sociated with an IBM 1800 computer.

There are a number of other automatic telescopes under construction, including at least three in the Soviet Union, and also an elaborate installation on Mount Haleakala, Maui, which will be partially devoted to infrared astronomy. However, the purpose of the present paper is not to catalogue every scheme that has been advanced to automate the optical observatory. Rather, an attempt has been made to illustrate the methods adopted by several groups in their efforts to solve specific astrophysical problems and increase the productivity of optical telescopes through automation.

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Pesticide Pollution Control

Suggestions are made for improving the quality of water subject to pollution by pesticides.

H. Page Nicholson

The Water Quality Act of 1965 amended the Federal Water Pollution Control Act to provide, among other things, for the establishment of water quality standards for interstate waters. The purpose of the act is to enhance the quality and value of our water resources and to establish a national policy for the prevention, control, and abatement of water pollution. Various pesticides now are recognized to be water pollutants and, as such, they must eventually be considered in the establishment of water quality standards.

Pesticide Pollution History

Water pollution by pesticides began to occur about 22 years ago when the organic insecticides developed just before and during World War II reached the public market. There followed an almost explosive expansion of chemical pesticides; today there are more than 650 basic kinds, of which at least 200 are of first importance. The tonnage of pesticides sold in the United States increased by 84 percent in the decade 1955–1965. Production of basic pesticides in the United States in 1965 totaled 437,500 tons (396,900 metric tons) (*1*).

Fish kills associated with rainfall runoff following application of chlorinated hydrocarbon insecticides in agriculture were the first evidence that a new form of water pollution was occurring. Sporadic scattered kills were climaxed in 1950 by the almost simultaneous occurrence of kills in 15 streams tributary to the Tennessee River in Alabama (*2*). Subsequent investigation indicated that the kills were caused by insecticides washed from cotton fields following passage of a storm front with its attendant intense thunder showers.

The first recovery and identification of a water-polluting pesticide was made in 1953 by personnel at the Robert A. Taft Sanitary Engineering Center in Cincinnati, Ohio, who recovered DDT from the Detroit River and Lake St. Clair (*3*). The first quantitation was achieved in 1957, when DDT was detected in the Mississippi River at Quincy, Illinois, and at New Orleans, in the Missouri River at Kansas City, and in the Columbia River at Bonneville, Oregon, in concentrations ranging from 1 microgram to 20 micrograms per liter (*4*). The quantitation procedure involved passage of several thousand gallons of water through activated carbon, extraction of adsorbed residue, column cleanup, and analysis by infrared spectroscopy. The advent in 1959 of gas-liquid chromatography for pesticide identification (*5*), quickly followed by other developments in instrumental analysis, made possible the first detailed evaluation of water pollution, by pesticides, resulting from runoff (*6, 7*). These studies were undertaken in 1959 by the U.S. Public Health Service's Division of Water Supply and Pollution Control, Pesticide Pollution Studies Laboratory, in Atlanta, Georgia. (The work of this Division has since been taken over by the Federal Water Pollution Control Administration of the U.S. Department of the Interior.) This activity, considerably expanded, continues at the Administration's Southeast Water Laboratory at Athens, Georgia.

Present Status of Knowledge

The two principal sources of water pollution by pesticides today are runoff from the land and discharges of industrial waste, either from industries that manufacture or formulate pesticides or from those that use these compounds in their manufacturing processes. Less important causes of pollution are (i) activities designed to control undesirable aquatic life, (ii) careless use of pesticides, and (iii) occasional accidents in transportation. Instances of careless use have been decreasing as a result of intensive educational campaigns sponsored by agricultural, con-

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The author is chief of the Water Contaminants Characterization and Rural Runoff Control Research activities of the Southeast Water Laboratory, Federal Water Pollution Control Administration, U.S. Department of the Interior, Athens, Georgia.

servation, water-pollution-control and public-health agencies and by the pesticide manufacturing industry.

It is now evident that pollution of water by pesticides occurs widely. Breidenbach and Lichtenberg (*8*) reported on the qualitative analysis of carbon adsorption samples taken at 101 sampling stations throughout the United States from May through December 1962. DDT was found in 32 samples from nine rivers: the Susquehanna, Delaware, Chattahoochee, Tennessee, Rio Grande, San Joaquin, Sacramento, Yakima, and Columbia. Dieldrin was recovered from the Savannah River. Identification by gas chromatography was confirmed by infrared spectroscopy.

In September 1964, in a further effort to determine the extent of pesticide pollution, Weaver *et al.* (*9*) conducted a synoptic survey of the U.S. river systems. Whole-water samples were collected from 56 rivers and from three of the Great Lakes. These samples were analyzed for chlorinated hydrocarbon insecticides by microcoulometric gas chromatography. In samples from 44 of the rivers and from Lake Michigan at Milwaukee, insecticides were found, at concentrations varying from 0.002 to more than 0.118 μg/liter. Of particular interest was the widespread occurrence of dieldrin, which was found in 39 rivers and in Lake Michigan. DDT, or its metabolite DDE, was found in 25 rivers. Endrin was found in 22 rivers and in Lake Michigan. The insecticides recovered in only one sample were DDD (TDE) and aldrin.

We at the Southeast Water Laboratory have studied in detail insecticide runoff from agricultural land, in situations where the watersheds and agricultural practices are well defined (*6, 7*). It was demonstrated that toxaphene, dieldrin, BHC, endrin, DDT, and parathion can enter watercourses along with runoff water, and that the addition of pesticides to a watercourse can be more or less continuous throughout the year. Rarely were any of these insecticides recovered from water in concentrations greater than 1 μg/liter.

Several instances of water pollution associated with pesticide manufacture are on record. Two examples will serve as illustrations.

A plant in Alabama which manufactured parathion and methyl parathion experienced a breakdown in its waste-treatment facility in May 1961 (*10*). Process wastes were discharged to the sewerage system of an adjacent city, and approximately 60 percent of the combined sewage and industrial waste was diverted, untreated, to a small stream during the breakdown. Fish, turtles, and snakes died along 28 miles (45 kilometers) of the stream, and lesser fish kills occurred in the Coosa River for approximately 90 miles downstream from its confluence with the creek. Samples of creek water, city sewage, and thickened sludge from the city's treatment plant were all found to contain parathion when analyzed by the Averill-Norris colorimetric procedure. For parathion, however, this method is not strictly definitive.

A second fish kill occurred in the creek in March 1966. The Southeast Water Laboratory recovered parathion in concentrations ranging from 2 to 51 μg/liter in nine of 12 river-water samples collected during the kill. Identification was made by electron-capture and microcoulometric gas chromatography. The identification of parathion was confirmed by infrared spectroscopy. Brain concentrations of acetylcholinesterase in moribund fish were found to be 68.0 to 71.5 percent below normal—a degree of depression sufficient to cause death.

The second example involved a plant in South Carolina that manufactures inorganic and organic phosphatic compounds, including a nematocide [*O,O*-diethyl,*O*-(2,4-dichlorophenyl) phosphorothioate], and a defoliant (*S,S,S,*-tributyl phosphorotrithioate). Wastes from this plant are discharged to the Ashley River, a tidal stream which is part of Charleston Harbor. The periodic occurrence of fish kills since 1961 prompted a request to the Southeast Water Laboratory for assistance. Investigation during June and July 1965 revealed evidence that the fish deaths were caused by inhibition of the activity of the enzyme acetylcholinesterase (*11, 12*). Although no inhibiting agent was recovered from the river, two were identified in waste-water samples from the plant. They were (i) the nematocide (one water sample contained 6.5 mg/liter and a matching sediment sample contained 300 mg/kg) and an oxidation product of the defoliant, *S,S,S,*-tributyl phosphorotrithioate (15 mg/liter in waste water and 5000 mg/kg in the mud) (*13*). The nematocide was identified by gas chromatography and infrared spectroscopy; the oxidation product of the defoliant was identified by the same means and the identification was confirmed by mass spectroscopy and nuclear-magnetic-resonance spectroscopy.

Frequently, indirect evidence can be obtained indicating the presence or past presence of a pesticide in the aquatic environment. Recovery of chlorinated hydrocarbon insecticides from tissues of aquatic organisms conclusively demonstrates exposure to these agents even when careful investigation may not reveal their occurrence in water or mud. Fish will store these chemicals in their tissues for long periods. My associates and I have analyzed livers from 30 fish caught alive in October 1964 from the Tombigbee-Alabama River complex north of Mobile, Alabama. These fish contained from one to five insecticides —DDT (detected 29 times; concentration, 0.018 to 47.1 mg/kg); dieldrin (11 times; concentration, 0.02 to 1.6 mg/kg); toxaphene (eight times; concentration, 0.1 to 10.9 mg/kg); endrin (five times; concentration, 0.01 to 0.7 mg/kg); BHC (four times; concentration, 0.002 to 0.8 mg/kg). The river mud at the collection sites contained DDT (0.05 to 410 mg/kg) at the time these fish were collected, but none of the other insecticides. Water samples collected periodically over the preceding 2 years frequently contained DDT (it was found in 23 of 58 samples, in concentrations ranging from 0.04 to 35.0 μg/liter), but other insecticides occurred rarely (four samples contained dieldrin in concentrations ranging from 2.3 to 4.0 μg/liter and seven contained BHC in concentrations of from 0.07 to 2.0 μg/liter). The sources of these insecticides were not determined. Keith (*14*) reported that single fish caught in California contained the insecticides DDT, DDD, dieldrin, and toxaphene and the epoxide of heptachlor.

Even aquatic animals far removed from areas where pesticides are known to have been used may have chlorinated hydrocarbon insecticide residues in their tissues. Sladen, Menzie, and Reichel (*15*) demonstrated that Adélie penguins and a crabeater seal, species that seldom range beyond the limits of the Antarctic ice pack, contained residues of DDT and its metabolites. Both of these animals feed on crustacea obtained from the sea. These analyses were made by gas chromatography, and the results were confirmed by thin-layer chromatography. DDT has been found in the oil of fish that live far out at sea and in fish caught off the coasts of North America, South America, Europe, and Asia (*16*).

It is evident that contamination of the aquatic environment by pesticides, primarily by the chlorinated hydrocarbon insecticides, is widespread. This is, in part, because these insecticides possess the characteristic of persistence —they degrade slowly in the environment. As a result, control of their distribution is lost soon after they are applied for the purpose of insect control. Persistence, one of the factors that has made the chlorinated hydrocarbon insecticides so desirable from the standpoint of insect control, then becomes an undesirable attribute.

In part, widespread environmental contamination by chlorinated hydrocarbon insecticides is a result of the extensive use made of these compounds. This group constituted approximately 52 percent of all insecticides and 30 percent of all pesticides produced in the United States in 1965 (*1*). DDT retained its position of some years' standing as the pesticide produced in greatest quantity, an estimated 70,000 tons (63,000 metric tons).

Significance

What can be said about the significance of water pollution by pesticides?

An Advisory Committee appointed by the Public Health Service in 1962 considered insecticides while reviewing drinking water standards and concluded that the information available was not sufficient to permit establishment of specific limits (*17*). The concentrations of chlorinated hydrocarbon and organophosphorus insecticides, where tested, were below levels which would constitute a known health hazard. However, the committee recommended that the situation be kept under surveillance and that action be taken to keep concentrations of these chemicals in drinking water to a minimum.

The death of fish and other forms of aquatic life from exposure to acutely toxic concentrations of pesticides is obviously undesirable. Occurrences of this type generally are local, readily apparent, and sporadic, with partial or total repopulation quickly occurring. They are generally associated with massive runoff from the land, careless use of pesticides, and accidental discharges of industrial waste or other accidents.

Widespread, long-term contamination of the environment is much more difficult to evaluate and is a matter of great public concern. It is caused primarily by insecticides of the chlorinated hydrocarbon group. Biologically speaking, pollution of water by pesticides in any form is undesirable. What are feared in long-term pollution are ecological effects so complex that it is almost impossible to relate cause and effect. For example, we do not know whether the occurrence of DDT, in milligram or microgram amounts per kilogram, in Antarctic aquatic animals and in fishes from all the seas of the world is harmful or harmless. We cannot at this time explain how DDT became so universally distributed. It has been speculated that DDT entered the oceans through runoff from the land by way of rivers, and from the air, but surely the tremendous dilution that would have occurred through mixing with waters of the sea would prevent direct exposure of most of these fish and other animals to measurable quantities. The term *biological magnification* has been coined to explain the phenomenon that is thought to occur. Insecticides, such as DDT, may be absorbed directly by living organisms and also, it is believed, may adsorb on, or be absorbed into, lower forms of life or inert foods that are subsequently consumed by larger organisms. Any DDT beyond that which can be excreted or metabolized goes into storage and can accumulate, to a degree. It may then be passed on to secondary consumers and passed up the food chain until it reaches higher forms of life (such as the Antarctic penguins). It is feared that somewhere along the line unrecognized damage is being done that may upset the ecological system. The same fears, it is well to note, apply to other of man's waste products.

Several instances have been reported of poisoning of fish-eating birds believed to have occurred as a final step in the biological magnification process. Hunt and Bischoff (*18*) attributed the deaths of grebes, and the decline in the number of their surviving offspring, on Clear Lake in California, to poisoning by DDD concentrated in fish. Investigation showed the following concentrations of DDD in samples from the lake taken 13 months after application of the DDD to the lake for gnat control: in plankton, 10 mg/kg; in fat from plankton-eating fish, 903 mg/kg; in fat from carnivorous fish, 2690 mg/kg; and in fat from fish-eating birds, 2134 mg/kg (*19*). The concentrations in fish-eating birds are said to be 100,000 times the concentrations in lake water after treatment.

Keith (*20*) reported an unusually high mortality of fish-eating birds between 1960 and 1962 at the Tule Lake National Wildlife Refuge in California, which he attributed, on the basis of circumstantial evidence, to ingestion of toxaphene accumulated in fish.

Burdick and his co-workers (*21*) performed a classic investigation in New York State, demonstrating that lethal amounts of DDT can be transmitted from female lake trout to their offspring through the egg. Complete loss of lake-trout fry occurred in the Lake George fish hatchery in 1955 and 1956. Trout eggs from Lake George were distributed to three other hatcheries to prove that hatchery procedures and the quality of the hatchery water were not at fault. The fry failed to survive when Lake George trout eggs were fertilized with milt from males from other lakes. The usual number of fry survived when the situation was reversed. Subsequent investigation of trout from this and other lakes showed that, when oil extracted from the eggs contained DDT equivalent to 2.9 mg or more per kilogram of fry, the fry died when the final contents of the yolk sacs were absorbed. There was no apparent correlation between the DDT content of the female lake trout and the survival of their offspring. Thus was demonstrated a very subtle adverse effect. We may assume that this effect is not generally and completely applicable to other fish species. Otherwise we would certainly be aware of drastic reductions in fish population in many of our rivers and lakes.

Allison and his co-workers (*22*) measured the effects on cutthroat trout of repeated exposure to DDT over a 20-month period. The effects of accumulation through contact were tested by exposure of the fish every 28 days to a 30-minute bath containing DDT. The effects of accumulation through food were tested by weekly feeding with a prepared diet. These workers concluded that there is a threshold for toxic effects around a dosage level of 0.1 mg of DDT per liter per month for accumulation through contact and around 0.3 mg per kilogram of fish for accumulation through weekly feeding of the prepared diet, under the experimental conditions described. At rates of exposure or at dosages higher than these, a marked increase in mortality was found, probably related to nonspecific stresses. Also, mortality among sac fry was higher. Mortality among adults and among sac-fry survivors was responsible for a reduction in reproductive potential. Cope (*23*) concluded that exposure to sublethal amounts of DDT increases fish mortality by reducing resistance to other stresses.

In the foregoing discussion of the significance of pesticides in the aquatic environment, emphasis has been placed on the chlorinated hydrocarbon insecticides because of (i) their persistence, (ii) their greater usage as compared with other classes of pesticide, and (iii) their widespread distribution in the environment. The information available is much too fragmentary to give a definitive picture of the impact of these compounds upon the aquatic environment. It is, however, sufficiently suggestive to alarm the ecologist.

Many studies have been made to cast light on the subject, but success has been limited. This failure is partly a result of the extreme complexity of ecological relationships. In part it is due to the failure of a large number of the investigators to take into account the physical chemistry of the toxicants they are studying and the fate of these chemicals within their experimental setups. The problem was clearly stated by Bowman *et al.* (*24*), who showed (i) that DDT quickly becomes deposited on the inner walls of a test container instead of remaining dissolved and homogeneously distributed throughout the water, and (ii) that appreciable amounts are also quickly lost to the atmosphere. DDT is a highly nonpolar (hydrophobic) substance, as are most of the chlorinated hydrocarbon insecticides, the others to lesser degrees. Investigators in the future will undoubtedly make use of modern analytical instrumentation to better define and to monitor their experimental procedures. They will doubtless make greater use of radioactive isotopes.

Water Quality Criteria

The desirability of establishing water quality standards for some of the pesticides is evident. However, it is currently not possible, or even desirable, to establish such standards for all pesticides. Many are of such a nature, or are used in such a way, that they could rarely become significant water contaminants. The use of others has been restricted by the U.S. Department of Agriculture, under its labeling authority, because these compounds are known to be excessively hazardous to aquatic life, or because their residues, when present in water used for irrigation, may result in crop damage.

There are, however, two groups of pesticides for which water quality criteria may be considered at this time. These are (i) the organophosphorus and carbamate insecticides and related acetylcholinesterase inhibitors, and (ii) a selected group of chlorinated hydrocarbon insecticides. Together they constituted somewhat less than half the total U.S. production of pesticides in 1965 (*1*).

Phosphorus Compounds and Carbamates

The organophosphorus and carbamate compounds include approximately 100 toxicants, mostly insecticides, but also include a few organophosphorus defoliants, nematocides, and miticides. They all have the ability to inhibit activity of the enzyme, acetylcholinesterase, which is involved in the transmission of nervous impulses. The degree of inhibition is related to the degree and duration of exposure and also to the inhibiting potential of the compound in question. Recovery may take place slowly over a period of approximately 3 weeks or less. Any further exposure during the recovery period results in additional inhibition. Thus, the effects of repeated sublethal exposures are cumulative, and measurable.

A criterion for evaluating water quality for fish subject to exposure to organophosphorus compounds and carbamate insecticides may be established, on the basis of the ability of these compounds to inhibit acetylcholinesterase activity in the brains of the fish. Thus, an effect would be measured, rather than a quantity of pesticide in water or fish. This method precludes the necessity for using a different analytical procedure for each of the approximately 100 compounds under consideration. The relatively simple colorimetric assay procedure developed by Hestrin (*25*) and applied to freshwater fish by Weiss (*26*) could be used. This method was slightly modified by Williams and Sova (*12*) and used by them for evaluating acetylcholinesterase activity in the brains of species of marine fish. An alternative and much more sensitive radioisotopic assay method also is available (*27*). The assay procedures can be used at modestly equipped laboratories and require about 2 hours to complete.

The degree of acetylcholinesterase inhibition (measured in fish brain tissue) that produces death in fish varies from about 40 to about 70 percent (*28*). The maintenance of a suitable aquatic environment would logically require that acetylcholinesterase-activity-inhibiting chemicals should not be permitted in a watercourse or other body of water in quantities that will produce an accumulated inhibition of this enzyme's activity approaching the lethal level. In pharmacological studies to determine acceptable levels of various toxic substances, it is common practice to use a 1000-to-1 or a 100-to-1 safety factor to allow for the incidental effects of other undefined conditions. Ten-percent inhibition in a sample of ten fish of the same species and size range is about the minimum level of detectability that is statistically significant (0.05). This value provides at best only a 7-to-1 safety factor, but the safety factor may be enhanced through choosing, for the study sample, the most sensitive species of fish abundantly present.

The classes of pesticides (and other chemicals) involved are not directly cumulative in the form of the parent compounds, or notably persistent, as the chlorinated hydrocarbon insecticides are. The danger of widespread contamination of the aquatic environment from their manufacture and use appears to be small, and adoption of a criterion of the type suggested can be reasonably expected to protect fishes.

The classes of acetylcholinesterase-activity-inhibiting pesticides that produce death in fish are as follows: aliphatic derivatives of phosphorus (example, malathion); aryl (phenyl) derivatives of phosphorus (example, parathion); heterocyclic derivatives of phosphorus (example, diazinon); and carbamate insecticides (example, carbaryl).

Chlorinated Hydrocarbon Pesticides

A second group for which water quality criteria may be considered includes those so-called chlorinated hydrocarbon insecticides that have long persistence and long residual effectiveness as economic poisons. This characteristic is responsible for the loss of control over these compounds after their application, and thus for their dispersal in the environment. Dispersal implies dilution, but in this instance dilution does not always prove to be corrective because the compounds have a remarkable affinity for the tissues of most animals, especially the fatty tissues, and accumulate in these tissues. This is the process defined above as "biological magnification." Damage is often difficult to prove, but is always a possibility. For these reasons steps should be taken to minimize pollution of water by chlorinated hydrocarbon insecticides, it being recognized at the same time that these insecticides play a legitimate and valued role in our society.

How then, with respect to content of the chlorinated hydrocarbon insecticides, can meaningful water quality requirements be established? What quantities can be declared harmless when discharged to a body of water, especially when the effects may be felt hundreds of miles from the place of entry? The establishment of limits implies the ability to detect the presence of the compound and to make meaningful evaluations. In the case of the chlorinated hydrocarbons—for example, DDT—the compounds may adsorb quickly on suspended sediment, on the bottom of the stream or lake, and on aquatic vegetation (*7*). Cope (*23*) reported that 2 weeks after the application, to aquaria, of C^{14}-labeled DDT at concentration of 20 μg/liter, the water contained 0.42 μg/liter, soil contained 6 μg/kg, and vegetation contained 15,600 μg/kg. Two weeks after fish were put into the aquaria they contained 1000 μg of the DDT per kilogram. Thus, chlorinated hydrocarbon insecticides that are actually or potentially active biologically and able to enter the food cycle of aquatic animals may be present in aquatic ecosystems without necessarily being detected by water analysis. Moreover, if all increment could be stopped instantly, the aquatic environment and its living components would not quickly be cleansed of these compounds. As Dustman and Stickel have pointed out (*29*), pesticide residues cannot disappear from the ecosystem by any means short of complete degradation.

Table 1. Suggested minimum detectable limits for selected chlorinated hydrocarbon insecticides, based on determination by electron-capture gas chromatography, for a sample volume of ~3.8 liters.

Insecticide	Amount (μg/liter) in effluent with associated suspended matter*
Chlorinated aryl hydrocarbons	
BHC	0.005
Lindane	.005
Heptachlor and/or heptachlor epoxide	.010
Aldrin	.010
Dieldrin	.010
Endrin	.020
Chlordane	.040
Toxaphene	.080
Strobane†	.080
Diphenyl aliphatics	
DDT	.020
DDD (TDE)	.020

* The values are minimum detectable limits under average conditions. In many instances lesser quantities can be detected and measured. † Registered trade name.

It is recognized that full control of pollution of water by chlorinated hydrocarbon insecticides is not now attainable. Runoff will be a continuing source for some years to come. Contributions from nonlocalized sources can be reduced through substitution, where feasible, of more readily biodegradable insecticides for those now in use. Withdrawal of some agricultural-usage recommendations because of undesirable side effects of the compounds recommended has indirectly provided some assistance toward this end. More assistance is anticipated from advances in agricultural technology tending to make application of very small quantities of pesticides more effective, and from changing concepts of pest control.

Point-source control, however, is possible now. Point sources can be most effectively monitored through analysis of waste effluents rather than analysis of stream water, mud, or any living component of the ecosystem. Point sources include manufacturers of basic pesticides, formulators, cooperage firms that reclaim used pesticide-containing drums, and firms that use the compounds in manufacturing—for example, manufacturers of wool products.

I suggest that quantities of insecticides discharged from point sources be kept at an established minimum—for example, at values less than the minimum detectable limit for a recognized analytical procedure (liquid-liquid extraction, column cleanup, and identification by electron-capture gas chromatography). Table 1 gives a list of selected chlorinated hydrocarbon insecticides with suggested minimum detectable limits for each. A standard sample would be required (1 gallon, or approximately 3.8 liters), and a standard sampling point (the point of entry of the effluent into the receiving body of water). The availability of supporting analytical procedures—such as infrared, mass, and nuclear-magnetic-resonance spectroscopy—for confirmation of questionable identifications should settle any technical argument about the quality of a waste effluent or the reliability of the analytical results reported. Prevention of point-source pollution, although not an answer to all problems of pollution by chlorinated hydrocarbon insecticides, would be an effective step in the right direction.

Summary

The suggested methods for evaluating water quality with respect to its content of selected groups of pesticides are based on interpretation of generally available information. They are intended to provide a base for discussion leading to the development of public policy for the inclusion of such values in water quality standards.

In suggesting these parameters, an attempt has been made to simplify, to apply common denominators. Therefore, an effect—10-percent depression of acetylcholinesterase concentration in fish brain—is suggested as an upper limit for a large group of organophosphorus and carbamate compounds. As a means of measuring the suitability of the water environment, this concept is superior to that of establishing values for water content of each of the approximately 100 compounds involved.

The selection of means for measuring the effect of chlorinated hydrocarbon insecticides is more difficult and presents problems, as follows.

1) No single group effect exists on which to base a measurement.

2) Runoff from land is of greater significance with these insecticides than with acetylcholinesterase inhibitors because of their persistence. The transport mechanism in runoff is poorly understood.

3) The concentrations that will have no effect on most aquatic animal life under conditions of long-term exposure are not known, and the place where effects will be felt cannot always be predicted.

4) Monitoring to detect the presence of chlorinated hydrocarbon insecticides in water is not sufficient for purposes of water-pollution control, because of the ability of these insecticides to con-

centrate in various components of the aquatic ecosystem (in mud, on plants, and in aquatic animal life).

5) Monitoring a combination of streamwater, mud, and aquatic life is inadequate because of the persistence of these compounds, which makes it difficult to relate the presence of the insecticides to the time and place of their introduction.

A suggested partial solution of these problems would be the establishment of minimum permissible concentrations in point-source discharges containing such materials. This would be an attainable objective that would result in reduction of the concentrations of such persistent materials.

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East Africa: Science for Development

The impact of science in a developing region reveals long-term personnel and research requirements.

Thomas R. Odhiambo

There is agreement among political leaders, administrators, and scientists that accelerated science development in Africa can best be effected by the systematic application of the great corpus of scientific knowledge to problems in the social and economic fields. Numerous international conferences have been convened since the beginning of the decade to discuss science as a modernizing tool in the newer countries. Three of these stand out: the International Conference on Science in the Advancement of New States, held in August 1960 at the Weizmann Institute of Science, Rehovoth, Israel; the United Nations Conference on the Application of Science and Technology for the Benefit of the Less Developed Areas, held in February 1963 in Geneva; and the International Conference on the Organization of Research and Training in Africa in Relation to the Study, Conservation and Utilization of Natural Resources held in July 1964 in Lagos, Nigeria. In general, one may conclude from the resolutions approved at these conferences that great emphasis is laid on the application of existing scientific knowledge to the solution of pressing economic and social problems of the developing countries. Indeed, science is thought of very much in terms of a *tool.*

This concept of science as a modernizing tool immediately raises a number of important issues. What is the cultural framework of these societies? Have they an educational program to match the heavy high-level manpower requirements that a modern industrial country demands? What type of science policy and science administration should these countries adopt? Is it valid to believe that only the technological results of science are important to developing countries? What should be our priorities in research? In considering these questions, I cannot claim to speak for the whole of Africa, let alone all "the newer developing countries." The only developing region that I know passably well is East Africa. My attitude and remarks will therefore be heavily colored by my experience in East Africa.

Administration of Science

The administrative structure for science in East Africa and in the newly independent African countries is largely a legacy of the colonial interlude. No important structural changes have as yet been implemented to give it a new look

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The author is senior lecturer in zoology at the University College of Nairobi, Nairobi, Kenya, and honorary general secretary of the East African Academy.