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Original Paper

Incidence of and Survival from Upper Aerodigestive Tract Cancers in the U.K.: the Influence of Deprivation

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The aim of the study was to investigate the effect of social deprivation on the incidence of and survival from upper aerodigestive tract (UAT) cancers in the U.K. Incidence was calculated on 25 903 cases of malignant upper aerodigestive tract cancers collected from four cancer registries in the U.K. for the period 1984–1993. A Cox proportional hazard model was used to determine the influence of deprivation, measured in Carstairs quintiles for crude and cause-specific survival on 17 393 of these cases. Patients with UAT cancers who were younger, males or of South Asian origin were more likely to live in a deprived area than in an affluent area. The incidence of UAT cancers in a district was correlated with deprivation score for the district for both men ($r=0.78$) and for women ($r=0.60$). People who lived in deprived areas had a relative risk of 1.25 (95% confidence interval (CI): 1.15–1.35) of dying from their cancer and of 1.24 (95% CI: 1.13–1.35) of dying from all causes compared with people who lived in affluent areas. People living in deprived areas were more likely to get UAT cancer and were more likely to die from their cancer than people living in affluent areas. © 1999 Elsevier Science Ltd. All rights reserved.

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INTRODUCTION

DEPRIVED PEOPLE have a greater incidence of many cancers [1,2] including upper aerodigestive tract (UAT) cancers [3] (cancers of the mouth, larynx, pharynx and associated structures). Tobacco and alcohol are the major risk factors for UAT cancers [4–8], and diet may play a protective role [9–11]. All these factors are associated with social deprivation and are interrelated [12–15].

Deprived populations have increased mortality due to a combination of material, social and behavioural factors [16]. Deprivation has been associated with a higher mortality from some cancers, including oral cancers [17,18]. This increase in mortality is partly due to differences in incidence and partly due to differences in survival [19]. Stage at diagnosis

appears to be the most important factor in contributing to the social class difference in survival [19].

The Carstairs deprivation score is a U.K. based composite measure of deprivation of a residential area which is based on four variables from census information: overcrowding (households with 1 or more people per room), social class, unemployment in men and car ownership [20]. It is a measure of the material deprivation of the population of an area and is particularly closely associated with mortality and morbidity [20].

The aim of this study was to investigate the effect of deprivation on the incidence of and survival from UAT cancers in four cancer registry areas in the U.K.

MATERIALS AND METHODS

In a multiregional collaborative study, data were collected from the Thames, West Midlands, West of Scotland and Yorkshire cancer registries in the U.K. on all cases of UAT cancers (ICD10: C00–C14, C30–C32) diagnosed from 1984 to 1993. This included cancers of the lip, mouth, pharynx,

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larynx and salivary glands. The records were recoded from ICD9 to ICD10. This included all people registered with malignant UAT cancers resident in these regions and 40% of the cases registered in the U.K. during this period.

A Carstairs score and U.K. based Carstairs quintile was allocated to each individual based on their enumeration district of residence, which was found by linking with their postcode. The data were based on the 1991 census. A mean Carstairs score was also calculated for each district health authority. Two ethnic categories of South Asian (Indian, Pakistani and Bangladeshi) and 'other' were determined using surname and place of birth. Tumour, node and metastases (TNM) staging was not available so an 'extent of spread' index was used to categorise cancers into those confined within their organ of origin, those with local spread, those with lymph node involvement and those with distant metastases (Table 1). Cancer registry staff assigned this index from information in clinical and pathology notes.

Cancer incidence was based on populations from the 1991 census, using World age-standardisation (when the data were analysed using the European age-standardised rate, results were similar; data not shown). There were 25 903 cases available for the analysis.

Both crude survival (from all causes of death) and corrected survival (from death by the cancer) based on information from death certificates were analysed. Analysis of corrected survival produced similar results to those of relative survival, indicating that death certificates give a reliable source of cause of deaths for groups of UAT cancer patients. The analysis of incidence and the multivariate analysis of survival were based on 17 393 cases from the Thames and West Midlands cancer registries where the information was

75% complete. Death certificate only (DCO) registrations and cases with missing information were excluded from the analysis. Cases not identified as dead were assumed to be alive. Due to the prevalence of local spread, multiple primary tumours and metastases in advanced UAT cancers, death from any cancer was considered as death from the incident cancer.

The SPSS package was used for analysis with a chi-squared test to assess differences in demographic factors and the Kaplan-Meier method to analyse survival. Multivariate analyses were undertaken using a forward stepwise Cox proportional hazard model to determine prognostic significance of deprivation. The multivariate analysis was undertaken for each site group (mouth, larynx and pharynx) entering the variables in the order: extent of spread, site and subsite (for mouth and pharynx) age, and deprivation. Ethnicity and sex were not found to be predictive of survival and were not included in the multivariate analysis. Marital status was correlated with deprivation and was less predictive of survival than deprivation and so was excluded.

RESULTS

Social deprivation was associated with age, sex, ethnicity, site and geographical area. UAT cancer patients living in deprived areas had a younger age profile than groups living in affluent areas; 34% of patients living in deprived areas (Carstairs quintile 4/5) were under 60 years of age compared with 29% of patients living in affluent areas (Carstairs quintile 1 $P<0.001$, Table 1). Seventy-one per cent of patients living in deprived areas were male compared with 67% of patients living in affluent areas ($P<0.001$, Table 1). Two thirds of patients of South Asian origin lived in deprived areas com-

Table 1. Association of deprivation with other tumour and demographic factors

	Carstairs quintile 1 (n=5159)		Carstairs quintile 2/3 (n=10 040)		Carstairs quintile 4/5 (n=10 704)		Total (n=25 903)
	n	(%)	n	(%)	n	(%)	n (%)
Sex							
Male	3431	(66.5)	6933	(69.1)	7637	(71.3)	18 001 (69.5)
Female	1728	(33.5)	3107	(30.9)	3067	(28.7)	7902 (30.5)
Ethnic origin							
South Asian	41	(0.8)	116	(1.2)	303	(2.8)	460 (1.8)
Other	5118	(99.2)	9924	(99.8)	10 401	(97.2)	25 443 (98.2)
Age group at diagnosis							
<60 years	1523	(29.5)	2843	(35.3)	3677	(45.7)	8043 (31.1)
60-74 years	2240	(18.8)	4655	(46.4)	5048	(42.3)	11 943 (46.1)
75+ years	1392	(27.0)	2536	(25.3)	1971	(18.4)	5899 (22.8)
Not known	4	(0.1)	6	(0.1)	8	(0.1)	18 (0.1)
Site							
Lip C00	227	(4.4)	395	(3.9)	392	(3.7)	1014 (3.9)
Mouth C01-C06	1488	(28.8)	2783	(27.7)	3021	(28.2)	7292 (28.2)
Salivary glands C07-C08	408	(7.9)	633	(6.3)	495	(4.6)	1536 (5.9)
Other pharynx C09-10, C12-14	1161	(22.5)	2280	(22.7)	2435	(22.7)	5876 (22.7)
Nasopharynx C11	187	(3.6)	348	(3.5)	354	(3.3)	889 (3.4)
Larynx C32	1688	(32.7)	3601	(35.9)	4007	(37.4)	9296 (35.9)
Extent of spread							
Confined to organ or origin, level 1	1284	(24.9)	2133	(21.2)	1327	(12.4)	4744 (18.3)
Local spread, level 2	659	(12.8)	1287	(12.8)	1166	(10.9)	3112 (12.0)
Local lymph nodes involved, level 3	860	(16.7)	1778	(17.7)	2010	(18.8)	4648 (17.9)
Metastatic spread, level 4	296	(5.7)	615	(6.1)	511	(4.8)	1422 (5.5)
Not known	2060	(39.9)	4227	(42.1)	5690	(53.2)	11 977 (46.2)

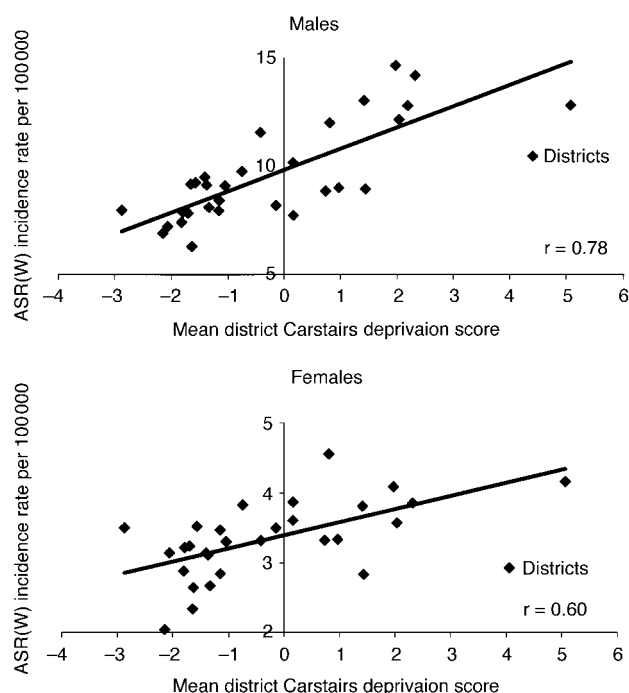
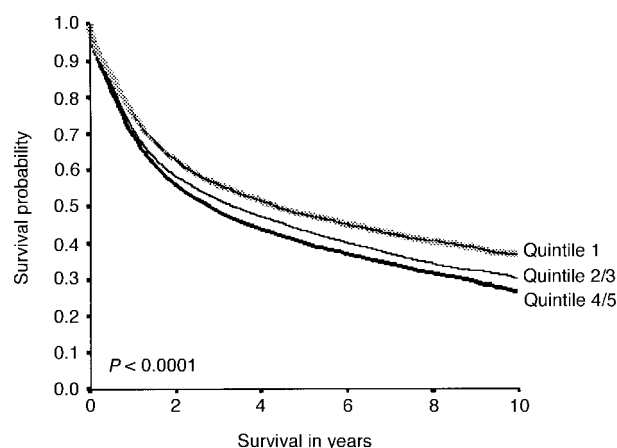


Fig. 1. District age-standardised incidence rate and mean Carstairs deprivation score. Thames and West Midlands.

pared with 41% of other ethnic groups. Patients living in affluent areas had a higher proportion of salivary gland cancers and a lower proportion of laryngeal cancers ($P < 0.001$) than patients living in deprived areas. Patients living in deprived areas had a higher proportion of cancers with lymph node involvement than patients living in affluent areas ($P < 0.001$ Table 1). Twenty three per cent of patients with complete information for survival analysis lived in affluent areas compared with 20% of those with some missing information (DCO or missing clinical information).

Social deprivation was associated with increased incidence of UAT cancers. The age-standardised incidence rate of UAT cancers (for the combined years 1984–1993) for a district health authority was strongly correlated with the mean Carstairs score for that district. The regression co-efficient was higher for men ($r = 0.78$) than for women ($r = 0.60$, Figure 1). Both deprivation and incidence were highest in the West of Scotland and lowest in South East England (results not shown).

(a) Crude survival according to Carstairs deprivation score



(b) Corrected survival according to Carstairs deprivation score

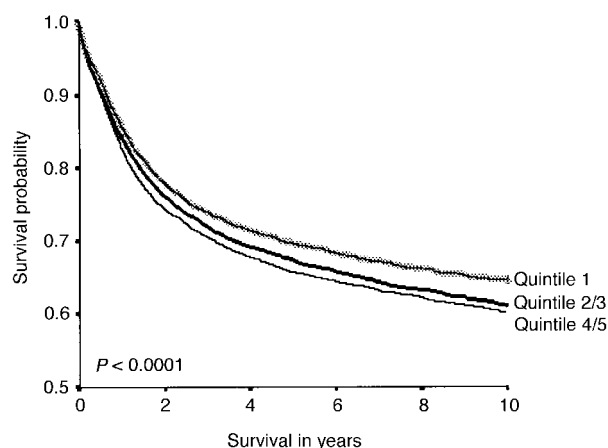


Fig. 2. (a) Crude survival according to Carstairs deprivation score. (b) Corrected survival according to Carstairs deprivation score.

Patients living in affluent areas had better crude and corrected survival than patients living in deprived areas (Table 2). Patients in Carstairs quintile 1 had a 48.4% 5 year crude survival rate for UAT cancers compared with 40.6% for quintiles 4 and 5 (Table 2, Figure 2a). Patients living in affluent areas were also less likely to die of their cancer than people living in deprived areas with 5-year cause-specific

Table 2. Survival of people with UAT cancers by deprivation. All sites

		Crude survival		Cause-specific survival	
% Surviving		% Surviving	95% CI	% Surviving	95% CI
1-year survival	Quintiles 1	75.3	(74.1–76.5)	85.5	(84.5–86.5)
	Quintile 2/3	70.9	(69.9–71.9)	82.4	(81.6–83.2)
	Quintile 4/5	69.3	(68.3–70.3)	84.0	(83.3–84.7)
2-year survival	Quintile 1	63.3	(62.6–63.9)	77.8	(76.8–78.8)
	Quintile 2/3	58.6	(58.3–58.9)	74.2	(73.2–75.2)
	Quintile 4/5	56.4	(56.1–56.7)	76.1	(75.5–76.7)
5-year survival	Quintile 1	48.4	(47.7–49.1)	69.5	(68.1–70.9)
	Quintile 2/3	44.0	(43.8–44.2)	65.7	(64.7–66.7)
	Quintile 4/5	40.6	(40.4–40.8)	67.3	(66.9–67.7)

CI, confidence interval.

Table 3. Multivariate analysis of survival and deprivation. Site, extent of spread and age in model.

Risk of death relative to Carstairs quintile 1			Crude survival		Corrected survival	
Site	Number of cases analysed/ of total number of cases		Relative risk of death	95% CI	Relative risk of death	95% CI
All sites	12 898/17 393	Quintile 2/3	1.16	(1.07–1.26)	1.17	(1.08–1.26)
		Quintile 4/5	1.24	(1.13–1.35)	1.25	(1.15–1.35)
Mouth C01-C06	3 396/4 780	Quintile 2/3	1.15	(1.06–1.25)	1.17	(1.02–1.33)
		Quintile 4/5	1.25	(1.15–1.35)	1.28	(1.11–1.47)
Larynx C32	4 120/6 228	Quintile 2/3	1.22	(1.12–1.33)	1.23	(1.08–1.40)
		Quintile 4/5	1.40	(1.28–1.52)	1.41	(1.24–1.62)
Pharynx C09,C10, C12-C14	3 023/4 078	Quintile 2/3	1.11	(1.02–1.11)	1.17	(1.04–1.31)
		Quintile 4/5	1.21	(1.11–1.32)	1.31	(1.17–1.47)

CI, confidence interval.

survival rates of 63.4% and 55.9% for quintiles 1 and 4/5, respectively (Table 2, Figure 2b).

Deprivation was a risk factor for survival in multivariate analysis of UAT cancers and of each site group when site, extent of spread, and age were controlled for. Marital status, sex and ethnic group were less predictive of survival than deprivation. Patients living in deprived areas were 25% (95% CI: 15–35%) more likely to die of their cancer and 24% (95% CI: 13–35%) more likely to die of any cause than people living in affluent areas (Table 3). Patients living in deprived areas had an increased risk of dying from their cancer of 41% (95% CI: 24–62%) for cancers of the larynx, 28% (95% CI: 11–47%) for mouth cancer and 31% (95% CI: 17–47%) for cancer of the pharynx relative to people living in affluent areas (Table 3). A multivariate analysis using only the Thames data (89% complete) produced very similar results but with wider confidence intervals.

DISCUSSION

UAT cancer patients who were younger, male and of South Asian origin were more likely to live in a deprived area than an affluent area. A smaller proportion of cases defined as Carstairs quintiles 4 and 5 were over the age of 75 years. These findings reflect the different age, gender and ethnic composition populations in affluent and deprived areas.

This study confirms the findings of others that the incidence of UAT cancers is associated with deprivation [3, 17, 18]. There was a clear correlation between the district deprivation score and the incidence of UAT cancers in the district. This correlation can be explained by the social class distribution of risk factors, such as smoking and diet, which are also risk factors for other cancers, coronary heart disease and stroke. A common risk factor approach to reducing these risk factors in deprived communities could not only reduce incidence of UAT cancers but also reduce inequalities in health.

There is evidence for some cancers that people with social and material deprivation may present later for treatment and that this may contribute to the decreased survival [19]. Although deprivation was associated with the extent of spread at presentation, it had an independent effect on both crude and corrected survival in multivariate analysis. Differences in survival from cancer may be due to differences in underlying health status and health risk, reduced material and social support or differences in treatment. There was no information on alcohol or tobacco use of patients in this study. The increased use of tobacco use in deprived populations may

account in part for the increased incidence and reduced survival of people living in deprived areas. The greater proportion of missing data from deprived populations may also have influenced the results. DCO cases has been shown to be associated with reduced survival [21, 22] and in this study were more likely to have lived in a deprived area. It is likely that inclusion of these cases would have strengthened rather than weakened the association between deprivation and survival. The results suggest that people living in deprived areas are more likely to get UAT cancer and more likely to die from it than people living in affluent areas.

1. Doll R, Peto R. The causes of cancer: quantitative estimates of avoidable risks of cancer in the United States today. *J Nat Cancer Inst* 1991, **66**, 1191–1308.
2. Pollock AM, Vickers N. Breast, lung and colorectal cancer incidence and survival in South Thames Region, 1987–1992; the effect of social deprivation. *J Public Health Med* 1997, **19**, 288–294.
3. Thorne P, Etherington D, Birchall MA. Head and neck cancer in South West of England: influence of socio-economic status on incidence and second primary tumours. *Eur J Surg Oncol* 1997, **23**, 503–508.
4. Sankaranarayanan R, Masuyer E, Swaminathan R, Ferlay J, Whelan S. Head and neck cancer: a global perspective on epidemiology and prognosis. *Anticancer Res* 1998, **18**, 4779–4786.
5. Tobias JS. Cancer of the head and neck. *Br Med J* 1994, **308**, 961–966.
6. Goud ML, Mohapatra SC, Mohapatra P, Gaur SD, Pant GC, Knanna MN. Epidemiological correlates between consumption of Indian chewing tobacco and oral cancer. *Eur J Epidemiol* 1990, **6**, 219–222.
7. Andre K, Schraub S, Mercier M, Bontemps P. Role of alcohol and tobacco in the aetiology of head and neck cancer: a case-control study in the Doubs region of France. *Eur J Cancer Part B Oral Oncol* 1995, **32B**, 301–309.
8. Ogden GR, Wight AL. Aetiology of oral cancer: alcohol. *Br J Oral and Maxillofacial Surg* 1988, **36**, 247–251.
9. Kune GA, Kune S, Field B, et al. Oral and pharyngeal cancer, diet, smoking, alcohol, and serum vitamin A and beta-carotene levels: a case control study. *Nutrition and Cancer* 1993, **20**, 61–70.
10. Hertog MG, Bueno-de-Mesquita HB, Fehily AM, Sweetnam PM, Elwood PC, Kromhout D. Fruit and vegetable consumption and cancer mortality in the Caerphilly Study. *Cancer Epidemiol Biomarkers Prev* 1996, **5**, 673–677.
11. Marshall JR, Boyle P. Nutrition and oral cancer. *Cancer Causes Control* 1996, **7**, 101–111.
12. La Vecchia C, Negri E, Franceschi S, Parazzini F, Decarli A. Differences in dietary intake with smoking, alcohol, and education. *Nutrition and Cancer* 1992, **17**, 297–304.
13. Freng A, Daae LN, Engeland A, et al. Malignant epithelial tumours in the upper digestive tract; a dietary and socio-medical case-control and survival study. *Eur J Clin Nutr* 1998, **52**, 271–278.

14. Frankel S, Gunnell DJ, Peters TJ, Maynard M, Davey Smith G. Childhood energy intake and adult mortality from cancer; the Boyd Orr Cohort Study. *Br Med J* 1998, **316**, 499–504.
15. Hart CL, Smith GD, Blane D. Inequalities in mortality by social class measured at 3 stages of the lifecourse. *Am J Public Health* 1998, **88**, 471–474.
16. *Our Healthier Nation: a contract for health consultation paper*. 1998, London, HMSO.
17. MacFarlane GJ, Sharp L, Porter S, Franceschi S. Trends in survival from cancers of the oral cavity and pharynx in Scotland: a clue as to why the disease is becoming more common? *Br J Cancer* 1996, **73**, 805–808.
18. O' Hanlon S, Forester DP, Lowry J. Oral cancer in North East of England: incidence, mortality trends and the link with material deprivation. *Community Dentistry Oral Epidemiol* 1997, **25**, 371–376.
19. Auvinen A, Karjalainen S. Possible explanations for social class differences in cancer patient survival. *IARC scientific publications* 1997, **2138**, 377–397.
20. Carstairs V, Morris R. Deprivation: explaining differences in mortality between Scotland and England and Wales. *Br Med J* 1989, **299**, 886–889.
21. Pollock AM, Vickers N. The impact on colorectal cancer survival of cases registered by 'death certificate only': implications for national survival rates. *Br J Cancer* 1994, **70**, 1229–1231.
22. Pollock AM, Vickers N. Why are a quarter of all cancer deaths in south-east England registered by death certificate only? Factors related to death certificate only registrations in the Thames Cancer Registry between 1987 and 1989. *Br J Cancer* 1995, **71**, 637–641.

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