relates to the period 1962–1999 when construction activity would have been intense. This activity was reliant on an influx (which included this author) of itinerant workers to the communities hosting the construction sites and the population mixing associated with this influx has been associated with the incidence of childhood leukaemia (Kinlen *et al,* 1995; Kinlen and Doll, 2004).

Kinlen *et al* studied the incidence of childhood leukaemia within 10 km of large, rural, construction sites and five of their chosen locations (Drax, West Burton, Longannet, Pembroke and Fawley) housed power stations and supported an on site construction work force of more than 2000 during the first decade of Bunch's study.

To quote from Kinlen's abstract:

> A 37% excess of leukaemia and non-Hodgkin's lymphoma at 0–14 years of age was recorded during construction and the following calendar year. The excesses were greater at times when construction workers and operating staff overlapped (72%), particularly in areas of relatively high social class. For several sites the excesses were similar or greater than those near the nuclear site of Sellafield (67%), which is distinctive in its large workforce with many construction workers.

The reader may object that Bunch *et al* were considering the impact of power lines, not power stations but all of the power stations under consideration were connected to the National Grid and powerlines would, necessarily, have run through the areas which Kinlen *et al* identified as having an elevated relative risk of childhood leukaemia.

He concluded that:

> Overall these findings provide further support for the hypothesis that rural population mixing is conducive to the transmission of the underlying infective agent(s) among susceptible people so as to increase the incidence of childhood leukaemia and non-Hodgkin's lymphoma.

The consumption of electricity in the UK grew by a factor of nearly three between 1962 and 2008 (Department of Energy and Climate Change (DECC), 2013), whilst Bunch *et al* show that the relative risk of leukaemia declined, from 4.5 to 0.71, over the same period. There can be little debate over their observation that magnetic fields cannot provide an explanation for their findings.

Bunch *et al* did not consider the relatively short term impact of the influx of powerline and power station construction workers but rather suggest that the result may be due to the *'changing population characteristics of people living near powerlines.'* Power stations are nodes on the powerline network and, of course, the lines are remote from them for much of their length. The authors mention *'changes to the types of houses built near powerlines or the characteristics of people living in them'* as possible causes of their findings. These changes would have taken time but the maximum relative risk of leukaemia was found in the early years of the study when the influx of contractors would have been at its height.

Kinlen's data show that mixing can enhance the risk of leukaemia in the relatively short term (1970–75 at Drax Phase 1, 1968–73 at Longannet, 1966–68 at West Burton). His cases were drawn from the same registry as Bunch's and it would be of great interest to locate the totality of Bunch *et al's* exposed cases with respect to the power stations as well as the powerlines.

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Comment on 'The NQO1 polymorphism C609T (Pro187Ser) and cancer susceptibility: a comprehensive meta-analysis'

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Sir,

I read the article by Lajin and Alachkar (2013) with great interest, which appeared in your journal as *Br J Cancer*. The study aimed to evaluate the impact of the NQO1 gene polymorphism with cancer risk. Although the study provides preliminary evidence to consider NQO1 polymorphism as a risk factor for cancer; however, after careful reading of the article, a few important issues came out that must be addressed for further actions.

First, it appears that the authors somehow missed the statistical power in this study. Sample sizes remain a major issue in genetic case–control studies analysing the association of polymorphism with disease susceptibility. The authors did neither mention the statistical power of individual studies nor their overall meta-analysis. Hence, the study should obtain an adequate statistical power (80%) to estimate significant association accurately, which remains a primary criterion to perform such studies, especially from the venous blood of study subjects. Underpowered studies usually lead to false-positive associations and misinterpretations (Hattersley and McCarthy, 2005). The authors also failed to mention the incidence rate of various cancers in the said study. The individual studies recruited in the present meta-analysis achieved the required statistical power is questionable and did not discuss in the text.

Second, the authors mention the sample size of Malik *et al* as 107 gastric cancer cases and 195 controls (Malik *et al*, 2011). But the exact number of gastric cancer cases is 108 in the study by Malik *et al.* All these points suggest a thorough examination of the association observed in the said study, and must be clarified before concluding that NQO1 gene polymorphism is a potential marker of cancer.

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