Parental educational attainment as an indicator of socioeconomic status and risk of childhood cancers

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BACKGROUND: Little has been reported on socioeconomic (SES) patterns of risk for most forms of childhood cancer.

METHODS: Population-based case–control data from epidemiological studies of childhood cancer conducted in five US states were pooled and associations of maternal, paternal and household educational attainment with childhood cancers were analysed. Odds ratios (ORs) and 95% confidence intervals were estimated using logistic regression, controlling for confounders.

RESULTS: Although there was no association with parental education for the majority of cancers evaluated, there was an indication of a positive association with lower education for Hodgkin’s and Burkitt’s lymphoma and Wilm’s tumour, with the ORs ranging from 1.5 to > 3.0 times that of more educated parents. A possible protective effect was seen for lower parental education and astrocytoma and hepatoblastoma, with ORs reduced by 30 to 40%.

CONCLUSIONS: These study results should be viewed as exploratory because of the broad nature of the SES assessment, but they give some indication that childhood cancer studies might benefit from a more thorough assessment of SES.

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There is ample evidence that poorer socioeconomic circumstances are linked to poorer health (Seeman and Crimmins, 2001; Galobardes et al, 2007). Socioeconomic status (SES) is complex, incorporating aspects of both availability of resources (education, income and wealth) and standing in the hierarchy of a society. SES measures may indicate a variety of health-related factors, including occupational exposures, dietary patterns, residential environmental exposures, exposures to infectious agents, and the knowledge of and adherence to healthy lifestyles (Krieger et al, 1997; Braveman et al, 2005; Galobardes et al, 2007).

A possible role of parental SES in childhood cancers has been investigated for childhood leukaemias, but without conclusive results, with associations varying by study design, time period, SES measure used and whether this was at the individual or family level vs neighbourhood or higher grouping (Poole et al, 2006; Adam et al, 2008). There is little published data on SES patterns of risk for other forms of childhood cancer, most studies tending to consider SES data, typically parental education, only to control for potential confounding. Using a large pooled data set of childhood cancers from five US states, we conducted an exploratory analysis of the effect of SES, as estimated by parental educational attainment, on incidence of specific childhood cancers.

MATERIAL AND METHODS

Details of the study population have been published elsewhere (Puutala et al, 2009). Briefly, population-based case–control data from studies conducted in California, Minnesota, New York (excluding New York City), Texas and Washington states (Reynolds et al, 2002; McLaughlin et al, 2006; Podvin et al, 2006; Puutala et al, 2008; Carozza et al, 2009) were pooled, with birth dates of subjects ranging from 1970 to 2004. For each study, cases of childhood cancer identified in the population-based state cancer registry were matched to state vital records to capture birth certificate data (Jarv, 1995). Cases were classified according to the International Classification of Childhood Cancer third edition (Steliarova-Foucher et al, 2005). Controls (frequency matched on delivery year in all states except California, where data were individually matched) were randomly selected from the state birth records, with case/control ratios ranging from 1:1 to 1:10. The final pooled data set consisted of 17 672 cases and 57 966 controls. Human Subjects Protection Committee approvals were obtained from the institutional review boards of all the participating institutions including each state’s health department.
Parental educational data were not available for all years in three states (California, Texas and Washington). Study subjects born in years for which there was no collection of parental educational data were excluded from the pooled data set, resulting in a total of 12,665 cases and 39,472 controls available for analysis. New York state collected education as a categorical variable between 1988 and 1990, which resulted in 643 cases and 1438 controls with maternal education, and 619 cases and 1261 controls with paternal education in categories only. Maternal and paternal education was assigned according to the highest completed level of education.

Table 1  Selected characteristics of cases and controls

| Characteristics          | Category          | Cases | %   | Controls | %   |
|--------------------------|-------------------|-------|------|----------|-----|
| Parental characteristics |                   |       |      |          |     |
| Maternal race/ethnicity  |                   |       |      |          |     |
| Non-Hispanic white       | 7292              | 67.6  | 22172| 70.2     |
| Non-Hispanic black       | 720               | 6.7   | 2078 | 6.6      |
| Non-Hispanic Asian       | 443               | 4.1   | 1545 | 4.9      |
| Non-Hispanic other       | 59                | 0.6   | 384  | 1.2      |
| Hispanic                 | 2267              | 21.0  | 5414 | 17.1     |
| Missing                  | 1884              |       | 7879 |          |
| Paternal race/ethnicity  |                   |       |      |          |     |
| Non-Hispanic white       | 6806              | 69.0  | 20137| 70.8     |
| Non-Hispanic black       | 549               | 5.6   | 1664 | 5.9      |
| Non-Hispanic Asian       | 383               | 3.9   | 1308 | 4.6      |
| Non-Hispanic other       | 58                | 0.6   | 262  | 0.9      |
| Hispanic                 | 2067              | 21.0  | 5093 | 17.9     |
| Missing                  | 2802              |       | 11008|          |
| Maternal age             |                   |       |      |          |     |
| < 20 years               | 1240              | 9.8   | 4119 | 10.4     |
| 20 – 24 years            | 2972              | 23.5  | 9839 | 24.9     |
| 25 – 29 years            | 4049              | 32.0  | 12473| 31.6     |
| 30 – 34 years            | 2957              | 23.4  | 8883 | 22.5     |
| 35+ years                | 1443              | 11.4  | 4148 | 10.5     |
| Missing                  | 4                 |       | 10   |          |
| Paternal age             |                   |       |      |          |     |
| < 20 years               | 348               | 3.1   | 1104 | 3.2      |
| 20 – 24 years            | 1792              | 15.9  | 5964 | 17.1     |
| 25 – 29 years            | 3380              | 30.0  | 10453| 29.9     |
| 30 – 34 years            | 3209              | 28.5  | 9785 | 28.0     |
| 35+ years                | 2551              | 22.6  | 7621 | 21.8     |
| Missing                  | 1385              |       | 4545 |          |
| Level of maternal education |               |       |      |          |     |
| < 12 years               | 2482              | 20.2  | 7246 | 19.2     |
| 12 years                 | 4346              | 35.4  | 13586| 36.1     |
| 13 – 16 years            | 4381              | 35.7  | 13815| 36.7     |
| 17 years or more         | 1053              | 8.6   | 3017 | 8.0      |
| Missing                  | 403               |       | 1808 |          |
| Level of paternal education |             |       |      |          |     |
| < 12 years               | 1868              | 16.8  | 5013 | 15.0     |
| 12 years                 | 3937              | 35.4  | 12006| 35.9     |
| 13 – 16 years            | 3997              | 35.9  | 12402| 37.1     |
| 17 years or more         | 1330              | 12.0  | 4005 | 12.0     |
| Missing                  | 1533              |       | 6046 |          |
| Child’s characteristics  |                   |       |      |          |     |
| Age at diagnosis (cases) |                   |       |      |          |     |
| 28 days – 4 years        | 8723              | 68.9  |      |          |
| 5 – 9 years              | 2263              | 17.9  |      |          |
| 10 – 14 years            | 1679              | 13.3  |      |          |
| Mean age in years (s.d.) | 4.5 (3.9)         |       |      |          |
| Sex                      |                   |       |      |          |     |
| Male                     | 7002              | 55.3  | 20860| 52.9     |
| Female                   | 5661              | 44.7  | 18603| 47.1     |
| Missing                  | 2                 |       | 9    |          |
| Plurality                |                   |       |      |          |     |
| Singleton                | 12401             | 97.9  | 38533| 97.6     |
| Multiple                 | 260               | 2.1   | 928  | 2.4      |
| Missing                  | 4                 |       | 11   |          |
| Birth order              |                   |       |      |          |     |
| First                    | 5096              | 41.1  | 15564| 40.7     |
| Second                   | 4079              | 32.9  | 12424| 32.5     |
| Third                    | 1992              | 16.0  | 6236 | 16.3     |
| Fourth                   | 781               | 6.3   | 2400 | 6.3      |
| Fifth or higher          | 465               | 3.7   | 1643 | 4.3      |
| Missing                  | 252               |       | 1205 |          |
| Mean (s.d.)              | 2.0 (1.2)         |       | 2.1 (1.3) |
Household education level was determined as either the highest parental education level attained or the highest education level of one parent if the other parent’s information was not listed. Educational attainment was analysed by categories typically associated with credentials: <12 years (did not complete high school degree); 12 years (completed high school degree); 13–16 years (Associates or Bachelor degree equivalents); 17 years or more (graduate or professional training). Analyses were also performed to evaluate associations for children whose birth certificate included only maternal data (i.e., missing all paternal data), which potentially indicated a household of low SES at the time of birth (Tan et al., 2004). Child-level characteristics retrieved from birth certificate data, included in the evaluation of potential confounding variables, were birth weight, gestational age, plurality, sex, birth order, year of birth, maternal age and maternal race/ethnicity as well as state of birth. ORs for educational attainment of mother, father and household are presented for all cancers combined and for International Classification of Childhood Cancer third edition cancer sites with >200 cases in the data set (excluding heterogeneous ‘other’ and unspecified tumour categories). As higher SES is generally beneficial when considering health outcomes, educational attainment of 17 years or more served as the referent category in the models. To assess the association between the different types of educational measures, overall and by racial/ethnicity groups. Additionally, ORs and 95% CIs were calculated for children with only maternal information recorded on the birth certificate and for children having data for both parents.

To evaluate whether associations varied according to age at diagnosis, age-specific ORs and 95% CIs also were calculated for cancer types with sufficient number of study subjects. Categories of 0–4 and 5–14 years of age were created for most cancers. As higher SES is generally beneficial, age-specific associations were used as the referent category in the models. To assess the association between the different types of educational measurement (maternal, paternal and household), Pearson’s correlation coefficient was calculated for each pair. Correlation was calculated overall and within maternal race/ethnicity groups. Additionally, ORs and 95% CIs were calculated for children with only maternal information recorded on the birth certificate compared with children having data for both parents.

Table 2 Correlations between education measures, overall and by racial/ethnic group

| Measure | 1  | 2  | 3  |
|---------|----|----|----|
| All     |    |    |    |
| 1. Maternal education (continuous) | 1.00 |    |    |
| 2. Paternal education (continuous) | 0.71 | 1.00 |    |
| 3. Household education (continuous) | 0.89 | 0.88 | 1.00 |
| Non-Hispanic white* |    |    |    |
| 1. Maternal education (continuous) | 1.00 |    |    |
| 2. Paternal education (continuous) | 0.61 | 1.00 |    |
| 3. Household education (continuous) | 0.85 | 0.84 | 1.00 |
| Non-Hispanic black* |    |    |    |
| 1. Maternal education (continuous) | 1.00 |    |    |
| 2. Paternal education (continuous) | 0.65 | 1.00 |    |
| 3. Household education (continuous) | 0.88 | 0.84 | 1.00 |
| Hispanic* |    |    |    |
| 1. Maternal education (continuous) | 1.00 |    |    |
| 2. Paternal education (continuous) | 0.65 | 1.00 |    |
| 3. Household education (continuous) | 0.88 | 0.84 | 1.00 |

*aMaternal race/ethnicity.

RESULTS

Cases and controls varied slightly in maternal and paternal race/ethnicity, with more cases than controls having Hispanic parents (Table 1). Race/ethnicity reporting was also more complete for cases than for controls. The average age at diagnosis was 4.5 years and slightly more than half of both cases and controls were males. Almost all the children were singletons and 41% were the first live birth for the mother. The majority of parents were non-Hispanic white. Approximately one-third of the mothers were less than 25 years of age at the time of child’s birth, with fathers being slightly older.

When considering all study subjects together, the three measures of educational attainment were strongly correlated, however, this masked substantial differences by race/ethnicity (Table 2). Correlation between maternal and paternal education was lowest among non-Hispanic black parents ($r = 0.45$).

DISCUSSION

For the majority of childhood cancers evaluated, no association, either increasing or decreasing, was observed with the level of...
There was an indication of a positive association with lower educational attainment for both Hodgkin’s and Burkitt’s lymphomas and for Wilm’s tumour, with ORs of 1.5 to greater than 3.0 times that of more educated parents. In contrast, a protective effect of lower parental education was suggested for both astrocytoma and hepatoblastoma, with ORs being reduced by 30% to 40% compared with those whose parents were more educated. A significantly increased association was seen for rhabdomyosarcoma only when comparing children whose birth certificates had no paternal data with those that did.

The main strength of this study is the large sample size allowing the assessment of SES in many specific histological types; it

| Table 3 | ORs for educational status and childhood cancer, by ICCC3 major category (adjusted for maternal age, maternal race/ethnicity, parity, state, birth year, gestational age and birth weight) |
|---------|----------------------------------------------------------------------------------|
| Education level (in years) | All cancers | Leukemia | Lymphoma | CNS |
| Maternal | | | | |
| <12 | 1.01 (0.91, 1.13) | 1.04 (0.88, 1.24) | 1.38 (0.96, 1.99) | 0.86 (0.70, 1.05) |
| 12 | 1.00 (0.91, 1.10) | 1.08 (0.93, 1.25) | 1.08 (0.79, 1.49) | 0.83 (0.70, 0.98) |
| 13–16 | 0.97 (0.89, 1.06) | 1.06 (0.92, 1.22) | 1.11 (0.82, 1.52) | 0.81 (0.69, 0.96) |
| >17 | Reference | Reference | Reference | Reference |
| Paternal | | | | |
| <12 | 1.12 (1.00, 1.24) | 1.09 (0.93, 1.28) | 1.21 (0.85, 1.73) | 1.07 (0.88, 1.31) |
| 12 | 1.07 (0.98, 1.17) | 1.07 (0.94, 1.22) | 1.18 (0.88, 1.58) | 0.92 (0.78, 1.09) |
| 13–16 | 1.05 (0.96, 1.14) | 1.03 (0.91, 1.17) | 1.19 (0.90, 1.58) | 0.99 (0.85, 1.16) |
| >17 | Reference | Reference | Reference | Reference |
| Household | | | | |
| <12 | 1.05 (0.95, 1.16) | 1.04 (0.90, 1.22) | 1.26 (0.90, 1.76) | 0.95 (0.78, 1.16) |
| 12 | 1.03 (0.95, 1.12) | 1.02 (0.90, 1.15) | 1.00 (0.77, 1.31) | 0.92 (0.79, 1.08) |
| 13–16 | 1.00 (0.93, 1.08) | 1.01 (0.90, 1.13) | 1.15 (0.90, 1.46) | 0.94 (0.82, 1.07) |
| >17 | Reference | Reference | Reference | Reference |

Sympathetic nervous system

| Education level (in years) | Retinoblastoma | Renal tumours | Hepatic tumours |
| Maternal | | | |
| <12 | 0.94 (0.70, 1.26) | 1.27 (0.83, 1.94) | 1.26 (0.90, 1.78) | 0.85 (0.48, 1.49) |
| 12 | 1.03 (0.81, 1.32) | 1.17 (0.79, 1.72) | 1.29 (0.95, 1.73) | 0.70 (0.43, 1.14) |
| 13–16 | 1.04 (0.83, 1.31) | 1.13 (0.78, 1.65) | 1.22 (0.91, 1.62) | 0.67 (0.42, 1.06) |
| >17 | Reference | Reference | Reference | Reference |
| Paternal | | | |
| <12 | 1.07 (0.81, 1.42) | 1.48 (0.98, 2.24) | 1.38 (1.00, 1.91) | 1.01 (0.56, 1.81) |
| 12 | 1.10 (0.88, 1.38) | 1.45 (1.01, 2.10) | 1.32 (1.01, 1.73) | 1.00 (0.61, 1.64) |
| 13–16 | 1.08 (0.87, 1.34) | 1.31 (0.91, 1.87) | 1.13 (0.87, 1.47) | 0.93 (0.58, 1.49) |
| >17 | Reference | Reference | Reference | Reference |
| Household | | | |
| <12 | 1.00 (0.77, 1.32) | 1.20 (0.82, 1.75) | 1.25 (0.91, 1.71) | 0.94 (0.54, 1.62) |
| 12 | 1.07 (0.87, 1.32) | 1.29 (0.94, 1.78) | 1.32 (1.03, 1.70) | 0.95 (0.61, 1.49) |
| 13–16 | 1.02 (0.84, 1.23) | 1.17 (0.86, 1.58) | 1.21 (0.96, 1.52) | 0.80 (0.53, 1.21) |
| >17 | Reference | Reference | Reference | Reference |

Malignant bone

| Education level (in years) | Soft-tissue sarcomas | Germ cell | Other carcinomas |
| Maternal | | | |
| <12 | 0.88 (0.49, 1.61) | 1.03 (0.71, 1.49) | 1.08 (0.66, 1.79) | 1.56 (0.72, 3.36) |
| 12 | 0.71 (0.44, 1.14) | 0.96 (0.70, 1.32) | 1.01 (0.65, 1.57) | 1.23 (0.66, 2.28) |
| 13–16 | 0.69 (0.44, 1.09) | 0.92 (0.68, 1.24) | 0.83 (0.54, 1.28) | 0.79 (0.42, 1.46) |
| >17 | Reference | Reference | Reference | Reference |
| Paternal | | | |
| <12 | 1.40 (0.77, 2.55) | 1.09 (0.75, 1.59) | 0.97 (0.60, 1.57) | 0.75 (0.31, 1.77) |
| 12 | 0.94 (0.58, 1.52) | 1.07 (0.79, 1.45) | 1.02 (0.68, 1.53) | 1.37 (0.79, 2.37) |
| 13–16 | 1.15 (0.74, 1.80) | 1.14 (0.85, 1.51) | 0.96 (0.65, 1.42) | 0.75 (0.42, 1.31) |
| >17 | Reference | Reference | Reference | Reference |
| Household | | | |
| <12 | 1.23 (0.68, 2.24) | 1.16 (0.82, 1.66) | 1.00 (0.63, 1.59) | 1.51 (0.70, 3.27) |
| 12 | 0.89 (0.57, 1.37) | 1.05 (0.80, 1.39) | 1.03 (0.71, 1.50) | 1.50 (0.88, 2.55) |
| 13–16 | 0.94 (0.64, 1.38) | 1.07 (0.84, 1.38) | 0.84 (0.59, 1.19) | 0.86 (0.51, 1.43) |
| >17 | Reference | Reference | Reference | Reference |

Abbreviations: CI = confidence interval; CNS = central nervous system; ICCC3 = International Classification of Childhood Cancer third edition; OR = odds ratio.
interpretation of our results must also be viewed within the context of limited but pertinent information about SES conveyed by parental educational level. Numerous studies have confirmed that the health and welfare of children is associated with parental educational attainment, with parental education level often a stronger predictor of children's health than family income, number of parents or size of the family (Zill, 1996). With respect to any SES-related risk factors operating in childhood cancers and whether they are captured by educational measures, it seems reasonable to hypothesize that education can influence the likelihood of particular occupational exposures of either parent, for example, through the employment opportunities available to those with and without high school diplomas. Specific health behaviours are influenced by educational level as well, such as awareness of and access to prenatal care, smoking tobacco products, specific dietary choices and compliance with immunization schedules (Zill, 1996). All these factors may be associated with risk for specific types of childhood cancers, with components of one or more acting either as initiators and/or promoters of carcinogenesis.

There is no single gold standard indicator of SES (Krieger et al., 1997; Daly et al., 2002; Braveman et al., 2005; Galobardes et al., 2006) and education is a commonly used indicator of overall SES in epidemiology; it is an attempt to capture knowledge-related assets (Galobardes et al., 2006). Household educational attainment has been recommended for studies of child development (Hauser, 1994) and may be the most appropriate approach for childhood diseases. A simple determination of years of schooling completed, however, contains no information about its quality, which may be important for health outcomes specifically related to knowledge, cognitive skills and analytical abilities. Quality of education may be less important, however, as a broad indicator of SES (Galobardes et al., 2006), as we propose in this study. Finally, our analysis considers SES around the time of birth, but for some cancers, SES during childhood or nearer to the time of diagnosis might be more relevant.

Our findings are consistent with a long-recognized association between increased Hodgkin's lymphoma incidence in childhood and markers of lower socioeconomic class (Correa and O'Connor, 1971; Gutensohn and Shapiro, 1982). In contrast, Hodgkin's lymphoma in young adults has been associated with markers of higher early childhood SES, which has been thought to reflect responses to infection, with the more protected environment during childhood or nearer to the time of diagnosis might be more relevant.

Abbreviations: ALL = acute lymphoblastic leukemia; AML = acute myeloid leukemia; CI = confidence interval; CNS = central nervous system; NHL = non-Hodgkin's lymphoma; OR = odds ratio; SES = socioeconomic status. *Observations with some paternal data as referent; adjusted for maternal age group, maternal race/ethnicity, sex, state, birth year, birth order, birth weight and gestational age.

Table 4 Risk of childhood cancer associated with missing paternal data as SES indicator, by cancer type

| Cancer type                  | No. of cases with no paternal data | OR 95% CI |
|-----------------------------|-----------------------------------|-----------|
| Total cancers               | 920                               | 0.95 0.86 1.04 |
| Leukemia                    | 262                               | 0.86 0.74 1.01 |
| ALL                         | 209                               | 0.86 0.72 1.03 |
| AML                         | 38                                | 0.82 0.56 1.20 |
| Lymphoma                    | 71                                | 0.80 0.59 1.09 |
| Hodgkin's lymphoma          | 22                                | 1.06 0.60 1.87 |
| NHL                         | 27                                | 0.86 0.55 1.37 |
| Burkitt's                   | 9                                 | 0.47 0.20 1.12 |
| CNS                         | 183                               | 0.86 0.72 1.04 |
| Ependymoma                  | 18                                | 0.64 0.36 1.13 |
| Astrocytoma                 | 86                                | 1.00 0.76 1.31 |
| Intracranial embryonal      | 39                                | 0.81 0.55 1.17 |
| Other gliomas               | 26                                | 0.88 0.54 1.44 |
| Sympathetic nervous system  | 94                                | 1.10 0.87 1.40 |
| Neuroblastoma               | 93                                | 1.10 0.86 1.40 |
| Retinoblastoma              | 49                                | 1.01 0.71 1.42 |
| Renal tumours               | 81                                | 0.99 0.75 1.29 |
| Wilms tumor                 | 79                                | 0.97 0.74 1.28 |
| Hepatic tumours             | 30                                | 1.23 0.77 1.97 |
| Hepatoblastoma              | 24                                | 1.14 0.68 1.90 |
| Bone tumours                | 25                                | 1.10 0.66 1.85 |
| Osteosarcoma                | 15                                | 0.90 0.43 1.89 |
| Ewing's sarcoma             | 6                                 | 0.87 0.34 2.22 |
| Soft-tissue sarcomas        | 73                                | 1.40 1.05 1.87 |
| Rhabdomyosarcoma            | 45                                | 1.59 1.11 2.29 |
| Germ-cell tumours           | 30                                | 0.91 0.58 1.43 |
| Extrarenal germ-cell tumours| 8                                 | 0.95 0.42 2.18 |
| Gonadal germ-cell tumours   | 15                                | 1.06 0.58 1.91 |
| Other epithelial            | 15                                | 1.04 0.54 2.01 |

Therefore adds appreciably to the information on rare childhood cancers. Additional strengths include our use of population-based cancer surveillance data, of prospectively collected birth records and use of population-based passive control selection.

Several limitations should be noted, including the effects of multiple comparisons; significant results in the subgroup analyses in particular could be because of chance alone. There is also the potential for error in birth certificate-based data, as items on these records have varying levels of accuracy. In a study comparing mothers' responses in a national natality survey with those on linked birth certificates, a 77% concordance was reported on maternal education overall between the two data sources, with an additional 15% reporting a difference of only 1 year (Querec, 1980). A 72% concordance was found for reported paternal education. Higher agreement was found when considering the education data in categories, with 90% agreement for 12 years (i.e. high school diploma) of maternal education and 86% for the same cut-point in paternal education. The report concluded that grouping educational data into categories marked by certificates or diplomas was likely to result in the least bias. Most variables used to adjust for confounding, such as maternal age, birth plurality and birth weight, have consistently been found to be accurate in validation studies (Northam and Knapp, 2006).

Finally, our analysis considers SES around the time of birth, but for some cancers, SES during childhood or nearer to the time of diagnosis might be more relevant.
There is little variation in the incidence of Wiln's tumour between developed and developing countries (Bunin, 2004), although an ecological study found evidence of increased Wiln's tumour incidence in more-affluent areas (McNally et al., 2003). These results are in contrast to ours; the methodologies used were also quite different and do not allow for direct comparisons. The aetiology of childhood brain tumours remains unclear. The International Agency for Research on Cancer has assembled the largest case–control population for the study of childhood brain tumours to date. With the exception of farm-related exposures and some paternal occupations, little evidence of an association with any environmental factor has been reported (McCredie et al., 1999; Filippini et al., 2002; Efird et al., 2003; Cordier et al., 2004; Myoller et al., 2004; Cardy et al., 2006). Current hypotheses of astrocytoma aetiology do not posit a protective effect of lower SES (Baldwin and Preston-Martin, 2004) and therefore, our finding of lower incidence among children born to mothers with lower educational attainment should be interpreted with reasonable caution.

One study found that parental occupational exposures may increase the risk of hepatoblastoma in offspring (Buckley et al., 1989), but the relevant exposures (e.g. metals, petroleum products, paints/pigments) would more likely be encountered in lower education level/manual labour jobs. The association of parental smoking with hepatoblastoma (Pang et al., 2003; Sorahan and Lancashire, 2004) may partly reflect uncontrolled confounding from low birth weight (Spector and Ross, 2003), with which there is convincing evidence of a strong inverse association (Spector et al., 2008). However, as smoking rates are generally higher among lower SES groups (Orleans, 2003), confounding by smoking would not explain the protective effect suggested for maternal education seen in our data. Our finding of an increased rhabdomyosarcoma OR for children with no paternal data on the birth certificate (i.e. potentially lower SES) mirrors the inverse association of rhabdomyosarcoma with increasing SES indicators found in a small case–control study (Grufferman et al., 1982). However, there was no such pattern when specific levels of parental education were evaluated.

Our finding of no pattern of association with childhood leukaemia is of note, given that it has probably been most thoroughly studied for SES associations. Results have been heterogeneous and have varied by measures of SES used, calendar time of study, geographic location and study design (Poole et al., 2006; Adam et al., 2008) there is no clear evidence of an association between SES and childhood leukaemia.

Our findings suggest that deprivation patterns, that are generally seen when comparing nations of differing economic levels (e.g. Hodgkin’s and Burkitt’s lymphoma), may be reproduced within the social strata of one nation. Our results should be viewed as exploratory because of the broad nature of the SES assessment, but they do suggest that studies of childhood cancers might benefit from a more thorough evaluation of the child’s SES.

Supplementary Information accompanies the paper on British Journal of Cancer website (http://www.nature.com/bjc)

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