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Wallace

COMMENTS

TCDD in the Environment

In the introduction to their paper, Hawkes and Norris (1977) noted that serious concern had arisen over the possible toxic effects of use of 2,4,5,-T[1] because TCDD[2] is present as a contaminant. 2,4,5,-T has been acknowledged as a teratogen as potent as thalidomide, but TCDD (dioxin) is about 10^5 times more potent than either (Steinfeld 1970). Hawkes and Norris further state that their study determined "the toxicity of TCDD to rainbow trout (*Salmo gairdneri*) and the possible implications of this type of exposure." I believe that the article is misleading in regard to both rainbow trout in particular and the TCDD controversy in general.

Hawkes and Norris estimate that the entry level of TCDD to streams in areas sprayed with 2,4,5-T is 0.0006–0.0085 mg/m² of stream bottom per year, based on concentrations of dioxin on organic litter. Reasoning from their laboratory results, the authors conclude that potential environmental exposures are safe for rainbow trout. However, they cannot guarantee that the low dosages implied by these low average entry values are realistic. There are several reasons why they cannot.

Herbicides for brush control are sprayed from airplanes, trucks, or backpacks. The deposition area and rates depend on both the control of the spray device and the local and continental weather parameters. The only time DDT[3] was used in the United States in recent years, when it was sprayed from aircraft to control a moth infestation in the Pacific Northwest during summer 1974, the chemical rained down on New York State (Peakall 1976). Application by careful backpack spraying is similar to Mirex[4] application for control of fire ants (*Solenopsis* sp.); in states where Mirex is used, elevated concentrations are being found in aquatic organisms (Waters et al. 1977).

Relatively water-insoluble organochlorines bind to soil as well as to organic matter. Soil erosion in areas of depleted plant coverage, of heavy equipment or off-road traffic, or of new roadbeds and other development does not occur steadily over a long period, but suddenly and massively during storms and thaws. A recent study on use of herbicides for rights-of-way maintenance showed periodic erosion occurring under freshet conditions (Carvell and Johnston 1978). Local temporary concentrations of eroded particles in water bodies can be high and can expose aquatic organisms to high concentrations of chemicals absorbed on the particles. The particles then settle out on the bottom generally in high localized concentrations and form a source of contamination for resident organisms, especially the epibenthic invertebrates and demersal fish. Further, floods, strong currents, and unusual turbulence which stir up sediment cause transport of contaminated particles into previously unaffected areas. For example, in spring 1976, a single heavy flood moved PCB[5]-laden sediment over dams and down into the tidal sector of the Hudson River, approximately 24 km below the southernmost dam (NYSDEC 1976). Dredging and navigation also expose planktonic organisms and fish to contaminated sediments. Land disposal of contaminated dredge spoils merely recycles the absorbed organochlorines.

Hundreds of tons of 2,4,5-T enter the environment every year in the United States alone, much of which must result in TCDD-contaminated aquatic sediments. Table 1 gives an example for 1974.

For the above reasons, I conclude that an average annual entry value of any ring-structured organochlorine per m² of stream bottom is useless in the estimation of the exposure of fish to the chemical.

Biomagnification must also be considered. Recent research has shown that fish, birds, and mammals sometimes accumulate pesticides in unexpectedly high amounts along environmen-

[1] 2,4,5-trichlorophenoxyacetic acid.
[2] 2,3,7,8-tetrachlorodibenzo-*p*-dioxin.
[3] Dichlorodiphenyltrichloroethane.
[4] Dodecachlorooctahydro-1,3,4-methano-2H-cyclobuta [cd] pentalene.
[5] Polychlorinated biphenyls.

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tal pathways that are still undescribed (Waters et al. 1977). Because of their hydrophobia and very slow-to-nonexistent rates of metabolism, ring-structured organochlorines exhibit biomagnification in aquatic organisms both by direct uptake and by food chain concentration effects. Direct uptake or concentration by herbivores is generally between 1,000 and 10,000 times the ambient levels (Nisbet and Sarofim 1972; Warlen 1974; Waters et al. 1977; Zitko et al. 1972). TCDD presents a special problem in that the common detection methods are not sensitive enough to find it at ambient concentrations which may result in toxicity after biomagification (Zitko et al. 1972). Its extreme hydrophobia keeps it out of the water column, limiting the organisms likely to accumulate it to the bottom-oriented groups (Matsumura and Benezet 1973) except during periods of high sediment load in the water column.

Another complication of TCDD biomagification and toxicity is the presence of 2,4,5-T or other organic pollutants which may act synergistically with the dioxin. Synergism has been demonstrated between organochlorines (Lichtenstein 1972), between organochlorines and organophosphates (Franklin 1976), and between TCDD and known chemical carcinogens (Lucier et al. 1978). Greater concentrations of TCDD may be accumulated, and the toxic effect of a given dose of TCDD is probably greater, in the presence of other organochlorines than if the dioxin were present alone. Laboratory exposures to single toxins may not reflect the chemical environment in nature.

Hawkes and Norris found histological evidence of liver degeneration in a rainbow trout fed TCDD at a concentration of 2.3 ng/g food. However, histology is a relatively insensitive tool in testing for TCDD toxicity; testing for activation or deactivation of certain enzyme systems is more appropriate. A brief summary of important results of studies of dioxin and related compounds follows.

A measure of the toxicity of a chemical is how low a dose will induce such detoxifying enzymes as aryl hydrocarbon hydroxylase (AHH). TCDD induced AHH in mice at extremely low doses (1 nmole/kg body weight for sensitive strains; 10 nmole/kg for nonsensitive strains) (Poland and Kende 1976) and in the little skate *Raja erinacea* at low doses (Bend et al. 1976). Juvenile coho salmon (*Oncorhynchus kisutch*) showed enhanced AHH activity after low dosage with pentachlorobiphenyl, a close relative of TCDD (Gruber et al. 1976). The action of TCDD as a carcinogen and a co-carcinogen has been attributed to its induction of AHH (Kouri et al. 1973; Lucier et al. 1978; Rice 1978).

Doses of TCDD as low as 10 μg/kg caused detectable effects on kidney function of laboratory rats: decreased filtration rates and decreased capacity for renal transport (Pegg et al. 1976).

All ring-structured organocholorines adversely affect the female mammalian reproductive system by inducing uterine enzymes or by mimicking the steroid hormone estradiol and binding to uterine enzyme receptors (Kupfer and Bulger 1976). The lipids of the human uterus act as a semifilter for organochlorines and differentially concentrate them, although all organochlorines studied have been found to cross the placenta (Polishok et al. 1977). TCDD-contaminated 2,4,5-T was applied in Viet Nam under the name of Agent Orange; statistically significantly more human stillbirths occurred in sprayed areas than in unsprayed areas (Meselson et al. 1972). TCDD causes miscarriages in humans (Norman and Hay 1976) and fetal deformations in mice (Smith et al. 1976). Organochlorines also affect the reproduction and embryological development of some aquatic invertebrates (Bresch and Arendt 1977).

TCDD affects the human immunological system by causing a decline in white blood cell count (Hay 1976).

TABLE 1.—*Reported usage of 2,4,5-T in the United States in 1974.*[a]

Use category	Metric tons applied
Agriculture	
Rangeland and pastures	439–1,040
Rice	7–100
Nursery crop	5
Federal and state agencies	147
Rights-of-way	1,800
Forestry[b]	23
Miscellaneous industry	299
Total	2,720–3,410

[a] 2,4,5-T = 2,4,5-trichlorophenoxyacetic acid. Source: USEPA (1978).

[b] It is unclear from the USEPA (1978) text whether or not "Forestry" is actually included in the "Federal and state agencies" category.

PCB-ARCH-EXT0375939

TCDD has been linked to personality changes in humans (Anonymous 1976).

The young rainbow trout used by Hawkes and Norris are among the least likely fish to accumulate organochlorines. Fatty fish such as Atlantic menhaden *Brevoortia tyrannus* (Warlen 1974) and long-lived fish such as American eel *Anguilla rostrata*, striped bass *Morone saxatilis*, and brown trout *Salmo trutta* (NYSDEC 1975, 1976), and carp *Cyprinus carpio*, channel catfish *Ictalurus punctatus*, and largemouth bass *Micropterus salmoides* (USEPA 1976) accumulate these molecules efficiently. The rainbow trout are also not demersal and would inhabit the water stratum with the lowest TCDD concentration. Therefore, the data presented for rainbow trout are a "best-case" for 2,4,5-T impact assessment and should not be used in decision-making about effects of herbicidal brush control on fish in general.

Testing rainbow trout too young to have accumulated their most probable levels of the organochlorines which humans would encounter in fish to be eaten does nothing to allay the central point of the controversy, namely that spraying hundreds of tons of dioxin-contaminated 2,4,5-T each year may end in toxic reactions in humans and in the animals on which humans depend.

The United States Environmental Protection Agency's Pesticide Programs have issued a "Rebuttable Presumption against Registration and Continued Registration of Pesticide Products Containing 2,4,5-T." Much of the foundation of this Presumption consists of data on the toxic effects of TCDD on both animals and humans. Producers and users of the herbicide had until June 5, 1978 to submit rebuttal evidence (USEPA 1978).

References

Anonymous. 1976. Seveso. Lancet 2 (7980):297.

Bend, J. R., R. J. Pohl, E. Arine, and R. M. Philpot. 1976. Hepatic microsomal and solubilized mixed function oxidase systems from the little skate, *Raja erinacea*, a marine elasmobranch. Hoppe-Seyler's Zeitschrift fuer Physiologische Chemie 357:1021–1022.

Bresch, H., and U. Arendt. 1977. Influence of different organochlorine pesticides on the development of the sea urchin embryo. Environmental Research 13:121–128.

Carvell, K. L., and P. A. Johnston. 1978. Environmental effects of right-of-way management on forested ecosystems. Report EA-491, Research Project 103-3, prepared for Electric Power Research Institute by the Division of Forestry, West Virginia University, Morgantown, West Virginia, USA.

Franklin, M. R. 1976. Methylenedioxyphenyl insecticide synergists as potential human health hazards. Environmental Health Perspectives 14:29–37.

Gruber, E. H., T. Hruby, and N. L. Karrick. 1976. Sublethal effects of structurally related tetrachloro-, pentachloro-, and hexachlorobiphenyls on juvenile coho salmon. Environmental Science and Technology 10:1033–1037.

Hawkes, C. L., and L. A. Norris. 1977. Chronic oral toxicity of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) to rainbow trout. Transactions of the American Fisheries Society 106:641–645.

Hay, A. 1976. Seveso: the aftermath. Nature (London) 263:538–540.

Kouri, R. E., R. A. Salerno, and C. E. Whitmire. 1973. Relationships between aryl hydrocarbon hydroxylase inducibility and sensitivity to chemically induced subcutaneous sarcomas in various strains of mice. Journal of the National Cancer Institute 50:263–268.

Kupfer, D., and W. H. Bulger. 1976. Interactions of chlorinated hydrocarbons with steroid hormones. Federation Proceedings, Federation of American Societies for Experimental Biology 35:2603–2608.

Lichtenstein, E. P. 1972. PCB's and interactions with insecticides. Environmental Health Perspectives Experimental Issue 1:151–154.

Lucier, G. W., C. S. Dieringer, W. Powell-Jones, E. Lui, and C. A. Lamartiniere. 1978. Metabolic activation/deactivation reactions during perinatal development. Conference on Pollutants and High Risk Groups, June 5–6, sponsored by the United States Environmental Protection Agency and Conducted by the Division of Public Health, University of Massachusetts, Amherst, Massachusetts, USA.

Matsumura, F., and H. J. Benezet. 1973. Studies on the bioaccumulation and microbial degradation of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. Environmental Health Perspectives 5:253–258.

Meselson, M. A., A. H. Westing, J. D. Constable, and J. E. Cook. 1972. Preliminary report of the Herbicide Assessment Commission (of the American Association for the Advancement of Science). Congressional Record 118 (1972):S3226–3233.

Nisbet, J. C. T., and A. F. Sarofim. 1972. Rates and routes of transport of PCB's in the environment. Environmental Health Perspectives Experimental Issue 1:21–38.

Norman, C., and A. Hay. 1976. Seveso: the problems deepen. Nature (London) 264:309–310.

NYSDEC (New York State Department of Environmental Conservation). 1975. PCB moni-

PCB-ARCH-EXT0375940

toring in the u[illegible] Hudson River Basin. October 31. Report SAN-p14 (10/75), Albany, New York, USA.

NYSDEC (New York State Department of Environmental Conservation). 1976. Hudson River PCB monitoring. Data summary, past, present, proposed. Division of Pure Waters, Division of Fish and Wildlife, October 26 (unnumbered), Albany, New York, USA.

Peakall, D. B. 1976. DDT in rainwater in New York following application in the Pacific Northwest. Atmospheric Environment 10:899–900.

Pegg, D. G., W. R. Hewett, K. M. McCormack, and J. B. Hook. 1976. Effect of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin on renal function in the rat. Journal of Toxicology and Environmental Health 2:55–65.

Poland, A., and A. Kende. 1976. 2,3,7,8-tetrachlorodibenzo-*p*-dioxin: environmental contaminant and molecular probe. Federation Proceedings, Federation of American Societies for Experimental Biology 35:2404–2411.

Polishuk, Z. W., D. Wasserman, M. Wasserman, S. Cucos, and M. Ron. 1977. Organochlorine compounds in mother and fetus during labor. Environmental Research 13:278–284.

Rice, J. 1978. Transplacental carcinogenicity. Conference on Pollutants and High Risk Groups, June 5–6, sponsored by the United States Environmental Protection Agency and conducted by the Division of Public Health, University of Massachusetts, Amherst, Massachusetts, USA.

Smith, F. A., B. A. Schwetz, and K. D. Nitschke. 1976. Teratogenicity of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in CF-1 mice. Toxicology and Applied Pharmacology 38:517–523.

Steinfeld, J. 1970. Testimony of the Surgeon General before the Commerce Committee of the 91st Congress: effects of 2,4,5-T on man and the environment. Second Session, April 7 and 15. Available in booklet of transcript published by the Commerce Committee and in Congressional Record.

USEPA (United States Environmental Protection Agency). 1976. Review of PCB levels in the environment. EPA-560/7-76-001, Office of Toxic Substances, Washington, District of Columbia, USA.

USEPA (United States Environmental Protection Agency). 1978. Rebuttable presumption against registration and continued registration of pesticide products containing 2,4,5-T. Pesticide Programs. Federal Register 43 (78):17116–17157.

Warlen, S. M. 1974. Accumulation of organochlorine insecticides by Atlantic menhaden, *Brevoortia tyrannus*, from the Newport River Estuary. Journal of the Elisha Mitchell Scientific Society 90:108–109.

Waters, E. M., J. E. Huff, and H. B. Gerstner. 1977. Mirex, an overview. Environmental Research 14:212–222.

Zitko, V., O. Hutzinger, and P. M. K. Choi. 1972. Contamination of the Bay of Fundy-Gulf of Maine area with polychlorinated biphenyls, polychlorinated terphenyls, chlorinated dibenzodioxins, and dibenzofurans. Environmental Health Perspectives Experimental Issue 1:47–50.

Deborah Wallace

Power Authority, State of New York[6]
10 Columbus Circle
New York, New York 10019

We are disturbed by Wallace's comments on our paper (Hawkes and Norris 1977) for two reasons: (1) she apparently did not read our paper carefully because she feels we made inferences or drew conclusions that we did not; (2) she appears afraid we will say TCDD in the aquatic environment is acceptable. Many of her comments go substantially beyond the scope of our paper. Unfortunately, her comments pertinent to our paper are intermixed with those that address broader issues. The scope of our study is clearly stated in the title and the introduction: to determine the chronic oral toxicity of TCDD to rainbow trout and to consider the possible implication of this type of exposure in terms of the specific observations of toxicity we cited in the introduction, the methods, and the results sections.

Obviously there are other types of exposure regimes that could have been used. However, after careful evaluation, we determined that exposure via the food (which we report in the subject paper) and via water (which we report in Miller et al. 1973, Norris and Miller 1974, and a manuscript by Miller and Norris submitted for publication) are most important. Equally obvious, there are other types of biological responses we could have studied, but surely the measurements of survival, growth, feeding, behavior, disease, and internal organ pathology which we included in our study, are of substantive importance.

The guarantees Wallace wants that our calculated levels of TCDD entry into aquatic systems will not be exceeded are not possible. Our reasoning and the data we used are clearly presented. Wallace infers our values are low, but

[6] For identification only. This Comment does not represent a technical or policy position of the Power Authority.

PCB-ARCH-EXT0375941

offers no data concerning TCDD to support her contention. She cites papers dealing with DDT, Mirex, and PCB, none of which appear relevant to the scenario we presented. Her concerns about erosion are not well founded. Sheet erosion on forest land is unusual because in most areas the infiltration capacity of the land exceeds rates of precipitation (Dyrness 1967). Studies by Norris (1967) and Norris et al. (1978) did not detect 2,4,5-T in forest streams flowing from sprayed forest areas in Oregon or West Virginia during periods of heavy rain the first year after application. These data and concepts argue against Wallace's conclusion that erosion will be a significant source of TCDD in forest streams.

Even if erosion occurs, the contribution of TCDD from these areas would be quite limited because of the short persistence (half-life, approximately 50 hours) of TCDD on soil when exposed to sunlight (Crosby and Wong 1977). TCDD binds tightly to soil and organic matter (Helling et al. 1973). When absorbed it is not readily available to fish. If it is accumulated by processors and enters the food chain, exposure may occur. That was exactly the thrust of our study, to determine the toxicity of TCDD in food to fish.

Wallace states that "hundreds of tons of 2,4,5-T enter the environment every year in the United States alone, much of which must result in TCDD-contaminated aquatic sediments." Data supporting her contention are lacking. Shadoff et al. (1977) did not find detectable (less than 3 ng/kg in mud, 0.1 ng/liter in water) levels of TCDD in water or mud from rice and range areas treated regularly with 2,4,5-T.

Wallace also is concerned because we did not consider TCDD bioaccumulation. The physical-chemical properties of TCDD indicate bioaccumulation is possible. Laboratory studies of aquatic systems which have a substantive and continuing supply of TCDD show accumulation ratios (TCDD concentration in biota relative to concentration in water) of 1,000 to 10,000 times (Matsumura and Benezet 1973; Isensee and Jones 1975). In the field, however, neither a substantive nor a continuing source of TCDD will exist in aquatic systems in areas where 2,4,5-T is likely to be used. Miller and Norris report (in a manuscript submitted for publication) that TCDD exposure via the water results in the accumulation and persistence of easily measurable levels of TCDD in fish in the laboratory. Thus, if the bioaccumulation Wallace is concerned about is occurring to a significant degree, evidence in the form of TCDD residues in fish should be common. Shadoff et al. (1977) did not find detectable (less than 2 to 12 ng/kg) TCDD in fish (including catfish) from areas receiving drainage water from both range and rice areas which were sprayed with 2,4,5-T on a regular basis. The United States Forest Service (1978) reported that a survey for TCDD in forest animals conducted by the United States Environmental Protection Agency showed no detectable levels of TCDD in five samples of fish collected less than 1.6 km downstream from several spray units treated with 2,4,5-T on western forest lands. From these data we conclude TCDD can bioaccumulate in aquatic biota, but it does not appear to be doing so in this country at detectable levels.

Wallace is correct that it is difficult to analyze TCDD at concentrations less than 10 ng/kg in biological material. However, if it biomagnifies to toxic levels as she alleges, detection will be simplified. We found no effect on fish in our study exposed for 105 days to concentrations as high as 2.3 ng/g. Measuring TCDD at 2.3 ng/g in biological material is relatively easy in a modern pesticide analytical laboratory.

Wallace is also concerned that we underestimated the toxicity of TCDD because of possible "synergistic effects from other pollutants." While we did not specifically test this effect, it is in fact an inherent part of our test because the fish, their food, and the stream water in our system all probably contained the usual complement of persistent organochlorine pesticides Wallace wants included in our tests.

Wallace is mistaken about the potency of TCDD as a teratogen. Toxicologists class TCDD as a relatively weak teratogen because the teratogenic dose is nearly equal to the lethal dose (Dost 1978). Substantial discussion and references are offered to indicate enzyme induction is a more sensitive tool than the gross histopathology we performed. We agree, in part, but enzyme induction was beyond the scope of our study. Interestingly, the TCDD concentration of 1 nmole/kg that Wallace cited for aryl hydrocarbon hydroxylase induction is equivalent to 0.32 ng TCDD per gram; this is about 10 times greater than the daily intake of 0.036 ng/g we found did not have an effect in

PCB-ARCH-EXT0375942

our test. The 10 μg/kg (0.01 μg/g) she indicates affects kidney function is about equal to the highest level in our test (0.02 μg/g), which we found caused heavy mortality in trout. Thus, it appears to us that the indicators of toxicity we used were reasonably sensitive and our exposure levels bracketed the values reported by others to cause toxic effects in other types of animals. In any case, if enzyme induction, kidney malfunction, or incipient carcinogenicity were occurring in the fish in our test, these anomalies did not affect survival, growth, feeding behavior, disease, or liver pathology after 105 days exposure to TCDD.

Inferences about possible human health effects caused by TCDD in Vietnam (Agent Orange) and in Seveso, Italy (from an industrial explosion) are not relevant to our paper.

Wallace argues rainbow trout are a poor choice for a test species. We disagree. Rainbow trout or a similar species are common inhabitants of forest streams. Certainly other species could and should be tested, but to propose that our data "should not be used in decision-making about effects of herbicidal brush control on fish in general" is absurd. Is Wallace seriously suggesting these decisions for range and forest areas be based on tests with the Atlantic menhaden in lieu of trout?

Wallace's final comments we believe illustrate the motivation for her thrust at our paper. She indicates that tests with young rainbow trout do nothing to allay the critical point of the controversy, namely, that "spraying hundreds of tons of dioxin-contaminated 2,4,5-T each year may end in toxic reactions in humans and in the animals on which humans depend." We neither stated nor inferred such a conclusion. It is clearly beyond the scope of our paper. We note here, however, that 2,4,5-T has in fact been an important tool in forestry and agriculture for 30 years in the United States. Despite this prolonged and extensive pattern of use, the ecological disaster suggested by Wallace has not materialized.

We made every effort to conduct our study and report our results in a manner that was creditable scientifically and that did not engage in the rhetoric of the 2,4,5-T controversy. We reaffirm our finding that more than 2.3 ng TCDD/g is required in the food of young rainbow trout to produce measurable changes in their survival, growth, feeding behavior, disease, or liver pathology. We doubt that these levels occur in streams that drain the forest and rangelands of the United States treated (according to the label directions) with 2,4,5-T.

References

Crosby, D. G., and A. S. Wong. 1977. Environmental degradation of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). Science 195:1337–1338.

Dost, F. N. 1978. Toxicology of phenoxy herbicides and hazard assessment of their use in reforestation. United States Department of Agriculture Forest Service, California-Pacific Region, Sacramento, California, USA.

Dyrness, C. T. 1967. Erodibility and erosion potential of forest watersheds. Pages 599–611 *in* W. E. Sopper and H. W. Lull, editors. International symposium on forest hydrology. Pergamon, Oxford, England.

Hawkes, C. L., and L. A. Norris. 1977. Chronic oral toxicity of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) to rainbow trout. Transactions of the American Fisheries Society 106:641–645.

Helling, C. S., A. R. Isensee, E. A. Woolson, P. D. J. Ensor, G. E. Jones, J. R. Plimmer, and P. C. Kearney. 1973. Chlorodioxins in pesticides, soils and plants. Journal of Environmental Quality 2:171–178.

Isensee, A. R., and G. E. Jones. 1975. Distribution of 2,3,7,8-tetrachlorodiobenzo-*p*-dioxin (TCDD) in aquatic model ecosystems. Environmental Science and Technology 9:668–672.

Matsumura, F., and H. J. Benezet. 1973. Studies on the bioaccumulation and microbial degradation of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. Environmental Health Perspectives 5:253–258.

Miller, R. A., L. A. Norris, and C. L. Hawkes. 1973. Toxicity of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) in aquatic organisms. Environmental Health Perspectives 5:177–186.

Norris, L. A. 1967. Chemical brush control and herbicide residues in the forest environment. Pages 103–123 *in* Proceedings of the Herbicide and Vegetation Management Symposium, Oregon State University, Corvallis, Oregon, USA.

Norris, L. A., and R. A. Miller. 1974. The toxicity of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) in guppies (*Poecilia reticulatus* Peters). Bulletin of Environmental Contamination and Toxicity 12:76–80.

Norris, L. A., M. L. Montgomery, C. A. Troendle, and J. M. Kochenderfer. 1978. 2,4,5-T persistence in a West Virginia watershed. Weed Science 83:38. (Abstract.)

Shadoff, L. A., R. A. Hummel, L. Lamparski, and J. H. Davidson. 1977. A search for 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) in an environment exposed annually to 2,4,5-trichlorophenoxyacetic acid ester (2,4,5-T) herbicides.

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Bulletin of Environmental Contamination and Toxicology 18:478–485.

United States Forest Service. 1978. Vegetation management with herbicides, volume 1. Final environmental impact statement. Pacific Northwest Region, Portland, Oregon, USA.

Logan A. Norris
Clifford L. Hawkes[1]

United States Department of Agriculture Forest Service Pacific Northwest Forest and Range Experiment Station Forestry Sciences Laboratory, Corvallis, Oregon 97331

[1] Present address: United States Department of Agriculture Forest Service, Rocky Mountain Forest and Range Experiment Station, Forest Research Laboratory, South Dakota School of Mines, Rapid City, South Dakota 57701.

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