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Historical Perspectives

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All organisms are greatly influenced by alterations in their environments. Change threatens some species, while survival of others is enhanced. In pursuing their own interests, humans have considerably altered the earth's environment and have decreased the probability of survival for many other species. There is a question whether humans as environmental manipulators have increased or decreased their own chance of survival. It might prove to be that humans would have persisted longer as a species if, as all other organisms on earth, they had pursued the course of adaptation rather than manipulation.

As agriculturalists, humans changed the physiognomy of continents, especially in the Northern Hemisphere. Centuries of intensive land use and habitat alteration certainly have had a greater influence on other organisms than anything else humans have done. This process of change continues today at an accelerating rate, particularly in the developing countries of the Southern Hemisphere. The Industrial Revolution ultimately led to a different kind of influence on the earth's environment. During only the last 40 years, increasing technological pollution has seriously degraded the quality of natural environments. Entire continents have been affected, and pollutants now contaminate the oceans, the atmosphere, and thus the entire biosphere.

The development and intensive use of synthetic chemicals, heavy metals, oil, and petrochemical products are responsible for today's most serious pollution problems. These activities also created most of the increased wealth in the world today. Therefore, current practices will con-

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tinue and pollution problems will intensify. Regulatory and technological controls offer the best hopes for limiting the impact of pollution on the environment.

Broad concern about the environmental effects of pollution began only recently. In the United States, it came with the realization that chemicals used to control rodents and predators on western rangelands could also poison other animals (Pierce and Clegg 1915, Lindsdale 1931, Ward 1931). With the increased use of inorganic and botanical pesticides, studies were begun to determine mortality of wildlife in treated areas. Evaluations were made of the use of arsenicals for control of grasshoppers (Pollack 1929, Whitehead 1934), gypsy moths (Frost 1938), and aquatic weeds (Surber and Meehan 1931). Similar studies were made of pyrethrums (Leonard 1942), mercury, cryolite, ryania, and copper sulfate (Rudd and Genelly 1956) used in agriculture. Concern for wildlife increased with the development of methods for the aerial application of pesticides to agricultural lands and forests (Tragardh 1935). Until 1945, rodenticides and arsenicals were the principal pesticides used in the United States. Conflicts between pest control and wildlife were identified and discussed (Strong 1938, Zimmerman 1938). Few intensive studies were conducted, but wildlife mortality was documented (Rudd and Genelly 1956). The abundance of some species decreased after pesticide treatments, and indirect effects through reduction in food supplies were suspected. However, considering what was to come, these programs did not cause many serious problems.

World War II brought on intensive research into the use of chemicals for both positive and negative purposes. The insecticidal properties of DDT were discovered, and during and after the war, DDT was used to control disease vectors threatening military personnel and civilian populations. Research on DDT also identified the potential insecticidal and herbicidal properties of other related chlorinated hydrocarbons. Military research on nerve gases during the war led to the development of the organophosphates for insect control. Similarly, research on rodent control to protect troops and cities from disease produced the new rodenticide, Compound 1080 (Aceto Chemical Co., Flushing, NY), which later was adapted for use in controlling predators.

At the end of World War II, a variety of new, synthetic chemicals was available for control of pests that had plagued humans since crops were first planted. In 1945, private firms in the industrialized countries began to manufacture and market these new chemicals. Agricultural acreage had greatly increased in the United States during the war; and elsewhere in the world, normal agricultural production had to be quickly reestablished. This coincidence of need and availability of pesticides created a large and instant demand for products and an explosive increase in their use

throughout the world. Basic toxicity and efficacy data were rapidly developed, but widespread use was begun without knowledge of the ultimate fate and effects of the chemicals in the environment.

THE EARLY YEARS

Fortunately, biologists were alert to the potential hazards of the new synthetic pesticides. In 1946, the U. S. Fish and Wildlife Service conducted field studies on pesticides in seven states and ran numerous laboratory trials (Nelson and Surber 1947). Most research was on DDT because more of it was being used than all of the other insecticides combined. Many publications appeared in 1946, including those on the effects of DDT for control of mosquitoes (Couch 1946) and gypsy moths (Hotchkiss and Pough 1946), DDT effects on birds (Kozlik 1946; Stewart, Cope, Robbins, et al. 1946) and nestlings (Mitchell 1946), DDT effects on mammals (McDermid 1946, Stickel 1946), and general discussions of the biological effects of DDT (Coburn and Treichler 1946, Cottam and Higgins 1946, Storer 1946).

Some interesting characteristics of DDT were discovered during initial studies. The accumulation of DDT in adipose tissue of animals was demonstrated (Woodard, Ofner, and Montgomery 1945; Kunze, Nelson, Fitzhugh, et al. 1949). Milk from mammals exposed to DDT was found to contain residues (Woodard, Ofner, and Montgomery 1945), and chickens fed DDT were shown to lay contaminated eggs (Rubin, Bird, Green, et al. 1947). Jones (1952) defined the stability of DDT; Finnegan, Haag, and Larson (1949) described the distribution and elimination of residues from animal tissues, and the estrogenic activity of DDT was discovered by Fisher, Keasling, and Schueler (1952).

By 1950, 15 insecticides and fungicides were in common use (Bunyan and Stanley 1983). As applications of other chlorinated hydrocarbon insecticides increased, wildlife studies were broadened to give them scrutiny along with DDT (Baumgartner 1948, Post 1949, Barnett 1950). DDT, aldrin, dieldrin, chlordane, and toxaphene sometimes caused wildlife mortality when used in grasshopper programs (Harris 1951, Eng 1952), in orchards (Odum and Norris 1949, Kelsall 1950), in cotton (Young, Hulse, and Moe 1952; Eyer, Faulkner, and McCarty 1953), and in rice (Rudd and Genelly 1955). The acute toxicity and gross effects of these chemicals on reproduction were assessed in laboratory studies (Dahlen and Haugen 1954, DeWitt 1955, Genelly and Rudd 1956).

The first widespread use of organophosphate insecticides began in the 1950s. Field applications were evaluated (Barnett 1950; Mohr, Telford, Peterson, et al. 1951), and the first of many poisonings of geese with

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organophosphates was reported (Livingston 1952). Organophosphate persistence was evaluated (Fahey, Hamilton, and Rings 1952), and the technique of cholinesterase measurement was used to monitor exposure (Stearns, Griffiths, Bradley, et al. 1951). Organophosphates, in general, were more toxic than the chlorinated hydrocarbon insecticides, and concern over human hazard restricted their acceptance. Their use increased with knowledge of how to monitor human exposure and how to safely apply the chemicals by air. In these early programs, the higher toxicity of the organophosphates did not result in more frequent wildlife deaths, as some had feared.

By 1955, several hundred papers had been published on the effects of pesticides on wildlife. Acute toxicity data existed for many chemicals against laboratory animals, fishes, and a few species of wild birds. In general, birds and fishes appeared more susceptible to pesticide poisoning than mammals, reptiles, and amphibians (see discussion by Walker 1983). Insecticides were shown to be more toxic to wildlife than other pesticides, although the toxicity of insecticides differed considerably among compounds and wildlife species. Field experience showed that the acute toxicity of pesticides did not necessarily depict their hazards to wildlife when used in the field. Some highly toxic pesticides did not persist long enough to be hazardous, while less toxic materials persisted and had serious sublethal effects. Field studies had identified the insecticides and rates of applications that were most hazardous to wild animals, and the potential for reproductive effects was documented in both field and laboratory investigations.

Robert L. Rudd and Richard E. Genelly of the University of California compiled the available knowledge on pesticides in their encyclopedic reference book, *Pesticides: Their Use and Toxicity in Relation to Wildlife* (Rudd and Genelly 1956). This book was the first classic publication in the wildlife pesticide field, and it continues to be a valuable reference today.

MAJOR PROGRAMS

From the beginning, biologists were especially concerned about pesticide programs that each year treated extensive areas of important wildlife habitat. Improved techniques for aerial application of pesticides enabled treatment of thousands of hectares of forests and rangelands. Equally disturbing was the widespread use of toxic seed dressings and the multiple treatments (up to 20) being applied each year to some wetlands for mosquito control.

Insect Control in Forests

High rates of DDT (2–7 kg/ha) were applied to forests for control of spruce budworm (Hope 1949, Cope and Springer 1958), Dutch elm disease (Benton 1951), and Japanese beetles (Fleming and Hawley 1950). These programs caused bird mortality, reduced populations, and sometimes affected reproduction (Adams, Hanavan, Hosley, et al. 1949; Langford 1949). Use of DDT in control of Dutch elm disease consistently caused high mortality of birds (especially robins), and some populations were severely decimated in local areas (Hickey and Hunt 1960; Wallace, Etter, and Osborne 1964; Wurster, Wurster, and Strickland 1965).

During the 1950s, concern over the mortality of fishes and forest wildlife resulted in a reduction in application rates of DDT. Satisfactory insect control was achieved with much less DDT, and the incidence of bird and fish mortality decreased (Hoffman, Janson, and Hartkorn 1958). Continued use of DDT in forests undoubtedly contributed to global contamination with DDT residues, but that problem had not yet been identified in the 1950s.

Biologists in the United States (Pillmore and Finley 1963) and Canada (Fowle 1966, Pearce 1971) worked with forest entomologists (Benedict and Baker 1963) for over two decades to make forest applications safer for wildlife. Gradually, the less toxic chemicals malathion, carbaryl, and the synthetic pyrethrins replaced DDT in many programs, but use of some organophosphates such as phosphamidon still caused bird mortality (Finley 1965, Fowle 1972). Thorough evaluation of the most recent, and one hopes the last, use of DDT in forests again clearly illustrated its lethal and sublethal effects on wildlife (Herman and Bulger 1979; Henny, Maser, Whitaker, Jr., et al. 1982).

Fire Ant Eradication

A program to eradicate the imported fire ant in the southeastern United States was begun in the 1950s and is still underway. Applications of insecticides in baits were made to extensive areas of woodlands, pastures, and farms during most years. Heptachlor and dieldrin were used during the first decade of this program, and biologists documented high bird mortality and population decreases after treatments (Clawson and Baker 1959, Rosene 1965). Residues of heptachlor epoxide were found in many species of birds (Stickel, Hayne, and Stickel 1965), including woodcock, whose eggs on Canadian breeding grounds also contained residues (Wright 1965).

Mirex (Allied Chemical Corp., Hopewell, VA) was later substituted in the fire ant program, but it also was highly persistent, accumulated

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in wildlife (Oberheu 1972), and was toxic to terrestrial and aquatic life (Galbreath 1965, Baker 1967). Kepone (Allied Chemical Corp., Hopewell, VA) bait has since been used, and new, less hazardous materials are currently being tested for this program.

The history of the program to eradicate the fire ant illustrates the hazards to animals of large-scale, blanket treatments of farmlands, woodlots, and wild areas that in the aggregate constitute important habitats for wildlife. Many species are exposed, and this increases the probability that some will be vulnerable to either mortality or sublethal effects. Using bait formulations is especially hazardous, as they are often highly palatable to birds that feed on the ground.

Seed Dressings

In the large programs involving the use of insecticides as seed dressings, mortality was the primary effect on wildlife. During the 1950s and early 1960s, seeds were commonly treated with aldrin, dieldrin, heptachlor, and mercurial fungicides in the United States and western Europe. Extensive areas were planted with treated seeds. The seeds of many agricultural crops were treated as were most seeds planted in forests and rangelands. Such practices caused extensive primary and secondary poisoning of birds, the evidence of which was annually compiled and published in the United Kingdom (Bunyan and Stanley 1983, Walker 1983).

The hazard of seed dressings is rather straightforward. Poisons are put on seeds; wild animals that eat the seeds are poisoned and they die. Scavengers that feed on the dead animals can also be killed. Still, it took 25 years to ban most hazardous seed dressings, and that experience is indicative of the resistance that was usually encountered in changing use practices once they became established. Aldrin-treated rice seeds were still killing birds in Texas in the early 1970s (Flickinger and King 1972) and heptachlor on seeds killed geese in Oregon in the late 1970s (Blus, Henny, Lenhart, et al. 1979).

Mosquito Control

Mosquitoes are a nuisance to humans, and adults and larvae are controlled over large areas of the United States. Wetlands are commonly treated, which subjects a variety of wildlife species to exposure. Heavy applications of DDT were made to marshlands and estuaries for many years, and wildlife effects were commonly documented (Tarzwell 1947, Knedel 1951, Springer and Webster 1951, Hanson 1952). As multiple treatments often

were made each year, this use of DDT greatly contributed to the contamination of aquatic food chains that would later be identified. Within a decade, such intensive use of DDT resulted in the development of resistant mosquito populations. Other chlorinated hydrocarbons were effective for only short periods before resistance developed. Organophosphate insecticides then became the only effective means of controlling mosquitoes. Tests with wildlife (Keith and Mulla 1966, Hill 1971) indicated the hazards of organophosphate insecticides like fenthion, but the low toxicity of fenthion to mammals made it safer for humans to use than other insecticides such as parathion. Fenthion came into wide use and repeatedly has caused bird losses throughout the United States (Seabloom, Pearson, Oring, et al. 1973; DeWeese, McEwen, Settini, et al. 1983).

In California, mosquito populations ultimately developed a resistance to all insecticides. This set off a rapid search for alternate means of mosquito control. As has happened whenever use of chemicals was restricted, entomologists quickly found promising alternatives. Synthetic juvenile hormones that interrupt larval development are now on the market. Also BTI (*Bacillus thuringiensis var. israelensis*) and certain chemical amines were found effective in controlling mosquitoes and are being developed for widescale use.

Grasshopper Control

Grasshoppers have been controlled on western rangelands in the United States nearly every year since 1934; areas treated annually have averaged about 800,000 ha (McEwen 1982). Many chlorinated hydrocarbon insecticides are effective against grasshoppers, and most have been applied to control grasshoppers and other rangeland insect pests. Such uses consistently killed birds, and sometimes mammals, fishes, reptiles, and amphibians. Applications of chlorinated hydrocarbons were discontinued after 1965 in the major control programs, but their use has continued in some states. Wildlife hazards and relationships were thoroughly documented, and wildlife ecologists worked with entomologists to demonstrate the greater safety of malathion and carbaryl to wildlife (McEwen 1982).

The newest approach for grasshopper control is most encouraging because of its safety to wildlife. Combinations of carbaryl bait and a bait containing spores of protozoans are aerially applied. The instant action of the insecticide coupled with the residual effect of the parasite give good grasshopper control without poisoning wildlife.

The reduction of grasshoppers itself adversely affects wildlife, as grasshoppers are a principal food of rangeland birds, especially during the birds'

breeding season. Control of grasshoppers essentially diverts energy produced on rangelands from food chains of small insectivores into the food chains of herbivores, especially livestock. Such indirect effects of grasshopper control may have greater influences on wildlife populations than the mortality caused by insecticide use.

Herbicide Programs

More herbicides have consistently been used each year than any other type of pesticide. Herbicides account for two-thirds of the pesticides used in the United States (Maddy 1983). Most of this use is to control weeds in crops, but considerable amounts are applied to forests, rangelands, and other important wildlife habitats. Laboratory trials have shown that most herbicides are not highly toxic to birds and mammals, and field investigations have seldom shown lethal effects (Rudd and Genelly 1956).

The greatest concern about the use of herbicides has been their possible indirect effects through altering habitats. Indeed, herbicides are widely used by wildlife biologists to manage vegetation for the benefits of wildlife. Still, herbicides applied to eliminate brush (Goodrum and Reid 1956), to maintain right-of-ways (Bramble and Byrns 1958), and to control marsh vegetation (Hanson 1952) have adversely influenced wildlife populations.

Indirect effects of herbicides on wildlife are difficult to assess. Usually, treated habitats already have been disturbed and the availability of food and other life requirements of animals are seldom clearly understood either before or after herbicide treatments. In one study where food supplies, food habits, and population changes could be measured, the herbicide 2,4-D reduced food abundance, which in turn altered food habits and severely reduced numbers of pocket gophers (Keith, Hansen, and Ward 1959).

The influence on wildlife of dioxins, the highly toxic contaminants in certain herbicides, was never thoroughly evaluated. They had great potential to adversely influence animals, but working with dioxins was extremely hazardous. Now herbicides containing dioxins have been banned.

Evaluations of the major programs concentrated on abundant species that occurred in treated areas. Biologists reasoned that if pesticides caused mortality or reproductive problems, those effects should be most obvious in animals directly exposed to applications. Studies often documented the death of wildlife and enabled modification of programs so as to reduce or prevent further mortalities. Important effects on reproduction were seldom observed. This work accomplished a lot, but biologists were slowly learning that serious problems had developed elsewhere.

RESIDUE CONTAMINATION AND BIOACCUMULATION

In the late 1950s, biologists investigated a mortality in western grebes at Clear Lake, California. Birds contained high levels of DDD in their adipose tissue and apparently had died from DDD poisoning. DDD had been applied to the lake to control midge larvae as the adult midges were a nuisance to humans that resided on the lake shore. But how had the birds been exposed? The ensuing research documented for the first time the phenomenon of bioaccumulation of insecticide residues in animal food chains. This pioneer work by Hunt and Bischoff (1960) initiated a line of research that ultimately led to the banning of most chlorinated hydrocarbon insecticides and thereby to saving a number of species endangered by those chemicals.

Subsequently, investigations of fish-eating bird mortality in marshlands in England (Prentt 1966) and the United States (J. O. Keith 1966) showed that birds had accumulated sufficient chlorinated hydrocarbon residues to kill them. These studies documented that insecticides used in agriculture were being transported in waste irrigation water into untreated natural habitats, where they accumulated in aquatic food chains and ultimately poisoned predaceous species.

Dispersal of Residues

Hooper and Hester (1955) measured pollution of streams by runoff water from agricultural fields, but consideration was given only to the immediate lethal effects on aquatic organisms. In the United States, nationwide surveys were conducted to determine the levels of pesticide contamination in streams receiving waste agricultural water. Results consistently showed only trace amounts of residues at most sampling stations. However, these findings were largely erroneous. Sampling was done with carbon filters that were easily clogged with detritus and sediments. Sand prefilters were used to divert material that was suspended in water. Measurements, therefore, were of pesticides in water solution; chlorinated hydrocarbons are relatively insoluble in water.

Later work showed that most pesticides transported in water are adhered to suspended organic debris (J. O. Keith 1966). Organic matter in water is fed upon by small invertebrates, and this initiates the contamination of aquatic food chains. This organic debris often contains higher residues (20–80 ppm) than those found in invertebrates, fishes, and the fat of birds; but it is present in only very small amounts. Bioaccumulation, therefore, must be viewed in terms of the quantity of pesticide accumu-

lated as well as the proportional amount in parts per million. In the final analysis, it is the number of molecules or weight of poison that becomes important.

Soil is the matrix that remains to become contaminated from repeated treatments of crops with pesticides. Residue levels in soil and their persistence after treatments were measured in many studies, such as one by Chisholm and Koblitsky (1959). Most studies implied that loss of contamination over time illustrated the rate of pesticide degradation in soil. That suggestion was often incorrect. Residues usually disappeared from soil by moving into the atmosphere and by being carried off in irrigation water.

Measurements of aerial applications consistently have shown that deposits on treated areas seldom exceed 50 percent, and often are as low as 10 percent, of the amount applied. Herbicides applied to rice fields in California were finally identified as the cause of death to fruit trees in orchards 30 km downwind. Probably the most spectacular finding was that residues of pesticides used in Africa were being transported on dust particles across the Atlantic Ocean to the Western Hemisphere in the southeasterly tradewinds (Risebrough, Huggett, Griffin, et al. 1968). Airborne fallout on Iceland also contained DDT and PCB residues (Bengtson and Sodergren 1974).

Residue Surveys

Evidence was gradually accumulated that residues of chlorinated hydrocarbons were dispersing widely and often persisting much longer than expected. Interestingly, this information primarily came from wildlife biologists and not from agricultural scientists. Surveys of residues in birds, mammals, fishes, reptiles, and amphibians were conducted throughout the United States and in more than 30 other countries. Results provided convincing evidence that insecticides in air and water were contaminating untreated wildlife habitats. The homerange of many species containing residues simply did not include areas where direct applications had been made. It was evident that not all animals received equal exposure. As a group, birds carried greater amounts of residues than mammals. Flesh-eating birds had higher levels than herbivores. Residues of DDT and its metabolites were consistently found in more samples and at higher levels than any other chlorinated hydrocarbons (Keith and Gruchy 1972). Residue levels of the more toxic compounds (e.g., endrin, dieldrin) were lower than those of slightly less toxic ones (e.g., chlordane, heptachlor). Apparently, the more toxic ones killed before high levels could be accumulated, whereas the less toxic ones accumulated and later sometimes caused

mortality when animals were stressed (Keith 1968). Mortality was the only real effect noted in most species during these early surveys, but levels in living birds were often high enough to cause concern about sublethal effects.

Migratory birds were thought to be exposed on wintering grounds and subjected to the hazards of accumulated residues while breeding after migration. This potential existed for snow geese (Flickinger 1979), white-faced ibis (Capen 1977), white pelicans (J. O. Keith 1966, Knopf and Street 1974), peregrine falcons (Cade, White, and Haugh 1968), woodcock (Wright 1965), and many other species. Measurement of exposure during migration proved difficult, but winter exposure in species showing breeding debility was finally shown for brown pelicans (Anderson, Jehl, Risebrough, et al. 1975) and peregrine falcons (Henny, Prescott, Riddle, et al. 1982). Risebrough, Menzel, Martin, Jr., et al. (1967) reported some of the first residues from marine fishes. Their findings that fishes in the oceans were being exposed to insecticides stimulated a number of additional surveys. Results showed that even fishes in the middle of oceans carried insecticide residues (Risebrough 1969). Subsequently, contaminations were found in marine mammals in Sweden (Jensen, Johnels, Olsson, et al. 1969), in Arctic ringed seals (Addison and Smith 1974), in British grey seals (Heppleston 1973), and in many other sea mammals. DDT and DDE were found in Antarctic mammals and birds (Tatton and Ruzicka 1967, Brewerton 1969), in birds of Greenland (Braestrup, Clausen, and Berg 1974), animals in Iceland (Bengtson and Sodergren 1974), and in eggs of green sea turtles from Ascension Island (Thompson, Rankin, and Johnston 1974).

Chlorinated hydrocarbons were being used throughout the world, but only a small proportion of each continent had received treatments. Even in California, which accounts for 10 percent of the world's annual pesticide use (Maddy 1983), only an average of 15 percent of the land is treated each year. The United Kingdom is an example of the exceptions: 85 percent of the land is in agriculture, and most is treated with pesticides. Dispersal of these persistent chemicals from areas of use had created a global contamination, the total effects of which were not yet clearly understood.

Residues in Ecosystems

Several ingenious studies helped to describe the fate and involvement of insecticides in ecosystems. Radioactive-labeled DDT was applied to a marsh, and levels in different substrates were analyzed over time (Meeks 1968). Hickey, Keith and Coon (1966) and J. A. Keith (1966) successfully documented the source and food chain contamination of DDT that influ-

enced herring gulls in Lake Michigan. The fate of malathion in a forest was studied with labeled insecticide (Giles 1970). Korschgen (1970) followed aldrin from the soil of treated fields into wildlife food chains, and Woodwell, Wurster, and Isaacson (1967) documented biological concentration of DDT in an estuarine environment.

Most studies of insecticide dynamics dealt with relationships within treated areas, and attention was primarily given to agricultural basins and other areas of intense and annual pesticide use. Animals were continuously exposed to residues, but it was seldom clear whether that exposure came primarily from current applications or from those made in the past. Where single applications were made to study areas, chemicals sometimes persisted and contaminated food chains for many years (Herman, Garrett, and Rudd 1969; Dimond, Belyea, Kadunce, et al. 1970; Forsyth, Peterle, and Bandy 1983), whereas in other instances residues appeared to quickly degrade and only briefly contaminate food chains (Keith 1968, Meeks 1968). This contrast was most evident in a study of two lakes treated with toxaphene to control undesirable fishes (Terriere, Kiigemagi, Gerlach, et al. 1966). Trout were successfully stocked in one lake within months following treatment, but in the other lake toxaphene killed introduced trout each year for 8 years. The process and rate of toxaphene degradation differed considerably between the two lakes.

Woodwell, Wurster, and Isaacson (1967) used the concept of half-life to describe the persistence of DDT, and others have continued to use this term. The concept is misleading and should not be applied to pesticides. Pesticides, unlike radioactive materials, do not have a predictable half-life. Pesticide degradation rates are governed by the kinds and intensity of factors that metabolize pesticides in the environments where residues occur. For instance, Brown and Brown (1970) convincingly showed that DDT persistence is exceptionally lengthened in the subarctic compared with its persistence in more temperate climates. It is known that the persistence and involvement of a pesticide can vary among environments, but not enough is known to enable a prediction of its fate and influence at a particular site. It becomes difficult, therefore, to generalize the rate of residue degradation, which is the basis for a half-life value.

A rather unusual method was used by biologists in an attempt to evaluate the cumulative effects of environmental contamination on bird populations. Life tables were developed for periods before and after the advent of modern pesticides. Comparisons were then made of the dynamics of populations with and without the influence of pesticides. Population problems were indicated in some species that had been shown in other studies to receive high exposure (Henny 1972, Franks 1973).

Residue Effects in Animals

Laboratory scientists became involved in diverse kinds of studies to evaluate the hazards of DDT and other persistent chemicals. Considerable effort was made to document the lethal toxicity of pesticides to wild animals. Handbooks were published of acute toxicity to fishes and invertebrates (Johnson and Finley 1980) and to birds and mammals (Tucker and Crabtree 1970; Hudson, Tucker, and Haegele 1984). Lethal levels of pesticides in the diet of birds were reported (Heath, Spann, Hill, et al. 1972; Hill, Heath, Spann, et al. 1975). Lethal toxicity data indicated the physiological susceptibility of animals to different poisons. Such information provided a valuable perspective on the potential for lethal effects on animals. Once pesticides are applied, however, this potential is modified by the ecological vulnerability of species to exposure. Species killed may not be the most susceptible ones on treated areas. Animals whose foods are contaminated become vulnerable and are most likely to be poisoned. Mortality reduces the abundance of animals; but it was ultimately found that chronic effects, which decrease survival and reproductive success, had a greater impact on populations than acute mortality.

Rates of uptake and loss of pesticides received considerable study in poultry (see review by Stickel 1973). Residues in poultry were a serious problem, as contaminated birds and eggs could not be marketed. Some of these studies misled biologists, as domestic hens often proved to be less susceptible than wild birds to the effects of pesticides.

Studies defined the residue levels in tissues that were indicative of pesticide poisoning. Brain residues best correlated with mortality (Bernard 1963; Stickel, Stickel, and Christensen 1966; Stickel and Stickel 1969). Residues in adipose tissue of birds were shown to be mobilized with fats during stress (Sodergren and Ulfstrand 1972), and this often caused mortality (Van Velzen, Stiles, and Stickel 1972). The various types of stress found to cause death in contaminated birds included starvation and weight loss (Bernard 1963), molting (Stickel, Stickel, and Spann 1969; Stickel and Rhodes 1970), and disturbance of captive birds (Stickel 1965).

The effects of many pesticides on reproductive success in penned birds were evaluated (Dahlen and Haugen 1954; DeWitt 1955; Genelly and Rudd 1956; Azevedo, Hunt, and Woods 1965; Jefferies 1967; Neill, Muller, and Schutze 1971; Haegele and Hudson 1973). These studies often showed that pesticide exposure resulted in decreased egg production and reduced hatchability. Other studies suggested that chlorinated hydrocarbons delayed ovulation (Jefferies 1967), increased estradiol metabolism (Peakall 1970b), stimulated production of liver microsomal enzymes that led to hydroxylation of steroids (Gillett, Chan, and Terriere 1966; Peakall 1967),

and altered thyroid secretion (Jeffries and French 1972) and adrenal structure (Lehman, Peterle, and Mills 1974). Wurster (1969), Stickel (1973), and Risebrough (1986) reviewed the relationships between pesticides and avian reproduction.

Wild animals contaminated with chlorinated hydrocarbon residues must often be faced with the additional stresses of disease and food shortage. In such situations, the combined effects could be additive and create a severe challenge to animals. Studies evaluating pesticide-disease interactions were made by Friend and Trainer (1974a and b) and Thompson and Emerman (1974). Keith (1978) tested the combined effects of DDE and food restriction on reproductive success. These studies showed that pesticides can seriously aggravate the debilities caused by natural limiting factors.

Concern among biologists was heightened by these accounts of environmental contamination and wildlife effects from pesticides. They reasoned that if pesticides killed some animals, then certainly survivors containing high residue levels could be subject to sublethal effects on reproduction and survival. All of these fears, backed by the evidence that caused this concern, were convincingly presented by Rachel Carson (1962) in her book, *Silent Spring*. This book successfully informed the public of the hazards of pesticides to wildlife and is now regarded as giving rise to the environmental movement that has become so dominant in decision-making processes today.

POPULATION STUDIES

Ultimately, it was the study of declining avian populations that provided definition of the specific debilities caused by exposure to chlorinated hydrocarbon insecticides. Biologists in several countries were investigating populations of birds that had slowly declined to critical levels. The species all carried residues of DDT, its metabolites, other chlorinated hydrocarbon insecticides, polychlorinated biphenyls, and other chemicals. Nesting success was lower than normal in all affected species, but the exact nature of the debility affecting birds was not clear (see discussion in Cade, White, and Haugh 1968). Species of concern included osprey (Ames 1966), bald eagles (Stickel, Chura, Stewart, et al. 1966), Bermuda petrels (Wurster and Wingate 1968), herring gulls (J. A. Keith 1966), and brown pelicans (Schreiber and DeLong 1969).

A British biologist, Derek Ratcliffe, had tenaciously struggled for 10 years to understand the relationships involved in the decline of sparrow hawks and peregrine falcons in the United Kingdom. This inquiry finally led him to considerations of raptor eggshells; measurements proved that shells of eggs laid after World War II were lighter in weight than those of

eggs laid before the war (Ratcliffe 1967). He postulated that the use of chlorinated hydrocarbon insecticides had somehow caused eggshell thinning in raptors.

Ratcliffe's ideas immediately stimulated an assessment by biologists of the incidence and cause of eggshell thinning in birds. Anderson and Hickey (1972) measured over 23,000 eggshells of 25 species. They found shell thinning in 22 of those species and illustrated that thinning was correlated with DDE residues in the eggs. Experimental work with mallards (Heath, Spann, and Kreitzer 1969) and American kestrels (Wiemeyer and Porter 1970) soon showed DDE, a metabolite of DDT, caused eggshell thinning. Since those early studies, it has become clear that DDE is the primary cause of shell thinning in many kinds of birds. Gallinaceous species were not affected. A tremendous amount of effort has gone into study of eggshell thinning, and results have been reviewed (see Cooke 1973, Stickel 1973). Eggshell thinning was definitely a major cause of low reproductive success and population decline in some species, but chlorinated hydrocarbons probably contributed to declines in other ways. Many adults of some contaminated species did not attempt to breed in some years, and desertion of eggs and nestlings was commonly observed. Preliminary studies suggested such behavior could be caused by DDT and polychlorinated biphenyls (Peakall and Peakall 1973, Haegeler and Hudson 1977). Later, intensive work more fully described the aberrant behavior and reduced productivity caused by chlorinated hydrocarbons (Keith 1978) and the hormonal basis for these debilities (McArthur, Fox, Peakall, et al. 1983). Continued investigations of this nature are needed to assess the potential for other persistent chemicals to adversely affect the behavior and breeding performance of birds.

Populations of several species of wild birds were severely affected by DDE, but other chlorinated hydrocarbons contributed to the decline of some species. Exposure often was from specific, identifiable sources. Osprey populations probably were influenced by DDT used for mosquito control. Bald eagle abundance decreased over much of North America due to contamination of agricultural waters with DDE and dieldrin; DDE caused eggshell thinning, while dieldrin caused direct mortality. Regional populations of brown pelicans probably were reduced by endrin poisoning (King and Flickinger 1977) and by DDE-caused eggshell thinning (Keith, Woods, and Hunt 1970). In both instances, chemicals originated primarily from manufacturing plants. White-faced ibis apparently received (and continue to receive) greatest exposure to DDE on their wintering grounds outside of the United States. Osprey, bald eagles, and pelicans eat fishes, whereas ibis eat earthworms. Their foods were easy to identify and sample for residues. The situation with peregrine falcons was more complex.

Dieldrin mortality reduced populations in the United Kingdom, but after dieldrin use was curtailed, DDE effects prevented population recovery (Bunyan and Stanley 1983). Peregrines feed on a variety of prey species, and the specific sources of contaminations in their prey have seldom been determined, either in Europe or the United States.

Field investigation of problems in avian populations will continue to offer one means of monitoring effects of chemical contaminations in the environment. It is not the best way, as problems often must be serious before they can be recognized. It is hoped that better predictive models can be perfected in the future to help identify problems before they become detrimental to wildlife populations.

AFTER THE BANS

Restrictions on the use of DDT and other organochlorine insecticides in the United States that were begun in the early 1970s resulted in a decreased exposure of some birds to DDE (Johnston 1974), to endrin (Blus, Henny, and Grove 1989), and to other harmful residues. Population productivity improved immediately in brown pelicans (Anderson, Jehl, Risebrough, et al. 1975). Wiemeyer, Bunck, and Krynitsky (1988) found residues in osprey eggs had declined by 1979 in New Jersey. Bald eagle reproductive success in the Chesapeake Bay area was improved by 1986 as DDE residues declined (Ohlendorf and Fleming 1988). Anderson and Gress (1983) followed the recovery of brown pelicans in California between 1970 and 1980. Dramatic increases in the annual number of breeding adults (750 to 2,244), the number of young fledged (1 to 1,515), and in productivity (0.002 to 0.68 young per nest) were documented.

However, peregrine falcons continued to accumulate pesticides in Latin America (Henny, Prescott, Riddle, et al. 1982), and their prey in the western United States were still contaminated in 1980 (DeWeese, McEwen, Hensler, et al. 1986). Five addled peregrine eggs collected in Arizona between 1978 and 1982 contained residues of 15 chlorinated hydrocarbons, and highest residues continued to be those of DDE (Ellis, DeWeese, Grubb, et al. 1989).

Eggshells of osprey in the Chesapeake Bay area remained 10 to 24 percent thinner than normal in 1977 and 1978 (Ohlendorf and Fleming 1988); however, organochlorine residues in eggs were lower than in earlier surveys. Between 1978 and 1980, single eggs collected from 220 nests of black-crowned night herons all contained DDE (Henny, Blus, Krynitsky, et al. 1984). As DDE residues increased in eggs (4 to 25 ppm), shell thinning increased (3 to 17 percent) and fledgling success in the 220 nests decreased (79 to 22 percent). In isolated Mississippi wetlands, even waterbirds resi-

dent in the United States had accumulations of DDE in eggs and showed eggshell thinning in 1984 and 1985 (White, Fleming, and Ensor 1988). High DDE residues also persisted in wildlife from New Mexico and Texas (White and Krynitsky 1986). White-faced ibis in Nevada were gravely influenced by DDE in 1985 and 1986 (Henny and Herron 1989). Forty percent of the nesting population was sufficiently contaminated to reduce breeding productivity by 20 percent.

These studies showed the continued presence of DDE and other insecticide residues in the United States long after use restrictions were imposed. Some contaminations probably are due to prolonged persistence of residues, others to illegal use of the insecticides. Residues in migratory birds could represent exposure in other countries of both predaceous and prey species. The use of chlorinated hydrocarbon insecticides has not been restricted in many countries, and residues will continue to create problems for local birds and also for migratory birds that visit those countries.

Documentation of chlorinated hydrocarbon effects on wildlife populations led to bans on some insecticides and to greater restrictions on use of others in the United States. This, in turn, prompted rapid development of the organophosphates, carbamates, synthetic pyrethroids, bacterials, synthetic juvenile hormones, and other types of insecticides to replace the banned materials.

Although registrations were canceled and use restrictions were imposed on the "dirty dozen" in the United States, considerable use of chlorinated hydrocarbons continued in other parts of the world during the 1980s. Information compiled by Hirano (1989) gives a good indication of world trends. Sales of chlorinated hydrocarbon insecticides decreased by 20 percent (\$500 to \$400 million) between 1983 and 1986, while organophosphate sales (\$1,600 to \$1,900 million) and carbamate sales (\$1,000 to \$1,100 million) increased 18 and 10 percent, respectively. Sales of the synthetic pyrethroids more than doubled (\$600 to \$1,300 million), while sales of other insecticides increased 38 percent (\$400 to \$550 million).

Many predicted that if chlorinated hydrocarbon insecticides were banned, greater wildlife mortality due to insecticides would occur. That is exactly what happened—with greater use of organophosphate and carbamate insecticides, reports of wildlife mortality increased (Seabloom, Pearson, Oring, et al. 1973; Zinkl, Rathert, and Hudson 1978; Stone 1979; Flickinger, Mitchell, White, et al. 1986; Hill and Fleming 1982; White, Mitchell, Kolbe, et al. 1982; White, Mitchell, Wynn, et al. 1982). Of the six organophosphate insecticides that made up 50 percent of total use between 1978 and 1981, five are highly toxic to wildlife. Carbofuran, the carbamate insecticide that received greatest use, also is extremely toxic to birds and mammals (Smith 1987).

Grue, Fleming, Busby, et al. (1983) found over 400 reports of wildlife mortality due to organophosphate poisoning. Of the unintentional poisoning in North America, 74 percent involved either diazinon, fenthion, parathion, or phosphamidon, while seed treatments with carbophenothion and chlorfenvinphos caused most losses elsewhere. Wildlife numbers involved in each incident ranged from a few individuals to estimates of 2.9 million.

The mechanisms of insecticide exposure that cause wildlife mortality have been clearly identified. Exposure can be dermal (Pope and Ward 1972; Guzman and Guardia 1978; Bruggers, Jaeger, Keith, et al. 1989) or through inhalation (Weeks, Lawson, Angerhofer, et al. 1977; Berteau and Chiles 1978), but most often it is caused by ingesting contaminated food. Birds often die after consuming contaminated vegetation (Stevenson 1972; Nettles 1976; Stone 1979; White, Mitchell, Kolbe, et al. 1982; White, Mitchell, Wynn, et al. 1982), contaminated insects (Mills 1973; Stehn, Stone, and Richmond 1976; DeWeese, Henny, Floyd, et al. 1979; White, King, Mitchell, et al. 1979; Bruggers, Jaeger, Keith, et al. 1989), or contaminated vertebrates (Mills 1973; Mendelssohn and Paz 1977; Hall and Kolbe 1980; Balcomb 1983; Bruggers, Jaeger, Keith, et al. 1989). Seed treatments pose hazards to granivores (Hamilton and Stanley 1975, Stromborg 1977, Stanley and Bunyan 1979, Smith 1987).

Wildlife mortality due to chlorinated hydrocarbon insecticides was reduced in the 1950s because research proved that pests could be controlled with considerably lower rates of applications than those first used. In the 1980s, documented evidence of mortalities shows that application rates now recommended for many of the more toxic organophosphate and carbamate insecticides are sufficient to kill wildlife (Smith 1987). It seldom will be possible to reduce rates at which these current insecticides are being applied without limiting efficacy.

In the United States and elsewhere, wildlife mortality has not often been a sufficient reason to restrict use or to cancel registration of a pesticide. Usually, a real threat to populations of rare or endangered species must be shown. This was the case recently in the United Kingdom. Use of carbophenothion as a seed treatment caused unexpected mortalities in geese wintering in Scotland. As a large proportion of the world's population of these geese were killed, carbophenothion use as a seed dressing in Scotland was banned (Hart 1990).

There is no evidence that mortalities due to organophosphate and carbamate insecticides are causing population declines in any wildlife species in the United States. The subject has not received adequate study, as most current research has been conducted under laboratory and pen conditions

provided adequate reason for concern about the threat of organophosphates and carbamates to wild populations of birds and mammals.

Grue, Fleming, Busby, et al. (1983) and Smith (1987) reviewed the sublethal effects of organophosphates and carbamates on wildlife. An abundance of laboratory and controlled studies have shown effects in birds ranging from reduced food consumption (Herbert, Peterle, and Grubb 1989) and general fitness of animals (McEwen and Brown 1966) to reduced reproductive performance and poor reproductive success (Grue, Fleming, Busby, et al. 1983; Peakall and Bart 1983).

Field studies of mammals have documented reduced productivity of adults and lower survival of young in areas treated with organophosphates (Giles 1970, Buckner and McLeod 1975). Dosing of wild nestling starlings with dicrotophos reduced cholinesterase levels and caused mortality, but there were no lasting effects on survivors (Stromborg, Grue, Nichols, et al. 1988). Other field studies have documented exposure of birds (minor reduction in cholinesterase levels) to organophosphates and carbamates in agricultural areas without mortality or other adverse effects (White and Seginak 1990; White, Seginak, and Simpson 1990). In contrast, a similar study clearly indicated severe exposure and considerable mortality in sage grouse exposed to agricultural sprays (Blus, Staley, Henny, et al. 1989). These inconsistencies appeared related to the relative toxicity of the insecticides and the susceptibility of the birds studied.

Laboratory studies have documented a unique effect of organophosphates on birds that would be very difficult to either recognize or evaluate in the field. Exposure of chickens has caused a delayed neurotoxicity characterized by axonal degeneration and demyelination of nerve bundles that produce symptoms of ataxia and paralysis (Davies and Richardson 1980). Recent studies have shown that the commonly used insecticides fenthion and fenitrothion are capable of causing these delayed, long-term effects (Farage-Elawar and Francis 1987, 1988).

Another class of insecticides in which there has been much interest are those that inhibit the synthesis of chitin in insects and thereby cause mortality (Marx 1977). Diflubenzuron has received most attention and study. It has a low toxicity to wildlife (Wilcox and Coffey 1978), and field studies suggest it can be safely used in wildlife habitats without adverse effects on birds and mammals (Bart 1975; Bruckner, McLeod, and Lidstone 1975; Richmond, Henny, Floyd, et al. 1979).

By 1986, pyrethroid insecticides were in use worldwide, and 25 percent of the insecticides used to protect crops were these relatively new, photostable, synthetic materials (Hirano 1989). Four are registered for use in the United States: permethrin, cypermethrin, deltamethrin, and fenvalerate (Day 1989). The pyrethroids are generally about 100 times more toxic to

insects than the other classes of insecticides (Elliott 1989) and are used for household, public health, and veterinary purposes as well as in the protection of cotton and other agricultural crops (Haya 1989).

The pyrethroids are lipophilic, are rapidly adsorbed onto materials in water, and readily contaminate aquatic organisms (Demoute 1989). They are extremely toxic to fishes (Elliott 1989, Haya 1989) and aquatic invertebrates (Coats, Symonik, Bradbury, et al. 1989), especially crustacea (Day 1989).

In contrast, the pyrethroids are quickly metabolized and have a low toxicity to birds and mammals (Elliott 1989). They are more rapidly excreted (within a few days) from birds and mammals than from insects and fishes (Demoute 1989). They do not persist or accumulate in the environment. With low vapor pressures, they are not readily carried in air currents and have not been found to be subject to aqueous leaching (Demoute 1989, Elliott 1989).

The severe toxicity of the pyrethroids to fishes and aquatic arthropods raises a question about their use in aquatic ecosystems. Fish mortality following pyrethroid applications has not been common, suggesting that adsorption to other materials in natural waters may protect fishes (Demoute 1989). Their effects on nontarget organisms have been reviewed (Mulla, Majori, and Arata 1979; Hill 1985; Natl. Res. Counc. Canada (NRCC) 1986; Smith and Stratton 1986).

SOME FINAL THOUGHTS

The indirect effects of pesticides on habitat quality have the potential to seriously influence wildlife populations, and more attention should be given to such possibilities (Bunyan and Stanley 1983). Many human activities contribute simultaneously to habitat changes, and it is difficult to isolate alterations due to pesticides and to identify their particular influence on animal populations. Little is known of the life requirements of most species, so these evaluations will demand additional ecological and life history information. Pesticide studies have been exciting because they often required the gathering of such data. Research on pesticides has contributed much new knowledge about the habits, behavior, reproduction, foods, and population dynamics of species.

Pesticide mortality has created a genetic resistance in a few vertebrate populations to some insecticides. This has occurred in frogs (Boyd, Vinson, and Ferguson 1963), pine mice (Webber, Hartgrove, Randolph, et al. 1973), and mosquitofish (Ferguson 1963). Resistant frogs and mosquitofish accumulate much higher residues when exposed to chemicals and pose a greater hazard to their predators (Rosato and Ferguson 1968). The fright-

ening potential of this process has not been thoroughly assessed, and further work is needed.

Wildlife mortality from the more toxic carbamate and organophosphate insecticides remains a problem. Use of these insecticides must be restricted, and additional data are needed to fully illustrate their hazards. It is generally accepted that carbamate and organophosphate insecticides neither persist in the environment nor accumulate in food chains. This assumption is sometimes false. Residues of organophosphates can persist for long periods in certain environmental substrates. As with chlorinated hydrocarbons, this occurs in situations where residues are isolated from factors that metabolize them. These relationships are poorly understood, as they have not been studied. Techniques are available to investigate the environmental fate of organophosphates, and such information should be generated.

Organophosphates can persist, and they also can accumulate in food chains. Concentrations have been measured in fishes (Mulla, Keith, and Gunther 1966), tadpoles (Hall and Kolbe 1980), and frogs (Fleming, de Chacin, Patee, et al. 1982). Some predators fed contaminated tadpoles and frogs in these studies died from organophosphate poisoning. Apparently, fishes and amphibians do not produce the enzymes that rapidly degrade organophosphates. Birds and mammals have such enzymes, and residues have not been found to accumulate in their bodies. Field and laboratory investigations of food chain accumulations of organophosphates and their hazards to birds and mammals are needed.

In the past, much effort was given to evaluating effects of polychlorinated biphenyls and mercury on wildlife. These chemicals were widely distributed in the environment and accumulated in animals. Mortality and other sublethal effects were documented (Bailey and Bunyan 1972; Heath, Spann, Hill, et al. 1972), and toxicity and sublethal exposure were evaluated in the laboratory (Dahlgren and Linder 1971, Heinz 1974). Seed dressing with mercury has been discontinued, and other uses of mercury and the uses of polychlorinated biphenyls are increasingly being regulated to prevent contamination of the environment and wildlife.

The problems created by heavy metals, oil pollution, and acid rain are subjects that deserve attention along with the remaining questions about pesticides. Although a number of biologists are working on toxic substances in the environment, new problems seem to arise before the older ones are completely solved. Still, it is evident that sufficient progress in identifying problems has been made to enable regulation of chemicals that pose the most serious threats to wildlife. Adequate regulations must be promulgated and vigorously enforced to ensure protection of the environment and wildlife resources from toxic substances.

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