NIH Public Access
Author Manuscript
*Cancer Causes Control*. Author manuscript; available in PMC 2009 December 1.

Published in final edited form as:
*Cancer Causes Control*. 2008 December ; 19(10): 1201–1207. doi:10.1007/s10552-008-9189-7.

# A CASE-CONTROL STUDY OF CHILDHOOD BRAIN TUMORS AND FATHERS' HOBBIES — A CHILDREN'S ONCOLOGY GROUP STUDY

**Andrea L. Rosso**[1,2], **Mary E. Hovinga**[1], **Lucy B. Rorke-Adams**[3,4], **Logan G. Spector**[5,6], and **Greta R. Bunin**[4,7]

1*Department of Epidemiology and Biostatistics, Drexel University School of Public Health, Philadelphia, PA*

2*Department of Neurology, Drexel University College of Medicine, Philadelphia, PA*

3*Department of Pediatrics, University of Pennsylvania School of Medicine, Philadelphia, PA*

4*Department of Pathology and Department of Neurology, Children's Hospital of Philadelphia, Philadelphia, PA*

5*Division of Epidemiology and Clinical Research, Department of Pediatrics, University of Minnesota, Minneapolis, MN*

6*University of Minnesota Cancer Center, Minneapolis, MN*

7*Division of Oncology, Children's Hospital of Philadelphia, Philadelphia, PA*

## Abstract

**Objective—**A comprehensive case-control study was conducted to evaluate parental risk factors for medulloblastoma (MB) and primitive neuroectodermal tumor (PNET). This analysis was conducted to evaluate associations between fathers' hobbies and risk of their children developing MB/PNET. The hobbies chosen for study were those with similar exposures as occupations associated with childhood cancers.

**Methods—**Cases were 318 subjects under 6 years of age at diagnosis between 1991-1997 and registered with the Children's Cancer Group. An equal number of controls were selected through random digit dialing and individually matched to cases.

**Results—**In multivariate analyses, a significant association was seen for lawn care with pesticides [during pregnancy: odds ratio (OR) = 1.6, 95% confidence interval (CI): 1.0, 2.5; after birth: OR = 1.8, 95% CI: 1.2, 2.8] and a weak association was seen for stripping paint [during pregnancy: OR = 1.4, 95% CI: 0.8, 2.6; after birth: OR = 1.4, 95% CI: 0.7, 2.6].

**Conclusions—**This study suggests that household exposures from hobbies, particularly pesticides, may increase risk of MB/PNET in children; previous research has been mostly limited to occupational exposures.

### Keywords

case-control studies; brain neoplasms; medulloblastomas; child preschool; infant; hobbies; pesticides

Correspondence should be addressed to: Greta Bunin Children's Hospital of Philadelphia 3535 Market St., Rm. 1472 Philadelphia, PA 19104.

NIH-PA Author Manuscript
NIH-PA Author Manuscript
NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

## INTRODUCTION

Little is known about the etiologies of brain tumors that develop in early childhood. Medulloblastoma (MB) and primitive neuroectodermal tumor (PNET), histologically similar tumors, are the second most common type of pediatric central nervous system (CNS) cancers and have their highest incidence between birth and four years (1). MB is the more common of the two, accounting for approximately 80% of the combined total (2).

Some genetic syndromes are known to greatly increase the risk of brain cancer in children, but these mutations are rare and account for fewer than 5% of all cases (3). Recent observational studies of childhood CNS tumors have reported associations for a number of environmental factors, including maternal diet and parental heat exposure (3-6), although the evidence is limited or inconsistent. In several studies, paternal occupational exposures, including ionizing radiation, solvents and pesticides (3,7-11), which could also be encountered during a father's participation in a hobby, have been implicated.

There are at least three pathways by which paternal exposures can lead to adverse outcomes in children: mutagenic or epigenetic changes to the sperm prior to conception, transfer of chemicals to the mother during conception or pregnancy, and direct transfer to the child after birth. All three of these modes have been shown in animal studies to result in adverse outcomes in offspring (12-14).

In this study, we examined the association between fathers' hobbies that could result in potentially toxic exposures with their children's risk of developing MB or PNET.

## MATERIALS AND METHODS

The study design and methods for subject selection have been described in detail elsewhere (5). Briefly, the study (Children's Cancer Group protocol E21) enrolled eligible participants who were diagnosed between July 1991 and November 1997 with MB or PNET before 6 years of age. All subjects were registered with the Children's Cancer Group (CCG), one of the four pediatric oncology cooperative groups that later merged to form the Children's Oncology Group (COG). At the time of the study, about 100 medical centers participated in the CCG and were estimated to diagnose and/or treat approximately half of all pediatric cancers in the US and Canada.

The institutional review boards of all participating institutions granted approval of the study. All subjects gave verbal informed consent to participate at the time of the telephone interview.

### Study Participants

A total of 558 eligible cases registered with CCG were identified. Some of these were excluded for reasons including: another diagnosed cancer before or at the time of MB/PNET diagnosis (n=3), inability to locate the family (n=48), lack of physician consent (n=35), pathologic review revealing misdiagnosis (n=28), biological mother not available (n=17), presence of a language barrier (n=16), lack of a household phone (n=13), residence outside of North America (n=6), no response from the institution (n=2) or refusal by the family (n=66). Random-digit dialing was used to enroll control mothers. Controls were matched to cases on area code, race, and date of birth (within six months for cases diagnosed at age less than one year and within one year for all others). Controls could not be found for 6 of the cases. The final sample included 318 case mothers (73% of eligible cases) and an equal number of controls. Information on the fathers of 283 cases (89% of the 318 cases) and 262 controls (82% of the 318 controls) was collected and analyzed.

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

## Data Collection

Data collection was accomplished through structured phone interviews conducted for all cases and controls by trained interviewers. The median time between the child's diagnosis and the interview was 1.6 years (range: 0.1-6.2 years). All mothers and as many fathers as possible were interviewed directly. When the father was not available for an interview, the mother often completed it in his place; 28 (10%) of the case fathers and 63 (24%) of the control fathers were interviewed by proxy. Information was collected on a variety of possible exposures and confounders potentially related to MB and PNET.

## Primary Exposure - Hobbies

Hobbies were selected based on associated exposures that have been hypothesized to cause brain tumors, including pesticides, solvents, paints and pigments, electromagnetic fields, and lead. The hobbies were stripping paint from furniture or woodwork; car repair; home repair or remodeling; painting (either artistic or home); pottery, ceramics, or glazing; photographic developing; gardening or lawn care using insecticides, bug, or weed killer; model building; silk screen printing or painting; electronics or ham radio operation; TV, radio, stereo or other electronic repair; sewing with a sewing machine; working with large power tools such as a lathe, table saw or band saw; target shooting or hunting. For questions regarding hobbies, fathers were asked whether or not they participated during the pregnancy or from the child's birth until the child's reference age and about their frequency of participation during each time period. For pregnancy exposures, fathers were asked about the time period starting the month before the pregnancy in order to capture very early pregnancy.

Not all hobbies had sufficient participation to allow analysis. Only those with more than 50 total participants in each time period were included in further analyses. Hobby participation during these two time periods was highly correlated, so it was not possible to include participation during both time periods in a single analysis.

## Statistical Analyses

Demographic attributes and potential confounders were compared between cases and controls by either t-tests for continuous variables or chi-square tests for categorical ones.

Logistic regression was used to determine crude odds ratios (OR), ORs adjusted for potential confounders and 95% confidence intervals (CI) for each hobby. Exposure was defined as a 'yes' answer to the question regarding a specific hobby. In analyses to investigate dose-response, frequency was categorized as never, less than once per month or once per month or more and the categories were analyzed as indicator variables. Analyses stratified by reference age (defined below) above or below the median of 23 months were performed to determine if risk differed for older compared to younger children.

Reference age was related to the age of diagnosis and represented an estimate of the age of onset for the tumor. Those with a diagnosis before 5 months of age were considered to have a reference age at birth. The reference ages for the remaining cases were defined as follows: 3 months for those diagnosed at 6-11 months; 6 months for those diagnosed 12 – 17 months; 9 months for those diagnosed at 18-23 months; age of diagnosis minus 12 months for those diagnosed at 24-59 months. Controls were assigned the reference age of their matched case. Children with a reference age at birth were excluded from all after birth exposure analyses.

In an attempt to determine whether recall bias might explain our results, an analysis was performed which excluded the least frequent participants (< 5 times during the time period). Recall bias is thought to operate in childhood diseases by more complete reporting by case parents compared to control parents (15). One possibility is that case parents report infrequent

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

or incidental exposures more completely than do control parents. It may be possible to assess the amount of recall bias that is present in the study by comparing the ORs with or without the least frequent participants.

Controls were matched to cases based on date of birth, race, and area code; however, not all case-control pairs had interviews for both fathers, resulting in a reduced sample size if only matched pairs were used. Therefore, unconditional logistic regression was performed on all subjects for whom a paternal interview was available with matching variables included as confounders. Child's race was defined as either white or other and area codes were categorized into one of five geographic regions. Due to the time lag between interviewing case fathers and finding and interviewing a matched control father, control interviews took place at a later date than those of their matched cases and controls were older on average. Inclusion of the three variables, the child's date of birth (a matching factor), the date of the father's interview and the child's age at interview, was not possible due to multicollinearity. Therefore, only two variables — the date of the father's interview and the child's age at interview — were included in analyses.

The following variables were also assessed for inclusion in the regression models: whether a proxy interview was conducted, gender of the child, father's smoking status, mother's smoking status, father's alcohol use, father's heat exposure, father's age at the child's birth, father's marital status, father's educational level, the size of the household, the number of household moves, father's income, and the season of the child's birth. Variables were included as confounders if they were associated with both the disease outcome and the exposure and if they were either significant ($\alpha$=0.05) in the regression or changed the OR by more than fifteen percent. The final model included father's race, geographic region, date of father's interview, child's age at interview, whether a proxy interview was conducted, the child's gender, father's smoking status and the child's birth season.

All analyses were performed using SPSS version 14.0 for Windows.

## RESULTS

Fathers of cases and controls were similar with respect to race, age at child's birth, residential mobility, alcohol use, marital status and educational attainment (Table 1). Cases were less likely to have had the mother complete a proxy interview for the father, were more likely to be male and more likely to come from smaller households. Fathers of cases smoked less and had higher incomes. Control fathers were interviewed later in the study and control children were older at the time of the interview. More cases than controls were born in summer compared to the fall, with winter and spring being intermediate.

Lawn care before and after the child's birth was associated with increased risk of MB/PNET development (during pregnancy: OR = 1.6, 95% CI: 1.0, 2.4; after birth: OR = 1.7, 95% CI: 1.1, 2.6; Table 2). Weak, non-significant associations were seen for stripping paint and hunting in at least one time period.

Analyses of frequency of hobby participation (see Table 3) did not show increased risk with increased frequency of participation.

For several of the hobbies, there was a trend towards higher risk for older children (those with a later reference age), particularly for exposures during the pregnancy. The most striking differences were seen for stripping paint during the pregnancy ($OR_{<23\ months}$ = 1.3, 95% CI: 0.6, 2.9; $OR_{>23\ months}$ = 1.9, 95% CI: 0.7, 5.1), lawn care (during pregnancy: $OR_{<23\ months}$ = 1.3, 95% CI: 0.7, 2.5; $OR_{>23\ months}$ = 1.9, 95% CI: 1.0, 3.7; after birth: $OR_{<23\ months}$ = 1.3,

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

95% CI: 0.7, 2.5; $OR_{>23\ months}$ = 2.0, 95% CI: 1.1, 3.8), and painting during the pregnancy ($OR_{<23\ months}$ = 0.9, 95% CI: 0.5, 1.7; $OR_{>23\ months}$ = 1.6, 95% CI: 0.8, 2.9).

In analyses to assess potential recall bias, there were generally no appreciable differences when including or excluding the least frequent participants (data not shown). The only hobby that showed much evidence of recall bias was lawn care during pregnancy ($OR_{with\ <5x\ group}$ = 1.6, 95% CI: 1.0, 2.4; $OR_{without\ <5x\ group}$ = 1.3, 95% CI: 0.7, 2.3).

## DISCUSSION

We found suggestive associations between several paternal hobbies and risk of a child developing MB or PNET in the first 6 years of life. The strongest associations were seen for lawn care with pesticides both during the pregnancy and after the child's birth; however, no dose-response relationship was observed and there was evidence that recall bias might partly explain the association. Possible weak associations were seen for stripping paint from furniture or woodwork and for hunting.

The presumed exposures of interest for these hobbies are: pesticides from lawn care, solvents from stripping paint, and lead and cleaning solvents from hunting. The results of this study are consistent with and support findings from other studies that have looked at parental pesticide exposure and childhood brain tumors. Many pesticides readily cross the blood-brain barrier and are intentionally neurotoxic. Laboratory studies have shown that some pesticides can cross the placenta, damage DNA and initiate tumorigenic changes in fetal cells (reviewed in Baldwin & Preston-Martin, 2004 (3)). A number of studies have found elevated risk for MB/PNET and other pediatric CNS cancers among children whose fathers have experienced pesticide exposures either occupationally or in the home (3,7,8). However, null associations have also been found for both occupational and residential exposures (7). Much of the research in this area has focused on occupational rather than residential exposures, but the strongest associations have been found for household pesticide use prior to or just after birth (3). Most of the positive risk estimates for residential pesticide use have been on the order of 1.5 – 2.5 (7), comparable to those found in this study.

The previous evidence for solvent and lead exposures has been less clear than that for pesticides. Mixed results have been found with some studies showing positive associations and others showing none (3,9-11). This study adds to the suggestive but inconclusive evidence regarding solvent related exposures. To our knowledge, no previous studies have looked at possible associations between lead exposure and MB or PNET development. Lead is known to cross the developing blood-brain barrier (3) and to induce potentially carcinogenic chromosomal aberrations (16). In addition, occupational lead exposure has been shown to increase risk of brain tumors in adults (17,18). Other research has shown significantly elevated levels of lead in the cerebral spinal fluid (CSF) of those with malignant tumors compared to control groups, with MB showing the highest mean CSF lead levels (19).

More frequent hobby participation was not associated with higher risk estimates. There are a number of possible reasons for the lack of a dose-response relationship, including that small sample sizes limited the ability to detect these differences. It is also possible that the lack of a dose-response relationship is indicative of bias in the results rather than a true positive association.

The analyses by age of the child found generally higher odds ratios for the children who were older at diagnosis. This suggests that the environmental exposures measured here may manifest themselves primarily in older children, rather than in children diagnosed shortly after birth, possibly due to a necessary latency period between exposure and disease onset. However, these

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

results must be interpreted with caution due to the small numbers and the lack of statistical significance.

The primary limitation of this study is the retrospective data collection based on paternal recall, which can result in recall bias or non-differential misclassification of exposure. There is some evidence from our analyses that recall bias was not a major concern here, but its effects on the results can not be ruled out. The presence of non-differential misclassification due to the time lag between exposures and reporting would generally be expected to bias the risk estimates towards the null.

Another limitation of this study was the broad nature of the questions. A father's participation in a hobby could result in exposure to many substances. In addition, the exposures encountered during a hobby may not be the same for all participants. The broad nature of these questions would have acted to dilute risk estimates that may otherwise have been positive for specific exposures. On the other hand, asking such broad questions related to hobbies, rather than very detailed questions related to specific exposures, may have resulted in more accurate recall on the part of the study subjects. Data were not available on maternal hobbies or for either parent's occupational exposures. Therefore, we can not account for exposures that may have resulted from maternal household activities or from either parent's work-related exposures. This may have added to misclassification by exposure.

Further consideration must be given to the population used in these analyses. Only children who had been patients at a medical center participating in the CCG were enrolled in this study. Selection bias would have occurred if children with MB/PNET seen at CCG institutions differed from other children with MB/PNET. However, data were not available to assess potential differences and thus, we cannot assess the likelihood of selection bias due to case ascertainment through CCG. Further selection pressures arise from paternal response rates and use of mothers as proxy respondents. The overall response rate from fathers of identified eligible cases was 65%. Proxy interviews were conducted for a sizable minority of the fathers, particularly among controls. Use of these interviews could be expected to increase the probability of misclassification of exposure. Analyses were performed excluding proxy interviews and no differences from the reported results were seen.

These data on father's hobbies prior to and after birth and his child's risk of developing MB or PNET in the first 6 years of life provide some suggestive results and directions for future research. Combined with data from other studies, these results are suggestive of an association between residential pesticide exposures and brain tumor risk in children. This association was unlikely to have been entirely explained by confounders or bias, although these must be considered as possible explanations for the results. This study also suggests a possible role for stripping paint and hunting which should be further explored in future studies.

## REFERENCES

1. Gurney, JG.; Smith, MA.; Bunin, GR. CNS and Miscellaneous Intracranial and Intraspinal Neoplasms ICCCIII. In: L.A.G., R.; Smith, MA.; Gurney, JG.; Linet, M.; Tamra, T.; Young, JL., et al., editors. Cancer Incidence and Survival among Children and Adolescents: United States SEER Program 1975-1995. National Cancer Institute, SEER Program; Bethesda, MD: 1999.
2. Surveillance, Epidemiology, and End Results (SEER) Program public-use data (1973-2001) [database on the Internet]. Cancer Statistics Branch, Surveillance Research Program, Division of Cancer Control and Population Sciences, National Cancer Institute. 2004. [cited. Available from: www.seer.cancer.gov.
3. Baldwin RT, Preston-Martin S. Epidemiology of brain tumors in childhood--a review. Toxicology and Applied Pharmacology 2004;199(2):118–31. [PubMed: 15313584]

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript


4. Bunin GR, Gallagher PR, Rorke-Adams LB, Robison LL, Cnaan A. Maternal Supplement, Micronutrient, and Cured Meat Intake during Pregnancy and Risk of Medulloblastoma during Childhood: A Children's Oncology Group Study. Cancer, Epidemiology, Biomarkers, and Prevention 2006;15(9):1660–7.
5. Bunin GR, Kushi LH, Gallagher PR, Rorke-Adams LB, McBride ML, Cnaan A. Maternal Diet during Pregnancy and Its Association with Medulloblastoma in Children: A Children's Oncology Group Study (United States). Cancer Causes and Control 2005;16:877–91. [PubMed: 16132798]
6. Bunin GR, Robison LL, Biegel JA, Pollack IF, Rorke-Adams LB. Parental Heat Exposure and Risk of Childhood Brain Tumor: A Children's Oncology Group Study. American Journal of Epidemiology 2006;164(3):222–31. [PubMed: 16775044]
7. Zahm SH, Ward MH. Pesticides and Childhood Cancer. Environmental Health Perspectives 1998;106 (S3):893–908. [PubMed: 9646054]
8. Pogoda JM, Preston-Martin S. Household Pesticides and Risk of Pediatric Brain Tumors. Environmental Health Perspectives 1997;105(11):1214–20. [PubMed: 9370522]
9. Feychting M, Plato N, Nise G, Ahlbom A. Paternal Occupational Exposures and Childhood Cancer. Environmental Health Perspectives 2001;109:193–6. [PubMed: 11266332]
10. Cordier S, Lefeuvre B, Filippini G, Peris-Bonet R, Farinotti M, Lovicu G, et al. Parental occupation, occupational exposures to solvents and polycyclic aromatic hydrocarbons and risk of childhood brain tumors (Italy, France, Spain). Cancer Causes and Control 1997;8(5):688–97. [PubMed: 9328190]
11. IARC. IARC monographs on the evaluation of carcinogenic risks to humans. Vol. volume 65. World Health Organization; Lyon, France: 1995.
12. Olshan AF, Faustman EM. Male-Mediated Developmental Toxicity. Annual Review of Public Health 1993;14(1):159–81.
13. Wyrobek AJ. Methods and concepts in detecting abnormal reproductive outcomes of paternal origin. Reproductive Toxicology 1993;7(Supplement 1):3–16. [PubMed: 8400638]
14. Friedler G. Paternal exposures: Impact on reproductive and developmental outcome. An overview. Pharmacology Biochemistry and Behavior 1996;55(4):691–700.
15. Neugebauer R, Ng S. Differential Recall as a Source of Bias in Epidemiologic Research. Journal of Clinical Epidemiology 1990;43(12):1337–41. [PubMed: 2254770]
16. Vainio H, Sorsa M. Choromsome Aberrations and Their Relevance to Metal Carcinogenesis. Environmental Health Perspectives 1981;40:173–80. [PubMed: 7023931]
17. Cocco P, Dosemeci M, Heineman EF. Brain Cancer and Occupational Exposure to Lead. Journal of Occupational and Environmental Medicine 1998;40(11):937–42. [PubMed: 9830598]
18. Rajaraman P, Stewart PA, Samet JM, Schwartz BS, Linet MS, Zahm SH, Rothman N, Yeager M, Fine HA, Black PM, Loeffler J, Shapiro WR, Selker RG, Inskip PD. Lead, Genetic Susceptibility, and Risk of Adult Brain Tumors. Cancer Epidemiology Biomarkers and Prevention 2006;15(12): 2514–20.
19. El-Yazigi A, Al-Saleh I, Al-Mefty O. Concentrations of Ag, Al, Au, Bi, Cd, Cu, Pb, Sb, and Se in Cerebrospinal Fluid of Patients with Cerebral Neoplasms. Clinical Chemistry 1984;30(8):1358–60. [PubMed: 6744584]

NIH-PA Author Manuscript
NIH-PA Author Manuscript
NIH-PA Author Manuscript



**Table 1**

Demographic characteristics of fathers of children diagnosed with medulloblastoma or PNET in the first 6 years of life and random digit dial controls; Children's Cancer Group, 1991-1997.

| | Cases (n=283) | | Controls (n=262) | |
|---|---|---|---|---|
| **Characteristic** | **N** | **%** | **N** | **%** |
| Proxy interview by mother | | | | |
| *Yes* | 30 | 11 | 65 | 25 |
| *No* | 253 | 89 | 197 | 75 |
| Child's gender | | | | |
| *Male* | 167 | 59 | 125 | 48 |
| *Female* | 116 | 41 | 125 | 48 |
| Father's race | | | | |
| *White* | 244 | 86 | 230 | 88 |
| *Other*[*] | 39 | 14 | 32 | 12 |
| Father's education | | | | |
| *High school or less* | 102 | 36 | 110 | 42 |
| *Beyond high school* | 181 | 64 | 152 | 58 |
| Father's marital status | | | | |
| *Married* | 242 | 86 | 226 | 86 |
| *Other* | 38 | 14 | 34 | 13 |
| Child's birth season | | | | |
| *Winter* | 64 | 23 | 64 | 24 |
| *Spring* | 73 | 26 | 59 | 23 |
| *Summer* | 76 | 27 | 58 | 22 |
| *Fall* | 70 | 25 | 81 | 31 |
| Mother smoked during pregnancy | | | | |
| *Yes* | 44 | 16 | 55 | 21 |
| *No* | 239 | 84 | 207 | 79 |

| | N | mean(SD) | N | mean(SD) |
|---|---|---|---|---|
| Father's income (x$10,000) | 276 | 4.4 (2.7) | 244 | 3.9 (2.4) |
| Father's age at child's birth | 282 | 30.7 (6.5) | 262 | 30.6 (5.8) |
| Household size | 264 | 3.8 (1.1) | 259 | 4.0 (1.3) |
| Number of moves | 279 | 1.0 (1.4) | 230 | 1.1 (1.3) |
| Father's smoking during pregnancy (cigarettes/day) | 274 | 4.2 (9.2) | 258 | 6.9 (11.8) |
| Father's alcohol consumption (drinks/month) | 283 | 11.6 (14.8) | 252 | 14.0 (20.0) |
| Child's age at interview (years) | 283 | 4.9 (2.1) | 261 | 5.6 (2.3) |
| Date interviewed (months since start of study) | 283 | 22.4 (10.5) | 262 | 30.0 (8.7) |

| | N | mean(SD) | N | mean(SD) |
|---|---|---|---|---|

*'Other' includes 23 non-Hispanic black, 35 Hispanic, 9 Asian and 4 other.

NIH-PA Author Manuscript
NIH-PA Author Manuscript
NIH-PA Author Manuscript

NIH-PA Author Manuscript NIH-PA Author Manuscript NIH-PA Author Manuscript



**Table 2**

Participation in hobbies, adjusted odds ratios and 95% confidence intervals for fathers of children diagnosed with medulloblastoma or PNET in the first 6 years of life and random digit dial controls; Children's Cancer Group, 1991-1997.

| | | Cases (n = 283) | | Controls (n = 262) | | | |
|---|---|---|---|---|---|---|---|
| **Hobby** | | **N** | **%** | **N** | **%** | **Adjusted OR[a]** | **95% CI** |
| Stripping paint | pregnancy | 40 | 14 | 26 | 10 | 1.4 | 0.8, 2.6 |
| | after birth | 38 | 14 | 21 | 8 | 1.3 | 0.7, 2.6 |
| Car repair | pregnancy | 131 | 47 | 122 | 47 | 1.0 | 0.6, 1.4 |
| | after birth | 121 | 45 | 117 | 47 | 0.9 | 0.6, 1.4 |
| Home repair | pregnancy | 84 | 30 | 89 | 34 | 0.8 | 0.5, 1.2 |
| | after birth | 76 | 28 | 86 | 34 | 0.7 | 0.5, 1.1 |
| Painting | pregnancy | 109 | 39 | 82 | 32 | 1.2 | 0.8, 1.8 |
| | after birth | 96 | 35 | 92 | 37 | 0.8 | 0.5, 1.2 |
| Lawn care | pregnancy | 105 | 37 | 67 | 26 | 1.6 | 1.0, 2.4 |
| | after birth | 111 | 41 | 70 | 28 | 1.7 | 1.1, 2.6 |
| Power tools | pregnancy | 39 | 14 | 41 | 16 | 0.9 | 0.5, 1.5 |
| | after birth | 38 | 14 | 40 | 16 | 0.8 | 0.4, 1.4 |
| Hunting | pregnancy | 71 | 25 | 61 | 24 | 1.3 | 0.8, 2.0 |
| | after birth | 68 | 25 | 54 | 21 | 1.4 | 0.9, 2.3 |

[a]Adjusted for father's race, geographic location, date of father's interview, child's age at interview, whether a proxy interview was conducted, the child's gender, father's smoking status and the child's birth season.

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

**Table 3**

Adjusted odds ratios and 95% confidence intervals for hobbies of fathers of children diagnosed with medulloblastoma or PNET in the first 6 years of life and random digit dial controls by frequency of participation; Children's Cancer Group, 1991-1997.

| Hobby | n - cases | n — controls | OR[a] | 95% CI |
|---|---|---|---|---|
| *During Pregnancy* | | | | |
| Stripping paint <1/mo | 32 | 21 | 1.5 | 0.7, 2.9 |
| >1/mo | 8 | 5 | 1.4 | 0.4, 4.8 |
| Car repair <1/mo | 78 | 77 | 0.8 | 0.5, 1.3 |
| >1/mo | 53 | 45 | 1.3 | 0.8, 2.3 |
| Home repair <1/mo | 48 | 43 | 0.8 | 0.5, 1.3 |
| >1/mo | 36 | 46 | 0.7 | 0.4, 1.3 |
| Painting <1/mo | 90 | 65 | 1.2 | 0.8, 1.9 |
| >1/mo | 19 | 16 | 1.1 | 0.5, 2.4 |
| Lawn care <1/mo | 82 | 49 | 1.7 | 1.0, 2.7 |
| >1/mo | 23 | 18 | 1.3 | 0.6, 2.7 |
| Power tools <1/mo | 17 | 16 | 0.8 | 0.4, 1.8 |
| >1/mo | 22 | 25 | 0.9 | 0.5, 1.9 |
| Hunting <1/mo | 38 | 38 | 1.0 | 0.6, 1.8 |
| >1/mo | 33 | 23 | 1.6 | 0.8, 3.1 |
| *After Birth* | | | | |
| Stripping paint <1/mo | 29 | 15 | 1.4 | 0.6, 2.9 |
| >1/mo | 7 | 5 | 1.1 | 0.3, 4.0 |
| Car repair <1/mo | 70 | 72 | 0.7 | 0.5, 1.2 |
| >1/mo | 50 | 44 | 1.3 | 0.7, 2.2 |
| Home repair <1/mo | 42 | 39 | 0.8 | 0.4, 1.3 |
| >1/mo | 34 | 44 | 0.7 | 0.4, 1.3 |
| Painting <1/mo | 80 | 75 | 0.9 | 0.6, 1.4 |
| >1/mo | 16 | 17 | 0.6 | 0.3, 1.3 |
| Lawn care <1/mo | 79 | 52 | 1.6 | 1.0, 2.7 |
| >1/mo | 31 | 18 | 1.8 | 0.9, 3.6 |
| Power tools <1/mo | 12 | 17 | 0.5 | 0.2, 1.2 |
| >1/mo | 24 | 23 | 1.0 | 0.5, 1.9 |
| Hunting <1/mo | 37 | 33 | 1.2 | 0.5, 2.0 |
| >1/mo | 30 | 21 | 1.6 | 0.8, 3.2 |

[a]For all, reference category is fathers who never participated; adjusted for father's race, geographic location, date of father's interview, child's age at interview, whether a proxy interview was conducted, the child's gender, father's smoking status and the child's birth season.