This is not informative and was not the quantity of interest in this study, but rather the median latency for those who died with mesothelioma. I acknowledge that the median latency of 23 years estimated in the study (30 years after excluding deaths within 10 years of first occupational exposure) will be restricted by the duration of follow-up, and so would increase as follow-up continues.

Second, if individuals who died from other causes or were alive at the end of follow-up were included as censored observations, then the estimated latency becomes dependent on the mesothelioma incidence rate. For example, if the incidence rate of mesothelioma was greater than the 37 cases per 100 000 person-years observed among the cohort, then the predicted median latency would be shorter even if the median latency for those who died with mesothelioma. I would like to thank the commenters for their thoughtful and constructive remarks, which highlight the challenges involved when latency is the outcome of interest.

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Frost G (2013) The latency period of mesothelioma among a cohort of British asbestos workers (1978–2005) – the opening year of the study was the last in which there was insufficient demand to meet the maximum demand for electricity (Department of Energy and Climate Change (DECC), 2013). The next decade saw a near doubling of demand which drove a frenzied programme of power station and power line construction, (National Grid Company; 2010) and, by the time that the 1973 oil crisis forced a slow down, a 50% margin of generating capacity over the peak demand had been established (Department of Energy and Climate Change (DECC), 2013).

Construction of the 400 kV supergrid did not begin until 1965 and it is noteworthy that Bunch et al’s maximum relative risk of 4.5 (0.97–20.83) would have died with mesothelioma) would be shorter even if the median latency for those who died with mesothelioma. I would like to thank the commenters for their thoughtful and constructive remarks, which highlight the challenges involved when latency is the outcome of interest.

**LETTERS TO THE EDITOR**

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

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Bunch et al (2014) studied the incidence of childhood leukaemia in relation to distance at birth from high-voltage powerlines over the period 1962–2008 and found that, for children born within 200 m, the relative risk fell from 4.5 (0.97–20.83) in the 1960s to 0.71 (0.49–1.03) in the 2000s.

The opening year of the study was the last in which there was insufficient capacity to meet the maximum demand for electricity (Department of Energy and Climate Change (DECC), 2013).
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).

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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