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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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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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