Why should plants be evergreen?

SIR — I read with interest Peter D. Moores's article "Why be an evergreen?" (*Nature* **313**, 703;1984). One factor referred to as a possible explanation for leaf longevity was leaf fall as a potential sterilizing agent. It was stated that many evergreens grow in impoverished soils. It has been argued in the past that plants growing in these conditions need to invest more heavily in chemical protection against phytophagous insects since growth on poor soils is slow, increasing vulnerability to attack. Many evergreens appear to be particularly rich in chemicals which defend them from phytophagous insects (terpenes associated with pine, for example). On the other hand, where growth is less costly, the alternative defence against phytophagous insects may be simply to shed leaves synchronously — that is, to adopt the deciduous habit to cleanse the tree annually of pests.

This hypothesis is open to an experimental test. If an evergreen is fumigated to remove phytophagous insects at the same time a deciduous species is coming into leaf, phytophagous insect population growth, measured as insect biomass per tree biomass, should be more rapid on the latter than the former. A favourable result would,however, be indicative rather than conclusive support of the hypothesis.

The hypothesis would be suspect if the maximum insect biomass achieved per tree biomass were higher on the evergreen. The reverse of this result would be consistent with the hypothesis whereas similar maxima would be the least informative outcome (although not inconsistent with the hypothesis).

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Assessing the risk of dioxin exposure

SIR — The health risk of exposure to 2,3,7,8-tetrachlorodibenzodioxin (dioxin) is a subject of considerable international interest which has been addressed recently in *Nature*[1] and *Science*[2,3]. As noted many times, understanding the nature of risk to humans is difficult because of the relative lack of information from epidemiological studies. Because of this, great emphasis has been placed on the results of two papers drawing on the mortality experience of workers exposed to dioxin at the Monsanto chemical plant in Nitro, West Virginia[4,5].

As with many occupational health studies, there are problems with these papers in the definition, and consistency of definition, of exposure. Zack and Suskind[4] report nine cancer deaths in the cohort of 121 workers exposed to tetrachlorodibenzodioxin in a specific trichlorophenol process accident, occurring in 1949. These nine deaths (Table 2 in the paper) give details of the individual's year of birth, year of hire by Monsanto, year of death and the cause of death. In the second paper, by Zack and Gaffey, published three years later[5], there are lists of death from cancer amongst workers at Monsanto who are described as having been exposed to the herbicide 2,4,5-T. Workers exposed to 2,4,5-T were also exposed to varying concentrations of dioxin and were classified on the basis of having contracted the sentinel indicator disease chloracne. Zack and Gaffey report 25 deaths from cancer in the controls, workers not exposed to 2,4,5-T. This is referred to in Table II of the paper[5].

In comparing the two papers, some men are listed as exposed to dioxin in one paper, but as not exposed in the other. In Table 2 in the Zack–Suskind paper, four cases are described as exposed to dioxin, whereas in the Zack–Gaffey paper, Table II reports the same four being not exposed (to 2,4,5-T and dioxin). Cases 1,2,5 and 7 in the Zack–Suskind Table 2 are those listed as being exposed to dioxin whereas in Table II of the Zack–Gaffey study, the same individuals are described (lines 6,7,9 and 22 of table) as being non-exposed. Judith Zack, an author on both papers, has reported that these individuals are one and the same[6].

In addition, a number of exposed individuals may not have been included in the second Monsanto study. In Monsanto records, there are some 19 individuals who died of circulatory disease or cancer whilst in employment at the company, and who meet the criteria for inclusion in the exposed group. No reason is given for not including these cases.

Determining exposure in the context of the Nitro plant is clearly difficult, since conditions may have produced general exposure to much of the work force. Those men exposed to the chemical in the 1949 trichlorophenol process accident are said to have been seriously exposed, on the basis of developing more severe chloracne, than the men exposed to dioxin in the course of making 2,4,5-T over several decades at Nitro. It may be the case that some of the more severe chloracne cases were the workers exposed to dioxin in the trichlorophenol process accident. However, there was a similar spectrum of chloracne incidence in both groups according to a study in 1953[7]. Of the 117 cases of chloracne determined in 1953 in individuals exposed to dioxin as a result of the 1949 accident, 10 workers had left the company, 80 were said to have recovered from the skin disease, 24 had a mild form of chloracne, and 3 had moderately severe disease. In other workers exposed during 2,4,5-T manufacture, 97 additional cases of chloracne were reported as having occurred, 51 of these cases were regarded as being clear of the disease, 36 were said to have a mild form and 3 to have moderately severe skin disease. Seven of the 97 men had left the company. Thus the spectrum of chloracne in the two groups seems similar. Another reason why it is inappropriate to separate these two exposed groups comes from information from a former manager of the Nitro plant[8]. In his deposition to court, Durland reported that some of the same workers were involved both in the clean-up after the trichlorophenol reactor accident and in making 2,4,5-T.

It may not be appropriate to classify degrees of dioxin exposure solely on the basis of chloracne as an indicator of relatively acute high-dose exposure to certain chlorinated hydrocarbons, including dioxin[9]. Other health effects — notably serious diseases of long latency, such as cancer — may be expected to result from lower and more chronic exposure[10-13]. Using one outcome variable to predict others is not advisable. Occupational studies usually use work histories, biological monitoring, or environmental measurements to determine, or approximate, exposure of subjects.

In view of the importance of the Monsanto cohort for determining human health risks of dioxin, it is our view that data from the company need to be reassessed. The *total* cohort of workers exposed to dioxin at Monsanto should be considered as a whole without making a distinction, as apparently done by Suskind, Zack and Gaffey, between workers exposed to dioxin in the trichlorophenol process accident or when making 2,4,5-T.

It may be that the overall number of deaths — from cancer, circulatory disease or other causes — is no greater than an appropriate control cohort. Data from a recent study by Moses *et al.*[14] on this same population suggests that there is an excess of cardiovascular-related deaths[15].

The epidemiological picture at Monsanto remains confused. We believe that more information will only be established by re-examining all of the data, and, given international concerns over dioxin, that this needs to be done urgently.

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