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Exposure to household painting and floor treatments, and parental occupational paint exposure and risk of childhood brain tumors: results from an Australian case-control study

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Abstract

Purpose

Childhood brain tumors (CBT) are the leading cause of cancer death in children, yet their etiology remains largely unknown. This study investigated whether household exposure to paints and floor treatments and parental occupational painting were associated with CBT risk in a population-based case-control study conducted between 2005 and 2010.

Methods

Cases were identified through all ten Australian pediatric oncology centers, and controls via nationwide random-digit dialing, frequency matched to cases on age, sex and state of residence. Data were obtained from parents in mailed questionnaires and telephone interviews. Information on domestic painting and floor treatments, and parental occupational exposure to paint, in key periods relating to the index pregnancy and childhood was obtained for 306 cases and 950 controls. Data were analyzed using unconditional logistic regression, adjusting for frequency matching variables and potential confounders.

Results

Overall, we found little evidence that parental, fetal or childhood exposure to home painting or floor treatments was associated with risk of CBT. There was, though, some evidence of a positive association between childhood exposure to indoor painting and risk of high-grade glioma (OR 3.31, 95% CI 1.29, 8.52) based on very small numbers. The OR for the association between CBT and paternal occupational exposure to paint any time before the pregnancy was OR 1.32 (95% CI 0.90, 1.92), which is consistent with the results of other studies.

Conclusions

Overall, we found little evidence of associations between household exposure to paint and the risk of CBT in any of the time periods investigated.

Keywords: child, brain tumors, paint, case-control, solvents, occupational exposures, household exposures

Abbreviations: CBT: childhood brain tumors, ALL: acute lymphoblastic leukemia, CATI: computer-assisted telephone interview, OR: odds ratio, CI: confidence interval.

INTRODUCTION

Childhood brain tumors (CBTs) are the second most common type of childhood cancer and, collectively, have the highest mortality among childhood malignancies. There are few known causes, with some genetic syndromes such as Neurofibromatosis Type 1 and ionising radiation to the head accounting for less than 5% of cases [1].

Previous reviews have indicated that parental exposure to a range of chemicals before pregnancy, and possibly similar exposures in the child, may increase the risk of CBT [1-3]. Some studies have specifically investigated the association between parental occupational exposure to paints and surface coatings and risk of CBT. In a review of this topic, Savitz and colleagues [4] reported that two out of four studies found positive associations. A further three studies published since the Savitz review also reported positive associations [5-7].

Household exposure to paints and surface coatings may also be important, since residents can be exposed for long periods and are unlikely to use protective clothing and masks. However, to our knowledge, only two previous studies have specifically investigated household exposures. A US study of paternal hobbies during pregnancy and after birth found no association with painting, but a weak positive association with paint stripping [8]. Another study reported no association between household exposure to paint during pregnancy and risk of astrocytoma or primitive neuroectodermal tumor (PNET) [9]. These findings were based on small numbers of exposed cases.

It is plausible that paint exposure may be a risk factor for CBT. Occupational exposure to painting has been classed as carcinogenic to humans because of the association with several adult cancers [10]; and some paints and other surface coatings contain chemical constituents including solvents (e.g., toluene) and other volatile organic compounds (VOCs) with carcinogenic potential [10]. Toluene (and other VOCs) are able to cross the blood-brain barrier due to their lipophilic properties [11,12]; thus, it is plausible that direct exposure to relevant compounds may lead to DNA damage in the developing brain. In addition, there is laboratory evidence that toluene causes DNA damage in the sperm of rats [13]; thus, it is plausible that paternal germ cell damage through preconception paint exposure may lead to cancer in the child [14,15].

The Australian Study of Childhood Brain Tumours (Aus-CBT) is a nationwide case-control study conducted between 2005 and 2010 that was designed to investigate environmental and genetic risk factors for CBT. The aim of this analysis was to investigate, in more detail than previous studies, whether parental or filial exposure to paint or other surface coatings such as floor treatments in the

home were associated with risk of CBT. We also examined associations with parental occupational exposure during relevant time periods.

MATERIALS AND METHODS

The design and methods of this study have been described in detail previously [16]. Briefly, cases were recruited from 10 paediatric oncology centres around Australia; they were eligible for inclusion if diagnosed between 2005 and 2010 and aged less than 15 years, and had a biological parent available who understood English sufficiently well to participate. In the complete years of recruitment for which registry data were available (to 2008), 97% of CBT cases were registered as having been treated at one of the participating hospitals (Personal communication, Australian Paediatric Cancer Registry November 2012). Approximately three controls per case were recruited by nationwide random-digit dialling and frequency-matched to cases by age, sex, and State of residence to ensure balance of these probable confounders of any effects of the major exposures of interest. Controls recruited in 2005 and 2006 were from a concurrently run case-control study of childhood ALL using identical recruitment methods [17]. Both Aus-CBT and Aus-ALL had Human Research Ethics Committee approval from participating hospitals.

Data Collection and exposure categorisation

Upon recruitment, mothers and fathers were mailed questionnaires requesting information about demographics, birth characteristics of the participating child, and basic (Yes/No) questions on the environmental exposures of interest, including residential house painting and floor treatments done in the 12 months before the index pregnancy (the ‘conception year’), during the pregnancy, and after the child was born (up to the time of diagnosis). Parents were also asked to list all occupations they had held from age 15 to the time of the child’s diagnosis. If household painting or floor treatments had been carried out, a comprehensive computer-assisted telephone interview (CATI) was performed to obtain detailed information, including the type of paint/treatment used, who did the painting/treatment, the number of rooms treated, and the number of occasions it was done. For floor treatments, parents were asked to specify the type of surface (floorboards/tiles/other), treatment types (stains/varnishes/sealants, adhesives, other) and number of rooms treated. Follow-up CATI questions were also asked when a parent listed an occupation involving potential exposure to an agent of interest, with detailed information being sought about the nature and timing of that exposure.

We assessed parental occupational exposure to paints qualitatively (*i.e.* exposed or non-exposed) based on the job characteristics obtained in the telephone interviews. These assessments were made based on *a priori* exposure rules, created by two of the authors (SP, DG). Subjects were classified

as being exposed to paints in a certain occupation if they were employed as a painter or as panel beater in a body shop, or when they indicated they had used paint. Jobs that were asked questions about paints included carpenter, cabinet maker, engineering technician, military, fisherman, shipping, mechanic, metal worker, railway worker, and teacher. Occupational parental exposure to paints was assigned for each job held from age 15. Exposures were examined for any time before the child's birth for both fathers and mothers, 'during pregnancy' (the year before the birth year) for mothers and 'the conception year' (two years before the birth year) for fathers.

Statistical Analysis

Odds ratios (OR) and 95% confidence intervals (CI) were estimated using unconditional logistic regression in PASW Statistics for Windows Version 18 (SPSS Inc., Chicago, IL, 2009). All models were adjusted for the frequency matching variables (child's age, sex, State of residence) so that confounding by these variables was fully taken account of in every analysis [18]. In addition, models were adjusted for variables meeting the empirical criteria for confounding; i.e. they were associated with the exposure in controls and with case/control status or study participation. For indoor painting and floor treatments, these were child's year of birth, ethnicity, parental education, maternal age group and professional pesticide treatments. For outdoor painting at home, they were child's year of birth, ethnicity, household income and maternal age group; and for paternal occupational painting, they were paternal education, year of diagnosis/recruitment, and paternal occupational exposure to diesel exhausts in the 2 years prior to birth.

A detailed description of household painting and floor treatment exposure metrics has been published previously [19]. Exposures were accumulated until the censoring date: date of diagnosis for the case families and the date of written questionnaire return for the control families (since controls were frequency matched - rather than individually matched - to cases, there was no matched case from which to create a pseudo date of diagnosis). For indoor painting, the main focus was the type of paint used ('water-based only' and 'any oil-based +/- water-based') and the relevant exposure period. We also examined painting carried out by someone other than the parents (often a professional painter) as this may result in different exposure intensity for people living in the home. Indoor painting was also investigated by CBT subtype. For outdoor painting, the main focus was on who had applied the paint (mother before/during pregnancy and father before pregnancy) as a child's high exposure to paint outside the house was considered unlikely because dilution of the exposure in outside air. In each case, the reference category was no exposure to that specific treatment (indoor painting, outdoor painting, or floor treatments respectively) from the 12 months before the pregnancy to the censoring date.

RESULTS

Between 2005 and 2010, 730 eligible CBT cases were identified, of whom 568 were invited to participate by their treating physician and 374 families consented (65.8% of invited, 51.2% of eligible). In the same time period, 3,624 eligible control families were identified, of whom 2,255 (62.2%) consented to participate. In accordance with our frequency matching criteria, 1,467 of these were recruited to the study.

Basic information on household paint and floor treatment exposure was available from written questionnaires for 306 (81.8%) case families and 950 (64.8%) control families. Detailed information on household paint and floor treatment exposure from the CATI (i.e. who painted, and location, frequency and type of paint) was available for 288 (77%) case families and 917 (62.5%) control families. Information about occupational exposure to paint was available for 247 (66%) case fathers and 302 (81%) case mothers, 799 (55%) control fathers and 941 (64%) control mothers.

The characteristics of case and control families are shown in Table 1. Control children were more likely to be female, of European ethnicity, and have an older mother, and less likely to have a parent without a secondary education. There was some overrepresentation of controls recruited in 2005-2006, as these were selected to match cases from the concurrent Aus-ALL Study.

Painting inside the house

We saw little evidence of an association between indoor painting in any time period and risk of CBT (Table 2), and little variation by paint type or number of rooms painted. Although several of the ORs for painting done by a person other than the parents appeared to be elevated, the estimates were imprecise due to small numbers and there was no evidence of a dose-response relationship. When any indoor painting was analysed by tumor subtype, the results for low-grade glioma and embryonal tumors were similar to the overall results, while the ORs for high-grade glioma were elevated for all time periods and particularly for painting after birth, although the estimates were imprecise (Table 3).

Painting outside the house

There was no evidence of any positive association of the mother painting the outside of the house before or during pregnancy or the father doing so before pregnancy with the risk of CBT, except for an elevated OR for maternal use of oil-based paints during pregnancy. However, this was based on only four exposed cases/controls (Table 4).

Floor treatments

The ORs for floor treatments in the home were mostly close to or below the null, and most estimates were imprecise due to small numbers (Table 5).

Occupational exposure to paints

ORs for paternal occupational exposure to paint were slightly elevated for exposure any time before the child's birth and in the conception year, although these results lacked precision (Table 6).

Maternal occupational exposure to paints was not analysed, as there were only two cases and five control mothers exposed any time before the child's birth.

DISCUSSION

We found little evidence that parental pre-conception, fetal or childhood exposure to indoor or outdoor painting or floor treatments was associated with an increased risk of CBT. Elevated ORs were seen for indoor painting in each time period and risk of high grade glioma, but these were based on small numbers. We also found little evidence that paternal occupational exposure to paint was associated with risk of CBT overall. Although the odds ratios were somewhat elevated for gestational exposure to indoor painting if done by a person other than the parents, the 95% CIs were wide; moreover, the ORs were higher for water-based than for oil-based paints and no dose-response relationship was evident, suggesting that this association was probably due to chance.

Our largely null findings are similar to those of the two previous studies of household paint exposure and the risk of CBT [9,8]. The positive associations we observed for high grade gliomas are interesting and require replication. It is biologically plausible that susceptibility to environmental exposures may vary by CBT subtype, although this may be a chance finding as it is a sub-group analysis based on small numbers.

The slightly elevated ORs we observed for paternal occupational paint exposure are similar to those of most previous occupational studies, as reported in a 1990 review [4]. Three additional studies published since the review have also reported at least some evidence of an increased risk of CBT with paternal occupational paint exposure: 1.50 (95% CI: 0.79, 2.66) [7], 3.65 (95% CI: 1.71, 7.80) [6], and 1.3 (95% CI: 0.5, 3.4) [5]. Another study had insufficient cases to analyze [20].

To our knowledge, our study is the first to comprehensively examine potential associations between CBT risk and household paint exposure to parents and children during relevant time periods, including the type of paint used, number of rooms painted and person doing the painting. Our findings suggest that the level of paint exposure associated with domestic painting is not associated with risk of most types of CBT. However, paternal occupational exposure to paints may be associated with an increased risk and is plausible, since it is likely to involve heavier exposure to

paint over longer periods than in domestic exposure and exposure to solvent-based agents used to clean painting equipment and painted surfaces. The most likely potential biological mechanism would be through DNA damage to the paternal germ cell in the period leading up to conception. However, the ORs for any occupational exposure to paint were similar to those for exposure during the conception year, suggesting that exposures further in the past may also be important. The compounds in modern paints that could plausibly underlie the observed associations include xylene, toluene and mineral spirits [21].

That it was population-based, with case recruitment from all 10 paediatric oncology centres in Australia, and control recruitment was undertaken by national random-digit dialling using state-of-the-art methods, are strengths of this study. During the recruitment period, around 90% of the Australian population had a landline telephone [22]. Detailed data on the specific nature of the domestic and occupational exposures of interest were collected in CATIs conducted by staff blinded to case/control status. We were also able to distinguish between exposure in different time periods relating to conception, gestation, and after the child's birth.

This study also had some limitations. Despite extensive efforts, participation fractions were less than ideal and, as previously reported, area-based measures of socioeconomic status (SES) were higher among participants than in the general population [16]. Further, among our study participants, a higher proportion of higher SES parents than lower SES parents reported painting their house or treating the floors in their home in the time periods of interest. Together, these facts indicate that these activities are likely to have been reported more by participants than would be the case in the general population. Given that both participation fractions and questionnaire completion were lower among controls than cases, these activities were probably overrepresented more among controls. ~~Although; the most likely effect of this would be~~ this would generally tend to lead to an an underestimation of the true ORs ~~for painting, in general, although the direction of any bias is~~ difficult to predict for the various categories of painting exposure examined in this study. Thus, the ~~positive associations we observed for a person other than the parents painting the home, and for indoor painting and risk of high grade glioma, may in fact be stronger.~~ We aimed to minimise the impact of selection bias by adjusting for factors related to the exposures of interest and to participation, including ethnicity, parents' education and area-based SES.

There is likely to have been error in the reporting of exposure, as parents were asked to recall details of treatments that occurred up to 15 years previously; however, for most families the period of recall would have been seven years or less. Although recall error is still possible, the main effect would be to bias the ORs to the null if it were non-differential for cases and controls. As in all case-

control studies, however, case parents' rumination about potentially harmful exposures may have led to over-reporting relative to control parents. It is therefore possible that the modest increases in some ORs were at least partly attributable to recall bias.

Indoor painting done by someone other than the parents – most likely a painting contractor – may be important, as more rooms are likely to be painted over a shorter timeframe; thus, the intensity of exposure for those living in the home may be higher, even if over a shorter period. Floor treatments, unlike painting of walls and ceilings, usually necessitate vacation of the rooms/house treated during the curing process; therefore, actual exposure may be less, which may partially explain the lower ORs observed for floor treatments at home.

In conclusion, apart from weak evidence of a positive association between childhood exposure to indoor painting and risk of high-grade glioma, we found little evidence that exposure to paints at home, or parental exposure at work, was associated with risk of CBT.

Consortium Statement

The Aus-CBT consortium conducted the study and the Telethon Institute for Child Health Research (TICHR), University of Western Australia, was the coordinating centre. Elizabeth Milne, Nicholas de Klerk, Carol Bower, Peter Dallas (TICHR), Bruce Armstrong (Sydney School of Public Health, University of Sydney), Frank van Bockxmeer (Royal Perth Hospital, University of WA), Rodney Scott and John Attia (University of Newcastle), Lin Fritschi (WA Institute for Medical Research), Lesley Ashton, Michelle Haber and Murray Norris (Children's Cancer Institute Australia for Medical Research, Lowy Cancer Research Centre, UNSW), Margaret Miller (Edith Cowan University) and Judith Thompson (WA Cancer Registry) were the research investigators.

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Conflict of Interest Disclosure: The authors declare that they have no conflict of interest.

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Table 1: Distribution of demographic and birth characteristics for Aus-CBT

Variable	Category	Case n	Case %	Control n	Control %
Any written questionnaire returned		306		950	
Mother's written questionnaire returned		302		941	
Father's written questionnaire returned		247		801	
Child sex	Female	123	40.2	450	47.4
	Male	183	59.8	500	52.6
Child age group	0-1	30	9.8	110	11.6
	2-4	85	27.8	304	32.0
	5-9	92	30.1	294	30.9
	10-14	99	32.4	242	25.5
Child state residence	NSW/ACT	103	33.7	286	30.1
	Victoria/Tasmania	86	28.1	251	26.4
	SA/NT	19	6.2	78	8.2
	WA	42	13.7	114	12.0
	Queensland	56	18.3	221	23.3
Birth year	1990-1998	86	28.1	229	24.1
	1998-2003	127	41.5	472	49.7
	2004-2010	93	30.4	249	26.2
Year of diagnosis/ Recruitment	2005-2006	109	35.6	418	44.0
	2007-2008	101	33.0	271	28.5
	2009-2010	96	31.4	261	27.5
Maternal age group	<25	47	15.4	87	9.2
	25-34	188	61.6	598	63.0
	35+	70	23.0	264	27.8
Paternal age group	<25	16	6.0	26	3.2
	25-34	154	57.0	436	54.2
	35+	100	37.0	342	42.5
Ethnic group ¹	European	186	60.8	682	71.8
	At least 50% European	76	24.8	176	18.5
	At least 50% non-European	12	3.9	30	3.2
	Indeterminate	32	10.5	62	6.5
Best Parental Education	Didn't complete secondary school	43	14.1	92	9.7
	Completed secondary school and/or trade qualification	101	33.0	303	31.9
	University/College	162	52.9	555	58.4
Father's education	Didn't complete secondary school	49	19.8	123	15.4
	Completed secondary school and/or trade qualification	101	40.9	375	46.8
	University/College	97	39.3	303	37.8
Household Income	Up to \$40, 000	50	16.4	129	13.3

	\$40,001-\$70,000	80	26.3	265	28.0
	\$70,001-\$100,000	80	26.3	251	26.5
	>\$100,000	94	31.0	302	31.9
Birth order	1	139	45.6	400	42.1
	2	100	32.8	331	34.9
	3+	66	21.6	218	23.0
Household pesticide treatment in year before pregnancy	No	229	75.6	754	80.1
	Yes	74	24.4	187	19.9
Paternal occupational exposure to diesel exhaust in the 2 years prior to birth	No	194	78.5	675	84.5
	Yes	53	21.5	124	15.5

¹European: at least 3 European grandparents; 50% European: 2 European grandparents; at least 50% non-European: 2 non-European grandparents and ethnicity of 2 other grandparents unknown; indeterminate: no 2 grandparents of same ethnicity (i.e European or non-European) and 2+ grandparents of unknown ethnicity.

NSW: New South Wales, ACT: Australian Capital Territory, SA: South Australia, NT: Northern Territory, WA: Western Australia.

Table 2: Household painting indoors and the risk of CBT

	Before pregnancy			During pregnancy			After birth			Any time		
	n cases/ controls	OR ¹	95% CI	n cases/ controls	OR ¹	95% CI	n cases/ controls	OR ¹	95% CI	n cases/ controls	OR ¹	95% CI
Any painting												
None inside ²	102/299	1.00	Referent	102/299	1.00	Referent	102/299	1.00	Referent	102/299	1.00	Referent
Any inside	132/432	0.94	0.68, 1.31	85/291	0.86	0.60, 1.23	146/450	0.91	0.66, 1.26	197/640	0.89	0.66, 1.20
Water only	82/277	0.92	0.63, 1.33	48/169	0.86	0.56, 1.31	86/254	1.01	0.69, 1.46	110/353	0.91	0.65, 1.27
Oil +/- water	35/126	0.94	0.58, 1.52	16/79	0.69	0.37, 1.28	43/147	0.92	0.58, 1.45	70/244	0.90	0.62, 1.33
Paint stripper	9/32	1.09	0.47, 2.49	2/13	0.48	0.10, 2.24	5/21	0.64 ⁴	0.23, 1.79	13/57	0.75	0.37, 1.50
Number of rooms painted												
Low ³	46/194	0.77	0.50, 1.17	50/184	0.84	0.55, 1.27	69/213	0.99	0.68, 1.45	75/270	0.86	0.60, 1.23
High	71/205	1.11	0.75, 1.63	13/64	0.63	0.33, 1.23	59/188	0.87	0.57, 1.33	78/266	0.89	0.61, 1.29
<i>p</i> trend			0.65			0.15			0.55			0.49
Painting done by only someone other than the parents												
Yes	14/31	1.32	0.65, 2.68	15/28	1.46	0.72, 2.96	39/95	1.20	0.75, 1.93	53/132	1.16	0.76, 1.77
Use of water-based paints	8/18	1.26	0.51, 3.12	9/14	1.64	0.66, 4.10	25/51	1.49	0.84, 2.64	31/70	1.26	0.75, 2.10
Use of oil paints (+/- water based)	3/10	0.84	0.21, 3.35	6/14	1.28	0.45, 3.61	13/41	0.89	0.44, 1.81	19/57	1.06	0.55, 2.06
Number of rooms painted												
Low ³	3/11	0.84	0.21, 3.22	11/13	2.18	0.89, 5.34	21/47	1.37	0.76, 2.48	29/77	1.17	0.70, 1.96
High	9/17	1.49	0.61, 3.61	4/15	0.81	0.25, 2.59	17/47	0.98	0.51, 1.88	19/49	1.02	0.55, 1.89
<i>p</i> trend			0.45			0.62			0.77			0.77

¹ ORs are adjusted for matching variables, maternal age group, child's ethnicity, child's year of birth group, parental education, and household pesticide use prior to pregnancy.

² The reference group for all analyses is no painting indoors from 1 year before the index pregnancy to the censoring date.

³ High room number is defined as: before and during pregnancy, more than 3 rooms; after birth, more than 5 rooms; any time, more than 7 rooms (cut point based on median in controls who had painting).

⁴ This OR is only adjusted for matching variables, the fully adjusted OR was not calculable due to low n.

Table 3: Painting Indoors and the risk of CBT by tumor subtype

	Before pregnancy				During pregnancy			After child's birth		
	Total cases	Exposed cases/controls	OR ¹	95 % CI	Exposed cases/controls	OR ¹	95 % CI	Exposed cases/controls	OR ¹	95 % CI
Low grade glioma	144	60/432	0.84	0.57, 1.22	37/291	0.79	0.52, 1.20	66/450	0.86	0.58, 1.25
High grade glioma	26	13/432	1.59	0.66, 3.83	10/291	1.86	0.78, 4.43	18/450	3.31	1.29, 8.52
Embryonal Tumors ²	71	32/432	1.19	0.70, 2.00	24/291	1.27	0.74, 2.16	38/450	1.30	0.77, 2.18

¹ORs adjusted for matching variables, maternal age group, child's year of birth group, ethnicity, parental education, household professional pesticide use pre-pregnancy. Reference group is no painting done within that period.

²Embryonal tumors comprise primitive neuroectodermal tumors, medulloblastomas, atypical teratoid rhabdoid tumors.

Table 4: Parental painting outside of the house and the risk of CBT

	Mother						Father		
	Before Pregnancy			During Pregnancy			Before Pregnancy		
	n cases/ controls	OR	95 % CI	n cases/ controls	OR	95 % CI	n cases/ controls	OR	95 % CI
No painting outside house	174/529	1.00	Referent	174/529	1.00	Referent	174/529	1.00	Referent
Any painting outside house	11/52	0.67	0.34, 1.35	5/29	0.60	0.23, 1.62	63/230	0.89	0.63, 1.26
Use of water-based paints	6/39	0.50	0.20, 1.22	1/24	0.15	0.02, 1.11	31/150	0.70	0.45, 1.11
Use of oil paints (+/- water based)	4/11	1.05	0.32, 3.49	4/4	3.38	0.81, 14.14	19/59	1.11	0.63, 1.97

¹ORs adjusted for matching variables, maternal age group, child's year of birth group, ethnicity. Reference group is no household painting outdoors from 1 year before the pregnancy to the censoring date.

Table 5: Household floor treatments and risk of CBT

	Before pregnancy			During pregnancy			After birth			Overall		
	n cases/ controls	OR ¹	95% CI	n cases/ controls	OR ¹	95% CI	n cases/ controls	OR ¹	95% CI	n cases/ controls	OR ¹	95% CI
Any Floor Treatments												
None ²	225/645	1.00	Referent	225/645	1.00	Referent	225/645	1.00	Referent	225/645	1.00	Referent
Any	40/137	0.87	0.58, 1.31	11/56	0.64	0.32, 1.26	39/174	0.65	0.44, 0.98	69/285	0.72	0.52, 1.00
Floorboards	31/102	0.90	0.57, 1.43	8/44	0.60	0.27, 1.32	34/139	0.71	0.46, 1.09	55/223	0.73	0.51, 1.04
Coatings ³	38/128	0.89	0.58, 1.35	10/50	0.66	0.32, 1.34	37/164	0.66	0.43, 1.00	66/264	0.75	0.54, 1.04
No. rooms treated												
Low	12/66	0.54	0.28, 1.05	3/35	0.28	0.08, 0.92	17/102	0.49	0.28, 0.86	23/144	0.48	0.30, 0.78
High ⁴	28/70	1.19	0.73, 1.95	8/20	1.36	0.58, 3.22	22/71	0.89	0.53, 1.51	45/133	1.00	0.68, 1.48
<i>p</i> trend			0.93			0.66			0.19			0.40

¹ ORs adjusted for matching variables, maternal age group, child's ethnicity, child's year of birth group, household income and professional pest control prior to pregnancy.

² The reference group for all analyses is no floor treatments from 1 year before the index pregnancy to the censoring date.

³ 'Coatings' include varnish, sealants, stains.

⁴ High number of rooms is defined as >2 for before/during pregnancy and >3 for after birth and overall.

Table 6: Father occupational exposure to the application of paint and risk of CBT

	N case/control	OR ¹	95% CI
No exposure before child's birth	192/661	1.00	Referent
Any exposure before child's birth	54/138	1.32	0.90, 1.92
Any exposure in the 'conception year' ^{2, 3}	18/48	1.25	0.69, 2.28

¹ORs adjusted for matching variables (child's age, sex, State of residence), paternal education, year of diagnosis/recruitment, paternal occupational exposure to diesel exhausts in the 2 years prior to birth.

²'Conception year' relates to the year 2 years before the birth year.

³Reference level is no occupational painting before the child's birth.

Erratum for: Exposure to household painting and floor treatments, and parental occupational paint exposure and risk of childhood brain tumors: results from an Australian case-control study

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After publication, it was noted by the authors that the dataset used for the analyses of occupational exposures in this study accidentally used incomplete occupational histories for 94 control fathers and 104 control mothers recruited in 2006, and these control parents were all treated as unexposed to occupational painting.

Results in Tables 1-5 **were unaffected by this error** as they concerned household rather than occupational paint exposure. Our conclusions regarding the association with household exposure to paint and floor treatments are consequently unaffected.

However, for occupational exposure, 15 control fathers and 1 control mother were mistakenly classified as being unexposed when they should have been classified as exposed (any time before the child's birth). The analyses presented in Table 6 in the published manuscript were re-run using the complete dataset to ensure correct exposure assignment (5 case fathers and 4 control fathers were additionally excluded from revised occupational analysis due to genuinely missing data). The updated Table 6 is presented in this erratum.

In the published manuscript, we commented in the results section that the ORs for paternal occupational exposure to paints were slightly elevated but lacked precision, and in the conclusion remarked that there was 'little evidence that exposure to paints at home, or parental exposure at work, was associated with risk of CBT'. The revised ORs for paternal occupational exposure to paints are only slightly different from those previously published so these statements are still correct. There were still too few occupationally exposed mothers to analyse (2 cases/6 controls). The sentence in the abstract concerning father's occupational exposure should now read "The OR for the association between CBT and paternal occupational exposure to paint any time before the pregnancy was 1.23 (95 %CI 0.85, 1.78), which is consistent with the results of other studies."

We offer our sincere apologies for any confusion we may have caused.

Table 6: Father's occupational exposure to the application of paint and risk of CBT

	n cases/controls	OR ^a	95% CI
No exposure before the child's birth	187/642	1.00	Referent
Any exposure before the child's birth	54/153	1.23	0.85, 1.78
Any exposure in the "conception year", ^{b,c}	18/52	1.26	0.70, 2.27

^a ORs adjusted for matching variables (child's age, sex, state of residence), paternal education, year of diagnosis/recruitment, paternal occupational exposure to diesel exhausts in the 2 years prior to birth

^b "Conception year" relates to the year 2 years before the birth year

^c Reference level is no occupational painting before the child's birth.