Pergamon

European Journal of Cancer, Vol. 35, No. 13, pp. 1816–1823, 1999

Printed in Great Britain
0959-8049/99/$ - see front matter

PII: S0959-8049(99)00173-2

Original Paper

Survival from Childhood Cancer in Yorkshire, U.K.: Effect of Ethnicity and Socio-economic Status

P.A. McKinney,[1] R.G. Feltbower,[1] R.C. Parslow,[1] I.J. Lewis,[2] S. Picton,[2] S.E. Kinsey[2] and C.C. Bailey[2]

[1]Paediatric Epidemiology Group, Centre for Health Services Research, University of Leeds, 32 Hyde Terrace, Leeds LS2 9LN; and [2]Paediatric Oncology & Haematology, St James' University Hospital, Leeds, U.K.

The effect of ethnicity and socio-economic status on the survival of a population-based cohort of 1979 children diagnosed with cancer between 1974 and 1995 was investigated. Ethnicity was assigned by computer algorithms and visual inspection as south Asian (or not) for each child, based on their full name. Socio-economic status was measured using the Carstairs index, based on census areas of case residence at diagnosis. 15 children (0.8%) were lost to follow-up. Log-rank tests showed survival from all cancers did not differ between south Asians and other children and no increased risk was observed for south Asians in any diagnostic category, although numbers were small. Increasing levels of deprivation were associated with significant trends of poorer survival from all cancers, leukaemias and brain tumours. Risk of death was typically higher for children from the most deprived areas although differences were not statistically significant after accounting for other factors including ethnicity. Taking all children with malignant disease together, neither ethnicity nor socio-economic status appear to influence survival after taking other factors into consideration. © **1999 Published by Elsevier Science Ltd. All rights reserved.**

Key words: survival, childhood cancer, ethnicity, socio-economic status
Eur J Cancer, Vol. 35, No. 13, pp. 1816–1823, 1999

INTRODUCTION

THE PROSPECTS of survival for children diagnosed with cancer have improved substantially in recent decades in the U.K., with approximately two-thirds of children surviving for at least 10 years [1]. However, certain types of cancer and specific characteristics of the children such as sex and age, as well as the treatment they receive and where it is delivered, continue to influence outcome and affect survival chances [2, 3].

Cancer incidence in U.K. children displays variation by ethnic group with a notable excess of lymphomas in children of Asian ethnic origin [4–7], but it is not known whether ethnicity influences survival in a U.K. setting. One study in the U.S.A. has shown black and caucasian children with similar 10-year survival rates, whereas historically black children had a significantly poorer rate of survival [8]. Investigations of ethnicity are subject to the potential confounding effects of levels of deprivation. This is illustrated by examination of U.K. 1991 decennial census data for Yorkshire, U.K., which clearly shows a positive association between areas with a high proportion of non-white households and raised levels of deprivation; these mainly occur in highly urbanised environments. This highlights the importance of an approach which examines both ethnicity and deprivation in an effort to disentangle their potentially separate effects.

The U.K. childhood (0–14 years) population comprises 5% of people whose ethnic group is described as south Asian (source: The 1991 Census, Crown Copyright. ESRC purchase) including, in the main, people described as Indian, Pakistani and Bangladeshi, which essentially reflects the geographical boundaries of their country of origin and not necessarily their ethnic group [9]. Within the U.K., south Asians reside principally within a small number of metropolitan urban areas, including West Yorkshire, where in 1991 they comprised 12% of children, the majority of whom will

Correspondence to P.A. McKinney, e-mail: p.a.mckinney@leeds.ac.uk

have been born in Britain. There is a particularly high concentration of south Asians in the Bradford district of West Yorkshire, where one-quarter of all children are south Asian. Bradford's Pakistani population, the largest of the south Asian group, became established in the early 1960s and is the largest ethnic community in northern England. The area of Pakistan from which this community migrated is highly localised to the northern Mirpur region [10]. Although some changes will have occurred in the composition and relative proportions of the south Asian populations of Yorkshire over the last three decades, it remains one of the few areas in the U.K. where there is an opportunity to study aspects of health and disease in a south Asian minority ethnic population.

Our study utilised follow-up data from a high-quality population-based incidence register of childhood cancers [11] to examine patterns of survival over a 22-year period. The putative influence of deprivation and whether the children's ethnic group was south Asian are specifically addressed.

PATIENTS AND METHODS

The Yorkshire Children's Tumour Register (YCTR) is a population-based register of childhood malignancies with a high level of completeness, for example between 1974 and 1995 over 80% of central nervous systems (CNS) tumours were histologically verified [11]. Demographic, diagnostic and clinical details are held for children diagnosed with a malignant disease prior to their fifteenth birthday whilst resident within the northern English counties of North Yorkshire, West Yorkshire and Humberside (the former Yorkshire Regional Health Authority), which comprise 22 District Health Authorities. The total population was approximately 3.5 million at the 1991 national census.

Clinical and personal details were confirmed by direct abstraction from hospital notes or post-mortem or coroner's reports. Particular attention was paid to pathological confirmation of diagnoses which were coded and categorised into the 12 major diagnostic groups detailed in Table 1 according to the International Classification of Childhood Cancer (ICCC) [12].

Cases are actively and systematically followed-up every 2 years, collecting information either from hospital consultants or General Practitioners. For those who are 'lost', details are requested on their status (alive, dead or embarked) from the primary care registration scheme of the National Health Service Central Register (NHSCR).

A survival analysis was performed on a dataset containing 1979 cases diagnosed between 1 January 1974 and 31 December 1995 and followed-up until 30 April 1998. The endpoint of interest was death from any cause, with date of diagnosis acting as the time-origin.

For each diagnostic group/subgroup, the following variables were investigated: sex (male or female); ethnicity (south Asian or not); age (in years) at diagnosis (0–4, 5–9, 10–14); period of diagnosis (1974–80, 1981–88, 1989–95); place of treatment (centralised or not); socio-economic status (Carstairs index—based on address at diagnosis); presenting white blood cell counts ($\times 10^9$ per litre) with categories 0–3, 3–10, 10–50 and 50 plus (for leukaemias only).

Ethnicity was assigned to individual children who were classified as south Asian (Pakistani, Indian, Bangladeshi) using the South Asian Names Analysis program Nam Pehchan [13] and South Asian Name & Group Recognition Algorithm (SANGRA) [14] in conjunction with careful visual inspection. Both algorithms look at first and second names; Nam Pehchan identifies part name (stem) or complete matches from a data dictionary of Asian names and is used by the City of Bradford Metropolitan Council and Bradford Health Authorities in West Yorkshire and it is particularly appropriate for the population in this study. SANGRA uses only full name recognition in a data dictionary, giving a highly specific identification and, in addition, all names were visually checked.

Place of treatment was classified as 'central' if the delivery of care was by a U.K. Children's Cancer Study Group (UKCCSG) consultant through attendance at a paediatric oncology tertiary referral (or shared care) centre either within the region (Leeds) or elsewhere in the U.K. In 1974, the first senior registrar for paediatric oncology was appointed and 18% of cases were centrally treated; this rose to 61% in 1978 following the appointment of the author C.C. Bailey as a consultant.

Individual children were assigned a deprivation score as a proxy for socio-economic status, based on the validated postcode of their address at diagnosis using the following census variables to calculate the Carstairs index [15]: percentages of unemployed male residents over 16 years of age, residents in social class 4 and 5, non-car ownership and overcrowding (persons in households with 1 or more persons per room). Each address was linked to its census electoral ward (EW) via the central postcode directory. The Carstairs index was calculated for each EW ($n = 536$) with diagnoses between 1974–1985 and 1986–1995 derived data from the 1981 and 1991 censuses, respectively. The Carstairs index was categorised into fifths of the entire study population, with scores ranging from -5.69 (most affluent) to 17.63 (least affluent).

Survival rates (in years) were calculated using Kaplan–Meier methods [16]. Initially, ethnicity and socio-economic status were investigated separately using log-rank tests to assess whether survivor functions differed across diagnostic groups. We restricted this analysis to groups where the number of deaths were greater than 40, because this ensured the statistical validity of the comparisons.

The data were then modelled using Cox's proportional hazards technique [17]. Hazard ratios (HR) and level of significance (10% or 1%) were reported. HR are the ratio of the hazards (probability of dying at time t, having survived to that time) for two different values of a covariate, and can be interpreted in a similar way to relative risks. To retain power, this was restricted to diagnostic groups where the number of deaths was not small relative to the number of covariates in the model, that is approximately 10 times as many deaths as the number of covariates were required [18]. As a result, parametric modelling was not performed for Hodgkin's disease (HD), ependymomas, retinoblastomas, renal tumours, hepatic tumours, bone tumours, soft-tissue sarcomas, germ cell tumours, carcinomas and other tumours. Lymphomas as a group were also omitted from the analysis due to differential survival between HD and non-Hodgkin's lymphoma (NHL). Each of the remaining diagnostic groups and the entire dataset were examined separately.

The level of significance was set at 10%, with a P value of 0.10 or less indicating a statistically significant effect. At this level, a potentially important effect was unlikely to be rejected, whilst allowing for the 'significance' of an individual variable to be enhanced in combination with other variables [19]. Missing values for place of treatment ($n = 153$), Carstairs socio-economic index ($n = 2$) and white blood cell

Table 1. *Frequency of cancers by diagnostic group and number of deaths of children (*0–14 *years) registered on the Yorkshire Children's Tumour Register (YCTR) between* 1974 *and* 1995

ICCC group*	Diagnostic group	Cases *n*	Deaths *n* (% of cases)	Lost to follow-up *n* (% of cases)
1–12	All cancers	1979	862 (43.6)	15 (0.8)
1	Leukaemias	614	276 (45.0)	3 (0.5)
1a	Acute lymphoblastic leukaemia (ALL)	479	184 (38.4)	2 (0.4)
1b	Acute myeloid leukaemia (AML)	112	73 (65.1)	1 (0.9)
2	Lymphomas	238	76 (31.9)	3 (1.3)
2a	Hodgkin's disease (HD)	101	13 (12.9)	2 (2.0)
2b–2e	Non-Hodgkin's lymphoma (NHL)	137	63 (46.0)	1 (0.7)
3	Central nervous system (CNS) tumours	455	237 (52.1)	4 (0.9)
3a	Ependymomas	42	22 (52.4)	0 (0.0)
3b and 3d	Astrocytomas and other gliomas	237	115 (48.5)	3 (1.3)
3c	Primitive neuroectodermal tumours (PNETs)	116	77 (66.4)	1 (0.9)
4	Neuroblastomas	152	99 (65.1)	0 (0.0)
5	Retinoblastomas	56	5 (8.9)	0 (0.0)
6	Renal tumours	109	29 (26.6)	1 (0.9)
7	Hepatic tumours	18	13 (72.2)	1 (5.6)
8	Malignant bone tumours	96	54 (56.3)	1 (1.0)
9	Soft tissue sarcomas	128	47 (36.7)	1 (0.8)
10	Germ cell tumours	61	15 (24.6)	0 (0.0)
11	Carcinomas	50	10 (20.0)	0 (0.0)
12	Others	2	1 (50.0)	1 (50.0)

*International Classification of Childhood Cancer [12].

counts for leukaemia ($n = 16$) were coded as a separate level to avoid casewise deletion.

To differentiate between the potential independent effects of ethnicity and socio-economic status, both were included in the regression model, together with other factors known a priori to be of prognostic importance, that is age at diagnosis (0–4, 5–9, 10—14 years), sex, period of diagnosis (1974–1980, 1981–1988, 1989–1995), place of treatment (centralised specialist clinic or not) and for leukaemias only white blood cell counts (0–3, 3–10, 10–50, $\geq 50 \times 10^9$ cells per litre). HR for each a priori factor were calculated adjusting for all the other a prior factors, within each diagnostic category.

Schoenfeld residuals [20] were plotted against rank survival time for each covariate, producing random scatters about zero. This demonstrated that the Cox proportionality assumption was valid [21].

Statistical analyses were performed using Stata [22] and SPSS 6.0 (log-rank test for trend).

RESULTS

15 children from the register were lost to follow-up representing 0.8% of all cases ($n = 1979$), although some years of survival may have been contributed prior to their censor dates to the total of 12 742 person-years of survival. Of those lost to follow-up 12 had emigrated, 1 was registered with the Armed Services and 2 were untraceable. The losses to follow-up were proportionate across the diagnostic groups (see Table 1).

Length of follow-up ranged from 0 days to 24 years (median 4 years 1 month). The 25th percentile survival time was 14.5 months (more than 50% of cases remained alive, hence median survival time cannot be calculated).

Table 1 shows for each diagnostic group the number of cases, deaths (from any cause), and those lost to follow-up. The 2- and 5-year survival rates for all cancers combined were 68.7 and 59.2%, respectively.

Table 2 shows the differences in the proportion of deaths between south Asians and non-south Asians by diagnostic group without adjusting for any other factors. No significant differences were observed.

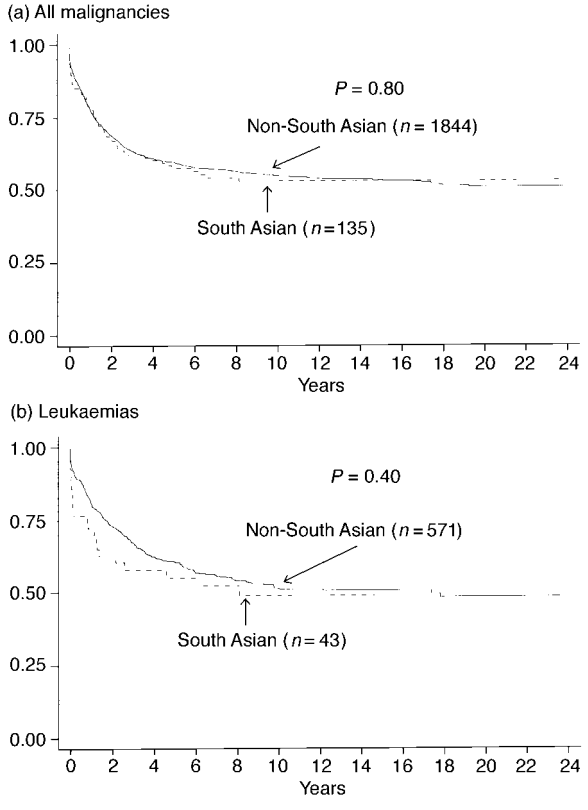

Figure 1. Kaplan–Meier survival curves (with log-rank *P* values) comparing South Asians with non-South Asians.

Table 2. Frequency of cancers and numbers of deaths by diagnostic group and ethnic group

Diagnostic group	Non-south Asian		South Asian		Log rank
	n Cases	*n* Deaths (%)	*n* Cases	*n* Deaths (%)	*P* value
All cancers	1844	803 (43.5)	135	59 (43.7)	0.80
Leukaemias	571	255 (44.7)	43	21 (48.8)	0.40
Acute lymphoblastic leukaemias (ALL)	447	172 (38.5)	32	12 (37.5)	0.98
Acute myeloid leukaemias (AML)	104	67 (64.4)	8	6 (75.0)	0.36
Non-Hodgkin's lymphoma (NHL)	122	55 (45.1)	15	8 (53.3)	0.41
Central nervous system (CNS) tumours	433	224 (51.7)	22	13 (59.1)	0.43
Astrocytomas and other gliomas	228	109 (47.8)	9	6 (66.7)	0.22
Primitive neuroectodermal tumours (PNETs)	108	73 (67.6)	8	4 (50.0)	0.20
Neuroblastomas	143	93 (65.0)	9	6 (66.7)	0.46
Bone tumours	90	50 (55.6)	6	4 (66.7)	0.71
Soft-tissue sarcomas	120	45 (37.5)	8	2 (25.0)	0.58

Kaplan–Meier survival curves are illustrated in Figure 1 for all cancers combined and leukaemias, comparing south Asians with non-south Asians. The curves indicate that there is no difference in survival for all cancers between south Asians and non-south Asians, although south Asians with leukaemia appear to do less well, particularly in the short term (<5 years)

The results of testing whether survival differed according to socio-economic status are given in Table 3 by diagnostic group. Significant trends were observed for all cancers combined, where the most deprived children had the poorest survival. This pattern was reflected in the leukaemias, mainly accounted for by acute lymphoblastic leukaemia (ALL), and the CNS tumours where the more deprived had the poorest chances of survival. A Kaplan–Meier survival curve (not shown) of children with leukaemia comparing the five socio-economic groupings suggested those in the poorest areas had consistently worse prognosis compared with other socio-economic groups, over the entire period of follow-up.

The data and results of the Cox regression modelling are given in Tables 4 and 5, respectively. It should be emphasised that in Table 4 the decrease in percentage dying associated with period of diagnosis—mostly attributable to improved survival—was also partly due to the shorter follow-up for more recent cases. Table 5 shows HRs for ethnicity (adjusting for socio-economic status and all other a priori factors) and socio-economic status (adjusting for ethnicity and all other a prior factors), as well as the risks for each of the other a priori factors (sex, age, period of diagnosis, place of treatment and white cell counts—mutually adjusting for each other). The columns indicate the results of the modelling conducted separately for each diagnostic group. For all cancers combined, or any diagnostic subgroup, there was no effect of either ethnicity nor socio-economic status on survival, apart from south Asians with primitive neuroectodermal tumours (PNETs) who had a significantly reduced risk of dying (HR = 0.32, $P = 0.05$).

Despite an increased risk of death being associated with more deprived areas in the univariate analysis (Table 3), the adjustment for other factors in the regression analysis removed the significance. No differences in survival for all cancers combined, between the most and least affluent, were evident for socio-economic status. Children with CNS tumours from areas of middle affluence were 1.62 times more likely to die than children from the most affluent areas ($P = 0.02$). Children with neuroblastomas in the second most affluent quintile had a reduced risk of dying compared with children in the most affluent areas (HR = 0.40, $P = 0.02$).

Table 3. Trends in survival by socio-economic status (Carstairs index) and diagnostic group

		Carstairs index (1 = most affluent, 5 = least affluent) % of cases that died					Log-rank test of trend
Diagnostic group	Cases	1	2	3	4	5	*P* value
All cancers	1977	42.3	38.6	43.4	45.2	48.0	0.05*
Leukaemias	614	40.0	45.4	39.3	46.5	56.2	0.04*
Acute lymphoblastic leukaemia (ALL)	479	31.7	42.6	31.8	41.8	47.4	0.10
Acute myeloid leukaemia (AML)	112	70.8	57.1	68.4	56.0	73.9	0.96
Non-Hodgkin's lymphoma (NHL)	137	47.6	54.8	41.7	44.0	41.7	0.34
Central nervous system (CNS) tumours	453	45.2	38.4	65.4	56.5	55.7	0.07*
Astrocytomas and other gliomas	237	44.1	38.6	55.3	51.2	54.5	0.22
Primitive neuroectodermal tumours (PNETs)	114	60.0	59.1	72.7	69.6	68.2	0.84
Neuroblastomas	152	76.9	45.2	71.0	77.4	57.6	0.98
Bone tumours	96	63.6	52.2	54.5	50.0	58.8	0.62
Soft-tissue sarcomas	128	41.4	25.0	20.0	55.2	36.4	0.29

*Significant at the 10% level.

Table 4. *Number of cases and the proportion of deaths by diagnostic group for ethnicity, socio-economic status and other prognostic factors included in the Cox regression modelling**

Variable	All cancers *n* (% died)		Leukaemia *n* (% died)		ALL *n* (% died)		AML *n* (% died)		CNS *n* (% died)		Astrocytomas *n* (% died)		PNETs *n* (% died)		Neuroblastomas *n* (% died)	
Sex																
Male	1089	43.5	349	48.7	274	43.4	60	65.0	248	50.4	121	44.6	68	69.1	74	66.2
Female	890	43.6	265	40.0	205	31.7	52	65.4	207	54.1	116	52.6	48	62.5	78	64.1
Age at diagnosis (years)																
0–4	889	43.2	333	42.3	262	34.7	56	66.1	142	54.9	62	43.5	44	63.6	121	64.5
5–9	525	45.3	155	40.6	130	39.2	23	43.5	168	57.7	93	54.8	47	66.0	16	62.5
10–14	565	42.5	126	57.1	87	48.3	33	78.8	145	42.8	82	45.1	25	72.0	15	73.3
Period of diagnosis																
1974–80	598	61.2	183	69.9	148	63.5	27	96.3	134	69.4	71	62.0	32	84.4	46	78.3
1981–88	666	42.8	209	41.6	167	33.5	34	70.6	158	55.1	84	57.1	35	77.1	42	66.7
1989–95	715	29.5	222	27.5	164	20.7	51	45.1	163	35.0	82	28.0	49	46.9	64	54.7
Centralised treatment																
Yes	1422	38.0	529	39.9	419	33.9	93	60.2	207	46.4	99	45.5	65	52.3	116	59.5
No	404	57.4	55	78.2	38	71.1	12	91.7	200	60.5	113	53.1	43	90.7	24	91.7
White cell count ($\times 10^9$ cells/l)																
0–3			95	28.4	83	25.3	11	45.5								
3–10			150	37.3	126	30.2	22	72.7								
10–50			214	45.3	165	40.6	40	57.5								
>50			139	60.4	92	53.3	36	72.2								
Ethnicity																
Non-Asian	1844	43.5	571	44.7	447	38.5	104	64.4	433	51.7	228	47.8	108	67.6	143	65.0
Asian	135	43.7	43	48.8	32	37.5	8	75.0	22	59.1	9	66.7	8	50.0	9	66.7
Carstairs index																
1, most affluent	397	42.3	126	40.0	101	31.7	24	70.8	104	45.2	59	44.1	25	60.0	26	76.9
2	394	38.6	119	45.4	94	42.6	21	57.1	86	38.4	44	38.6	22	59.1	31	45.2
3	396	43.4	135	39.3	110	31.8	19	68.4	81	65.4	38	55.3	22	72.7	31	71.0
4	392	45.2	129	46.5	98	41.8	25	56.0	85	56.5	41	51.2	23	69.6	31	77.4
5, least affluent	398	48.0	105	56.2	76	47.4	23	73.9	97	55.7	55	54.5	22	68.2	33	57.6

*See Table 5. ALL, Acute lymphoblastic leukaemia; AML, acute myeloid leukaemia; CNS, central nervous system tumours; PNETs, primitive neuroectodermal tumours.

Table 5. Hazard ratios (HR) of dying using multivariate Cox regression modelling by diagnostic group for ethnicity, socio-economic status and other prognostic factors: all factors mutually adjusted for each other

Variable	All cancers	Leukaemia	ALL	AML	CNS	Astrocytomas	PNETs	Neuroblastomas
Sex								
Male*	1	1	1	1	1	1	1	1
Female	1.01	0.79†	0.67†	0.79	1.14	1.47†	0.83	0.94
Age at diagnosis (years)								
0–4*	1	1	1	1	1	1	1	1
5–9	0.97	0.98	1.32	0.64	0.97	1.49	0.80	0.85
10–14	0.82†	1.34†	1.38	1.41	0.55‡	0.87	0.84	0.82
Period of diagnosis								
1974–80*	1	1	1	1	1	1	1	1
1981–88	0.72‡	0.61‡	0.56‡	0.46†	0.72†	0.90	0.91	1.07
1989–95	0.56‡	0.51‡	0.48‡	0.31‡	0.48‡	0.41‡	0.69	0.84
Centralised treatment								
Yes*	1	1	1	1	1	1	1	1
No	1.59‡	1.94‡	1.95‡	2.68†	1.18	0.89	3.00‡	5.85‡
White cell count ($\times 10^9$ cells/l)								
0–3*		1	1	1				
3–10		1.52†	1.28	1.50				
10–50		1.98‡	1.88†	1.17				
>50		3.44‡	3.37‡	1.87				
Ethnicity								
Non-Asian*	1	1	1	1	1	1	1	1
Asian	0.95	1.19	1.00	1.77	1.12	1.65	0.32†	0.46
Carstairs index								
1, most affluent*	1	1	1	1	1	1	1	1
2	0.90	1.01	1.24	1.02	0.91	0.96	0.85	0.40†
3	1.06	0.85	0.87	0.79	1.62†	1.43	1.27	0.86
4	1.01	0.90	1.05	0.78	1.17	1.01	0.97	1.33
5, least affluent	1.13	1.32	1.43	0.65	1.20	1.15	1.59	0.56

ALL, Acute lymphoblastic leukaemia; AML, acute myeloid leukaemia; CNS, central nervous system tumour; PNETs, primitive neuroectodermal tumours. *Baseline category. †Significant at 10% level. ‡Significant at 1% level.

DISCUSSION

Population-based survival rates for childhood cancer are published less frequently than results of clinical trials but are nonetheless important both from a clinical and public health perspective. This study was undertaken using the YCTR, a specialist register of children with cancer, which is considered to represent the population with respect to completeness and accuracy [11].

The purpose of the study was to investigate a prior the putative effects of ethnicity and deprivation on survival. As Yorkshire has one of the highest concentrations of south Asians in the U.K., the YCTR was an ideal dataset for the investigation. Despite small numbers and consequently reduced power in some of the diagnostic categories, the topic was considered to be of sufficient interest to warrant examination but our findings are interpreted with caution.

The designation of ethnicity based on a child's name and determining whether they are south Asian or not has advantages in that it allocates a category to an individual rather than a geographical area. The use of national decennial census data from small geographical areas is a more common approach to investigations of ethnicity and its relationship to cancer [23]. The computer algorithms applied to the YCTR for the allocation of south Asian ethnicity [13, 14] function slightly differently but taken together and in conjunction with the expert inspection accompanying SANGRA provide a classification of ethnicity which can be considered reliable. On a previous study, Nam Pehchan alone achieved an overall sensitivity and specificity of 90.5% and 99.4%, respectively [24]. However, no method which is limited to the recognition of an individual's name will be completely free of error. Also, although specific for flagging south Asians, the program will fail to identify children from mixed and other ethnic backgrounds, for example African-Caribbeans. Thus, comparisons for south Asians will be with an ethnically heterogeneous group of children, although caucasians are known to comprise 90% of the 0–14-year-old population in Yorkshire.

The designation of 'south Asian' may itself fail to identify a homogeneous group, as cultural, spatial and religious differences between Indian, Pakistani and Bangladeshi populations will determine differing lifestyle patterns. The proportional representation of these three groups will have varied over the time period of the study with the Indian population remaining fairly stable (1.4% and 1.8% of the total population of West Yorkshire at the 1981 and 1991 censuses, respectively), the Pakistani population increasing (1981: 2.9%, 1991: 4.1%) and the smallest Bangladeshi population rising most steeply (1981: 0.2%, 1991: 0.3%) but contributing the lowest absolute numbers [9]. Over the 22-year study period, the predominant south Asian minority ethnic group in West Yorkshire has been the Pakistani population where geographical spread has not differed between the 1981 and 1991 censuses. However, the classification of south Asian is not necessarily one that defines a population with common social characteristics or attributes and this should be borne in mind in the context of this study.

Leukaemia is the most common cancer in children, accounting for one-third of all paediatric malignancies [23]. We have shown that ethnicity (south Asian or not) is not related to survival for children diagnosed with any malignancy or leukaemia, an association which persists even after adjustment for socio-economic status. Modelling the data and allowing for additional prognostic variables did not demonstrate any independent effect for socio-economic status, although it was significant in the univariate analysis.

It has been shown in the U.S.A. that blacks with ALL have a poorer short-term survival than caucasian children, which is explained by over-representation of subtypes of leukaemia with unfavourable chromosomal abnormalities in black children [25]. Without adjusting for other factors, there was an apparent disadvantage in short-term survival for south Asian children with leukaemia but no significant difference beyond 5 years of follow-up. There is no evidence to indicate a predominance of poorer prognosis subtypes in south Asians.

Survival from childhood cancer is related to receiving the prescribed therapy. The most common treatment regimen for ALL includes oral therapy administered by parents for approximately the last 18 months of a 24-month treatment programme. The continuation of this 'maintenance' chemotherapy, continued to the second anniversary of diagnosis, is proven to be beneficial for survival [26]. Our study suggests it is highly unlikely that cultural or language differences are related to whether children receive therapy.

South Asian ethnicity did not influence the survival of children with CNS tumours, either in the univariate or multivariate analysis. The results of the multivariate modelling showed two independent factors associated with improved survival—being aged 10–14 years at diagnosis and being diagnosed in the most recent time period (1989–95). In contrast to the entire CNS group, the subset of PNETs did show a significant association between survival and being south Asian but the risk of dying was significantly reduced. The numbers, however, were very small (4 deaths out of 8 cases) and the reason why such a significant advantage is conferred in terms of survival is unclear. There is no reason to suspect south Asian children with poor prognosis PNETs have not been included due to misclassification of their diagnosis, as the proportion of histologically verified CNS tumours was actually higher (91%) amongst south Asians compared with the remaining children (84%). The degree of surgical resection of PNETs may be related to survival and has been debated [27, 28]. Our Yorkshire study was unable to investigate specific treatment modalities and therefore clarify whether the south Asian children had received particularly extensive surgery.

In the U.K., the uptake of healthcare services by minority ethnic groups is poorly documented and little attention has been focused on children [29], but very recently it has become clear that children and young people from minority ethnic groups do appear to receive a poorer quality of healthcare compared with the caucasian population [30]. Cultural differences may affect the way families seek medical help, for example, the speed at which medical intervention is sought may be a consequence of how well families will tolerate symptoms in the child before worrying and visiting their GP. However, late presentation of children to medical services does not appear to be present for the south Asian community, and it has recently been shown that south Asian children use GP services more than any other ethnic group [30].

Area-based measures of socio-economic status are unrelated to survival patterns of childhood cancer in Yorkshire. Estimates of deprivation by the Carstairs index are based on aggregated census data for a geographical area and potentially may not represent the environment experienced by an individual child [31]. The degree to which this potential misclassification of an individual occurs is impossible to assess but if present is most likely to dilute and therefore mask any effect which actually exists. Socio-economic circumstances are known to affect the health of populations [32] and the absence of an association may be explained by the necessity for specific multidisciplinary delivery of healthcare for children with cancer. This may well compensate for potential detrimental consequences of deprivation.

Over the study period increasing proportions of children have been treated at tertiary referral centres, such as Leeds, Yorkshire [33], where over 85% of children are now referred (C.C. Bailey, St James' University Hospital, Leeds, U.K.). The extensive support services, particularly health visitor and district nursing care, which are devoted to the clinical and social needs of the children is likely to mitigate against social inequalities, although in the general population socio-economic status *per se* may not be related to use of health services by those in the childhood age range [30].

One unexpected finding from our study was the significantly poorer survival of children with CNS tumours from areas of ''medium affluence''. This result may have emerged by chance due to lack of power, multiple testing or poor classification of deprivation by the areal method. Other plausible explanations are not obvious.

Stage of cancer at the time of presentation is an important prognostic factor for survival, which was not included for the solid tumour groups. Changes in the application of staging classifications over the 20-year period of data collection have resulted in inconsistencies in this information. If our results had indicated clear evidence of a link between poor survival and deprivation or being south Asian, a detailed survey of whether this might have been accounted for by late stage of disease at presentation would have been necessary, but the absence of an association did not warrant further study.

For children with leukaemia, a surrogate measure of stage of disease is the presenting white blood cell count [34, 35]. Findings were consistent with those from all major clinical trials showing presenting white blood cell count is an independent risk factor for ALL.

In conclusion, our analysis has failed to demonstrate any link between socio-economic status and ethnic group in relation to survival for all childhood cancers. Associations between high levels of deprivation and poorer survival were only present before adjustment for other prognostic factors but larger studies should be undertaken to evaluate this relationship further.

1. Stiller CA. Population-based survival rates for childhood cancer in Britain 1980–91. *Br Med J* 1994, **309**, 1612–1616.
2. Stiller CA, Bunch KJ. Trends in survival for childhood cancer in Britain diagnosed 1971–85. *Br J Cancer* 1990, **62**, 806–815.
3. Mosso ML, Colombo R, Giordano L, Pastore G, Terracini B, Magnani C. Childhood Cancer Registry of the province of Torino, Italy. *Cancer* 1992, **69**, 1300–1306.
4. Stiller CA, McKinney PA, Bunch KJ, Bailey CC, Lewis IJ. Childhood cancer and ethnic group in Britain—A United Kingdom Children's Cancer Study Group (UKCCSG) study. *Br J Cancer* 1991, **64**, 543–548.

5. Muir KR, Parkes SE, Mann JR, Stevens MCG, Cameron AH. Childhood cancer in the West Midlands: incidence and survival 1980-84 in a multiethnic population. *Clin Oncol* 1992, **4**, 177–182.
6. Powell JE, Parkes SE, Cameron AH, Mann JR. Is the risk of cancer increased in Asians living in the UK? *Arch Dis Child* 1994, **71**, 398–403.
7. Varghese C, Barrett JH, Johnston C, Shires M, Rider L, Forman D. High risk of lymphomas in children of Asian origin: ethnicity or confounding by socio-economic status. *Br J Cancer* 1996, **74**, 1503–1505.
8. Pui C-H, Boyett JM, Hancock ML, Pratt CB, Meyer WH, Crist WM. Outcome of treatment for childhood cancer in black as compared with white children: the St. Jude children's research hospital experience, 1962–1982. *J Am Med Assoc* 1995, **273**, 633–637.
9. Ratcliffe P, ed. *Ethnicity in the 1991 Census, Volume 3, Social Geography and Ethnicity in Britain: Geographical Spread, Spatial Concentration and Internal Migration*. London, HMSO, 1996 4–7, 81–82.
10. Rees P, Phillips D, Medway D. The socio-economic geography of ethnic groups in two northern British cities. *Environment and Planning A* 1995, **27**, 557–591.
11. McKinney PA, Parslow RC, Lane SA, *et al*. Epidemiology of childhood brain tumours in Yorkshire, UK 1974–95: changing patterns of occurrence and geographical distribution. *Br J Cancer* 1998, **78**, 974–979.
12. Kramárová E, Stiller CA, Ferlay J, et al., eds. *International Classification of Childhood Cancer* 1996. In IARC technical report No. 29. Lyon, IARC, 1996.
13. Bradford Health Authority and City of Bradford Metropolitan District Council. *NAM PEHCHAN*: South Asian Names (Indian Sub-continent) A computer program to identify and analyse south Asian names Version 1.1. City of Bradford Metropolitan District Council Computer Services Division, 1996.
14. London School of Hygiene and Tropical Medicine, SANGRA: South Asian Name & Group Recognition Algorithm. Version 1.1, 1999.
15. Carstairs V, Morris R. *Deprivation and Health in Scotland*. Aberdeen, Aberdeen University Press, 1991.
16. Kaplan EL, Meier P. Nonparametric estimation from incomplete observations. *J Am Stat Assoc* 1958, **53**, 457–481.
17. Cox DR. Regression models and life tables (with discussion). *J R Stat Soc B* 1972, **34**, 187–220.
18. Simon R, Altman DG. Statistical aspects of prognostic factor studies in oncology. *Br J Cancer* 1994, **69**, 979–985.
19. Palmer MKB, Machin D. *Survival Analysis: A Practical Approach*. Chichester, Wiley, 1995.
20. Schoenfeld D. Partial residuals for the proportional hazards regression model. *Biometrika* 1982, **69**, 239–241.
21. Collett D. *Modelling Survival Data in Medical Research*. London, Chapman and Hall, 1994.
22. StataCorp. Stata statistical software, release 5.0. College Station, TX, Stata corporation, 1997.
23. Bhopal RS, Rankin J. Cancer in minority ethnic populations: priorities from epidemiological data. *Br J Cancer* 1996, **74**(Suppl. XXIX), S22–S32.
24. Winter H, Cheng KK, Cummins C, Maric R, Silcocks P, Varghese C. Cancer incidence in the south Asian population of England (1990–92). *Br J Cancer* 1990, **79**, 645–654.
25. Kalwinsky DK, Rivera G, Dahl GV, Roberson P, George S, Murphy SB. Variation by race in presenting clinical and biologic features of childhood acute lymphoblastic leukaemia—implications for treatment outcome. *Leukemia Res* 1985, **9**, 817–823.
26. Rothwell D. Duration of chemotherapy in childhood acute lymphoblastic leukemia. *Med Pediatr Oncol* 1982, **10**, 511–520.
27. Jenkin D, Goddard K, Armstrong D, *et al*. Posterior fossa medulloblastoma in childhood: treatment results and a proposal for a new staging system. *Int J Radiat Oncol Biol Phys* 1990, **19**, 265–274.
28. Tait DM, Thornton-Jones H, Bloom HJG, Lemerle J, Morris-Jones P. Adjuvant chemotherapy for medulloblastoma: the first multicentre control trial of the International Society of Paediatric Oncology (SIOP). *Eur J Cancer* 1990, **26A**, 464–469.
29. Raleigh VS, Balarajan R. The health of infants and children among ethnic minorities. In Botting B, ed. The Health of our Children: Decennial Supplement. London, Office of Population Censuses and Surveys Series DS no 11 HMSO, 1995.
30. Cooper H, Smaje C, Arber S. Use of health services by children and young people according to ethnicity and social class: secondary analysis of a national survey. *Br Med J* 1998, **317**, 1047–1051.
31. McLoone P. *Carstairs Scores for Scottish Postcode Sectors from the 1991 Census*. Glasgow, Public Health Research Unit, 1995.
32. Sloggett A, Joshi H. Deprivation indicators as predictors of life events 1981–1992 based on the UK ONS longitudinal study. *J Epidemiol Community Health* 1998, **52**, 228–233.
33. Stiller CA. Centralisation of treatment and survival rates for cancer. *Arch Dis Child* 1988, **63**, 23–30.
34. Mastrangelo R, Poplack D, Bleyer A, Riccardi R, Sather H, D'Angio G. Report and recommendations of the Rome workshop concerning poor-prognosis acute lymphoblastic leukaemia in children: biologic bases for staging, stratification, and treatment. *Med Pediat Oncol* 1986, **14**, 191–194.
35. Chessells JM, Richards SM, Bailey CC, Lilleyman JS, Eden OB. Gender and treatment outcome in childhood lymphoblastic leukaemia: report from the MRC UKALL trials. *Br J Haematol* 1995, **89**, 364–372.

Acknowledgements—This work has been funded by the Candlelighters Trust, St James' Hospital, Leeds, U.K. The careful data collection and processing by Linda Proctor and Helen Lilley has been crucial and Dr Anthony Staines is thanked for his contribution in managing the register. Neuropathologists Dr James Ironside, Dr Sally Lane and Dr Lesley Bridges are thanked for their assistance with histopathological review. Dr Isabel dos Santos Silva of the London School of Tropical Medicine and Hygiene is thanked for the use of the SANGRA program. We are grateful to Professor David Forman of the Northern and Yorkshire Regional Cancer Registry Information Service and Mr Charles Stiller of the Children's Cancer Research Group in Oxford for supplying data for the cross-check. Programming assistance and helpful discussions from Graham Law and secretarial support from Sheila Jones were much appreciated. The help of the staff at the NHSCR at Southport and the contribution of many consultants and medical record staff throughout the region is gratefully acknowledged. We also thank Professor Ray Cartwright for comments on an earlier draft.