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Residential distance at birth from overhead high-voltage powerlines: childhood cancer risk in Britain 1962–2008

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Background: We extend our previous study of childhood leukaemia and proximity to high-voltage powerlines by including more recent data and cases and controls from Scotland, by considering 132-kV powerlines as well as 275 and 400 kV and by looking at greater distances from the powerlines.

Methods: Case–control study using 53 515 children from the National Registry of Childhood Tumours 1962–2008, matched controls, and calculated distances of mother's address at child's birth to powerlines at 132, 275, and 400 kV in England, Wales and Scotland.

Results: Our previous finding of an excess risk for leukaemia at distances out to 600 m declines over time. Relative risk and 95% confidence interval for leukaemia, 0–199 m compared with >1000 m, all voltages: 1960s 4.50 (0.97–20.83), 2000s 0.71 (0.49–1.03), aggregate over whole period 1.12 (0.90–1.38). Increased risk, albeit less strong, may also be present for 132-kV lines. Increased risk does not extend beyond 600 m for lines of any voltage.

Conclusions: A risk declining over time is unlikely to arise from any physical effect of the powerlines and is more likely to be the result of changing population characteristics among those living near powerlines.

Exposure to power-frequency electric and magnetic fields (EMFs) and associated risks of various diseases have been studied for > 30 years. Laboratory evidence has been largely negative (IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2002; World Health Organization, 2007), and no plausible biological mechanisms have been identified (Swanson and Kheifets, 2006), though candidates remain, for example, 'free-radicals' (Atkins, 1976; Brocklehurst and McLauchlan, 1996). Epidemiological studies have provided inconsistent support for a link between EMFs and various diseases (IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2002; World Health Organization, 2007; Kheifets *et al*, 2010b; Elliott *et al*, 2013), but a relatively consistent association has been found for magnetic fields and childhood leukaemia (Ahlbom *et al*, 2000; Greenland *et al*, 2000; Kheifets *et al*, 2010a).

The epidemiological evidence for effects on health from exposure to power-frequency electric fields is largely negative (World Health Organization, 2007; Kheifets *et al*, 2010c). The remaining concern is principally with power-frequency magnetic fields, which are classified as 'possibly carcinogenic' (IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2002; World Health Organization, 2007), and with residential proximity to high-voltage powerlines, which are one source of such fields.

In 2005, we reported a relationship of childhood leukaemia risk 1962–95 in England and Wales (E&W) with distance of mother's usual residence at the time of birth of the child from the National Grid plc (NG) overhead powerlines (all 400/275-kV powerlines in E&W plus a small fraction of 132-kV lines) (Draper *et al*, 2005). Leukaemia risk was increased within 600 m of the lines, compared

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with those residing beyond 600 m. No corresponding pattern of risk was found for central nervous system (CNS) tumours or the grouping of other childhood solid tumours. A clearly separable relationship of increasing leukaemia risk with higher socio-economic status (SES) was also evident and persists in more recent analyses (Kroll *et al*, 2011b; Keegan *et al*, 2012). A subsequent report found an increased risk for leukaemia with calculated magnetic fields generated by the powerlines, based on very small numbers and not statistically significant but consistent with the relative risk of around 2 for fields $>0.4 \mu\text{T}$ found in pooled analyses (Kroll *et al*, 2010). The United Kingdom Childhood Cancer Study (UK Childhood Cancer Study Investigators, 2000b) reported a smaller study of childhood leukaemias and other cancers across England, Wales and Scotland, which both measured magnetic fields within the home from all sources and calculated distances to powerlines. It also reported elevated risks for leukaemia that were not statistically significant, though they were compatible with the pooled analyses for magnetic fields and with the Draper *et al* (2005) results for powerline proximity (UK Childhood Cancer Study Investigators, 1999, 2000a; Draper *et al*, 2005).

Attempts have been made to establish whether the report by Draper *et al* (2005) of an association of childhood leukaemia with residential proximity to overhead powerlines, specifically at distances out to 600 m, which had not previously been reported, could be replicated. A study in France (Sermage-Faure *et al*, 2013) found elevated risks within 50 m, confined to the higher-voltage lines and to younger children but not extending beyond 50 m; a study in Denmark (Pedersen *et al*, 2014) found no overall pattern of increased risk; and a study in California is in progress. We therefore report here the most powerful analysis it has been feasible for us to construct using methodology similar to that of our previous report. We have extended our previous study to include data relating to cases diagnosed up to the end of 2008 (and controls) in E&W and cases and controls from Scotland for the whole period 1962–2008. Importantly, the analyses have also been extended to include consideration of 132-kV overhead powerlines as well as 400/275 kV and to evaluate risk at distances between 600 and 1000 m.

This paper presents risk analyses for residential distance at birth from these powerlines, which may be of aetiological interest in its own right, not just as a surrogate for the magnetic fields produced by the powerlines. We will present risk analyses in relation to calculated magnetic fields separately.

MATERIALS AND METHODS

Cases and controls. The UK National Registry of Childhood Tumours (NRCT) has recorded nearly all childhood cancers (malignancies and brain/CNS tumours whose behaviour may be benign or uncertain) occurring since 1962 in those diagnosed under the age of 15 years while domiciled in England, Wales and Scotland (Britain) (Kroll *et al*, 2011a, 2012).

In the registration years considered complete at the time of this study (1962–2008), 57 067 tumours were registered on the NRCT as occurring in children in England, Wales and Scotland. The publicly available part of the birth registration information is obtained routinely for about 94% of all those registered on the NRCT (unless born outside Britain, adopted or because of failure to trace at the NHS Central Registers). We therefore obtained mother's residential address at birth for 53 515 children registered on the NRCT 1962–2008.

For every childhood cancer case whose birth registration could be traced, a birth record was also obtained for at least one control child (known to be unaffected by cancer as determined by absence

from the NRCT at the time of selection) matched for sex, approximate date of birth and birth registration sub-district. There were some 430 birth registration districts and 1700 sub-districts of varying sizes across England, Wales and Scotland over the period covered by our study.

For those registered with cancer from the year 2000, two control birth records were obtained. Previously one only was identified by systematic sampling up or down in the birth register from the case's entry, depending on whether the case's NRCT registration number was odd or even. Now two controls are selected by moving at least six entries up and down the birth register from the case and selecting the next matching birth. Controls are nearly always available within the same sub-district register with a date of birth within 6 months of the case's date of birth. A few controls may have died or emigrated before the date of diagnosis of their matched case, but otherwise we believe the controls are essentially unbiased in their availability, and any limitations have minimal implications. Hence we obtained birth registration data for 66 204 control children.

Calculation of distance from powerlines. Grid references of case/control mothers' residence at time of birth of the child and of overhead powerlines were used to calculate distances, using the same methods as we used previously (Draper *et al*, 2005; Swanson, 2008), with minor differences, described in the following paragraphs, mainly forced by the form of the new data available to us.

Grid references for the 400/275 kV and 143 km of the 132-kV transmission powerlines in E&W were provided by NG. We obtained grid references for the remaining 132-kV distribution lines in E&W from the then seven Distribution Network Operators (DNOs, listed in Acknowledgements), which between them covered the 12 geographically based licence areas into which E&W are divided. Data for 400/275/132-kV lines in Scotland were obtained from the two DNOs, which also cover the two Scottish licence areas. For 4 of the 14 licence areas covering Britain, grid references of pylons or poles with connections between them in the correct sequence were provided. For the remaining 10, we had only the pylon/pole grid references without connections, and we assigned connections automatically using the largely sequential numbering system for pylons, with manual checks and corrections where indicated by obvious discrepancies or implausibilities. We sought information on year of construction and, for 7 of the 14 licence areas (including both in Scotland), were able to identify a year of construction for $>80\%$ of spans. Across these seven licence areas, we had year of construction for 93% of 132-kV spans and, across the whole study area, 99% of 400/275-kV spans. For those 132-kV lines where we did have year of construction, 67% predated our study period, which therefore gives an indication of the error rate produced by assuming powerlines were present if no construction date was available. In total, this study includes data for 11 058 km of 400/275-kV and 15 810 km of 132-kV lines.

For 98% (117 583) of addresses, we were able to allocate a postcode, and for 94% (110 639) of these cases and controls, we were also able to add the Address-Point (Ordnance Survey, Southampton, UK) grid reference of their home. The Address-Point grid reference is a 0.1-m resolution grid reference located somewhere within the footprint of the building concerned; in this study, we did not attempt to locate where within the building it lay. For those homes (6% of subjects) where we had only the grid reference of the centroid of the postcode, if the distance calculated from the postcode centroid to the powerline was >1500 m, we could reliably assume that all the houses within that postcode were at least 1000 m from the line. If the distance calculated from the postcode centroid to the powerline was ≤ 1500 m, the distance was treated as missing because of the uncertainty involved, stemming from the spatial extent of the postcode.

We calculated, from the grid references using coordinate geometry, blind to case-control status, the shortest distance to the nearest overhead powerline that existed in the year of birth, assuming that the line did exist if the year of construction was not available, for England, Wales and Scotland, separately for 400/275- and 132-kV lines.

Measures of socioeconomic status. Because we have repeatedly demonstrated associations of childhood cancer risk (particularly for leukaemia) with different measures of SES, some distance analyses were repeated adjusting for this variable, to ensure that any distance relationship observed was not a simple consequence of a relation between distance and SES. To do this, we used quintiles of the Carstairs deprivation index to allocate a 1981 measure of SES to the census ward in which each case or control resided at birth. Census wards vary in geographical size and population, and there were 10 444 such wards in England, Wales and Scotland at the 1981 census.

Statistical analysis. We analysed the distance between residential address and powerline location for cases and matched controls using conditional logistic regression. Distance bands for analysis were those used in our previous study (Draper *et al*, 2005) and other studies (e.g., Sermage-Faure *et al*, 2013), together with pre-specified 100-m bands from 600 m out to 1000 m from the powerlines. The conditional logistic regression analyses were implemented in STATA Version 11 (StataCorp., College Station, TX, USA).

RESULTS

Supplementary Data Table S1 reports the results of using the distance and case-control data for E&W available in 2012, for the period 1962–95, against the results for the same period as analysed about 10 years ago and reported in 2005. Over the decade elapsing between construction of the two data files relevant to 1962–95, there have been changes in the definition of childhood cancer cases. These arise from the implementation of the third edition of the International Classification of Diseases for Oncology (ICD-O-3) and the related third edition of the International Classification of Childhood Cancer (ICCC-3). Overall, there has been an increase in the number of eligible cancer cases on the NRCT for 1962–95 (2% increase in eligible case-control pairs for leukaemia of which 1.5% arose from re-categorisation of myelodysplastic syndrome as a malignancy in ICC3-3; 2% increase for CNS tumours; 0.5% increase for other solid tumours).

In all subsequent analyses, there were some sporadic elevated or reduced risks for CNS tumours in some year/distance categories, and some of these are statistically significant. To a lesser extent, this is also the case for 'other solid tumours'. However, these variations in risks do not form any pattern, and we consider them likely to be chance findings. Our following presentation of results therefore concentrates on leukaemia; however, results for both CNS and other solid tumours are shown in the Supplementary Data Tables. There were occasional significant results in analyses of subsets of the data, for example, $1/d$, $1/d^2$ for leukaemia in E&W alone in the period 1996–2008, but they are counterintuitive, indicating increased risk with increased distance, and we are inclined to regard them also as due to chance. We have also conducted all analyses adjusting for SES, but this made no appreciable difference to any of the results (not shown), so, for simplicity, we present only the unadjusted findings.

Table 1 (expanded version at Supplementary Data Tables S2a–c) reports the distance analyses for cases (diagnosed in 1962–2008) and one or two matched controls resident in Britain in relation to 400/275-kV powerlines for distances out to 1000 m, with those resident ≥ 1000 m as reference. Exclusion of data for Scotland or the

data for second controls makes no difference (not shown). Over the whole period, there is no evidence of a distance effect for any of the three cancer groups. However, when stratified by decade of occurrence (1962–69 to 2000–08) there is evidence that leukaemia risk in those living within 600 m of a powerline was raised between 1962–79, declining thereafter. Elevated risk persisted in the 1980s but was not statistically significant. Even in the decades when the risk appears to be present, there is no evidence that it extended beyond the 600 m outer limit of the original analysis.

Supplementary Data Tables S3a–c reports the distance analyses for cases (diagnosed in 1962–2008) and matched controls resident in Britain in relation only to 132-kV powerlines for distances out to 1000 m with those resident ≥ 1000 m as reference. There are some indications of raised risks for leukaemia within 200 m, reducing (as for 400/275-kV lines) across the decades, but none of the risks are statistically significant. This analysis uses data relating to only the seven licence areas with $> 80\%$ information on construction date of the lines on the basis that it is better to restrict to more robust data even at the cost of statistical power. Results for all 14 licence areas (not presented) give even less indication of any effects.

Table 2 (expanded version at Supplementary Data Tables S4a–c) reports the distance analyses for cases (diagnosed in 1962–2008) and matched controls resident in Britain in relation to the nearest powerline of any of the voltages included in this study (across the whole of England, Wales and Scotland for 400/275-kV powerlines, but only those licence areas with $> 80\%$ known construction years for 132 kV). These overall risks within 600 m are also presented in Figure 1. Over the whole period, there is no excess risk, but risks in the earlier decades suggest an excess for leukaemia within 600 m, larger than in our original report, which was the aggregate over 1962–1995, and also largely based on 400/275-kV powerlines only (Draper *et al*, 2005).

DISCUSSION

Using the updated NRCT case-control data set and information from NG available in 2012, we were able to replicate the excess risk for childhood leukaemia reported in 2005 for distances within 600 m of 400/275-kV (and some 132 kV) overhead powerlines in E&W over the period 1962–95. The similarity of our present re-analysis and original findings, despite changes in cancer classifications, confirms that our methods are reproducible. Using more recent data up to 2008, we did not detect in the 1990s and 2000s the significant risks we previously noted despite having adequate power to do so. Our new results suggest the excess risk was in fact present only in the earlier decades, up to and including the 1970s and possibly the 1980s, but not in the later decades and is not evident when the cases and controls are analysed in aggregate across Britain from 1962 to 2008. These early risks presumably dominated the findings to 1995 we previously presented, although no temporal trend was then investigated (Draper *et al*, 2005). The excess does not appear to extend beyond the 600 m of the original analysis of predominately 400/275-kV lines. There is no evidence that the situation in Scotland is different to E&W. There are suggestions that elevated risks are also present for the 132-kV lines, possibly confined to smaller distances, and similarly only in the earlier decades, but none of the results for 132-kV lines within 600 m are statistically significant. There is no obvious increase in risk or pattern of risks with distance for tumour groups other than leukaemia.

Previous studies reported that leukaemia risk increased with affluence (Kroll *et al*, 2011b). Similarly, measures of SES based on paternal occupation have shown an association between higher social class and leukaemia risk (Keegan *et al*, 2012). The two measures (Carstairs deprivation score, occupational social class)

Table 1. Distance of address at birth from the nearest 400/275-kV line within each decade of diagnosis and in aggregate for cases diagnosed from 1962 to 2008 (and controls) in England, Wales and Scotland (expanded version available at Supplementary Tables S2a–c)

| Distance to line (m) | Leukaemia | | | | CNS/brain tumours | | | | Other solid tumours | | | |
|----------------------|-----------|----------|-------------|-----------|-------------------|----------|------|-----------|---------------------|----------|------|-----------|
| | Cases | Controls | RR | 95% CI | Cases | Controls | RR | 95% CI | Cases | Controls | RR | 95% CI |
| 1962–1969 | | | | | | | | | | | | |
| 0–199 | 5 | 1 | ∞ | | 0 | 3 | 0.00 | | 3 | 3 | 0.67 | 0.11–3.99 |
| 200–599 | 22 | 9 | 2.50 | 1.10–5.68 | 6 | 4 | 1.50 | 0.42–5.32 | 23 | 22 | 1.06 | 0.55–2.01 |
| 600–999 | 23 | 30 | 0.76 | 0.40–1.46 | 9 | 10 | 0.88 | 0.32–2.41 | 22 | 37 | 0.59 | 0.34–1.02 |
| ≥ 1000 (ref.) | 1088 | 1098 | | | 603 | 601 | | | 1570 | 1556 | | |
| Total | 1138 | 1138 | | | 618 | 618 | | | 1618 | 1618 | | |
| 1970–1979 | | | | | | | | | | | | |
| 0–199 | 22 | 7 | 2.83 | 1.12–7.19 | 4 | 10 | 0.44 | 0.14–1.44 | 11 | 18 | 0.44 | 0.18–1.06 |
| 200–599 | 73 | 79 | 0.91 | 0.65–1.29 | 51 | 42 | 1.22 | 0.79–1.90 | 93 | 79 | 1.12 | 0.81–1.56 |
| 600–999 | 123 | 112 | 1.17 | 0.88–1.55 | 59 | 76 | 0.73 | 0.50–1.07 | 132 | 120 | 1.21 | 0.92–1.58 |
| ≥ 1000 (ref.) | 3357 | 3377 | | | 2212 | 2198 | | | 3902 | 3925 | | |
| Total | 3575 | 3575 | | | 2326 | 2326 | | | 4138 | 4142 | | |
| 1980–1989 | | | | | | | | | | | | |
| 0–199 | 18 | 9 | 1.56 | 0.67–3.59 | 20 | 19 | 0.88 | 0.43–1.79 | 23 | 32 | 0.59 | 0.32–1.10 |
| 200–599 | 112 | 84 | 1.39 | 1.01–1.90 | 76 | 61 | 1.38 | 0.92–2.05 | 146 | 139 | 1.08 | 0.84–1.39 |
| 600–999 | 148 | 171 | 0.89 | 0.70–1.13 | 99 | 107 | 0.95 | 0.71–1.27 | 215 | 214 | 1.00 | 0.81–1.23 |
| ≥ 1000 (ref.) | 3336 | 3359 | | | 2370 | 2392 | | | 4592 | 4608 | | |
| Total | 3614 | 3623 | | | 2565 | 2579 | | | 4976 | 4993 | | |
| 1990–1999 | | | | | | | | | | | | |
| 0–199 | 33 | 42 | 0.71 | 0.43–1.17 | 27 | 20 | 1.16 | 0.60–2.24 | 39 | 39 | 0.87 | 0.53–1.43 |
| 200–599 | 129 | 152 | 0.90 | 0.69–1.17 | 86 | 96 | 0.88 | 0.63–1.22 | 183 | 186 | 1.00 | 0.80–1.25 |
| 600–999 | 169 | 168 | 0.99 | 0.78–1.25 | 138 | 136 | 1.12 | 0.86–1.46 | 234 | 256 | 0.95 | 0.78–1.15 |
| ≥ 1000 (ref.) | 3966 | 3997 | | | 2965 | 3046 | | | 5254 | 5368 | | |
| Total | 4297 | 4359 | | | 3216 | 3298 | | | 5710 | 5849 | | |
| 2000–2008 | | | | | | | | | | | | |
| 0–199 | 27 | 60 | 0.78 | 0.48–1.27 | 28 | 44 | 1.17 | 0.71–1.95 | 43 | 77 | 1.17 | 0.78–1.76 |
| 200–599 | 147 | 260 | 1.10 | 0.88–1.37 | 107 | 172 | 1.21 | 0.93–1.57 | 170 | 315 | 1.05 | 0.86–1.28 |
| 600–999 | 182 | 340 | 1.01 | 0.83–1.22 | 138 | 270 | 1.02 | 0.82–1.27 | 252 | 491 | 0.99 | 0.84–1.17 |
| ≥ 1000 (ref.) | 3650 | 7074 | | | 2970 | 5829 | | | 5078 | 9864 | | |
| Total | 4006 | 7734 | | | 3243 | 6315 | | | 5543 | 10747 | | |
| 1962–2008 | | | | | | | | | | | | |
| 0–199 | 105 | 119 | 1.00 | 0.75–1.34 | 79 | 96 | 0.96 | 0.69–1.34 | 119 | 169 | 0.85 | 0.65–1.10 |
| 200–599 | 483 | 584 | 1.08 | 0.95–1.24 | 326 | 375 | 1.15 | 0.97–1.35 | 615 | 741 | 1.05 | 0.94–1.18 |
| 600–999 | 645 | 821 | 0.99 | 0.88–1.11 | 443 | 599 | 0.98 | 0.86–1.12 | 855 | 1118 | 0.99 | 0.90–1.09 |
| ≥ 1000 (ref.) | 15397 | 18905 | 1.00 | | 11120 | 14066 | 1.00 | | 20396 | 25321 | 1.00 | |
| Total | 16630 | 20429 | | | 11968 | 15136 | | | 21985 | 27349 | | |

Abbreviations: CI = confidence interval; RR = relative risk.
 Includes all cases diagnosed in the decades 1962–2008 in England, Wales and Scotland and their controls with grid reference of birth address or reliable postcode-based distance ≥ 1500 m taken as ≥ 1000 m. Figures in bold are significantly different from 1 (P < 0.05).

are poorly correlated but converge in suggesting that, in unbiased national studies in the UK, childhood leukaemia risk is associated with affluence (Kendall *et al*, 2013a, b). However, adjusting our analyses using the Carstairs measure made very little difference to our results.

Our previous results for calculated magnetic fields (Kroll *et al*, 2010) demonstrated that magnetic fields could not be the sole or even the main explanation for the excess leukaemia risk we reported within 600 m (Draper *et al*, 2005). This is reinforced by results presented here to 2008, as it is implausible that any biophysical effect of magnetic fields would change over time. Nor have the fields the lines produce reduced over this period; using

data collected for our magnetic-field calculations, we calculated that the average loads carried by the relevant NG lines (which, distance-for-distance, determines the magnetic field) in fact increased by 76% from the 1960s to the 2000s.

The ‘corona ion hypothesis’ (Fews *et al*, 1999) notes that the air ions produced by the electric fields of powerlines blow away from the lines and attach to existing airborne pollutants, increasing the charge of the pollutants, and hence the rate at which they are absorbed when breathed in and the resulting dose to the body. This could produce an effect diminishing over time if the concentration of relevant leukaemogenic pollutants also reduced over time. Some, though not all, pollutants have indeed reduced over the relevant

Table 2. Distance of address at birth within each decade of diagnosis and in aggregate from the nearest line: 400/275 kV (all companies) or 132 kV (from companies with > 80% data) for cases diagnosed from 1962 to 2008 (and controls) in England, Wales and Scotland (expanded version available at Supplementary Data Tables S4a–c)

| Distance to line (m) | Leukaemia | | | | CNS/brain tumours | | | | Other solid tumours | | | |
|----------------------|-----------|----------|-------------|------------|-------------------|----------|------|-----------|---------------------|----------|-------------|-----------|
| | Cases | Controls | RR | 95% CI | Cases | Controls | RR | 95% CI | Cases | Controls | RR | 95% CI |
| 1962–1969 | | | | | | | | | | | | |
| 0–199 | 14 | 4 | 4.50 | 0.97–20.83 | 1 | 5 | 0.25 | 0.03–2.24 | 11 | 9 | 0.88 | 0.32–2.41 |
| 200–599 | 34 | 26 | 1.33 | 0.76–2.35 | 17 | 16 | 0.92 | 0.40–2.08 | 45 | 54 | 0.82 | 0.51–1.31 |
| 600–999 | 48 | 53 | 0.97 | 0.61–1.54 | 24 | 24 | 1.05 | 0.56–1.97 | 60 | 79 | 0.70 | 0.48–1.04 |
| ≥ 1000 (ref.) | 1011 | 1024 | 1.00 | | 565 | 562 | 1.00 | | 1464 | 1438 | 1.00 | |
| Total | 1107 | 1107 | | | 607 | 607 | | | 1580 | 1580 | | |
| 1970–1979 | | | | | | | | | | | | |
| 0–199 | 40 | 22 | 2.46 | 1.29–4.69 | 17 | 23 | 0.67 | 0.32–1.38 | 36 | 40 | 0.79 | 0.48–1.32 |
| 200–599 | 141 | 147 | 0.94 | 0.72–1.23 | 95 | 80 | 1.29 | 0.92–1.81 | 174 | 160 | 1.12 | 0.87–1.44 |
| 600–999 | 207 | 192 | 1.11 | 0.88–1.39 | 123 | 120 | 1.01 | 0.76–1.35 | 190 | 207 | 0.92 | 0.74–1.15 |
| ≥ 1000 (ref.) | 3131 | 3158 | 1.00 | | 2051 | 2063 | 1.00 | | 3659 | 3656 | 1.00 | |
| Total | 3519 | 3519 | | | 2286 | 2286 | | | 4059 | 4063 | | |
| 1980–1989 | | | | | | | | | | | | |
| 0–199 | 52 | 36 | 1.54 | 0.92–2.58 | 49 | 37 | 1.21 | 0.74–2.00 | 53 | 66 | 0.77 | 0.51–1.16 |
| 200–599 | 180 | 161 | 1.13 | 0.88–1.44 | 119 | 116 | 1.10 | 0.81–1.51 | 253 | 246 | 1.03 | 0.84–1.26 |
| 600–999 | 215 | 255 | 0.85 | 0.69–1.03 | 170 | 157 | 1.12 | 0.88–1.44 | 343 | 337 | 1.03 | 0.87–1.22 |
| ≥ 1000 (ref.) | 3122 | 3126 | 1.00 | | 2188 | 2229 | 1.00 | | 4248 | 4265 | 1.00 | |
| Total | 3569 | 3578 | | | 2526 | 2539 | | | 4897 | 4914 | | |
| 1990–1999 | | | | | | | | | | | | |
| 0–199 | 67 | 64 | 0.99 | 0.66–1.49 | 48 | 37 | 1.15 | 0.71–1.85 | 77 | 85 | 0.88 | 0.60–1.28 |
| 200–599 | 223 | 271 | 0.86 | 0.70–1.05 | 154 | 167 | 0.98 | 0.76–1.27 | 315 | 329 | 1.00 | 0.84–1.19 |
| 600–999 | 285 | 269 | 1.04 | 0.86–1.25 | 238 | 215 | 1.18 | 0.96–1.46 | 370 | 393 | 0.93 | 0.79–1.09 |
| ≥ 1000 (ref.) | 3688 | 3721 | 1.00 | | 2747 | 2849 | 1.00 | | 4905 | 4998 | 1.00 | |
| Total | 4263 | 4325 | | | 3187 | 3268 | | | 5667 | 5805 | | |
| 2000–2008 | | | | | | | | | | | | |
| 0–199 | 48 | 113 | 0.71 | 0.49–1.03 | 54 | 90 | 1.13 | 0.78–1.65 | 80 | 156 | 1.07 | 0.79–1.46 |
| 200–599 | 242 | 436 | 1.03 | 0.86–1.23 | 174 | 306 | 1.10 | 0.89–1.35 | 283 | 580 | 0.90 | 0.77–1.06 |
| 600–999 | 292 | 553 | 1.00 | 0.85–1.17 | 227 | 454 | 1.01 | 0.84–1.20 | 405 | 784 | 0.98 | 0.86–1.12 |
| ≥ 1000 (ref.) | 3417 | 6610 | 1.00 | | 2784 | 5452 | 1.00 | | 4764 | 9194 | 1.00 | |
| Total | 3999 | 7712 | | | 3239 | 6302 | | | 5532 | 10714 | | |
| 1962–2008 | | | | | | | | | | | | |
| 0–199 | 221 | 239 | 1.12 | 0.90–1.38 | 169 | 192 | 1.06 | 0.84–1.35 | 257 | 356 | 0.91 | 0.75–1.09 |
| 200–599 | 820 | 1041 | 0.99 | 0.89–1.10 | 559 | 685 | 1.09 | 0.96–1.24 | 1070 | 1369 | 0.98 | 0.89–1.07 |
| 600–999 | 1047 | 1322 | 0.99 | 0.90–1.08 | 782 | 970 | 1.07 | 0.97–1.20 | 1368 | 1800 | 0.96 | 0.88–1.04 |
| ≥ 1000 (ref.) | 14369 | 17639 | 1.00 | | 10335 | 13155 | 1.00 | | 19040 | 23551 | 1.00 | |
| Total | 16457 | 20241 | | | 11845 | 15002 | | | 21735 | 27076 | | |

Abbreviations: CI = confidence interval; RR = relative risk.
 Includes all cases diagnosed from 1962 to 2008 in England, Wales and Scotland (and their controls) with grid reference of birth address or reliable postcode-based distance ≥ 1500 m taken as ≥ 1000 m. Figure in bold is significantly different from 1 (P < 0.05), figure in bold and underlined is significantly different from 1 (P < 0.01).

time period (Murrells *et al*, 2010). However, no such leukaemogenic pollutants have been identified, analyses suggest any such effects are quantitatively insignificant (Advisory Group on Non-Ionising Radiation, 2004) and our previous results did not demonstrate the expected excess of leukaemia downwind of powerlines (Draper *et al*, 2005). We will present a better test of this hypothesis in a separate paper.

An alternative explanation (Draper *et al*, 2005; Swanson *et al*, 2006) is that the effect, when present, is not a direct physical effect but indirect, arising either as a consequence of the types of area powerlines have been built through or an effect existing powerlines have on the type of housing that is built nearby or the people who

come to live there. This could be hypothesised to produce the change over time we observe in one of two ways. Either it could be a transient effect lasting a decade or two from the construction of a powerline, which might produce the observed change given that most powerlines were built in the 1950s/1960s, with the 132-kV lines on average older. Alternatively, there could be a change in the types of houses built near powerlines, or the characteristics of the people living in them, over this period.

Another explanation of our previous results could be bias, perhaps in control selection (Draper *et al*, 2005; Swanson *et al*, 2006). We noted previously, and others emphasised (e.g. Kheifets *et al*, 2005), that fewer of our leukaemia controls lived near

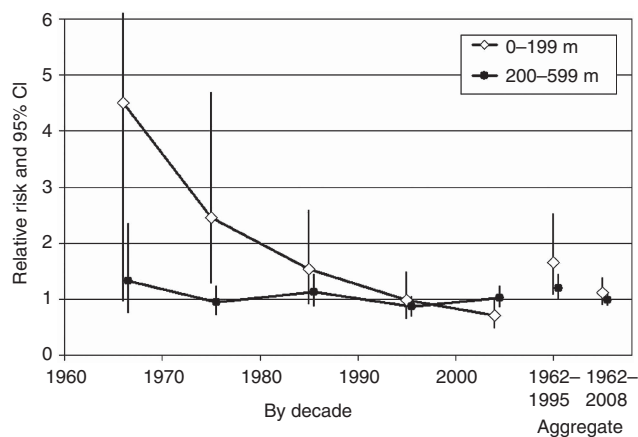


Figure 1. Relative risks of leukaemia by decade of birth in Britain, based on 132 (>80% year of construction information)/275/400-kV case-control exposure, by distance of residence from powerline. 1962–1995 is the aggregate result from our previous study (275- and 400-kV powerlines, E&W) as recalculated in this paper; 1962–2008, the aggregate result from this study, including 132-kV powerlines and Scotland.

powerlines than the controls for other cancers, perhaps suggestive of a biased control group, either through a selection bias or through chance. The data in the Supplementary Tables indicate that this applied for 275/400-kV lines in the earlier decades, coinciding with the increased risk, but applied much less to the 132-kV lines, where there were also suggestions of increased risks. No mechanism for such a bias has been identified, and it is even harder to think of a bias that reduces over time, but bias remains a possibility in principle. Chance must also always remain a possible explanation, perhaps particularly considering the multiple testing we have undertaken.

Of the two other studies published looking specifically at the same 600-m distances, one (Sermage-Faure *et al*, 2013) did not find elevated risks beyond 50 m and covered a more limited and more recent timespan (2002–2007). The other, Pedersen *et al* (2014), covering 1968–2006, found an elevated risk between 200 and 600 m for the highest voltage powerlines, but because this was not statistically significant and did not form part of a pattern of elevated risks, suggested it was likely to be a chance finding.

The change in the pattern of leukaemia risks (with distance) over time that we have observed, suggesting a real (if not obviously explicable) effect in the earliest period of observation but disappearing in later years, is not unique to non-ionising radiation EMF epidemiology. We have observed, in an as yet unpublished analysis, a similar effect of excess leukaemia risk (but few other increased tumour risks) declining over time in the vicinity of point sources of potential exposure to ionising radiation at Sellafield and at Dounreay. The most convincing explanation in that instance lies in the changing population characteristics of those living and working in the vicinity over time, rather than emanations from the nuclear plant.

Finally, this is the first ever investigation to consider specifically whether there are any risks to those living between 600 and 1000 m from overhead powerlines. Our results suggest little or no basis for concern. Although we reported risks in 2005 for those living within 600 m of 400/275-kV powerlines in the period 1962–95, the population living this close to powerlines is small. Therefore, given the size of the increased risk detected, very few childhood leukaemia cases per year could possibly be attributed to this residential feature. Clearly, the population living within 1000 m of a powerline is larger than that living closer, so it is reassuring that no risks have ever been evident outside 600 m.

CONCLUSIONS

We previously reported elevated risks for leukaemia in children born and diagnosed from 1962 to 1995 within 600 m of 400/275-kV powerlines. Our new data presented here suggest a lesser elevation may occur for the 132-kV powerlines. Increased risk does not extend beyond 600 m for lines of any voltage. Risk appears to have declined over the period from 1962 to 2008 in Britain, and in aggregate over that period, there is no significant excess risk.

The strengths of our study are its size, objective assessment of proximity to powerlines and freedom from any obvious source of bias. The result almost certainly cannot be produced by powerline-generated magnetic fields and is unlikely to be due to any other physical emanation from the lines. If the result is not due to study artefact or chance, the only remaining possibility seems to lie in changing population characteristics of people living near powerlines.

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