of the oceans was greater than it is today; however, his list of high sea levels confuses the issue, because it includes those (for example, at 81,000 and 100,000 years ago) that are thought by many to have been lower than today's (*2*). The last occasion when, by general agreement, absolute sea level was higher than it is today was about 120,000 years ago, and Emiliani's hypothesis should be examined in the light of events at that time.

What reason is there to believe that the Greenland Ice Sheet was seriously depleted during an interglaciation? The ice sheet appears to be well protected against a moderate temperature rise; it is mostly hemmed in by coastal mountains, so that only a narrow peripheral zone now lies beneath the snow line. Most of the ablation is by calving, not melting. Emiliani's hypothesis to account for melting of the Greenland Ice Sheet cannot be used as evidence that melting did, in fact, take place. Thus his sole evidence is high interglacial sea level, and here the possible role of Antarctic ice cannot be ignored, even if current astronomical hypotheses are unable to explain its melting at that time.

Emiliani summarily dismisses the Antarctic Ice Sheet as a factor in controlling interglacial high sea levels on the grounds that its situation in high latitudes and centered on the pole precludes significant interglacial ablation. This is probably valid for about nine-tenths of the ice sheet—that on the continent of East Antarctica—but not for the peculiar ice cover that is grounded far below sea level in West Antarctica. Emiliani ignores an earlier hypothesis (*3*) that sought to explain interglacial high sea levels by the deglaciation of West Antarctica. This hypothesis pointed out that fringing ice shelves, which are essential for the continued existence of an ice sheet grounded far below sea level, must consist of "cold" ice below the pressure melting point, and will rapidly disintegrate by calving if the average temperature of the warmest month rises above freezing point at sea level. An ice sheet on land, on the other hand, can consist of temperate ice at the pressure melting point and is able to survive under much warmer conditions. Thus the ice of West Antarctica is not only much less tolerant of summer warmth than is the ice cover of either East Antarctica or Greenland, but it would also disintegrate faster under adverse temperature conditions.

Significantly, there is evidence apart from high interglacial sea level that temperatures in Antarctica were too high for the survival of the West Antarctic ice during at least one Northern Hemisphere interglaciation (*3, 4*). [Unfortunately, because neither isotopic dates nor conclusive stratigraphic information is available, these warm episodes cannot be assigned to a particular interglaciation]. Disappearance of this ice would have raised sea level by 4 to 5 m. Much evidence indicates an absolute sea level of about + 6 m 120,000 years ago (*2, 3*), suggesting that if the high sea level was glacially controlled, it was mainly the result of the deglaciation of West Antarctica, not of Greenland.

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I misinterpret nothing. Absolute dating shows perihelion coincidence for Greenland, anticoincidence for Antarctica. So it must be Greenland. However, West Antarctica could contribute by responding to albedo changes produced by events in Greenland.

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8 May 1970

Herbicide Usage

Galston (*1*) has called for additional restrictions on use of phenoxy herbicides. His recommendation is based on preliminary evidence that these chemicals may be teratogenic (*2*). Galston says that a pregnant women might be continuously exposed to 1 mg of herbicide per kilogram of body weight per day as a result of water contamination from defoliation operations in Vietnam. His estimate is based on an application rate of 27 pounds of phenoxy herbicide per acre (1 lb/acre = 1.04 kg/hectare).

We have monitored a number of normal operational aerial spray projects in cool, temperate forests of the Pacific Northwestern United States where application rates of phenoxy herbicides do not exceed 4 lb/acre. Here, spray residues do not move into water in a constant process of contamination. During chemical brush-control operations, small amounts of stream contamination occur during aerial application, but once this water leaves the treatment area, movement of additional herbicides to the stream is negligible (*3*). A combination of rapid degradation (*4*) and resistance to leaching (*5*) prevents stream contamination by herbicide residues after heavy rains. The likelihood of chronic exposure of man or animals to phenoxy herbicide residues from forest spraying seems remote (*6*).

The treatments that showed the teratogenic capacity of the phenoxy herbicides consisted of repeated high doses in mice and rats. Data pointing to human health hazards are limited in scope and qauntity; more information is needed on the effects of phenoxy herbicides on humans. Toxicity is related to magnitude and duration of dose, and the assumption of chronic persistence and continuous flow of phenoxy herbicides into water is not supported by the available records. A long history of field use of phenoxy herbicides has demonstrated that they interfere little with the quality of the environment (*7*).

Regulatory agencies are under increasing pressure to restrict the use of some pesticides. An adequate assessment of hazard requires consideration of both the likelihood of exposure to a significant dose and the toxicity of the chemical. Any decision to further restrict the use of phenoxy herbicides in the United States must be based on careful consideration of risks, both of use and nonuse, of these valuable chemical tools.

The scientific community certainly should support restrictions on the hazardous use of any chemical. Equally, this community should support retention of chemical land management tools that research and long experience have demonstrated to be safe. Our studies of normal use of herbicides in Pacific Northwest forests indicate that further restrictions on these uses of the phenoxy herbicides are not justified.

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2 February 1970

Newton and Norris, while agreeing that more information is needed on the effect of phenoxyacetic acid herbicides on humans, are willing to sanction the continued use of these compounds because studies indicate that their normal use yields benefits without appreciable dangers. Until the currently unclear situation regarding the teratogenicity of 2,4,5-T is resolved, I cannot agree with them. The data upon which my view is based have recently been well summarized (*1*).

It is now suspected that a tetrachlorodibenzodioxin impurity is the teratogenic agent in some commercial preparations of 2,4,5-T, occurring as 27 parts per million in the sample tested for teratogenicity by the Bionetics Research Laboratories (*2*).

It has been claimed that some commercial samples of 2,4,5-T, which have less than 1 part per million of this contaminant (*3*), are not teratogenic; but independent tests by the Food and Drug Administration are needed to solidify this claim. If, in fact, it is the dioxin which is effective in inducing developmental malformations, then all previous analyses for detection of residual traces of phenoxyacetic acids are irrelevant. What we need now is a crash program to answer the following questions:

1) Are the phenoxyacetic acids themselves teratogenic?

2) If not, do commercial preparations of these compounds used in agricultural practice in the United States contain impurities, such as the dioxins, which *are* teratogenic?

3) Can the phenoxyacetic acids be degraded, either in the plant, in the soil, or by virtue of fire or bright sunlight into dioxin-like teratogenic agents?

4) Are the dioxins biodegradable? What is their half-life in the plant and in the soil?

5) Are there any dioxins or other potentially teratogenic relatives of the phenoxyacetic acids in the drinking waters around areas which have been extensively sprayed with 2,4-D and 2,4,5-T?

Until these questions are satisfactorily answered, I would recommend that we halt or at least seriously restrict the use of the phenoxyacetic acid herbicides. Certainly this would be inconvenient in lots of ways. For example, it would cause economic distress to the companies that manufacture the products; it would cause foresters, power companies, and land managers to seek other, temporary ways to control unwanted trees and brush. But this, I submit, is a relatively small price to pay while we are getting the hard data that we need to protect the health of the public.

While we are on the matter of teratogenic pesticides, why has no one raised a fuss about pentachloronitrobenzene (PCNB)? This compound is widely used as a soil fungicide for cotton, crucifers, potatoes, lettuce, peanuts, wheat, beans, tomatoes, peppers, and ornamentals. The same Bionetics Research Laboratory report which implicated 2,4,5-T as a teratogenic compound also showed that PCNB was teratogenic. Because 2,4,5-T is used in Vietnam, both the government and the scientific community have paid attention to it. Why has there been no corresponding interest in PCNB? Must we wait for definite proof of an abnormal birth before we are prepared to act? Have we learned nothing from the thalidomide tragedy?

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13 May 1970

Synthetic Juvenile Hormone and "Synthetic Juvenile Hormone"

I wish to draw attention to a growing problem of nomenclatural abuse in the field of insect hormones. With increasing frequency, many of the juvenile hormone (JH) mimics under biological investigation are referred to as synthetic juvenile hormone, juvenile hormone analogs, or juvenile hormone. For the most part they are none of these. It is not unusual to find titles and cursive texts to be in terms of JH, while careful examination of the experimental detail reveals that the substance under study is a structurally unrelated or unknown hormonomimetic. Among their many remarkable achievements in the field of insect hormones, Williams and his colleagues (*1*) described the preparation and JH-like properties of a product from the reaction of farnesoic acid with ethanolic hydrochloric acid. Unfortunately, the multicomponent material was subsequently referred to as synthetic juvenile hormone. It may be presumed that the complex and structurally unknown mixture contains no JH, which, as isolated from the cecropia moth, is now known to be methyl *trans,trans,cis*-10-epoxy-7-ethyl-3,11-dimethyl-2,6-tridecadienoate. Juvenile hormone has been synthesized by a variety of routes (*2*); synthetic JH thus exists and, further, will seemingly be increasingly available for biological study (*3*). By introducing the JH-active Williams mixture under the name "juvenile hormone, synthetic," one commercial firm now compounds the confusion (*4*).

I urge that terminology which is necessarily precise to the chemist be respected when hormonomimetic substances are described, and that the names "synthetic juvenile hormone" and "juvenile hormone, synthetic," when these refer to the JH-active Williams mixture, no longer be used.

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