46)	(34)
50-54	7.95	(5.54-11.42)	(130)	7.40	(5.01-10.94)	(97)	12.26	(2.49-60.39)	(17)
55-56	9.25	(5.21-16.41)	(17)	8.83	(4.66-16.74)	(13)	12.86	(1.64-101.0)	(2)
Parity									
1[a]	1.00	—	(510)	1.00	—	(442)	1.00	—	(40)
2	0.86	(0.76-0.96)	(969)	0.85	(0.75-0.97)	(822)	0.77	(0.51-1.16)	(76)
3	1.00	(0.88-1.14)	(695)	0.97	(0.84-1.12)	(574)	1.21	(0.78-1.88)	(73)
4+	1.08	(0.93-1.26)	(367)	1.07	(0.91-1.27)	(311)	1.00	(0.58-1.72)	(28)
Age at first birth									
<21[a]	1.00	—	(1,030)	1.00	—	(887)	1.00	—	(73)
21-23	0.71	(0.65-0.78)	(831)	0.69	(0.62-0.76)	(689)	0.90	(0.65-1.25)	(79)
24-26	0.51	(0.45-0.58)	(384)	0.52	(0.45-0.60)	(331)	0.58	(0.38-0.89)	(34)
27+	0.54	(0.46-0.62)	(296)	0.52	(0.44-0.61)	(242)	0.69	(0.43-1.13)	(31)
Education[b]									
Low[a]	1.00	—	(1,357)	1.00	—	(1,167)	1.00	—	(94)
Medium	0.72	(0.67-0.79)	(1,036)	0.71	(0.65-0.78)	(869)	1.00	(0.75-1.32)	(105)
High	0.56	(0.47-0.67)	(148)	0.51	(0.41-0.62)	(113)	0.82	(0.48-1.41)	(18)
Marital status[b]									
Other never-married[a]	1.00	—	(217)	1.00	—	(193)	1.00	—	(14)
Never-married with child/children	2.28	(1.82-2.85)	(144)	2.34	(1.84-2.97)	(127)	2.10	(0.90-4.94)	(10)
Married	1.17	(0.99-1.39)	(1,884)	1.14	(0.95-1.37)	(1,578)	1.46	(0.76-2.78)	(174)
Divorced/separated	2.30	(1.86-2.83)	(275)	2.34	(1.87-2.93)	(236)	1.66	(0.74-3.74)	(16)
Widowed	1.32	(0.83-2.10)	(21)	1.14	(0.66-1.95)	(15)	2.24	(0.60-8.35)	(3)
Place of residence									
Rural[a]	1.00	—	(1,344)	1.00	—	(1,118)	1.00	—	(123)
Non-rural	1.18	(1.09-1.28)	(1,197)	1.23	(1.13-1.34)	(1,031)	1.01	(0.77-1.32)	(94)

[a] Reference category.
[b] Categorization based on the last previous census.
[c] Number of cases.

levelling off. In the adenocarcinomas, there was a continually increasing trend with age.

No parity effect was seen in either histologic type. However, a clear effect of age at first birth was noted, most pronounced in the squamous cell carcinomas. In this group, the incidence was reduced by 48 percent from age at first birth < 21 to age at first birth 27+.

There was a gradual decrease in incidence with increasing educational level, only significant for squamous cell carcinomas. In this histologic subgroup, the incidence was reduced by 49 percent when comparing the categories of 'low' and 'high' education.

A strong effect of marital status was seen. In particular, the categories 'never-married with child/children' and 'divorced/separated' displayed a high risk of squamous cell carcinomas compared with categories of 'other never-married' or 'married.' Within this histologic subgroup, there was also a 23 percent higher risk for women living in a non-rural area compared with women living in a rural area.

Table 2 is based on models where exposures and cases at parity 0 level are included. It gives the RR of uterine cervical cancer (all histologic groups pooled) according to age at first birth and parity, with the nulliparous as

the reference category. All women with an early age at first birth had a higher incidence than the nulliparous, and most clearly so for the teenage mothers. Parous women with a first birth after age 24 did not have a cervical cancer incidence significantly different from the nulliparous. All parameter estimates were also close to one, with one minor exception. The declining incidence with increasing age at first birth appeared at all parity levels. When leaving out the nulliparous, inclusion of an interaction between parity and age at first birth did not improve the model fit significantly.

We also estimated separate models for the clinical stages I, II, III, and IV (International Federation of Gynecology and Obstetrics). For stage I, a less clearly protective effect of education appeared. Apart from that, no substantial differences between the different stages were found (not shown).

Discussion

This register-based analysis is another contribution to the exploration of reproductive factors as potentially important in the etiology of cervical cancer. In the present analysis, it was estimated that parity level had no independent impact on cervical cancer incidence. When it was included as the only reproductive variable, a significant adverse effect of multiparity showed up, but it disappeared when age at first birth was included in the model. At any parity level, early age at first birth increased the risk of cervical cancer. A few other sociodemographic variables (education, marital status, and place of residence) were included in the analysis, primarily as controls, for lack of more adequate indicators of the sexual and other lifestyle factors that have been incriminated in the etiology of cervical cancer.

Some studies have indicated an increased risk of cervical cancer in multiparous women. Brinton *et al*[9] found a significant linear trend in the risk with increasing number of pregnancies, persistent after adjustment for HPV status. The association with age at first birth in that study disappeared after adjustment for number of pregnancies and other risk factors. Our own estimates are closer to those reported by Kvåle *et al*,[12] who found no significant association between parity and cervical cancer after adjustment for age at first birth. However, these authors concluded that the effect of first birth was confined to squamous cell carcinomas. Other investigators also have found a significant effect of age at first birth.[11,15]

It has been argued that pregnancy *per se* may promote a malignant development in the cervix, or increase the chance of diagnosing a cancer. Cervical trauma during parturition, hormonal and nutritional changes during pregnancy, and high detection rates of HPV among pregnant women are possible biologic explanations for an association with pregnancy.[3] Pregnancy is known to suppress the immune system and to impose a changing hormonal profile on the women, *i.e.*, increasing levels of progesterone and estrogen. Both factors may enhance HPV infections and possibly increase the oncogenic potential of the virus.[16,17] Both an increased prevalence of HPV and a higher replication rate of viral DNA have been demonstrated during pregnancy.[18]

The role of dietary factors in relation to cervical cancer has been of much interest recently. In addition to the protective effect of vitamins (A, C, and E) and β-carotene, folate deficiency has been proposed as a possible risk factor for cervical cancer development.[19] Pregnancy is associated with a depletion of maternal folate stores,[20] which has been suggested as a possible explanation for effects of parity on cervical cancer risk.

Our study, as well as some others, suggests that it is primarily the age at first pregnancy that is important. About 30 years ago, Coppleson and Reid[21] emphasized that women are at highest risk for the initiation of cervical cancer during adolescence and after the first pregnancy and delivery. During these two periods, the cervix is more vulnerable due to the ectocervix being covered by the transformation zone. The columnar epithelium of the transformation zone thus is being exposed to the acidic vaginal secretions. This may increase the susceptibility of the cervix to infectious agents and stimulate a metaplastic process. Possibly, having a first birth in adolescence is particularly harmful because of an extraordinarily vulnerability of the columnar epithelium. Besides, age at first birth might be an indicator of age at first sexual intercourse, and consequently a potential surrogate of age at first HPV infection.

It has been suggested[22] that the two most common cervical malignancies, squamous cell carcinoma and adenocarcinoma, are distinct diseases with a different biology and epidemiologic profile. This analysis displayed a different time and age profile for the two histologic types, confirming prior studies.[23] The reproductive variables, however, seemed to affect the squamous cell carcinomas and the adenocarcinomas similarly. The parity variable seemed to be of no importance in either histologic type, and the effect of age at first birth tended to be similar, although most pronounced in the squamous cell carcinomas. These findings, to some extent, disagree with the study of Kvåle *et al*,[12] who found a statistically significant difference between the two histologic types with regard to age at first birth.

The sociodemographic variables controlled for exerted a strong net effect on the cancer incidence, as also documented in some other studies.[11,24,25] The educational level was related inversely to the cancer risk. An increased risk was seen in particular for women who had given birth when they were still never-married and for those who

Table 2. Relative risk (RR) and 95% confidence interval (CI) of uterine cervical cancer in Norwegian women according to age at first birth and parity; adjusted for other sociodemographic factors[a]

Parity	RR	No.[c]	Age at first birth											
			< 21			21-23			24-26			27+		
			RR	(CI)	No.[c]	RR	(CI)	No.[c]	RR	(CI)	No.[c]	RR	(CI)	No.[c]
0[b]	1.0	(329)												
1			2.83	(2.33-3.44)	(149)	1.84	(1.51-2.23)	(145)	1.09	(0.87-1.38)	(92)	1.06	(0.86-1.31)	(124)
2			2.00	(1.72-2.34)	(356)	1.24	(1.06-1.46)	(320)	0.84	(0.70-1.03)	(160)	1.01	(0.82-1.25)	(133)
3			1.97	(1.67-2.32)	(295)	1.56	(1.32-1.85)	(259)	1.20	(0.95-1.50)	(105)	1.01	(0.71-1.43)	(36)
4+			2.45	(2.05-2.92)	(230)	1.46	(1.17-1.83)	(107)	1.01	(0.68-1.50)	(27)	0.35	(0.11-1.09)	(3)

[a] Also time period, age and education were included in the regression model.
[b] Reference category.
[c] Number of cases.

were divorced/separated at the time of the previous census. Women who were married at that time, or who were never-married and childless, displayed the lowest risk. A fairly small excess risk associated with living in non-rural compared with rural areas was seen as well.

Educational level was considered to be a particularly interesting covariate. It is related strongly to the age at first birth, primarily because of the time conflict between schooling and parenting.[26] However, a low education level is also found to induce an earlier sexual debut in Norway as well as other countries.[27-29] Low education level also is associated with less efficient contraceptive use, which in turn influences the age at first birth as well as cervical cancer incidence. In the present study, an excess incidence for women who had entered motherhood at an early stage was found, even though it was controlled for in the lower educational level in this group.

It is also possible that low education level is associated generally with having multiple sex partners (extending beyond the young adult years), and thus leads to an elevated cancer incidence. However, the evidence for this is quite conflicting. A study of young Americans[30] showed a low number of partners among women with less education. The Norwegian Sexual Behavior Survey[31] revealed a low probability of having sex with others than the spouse or cohabiting partner among those with low education, but also showed that high age at debut (as among the better educated) was associated with fewer partners.

The causal relationships between marital status and reproductive variables are very complex, with changes in one variable being affected by actual as well as expected changes in the other. From the present perspective, the most important issue is that a low age at first birth may be a signal of a premarital birth as well as subsequent divorce.[32] A premarital birth is linked, in turn, to early sexual activity.[33] Nevertheless, the significant effect of age at first birth remained after control for marital status.

These two census-based control variables referred to the situation up to 10 years earlier. Some of the women categorized in the low education group may have reached a higher education level during this period. Similarly, some of the 'never-married' may be married in the month in focus, and some of the 'married' may be divorced. In principle, this leads to a dilution of the adverse effects of low education and divorce.

To summarize, educational level and marital status serve as indirect controls for some of the sexuality variables that are linked to age at first birth. However, we cannot claim that they are adequate controls that leave a pure effect of an early pregnancy itself. Nevertheless, this registry analysis has explored reproductive variables and cervical cancer risk at a population level, and has captured age at first birth as the important factor with regard to reproductive life.

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