average area-under-the-curve of 5 mg/ml [8]. In all other patients, a dose of 300 mg/m^2 was administered every 3 weeks. Unless there was disease progression or stable disease after the first two cycles, patients were treated up to maximum response followed by two consolidation cycles. 12 patients who received carboplatin at first relapse also received consolidation treatment with conventional polychemotherapy salvage regimens such as MINE (mitoxantrone, ifosfamide + MESNA, etoposide) or CEP (CCNU, etoposide, prednimustine). From April 1992 to December 1994, 32 patients (15 HD and 17 NHL) were enrolled in the study. Patient and treatment characteristics are shown in Table 1.

Overall, two complete remissions (CR), 1 patient with HD and 1 with NHL, and 1 partial remission (PR) were observed, for an overall response rate of 9%. Currently, the patient who achieved CR with HD is alive and without evidence of disease after 36 months following six doses of carboplatin and consolidation radiotherapy on the only site of nodal relapse. The other 2 responding patients experienced sensitive relapse after 5 and 7 months; both are alive and disease-free after salvage chemotherapy. 21 of 29 patients who did not respond to carboplatin received further chemotherapy with a response rate of 57%. The administration of carboplatin was globally well tolerated. Grade IV neutropenia occurred in four cycles (5%), with no hospitalisation due to infection. Grade three and four thrombocytopenia were rare (5%) and related to heavily pretreated patients (at least three previous regimen and/or extended fields radiotherapy) and for carboplatin doses based upon BSA (six cases) rather than on GFR (two cases). 2 patients were admitted to the hospital because of severe thrombocytopenia requiring platelet transfusion and careful observation. Nausea, vomiting, neuro- or nephrotoxicity were sporadic and mild.

In conclusion, clinical activity of carboplatin in pretreated malignant lymphomas seems very limited at standard doses, as only 3 of 32 patients attained objective response, for an overall response rate of 9%. Our data confirm previous negative results obtained with standard dose single-agent carboplatin in lymphomas [5]. However, the low non-haematological toxicity of carboplatin has permitted dose-escalation studies in both solid and haematological tumours [6, 7], and its activity in lymphomatous malignancies when used at higher doses should be further evaluated.

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Radon Exposure and Incidence of Paediatric Malignancies

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WE READ with interest the editorial by Parker and Craft [1] which commented on our paper *Radon in Devon and Cornwall and Paediatric Malignancies* [2] and provided a detailed critique of the background factors which had necessarily been only summarised in our report. A study like ours, limited to a decade, and regional rather than national, clearly may not have sufficient power to detect small excesses in incidence that might be due to radon. However, our analysis was provoked by a paper [3] which suggested that excess from radon could be high and, therefore, our study might be of sufficient power. This does not appear to be the case as we detected no excess risk for all childhood cancers in our study.

The data showed a significantly raised incidence of neuroblastoma and a non-significantly raised incidence of acute myeloid leukaemia (AML) in high radon postcode sectors in the decade 1976–1985. The editorial discussed whether our finding for neuroblastoma was likely to have been due to chance. We have now looked at the incidence of the two malignancies in the subsequent decade. Table 1 shows the incidence rates in Devon and Cornwall for AML and neuroblastoma as given in our original paper, incidences for the decade 1986–1995 and incidences for the two periods combined.

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Table 1. Incidence in postcode sectors with average radon levels ≥ 100 Bq m^{-3} compared with postcode sectors with average radon level < 100 Bq m^{-3} (aged less than 15 years, living in Devon and Cornwall)

	Incidence in postcode sectors with average radon level ≥100 Bq m^{-3} (with 95% CI)	Incidence in postcode sectors with average radon level <100 Bq m^{-3} (with 95% CI)	*P* value
Neuroblastoma (1976–1985)	12.2 (6.1–21.9)	3.6 (1.3–7.8)	0.02
Neuroblastoma (1986–1995)	8.9 (3.8–17.5)	8.3 (4.5–13.9)	0.9
Neuroblastoma (1976–1995)	10.6 (6.4–16.5)	5.9 3.6–9.2)	0.08
AML (1976–1985)	6.7 (2.4–14.5)	2.4 (0.7–6.1)	0.11
AML (1986–1995)	11.1 (5.3–20.4)	4.8 2.0–9.4)	0.08
AML (1976–1995)	8.9 (5.1–14.4)	3.6 (1.8–6.2)	0.02

Incidences are per million child years. The P value for the difference between the two incidences is shown (two-tail). AML, acute myeloid leukaemia. CI, confidence interval.

In the subsequent decade, 1986–1995, the incidence of neuroblastoma was similar in the ≥ 100 Bq m^{-3} and <100 Bq m^{-3} radon postcode sectors. Thus, the finding of a significant difference in the decade 1976–1985 is likely to be due to chance.

The higher incidence for AML in the high radon postcode sectors in the period 1976–1985 persisted into the next decade. For the combined period of 20 years, 1976–1995, the incidence of AML in these sectors was significantly increased compared with that in the low radon sectors. A previous study has suggested a correlation between radon exposure and AML [4].

We await the results of the national case–control study described in the editorial that may further elucidate whether exposure to radon has a causal role in any childhood cancer. For those living in Devon and Cornwall, this is particularly important. It should be remembered that in these two counties even in the 'low' radon postcode sectors the average radon levels in homes was 57 Bq m^{-3} which is considerably above the national average of 21 Bq m^{-3}.

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Very High Male Lung Cancer Incidence in Areas with Tobacco Industries

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IN THE 1970s, in the south-east of The Netherlands, the male lung cancer incidence rate was among the highest in Europe, whereas that for females was among the lowest [1]. However, the rates for men have been decreasing since 1983; those for women are still increasing. In a previous report (*Eur J Cancer* 1995, **31A**, 949–952), it was shown that the changes in lung cancer incidence reflect changes in smoking behaviour, considered to be the major risk factor [2], which occurred 20–25 years ago [3]. The traditional presence of tobacco-processing industries in the south-east of The Netherlands, concentrated mainly in a few rural communities (Valkenswaard, Bladel, Eersel and Tegelen), very likely influenced smoking behaviour. Between 1900

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