

## Dioxin exposure at Monsanto

SIR—Michael Gough<sup>1</sup> says that in our letter<sup>2</sup> we do not challenge the conclusions that there were no excesses of cancer or heart disease deaths in studies carried out on Monsanto workers exposed to dioxin. This is not true. The fundamental point of our letter is that because of problems in the design of the studies and improper exclusion of some workers, the findings must be treated with some scepticism.

While agreeing with our concern over the indices of dioxin exposure in the two Monsanto studies — because they permit one group to be referred to as dioxin-exposed in one study and as unexposed in the second — Gough suggests that three of the cancer deaths are “likely independent of dioxin exposure”. These were lung cancer cases, all smokers. Although there is no reported association between lung cancer and 2,3,7,8-TCDD exposure, given the cancer-promoting properties of this chemical<sup>3</sup>, an interaction between TCDD and initiators with respect to lung cancer must be considered<sup>4</sup>. In fact, interactions between TCDD and benzo(a)pyrene (a constituent of cigarette smoke) have been described in animals<sup>5</sup>.

Nineteen workers who died of circulatory disease or cancer were excluded from one epidemiology study by Drs Zack and Gaffey<sup>6</sup>. According to our records, obtained from Monsanto by lawyers for the plaintiffs in a trial against the company, these men should have been included as they were employed by Monsanto between 1955 and 1977. Employment in this period was the criterion for inclusion in the study cohort.

Gough criticizes us for not contacting the authors to find out why these 19 workers were excluded. In fact, we encouraged *Nature* to write to Dr Zack in January 1985 to get her comments on our original letter. She failed to reply to both the January letter and a reminder sent by the journal in February or March.

Gough seems to have misunderstood our point about treating the two Monsanto mortality cohorts — workers exposed to dioxin in the aftermath of the 1945 accident, and hourly paid employees exposed to dioxin during the manufacture of the herbicide 2,4,5-T (ref. 6). According to the 1953 Kettering laboratory study<sup>8</sup>, it is clear that workers in the 2,4,5-T process have the same type of symptoms reported by those exposed in the accident, although the severity of the complaints seems greater in the latter group. Our point is that both groups should be included in epidemiological analysis. Moreover, some of the workers employed on the 2,4,5-T process also helped to clean up after the accident. They had potential exposure to 2,3,7,8-TCDD in both circumstances.

Gough claims that no excess of cancer has been found in the Nitro workers. This is incorrect. A reanalysis of the data, presented by EKS<sup>9</sup> at the Dioxin 85 Symposium in Bayreuth in September 1985, indicates an excess mortality due to lung and bladder cancer. Similar results on this cohort were also found by an expert retained by Monsanto.

Workers at the Monsanto Nitro plant who were exposed to a bladder carcinogen *p*-aminobiphenyl developed bladder cancer as a result. Some of them may have been exposed to TCDD in 2,4,5-T. Given the cancer-promoting properties of the dioxin, an effect on the incidence and severity of bladder cancer in the Monsanto workers cannot be ruled out either.

We did not suggest that “low-level, intermittent exposures bear more risk than higher-level exposures”, but that health effects such as cancer may be expected to result from lower and more chronic exposure in the absence of chloracne. The report by Moses *et al.*<sup>10</sup> demonstrates that chloracne did not predict work exposure to 2,4,5-T in that nearly 50 per cent of workers with considerable work history with the herbicide never showed chloracne.

On the discrepancy between studies on the use of phenoxy herbicides and chlorophenols in Sweden<sup>11,12</sup> and New Zealand<sup>13</sup>, it is important to look at the design of each study. The control group in the New Zealand studies consisted of other cancers selected from the National Cancer Registry, which helped reduce recall and interviewer bias, two factors that could have influenced the Swedish findings. In his latest study, Hardell<sup>12</sup> used colon cancer cases as controls to reduce bias which may have occurred in his initial investigation. This study also found an increased incidence of soft-tissue sarcoma<sup>12</sup>.

A recent study by Kogan and Clapp<sup>14</sup> showed a higher than expected incidence of soft-tissue tumours in Vietnam veterans, whereas Greenwald *et al.*<sup>15</sup> found no significant association between service in Vietnam and soft-tissue sarcomas. This demonstrates the need to examine each report critically, including the investigations of the Monsanto workers.

As to an excess of deaths due to circulatory disorders in the Monsanto study<sup>6</sup>, the authors calculate proportionate mortality ratios (PMRs) of 137 and 131 for exposed and unexposed cohorts respectively. This excludes 19 additional cases referred to in our original letter<sup>2</sup>. Further, if the authors classified workers as unexposed to 2,4,5-T when they were clearly exposed to dioxin after the reactor accident, this results in misclassification.

Finally, Gough says optimistically that it is “possible to read the literature and

arrive at a cautious conclusion that dioxin has not caused early death and cancer in heavily exposed workers”. We consider this conclusion to be premature.

The purpose of our earlier letter was to put forward reasons why the Nitro data should be reassessed and the cohort followed further.

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## Dowsing explained

SIR—David Marks<sup>1</sup> asserts that there are no theories to account for paranormal effects. This is not true for dowsing. Serious dowsing claims such as those made by Soviet geologists<sup>2</sup>, which are difficult to account for in terms of the reception of normal sensory cues, may be explained by postulating human sensitivity to small magnetic field gradient changes<sup>3–5</sup>. The theory is supported by a series of tests involving 150 subjects<sup>6</sup>.

The magnetic theory predicts that dowers can achieve above-chance results only if the features they claim to detect are associated with magnetic gradients of at least one nanotesla per metre. This was not the case in Randi's recent experiments<sup>7</sup>, so his chance results are therefore consistent with the magnetic theory, which merits further investigation.

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