by Knox and Cummins. While we agree with these authors that cyclical patterns are difficult to interpret, we do not think that their use of birth data for a single calendar year constitutes an appropriate comparison for the cases (who were born over a 30 or 40-year period), since the corresponding distribution in the general population may very well have changed during this interval. For example, modern forms of birth control have given parents greater control over the time of conception, so that birth patterns in more recent years must have been influenced by such various factors as marriage, climate, race, etc. Therefore, we chose a case-control approach, as a preferred alternative, to assess the significance of the apparent seasonal pattern in our data.

The 86 Hawaii-born testicular cancer cases were individually matched on sex, race, and birth-year (±2 years) to four controls who had been randomly selected from the population for a 1975–80 survey. The distributions of the cases and controls by month of birth are reasonably similar (Figure 1) and the chi-square test with 11 degrees of freedom is not significant (p = 0·83). Thus we are unable to confirm the existence of a seasonal pattern for the birth dates of men with testicular cancer in Hawaii.

The study by Knox and Cummins was an attempt to explore the possibility that an infectious or iatrogenic exposure might explain the recent increase in incidence of testicular cancer. Our analysis, on the other hand, was prompted by the possibility that the seasonal pattern observed by Czeizel et al.2 for births with undescended testes (increase during March–May and decrease during August–December) might also be observed for patients with testicular cancer, since the two conditions have been strongly associated in epidemiological studies. First trimester exposure to abnormally high levels of endogenous sex hormones has been implicated in the aetiology of both cryptorchidism and testicular cancer. Consistent with an hormonal aetiology, this seasonal birth pattern might be explained by the effect in early pregnancy of seasonal changes in the mother's production of melatonin and/or pituitary gonadotropins related to the duration of daylight. Since Hawaii is located in the Tropics, the seasonal variations in duration of daylight are reduced; thus our results do not totally exclude the possibility that such an effect might be seen in testicular cancer cases. This interpretation should certainly be considered, along with a possible infectious aetiology, if cyclical birth patterns for testicular cancer are confirmed in more northern populations. The four-month cycle observed by Knox and Cummins in Britain would be too short to fit with the daylight hypothesis. However, these authors' findings cannot be properly interpreted without secular trend data on the monthly distribution of births in the British population.

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References

Parental occupations and cancer

SIR—The recent article by Arundel and Kinnier-Wilson1 serves as a focus to highlight once again the problems of studying the potential relation of parental occupational exposures to childhood cancer. As they point out in their first sentence, little is known about the aetiology of cancer in children, despite the importance of cancer as a cause of morbidity and death in childhood. They also explain the rationale for examining the relation to parental occupation. However, they go on to state that the 14 reports they will review were conducted by a similar method and have only the “minor differences” of source of information, type of case, and categorisation of parental occupation. We believe that these are major

**Distribution by month of birth of testicular cancer cases and controls, Hawaii, 1962–83.**

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**Figure 1:** Distribution by month of birth of testicular cancer cases and controls, Hawaii, 1962–83.
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differences in study design and method of data collection which could well explain the discrepancies in the findings of these reports. The authors' classification of these differences as minor may well have led them to what we believe to be an overstatement of the conclusion, ie, "... the positive results of Kantor et al., 2 Peters et al., 3 van Steensel-Moll et al., 4 along with those of Fabia and Thuy 2 establish the hypothesis of hydrocarbon and lead exposed jobs being potentially hazardous ... in pregnancy."

The expectation that Wilms' tumour and brain tumours and leukaemia and all cancers, respectively, will have the same risk factors seems somewhat unreasonable on both biological and epidemiological grounds. A factor of primary importance, namely, timing of exposure, ie, prenatal versus postnatal, has received inadequate attention and may well differ for different types of cancer and for age when a given childhood cancer is diagnosed. We must also realize that specific exposures are not actually measured in these studies, but rather associations are reported with parental occupations that are believed to have had the exposures reported. Further, occupation and occupational exposures are ascertained by different methods (records, interviews, birth certificates) in different studies. These last two considerations may well lead to potential severe inaccuracies in assessing exposure. With this in mind, the fact that differing occupations have been reported in positive association with different cancers, and that nearly as many studies show no such associations, the most likely interpretations might well be that each of these differing findings is explicable on the basis of chance or due to the differing methodologies used or different cancers studied.

We agree with Arundel and Kinnier-Wilson in their final statement that "large numbers of cases and better information on both histological diagnosis and parental occupation exposure will be necessary." We would add that by investigating hypotheses more clearly defined with respect to timing of exposure, and by restricting comparisons to specific cancers, more meaningful information will be obtained about the role of parental occupation in cancers in children.

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References


The authors reply as follows:

It is a matter of opinion whether the differences of methodology used in studies of the association between parental occupational exposures and childhood cancer should be called major or minor. However, I would take issue over two points raised by Drs Gold and Shaw concerning our recent paper in the Journal. 1

Firstly, they consider our expectation that different neoplastic categories (Wilms' tumour, brain tumours, and leukaemia) might have the same risk factors to be "somewhat unreasonable on both biological and epidemiological grounds." According to the Oxford Survey of Childhood Cancer approximately 5% of all children's neoplasms are caused by prenatal x rays, and ratios of spontaneous to radiogenic cancers are the same for four subgroups of haemopoietic neoplasms and six types of solid tumours. 2-4 Judging by these observations, the cancer effect of prenatal irradiation (the only certain cause of childhood cancer) is non-specific. Therefore it is reasonable to expect that this might also be true of other causes of these diseases.

Secondly, Drs Gold and Shaw seem to think that "a factor of primary importance, namely, timing of exposure, ie, prenatal or postnatal, has received inadequate attention." In the Oxford Survey the age distribution of x rayed and non-x rayed cases has been extensively studied. 5 The results show that the third trimester of pregnancy is a relatively late date for initiating a cancer which occurs within the first ten years of life. Therefore, although postnatal exposures to radiation may have a cancer effect, this will show not in childhood but in adolescent or early adult life. Again it is reasonable to suppose this to be true of other causes of cancer.

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