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TOXAPHENE HAZARDS TO FISH, WILDLIFE, AND INVERTEBRATES: A SYNOPTIC REVIEW

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SUMMARY

Toxaphene (chlorinated camphene, 67-69% chlorine) is a broad-spectrum insecticide which, until recently, was one of the most heavily-used agricultural chemicals on a global scale, especially against pests of cotton. It is extremely persistent in soil and water, with documented half-times of 9 to 11 years; however, in air and in warm-blooded organisms, toxaphene degradation is rapid with half-times of 15 and 3 days, respectively. Toxaphene is especially hazardous to nontarget marine and freshwater organisms, with death recorded at ambient water concentrations substantially below 10 ug/l, and adverse effects observed on growth, reproduction, and metabolism at water concentrations between 0.05 and 0.3 ug/l. Aquatic organisms readily accumulate toxaphene from the ambient medium and diet, sometimes spectacularly, retain it for lengthy periods, and biomagnify the chemical through food chains. These phenomena could account for the numerous fish kills recorded after toxaphene application, as well as the high residues measured in fish from the Rio Grande Valley in southern Texas and other locations of high agricultural use of toxaphene. Atmospheric vectors, including prevailing winds and rainfall, may transport toxaphene hundreds of kilometers from known point sources of application. This, in part, would explain the levels of 5 to 10 mg/kg whole body wet weight recorded in various species of fish from the Great Lakes.

Based on estimated environmental exposure levels, toxaphene does not appear to constitute a major threat to warm-blooded animals, including migratory birds and other wildlife, domestic poultry and livestock, small laboratory mammals, and humans. Wildlife typically contain low or nondetectable levels of toxaphene, except for some species of fish-eating raptors, and the frequency of occurrence is low when compared with that of other organochlorine agricultural compounds. However, toxaphene has been implicated as a human carcinogen and mutagen at relatively high test dosages and was associated with some bird kills following aerial applications.

In water, the concentration of toxaphene considered safe for protection of freshwater life is conservatively estimated to lie between 0.008 and 0.013 ug/l; for marine life, it is 0.07 ug/l. This is in sharp contrast to the current recommended drinking water criterion for human health protection of 5.0 to 8.8 ug/l. Similarly, residues in fish tissue in excess of 0.4 to 0.6 mg/kg wet weight may be hazardous to fish health and should be considered as presumptive evidence of significant environmental contamination, although fish may contain up to 5.0 mg/kg before they are considered hazardous to human consumers. At present, other existing criteria for human health protection, which range in various foods from 0.1 mg/kg for sunflower seeds to 7.0 mg/kg in meat, fats, and citrus fruits, also appear adequate to safeguard sensitive species of wildlife.

In 1982, the U.S. Environmental Protection Agency cancelled the registrations of toxaphene for most uses. However, current stocks of toxaphene may be used, with restrictions, through 1986. Furthermore, considerable, but unknown, quantities of toxaphene previously discharged into the environment over the past several decades may remain undegraded and potentially available to living resources. Accordingly, we recommend, to all natural resources managers, that toxaphene application is contraindicated if there is a history of extensive prior treatment with toxaphene in their jurisdictional areas, if alternative control methods are available, or if there is no clear threat to crop production or to the health of livestock and humans.

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TABLES

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INTRODUCTION

Environmental hazards and increasing public concerns associated with toxaphene (chlorinated camphene, 67-69% chlorine) are documented in a series of useful review articles (Pollock and Kilgore 1978; EPA 1980a; Cohen et al. 1982; Rice and Evans 1984). Although toxaphene was introduced in the mid-1940's as a new insecticide, only a few years elapsed before it was being used commercially on a large scale to effectively control a variety of pests. In the mid-1950's, toxaphene was first used in ponds, lakes, and streams as a piscicide. By 1966, toxaphene was the chemical of choice in fish eradication programs in Canada and second in the United States after rotenone (Lennon et al. 1970). Its use for this purpose was discontinued in the 1960's due to its lengthy persistence in water, high acute toxicity to aquatic biota, and significant bioaccumulation and biomagnification in various environmental compartments. By 1974, cumulative world use of toxaphene, mainly against insect pests of cotton, was estimated at 450,000 metric tons. Production of toxaphene declined from 1973 to 1980; however, annual consumption in 1980 was estimated at 105,000 tons, thus qualifying toxaphene as one of the most heavily-utilized agricultural chemicals worldwide. Until recently, toxaphene was extensively applied in California to control fruitworms on tomatoes, bollworm on cotton, and a wide range of pestiferous insects that infested alfalfa, broccoli, celery, beans, clover, lettuce, cauliflower, and pears. In time, toxaphene-resistant strains of cotton pests, including bollworm and lygus bug, appeared in California, Texas, Egypt, and India. In November, 1982, most registered uses of toxaphene were cancelled by the U.S. Environmental Protection Agency (EPA), although existing available stocks may be used through 1986 (EPA 1982). Prior to the EPA action, similar actions that banned or restricted toxaphene use had been implemented in a number of countries, including Canada, England, Sweden, Finland, Denmark, France, Switzerland, Hungary, Italy, Egypt, and Algeria (Cohen et al. 1982).

In this account, we briefly summarize available information on the environmental fate and effects of toxaphene, with special emphasis on gamefish, migratory birds, and their predators and prey. The recommendations on current and proposed safe limits for toxaphene residues in air, water, and biota are reviewed. This effort is part of a continuing series of synoptic reviews on contaminant hazards to natural resources and was prepared in response to informational requests from environmental specialists of the U.S. Fish and Wildlife Service.

ENVIRONMENTAL CHEMISTRY

The commercial production of toxaphene involves the reaction of camphene, chlorine activated by ultraviolet radiation, and certain catalysts to yield chlorinated camphene with a chlorine content of 67 to 69% by weight. This product is a relatively stable material composed of a mixture of structurally similar compounds and isomers. Of the 177 components, 26 have been isolated but only 10 have been identified; these 26 components comprise 40% of the toxaphene. Information on chemical properties and the fate and effects of the remaining components is missing or incomplete (Cohen et al. 1982). Several components that have been tested are more toxic to houseflies than the technical mixture, especially di-, tri-, and tetrachlorobornane compounds (Pollock and Kilgore 1978). Technical toxaphene is a yellow, waxy solid of empirical formula $C_{10}H_{10}Cl_8$ and an average molecular weight of 414. Toxaphene is soluble in water to 3 mg/l and is readily soluble in fats and organic solvents, based on its high partition coefficient of 10 to the power 3.3-6.4. Toxaphene has a tendency to adsorb on sediments and to bioaccumulate in aquatic organisms.

Because toxaphene consists of numerous compounds, it seems inappropriate and misleading to continue using the name toxaphene to describe this insecticide. We now know that chemical properties, such as solubility, toxicity, volatility, and other properties, are the sum of the individual contribution of many different compounds in differing relative amounts. A 50-fold difference between toxicities of toxaphene components can occur, and, with a wide range in the polarity of different fractions, there probably are also significant solubility differences. In addition, the composition of toxaphene changes with time, and residues in fat are not of the same composition as parent toxaphene (Pollock and Kilgore 1978). The metabolism of toxaphene has been an area of limited research activity, owing to the analytical difficulties involved in detecting a multicomponent substance (Pollock and Kilgore 1978). However, toxaphene has been reported more often in biological samples in recent years. This increased recognition is probably due to better analytical methods for toxaphene analysis (Ribick et al. 1982), greater awareness by analysts, and the continuing widespread use of toxaphene while use of potentially interfering organochlorine insecticides has slowly decreased.

Toxaphene was available as an emulsifiable concentrate, wettable power, or dust. The commercial product is relatively stable but may decay upon prolonged exposure to sunlight, alkalis, or temperatures above 120°C. Toxaphene is also known as chlorinated camphene, Synthetic 3956, Octachlorocamphene, Alltox, Geniphene, Toxakil (Negherbon 1959), polychlorocamphene, camphechlor, Clor Chem T-590, Cristoxo, Moto, Phenacide, Phenatox, Strobane-T, Toxon 63, and Vapotone (Johnson and Finley 1980). Chemically, it is known as a mixture of various chlorinated camphenes (Tucker and Crabtree 1970).

Toxaphene residues have been detected in various environmental compartments hundreds of kilometers distant from known applications of this insecticide. Prevailing winds, rainfall, and sediment runoff probably account for substantial portions of this transport. Rainfall, for example, has been implicated as a significant toxaphene vector in South Carolina estuaries (Harder et al. 1980). During and immediately after the summer use season, toxaphene levels in rain exceeded, by several times, the concentrations reported to produce bone damage in fish under controlled laboratory conditions. Toxaphene becomes sorbed to soils when it is used in agriculture; therefore, a major mode of toxaphene transport in areas planted continuously in cotton is through sediment loss in runoff (McDowell et al. 1981). Measurements indicated a linear relation between toxaphene yield and sediment yield in runoff water. Atmospheric transport of toxaphene is well documented. Air samples from the western North Atlantic contained measurable levels of toxaphene at distances up to 1,200 km from the nearest point source of application on land (Bidleman and Olney 1975). Similarly, Nationwide monitoring of toxaphene in fish showed increases during 1970-74 (Schmitt et al. 1981), especially in areas where the insecticide was not used, suggesting that atmospheric transport is essential to widespread distribution. Airborne toxaphene is resistant to photodecomposition; however, selective volatilization of toxaphene components is a major cause of degradation resulting in an estimated half-time of 15 days while in the atmosphere (Cohen et al. 1982).

Toxaphene degrades more rapidly in most environmental compartments than other chlorinated pesticides, such as DDT and dieldrin (Matsumura 1978). Toxaphene persistence and degradation in soil, water, and biota is modified by numerous and disparate biological and abiotic factors. In lakes, toxaphene persistence was significantly related to lake depth, stratification, and turnover, but not related to surface area, pH, temperature, sunlight, and oxygen (Cohen et al. 1982). Data from studies where toxaphene was used to control nongame fish in lakes suggest that it may persist in water from several months to more than 9 years. For example, two mountain lakes in Oregon that were treated with toxaphene in fish eradication programs remained toxic for 1 to 6 years (Terrierre et al. 1966). Davis Lake, a shallow lake rich in aquatic life, which was treated with 88 ug/l toxaphene, could be restocked with rainbow trout (*Salmo gairdneri*) within 1 year when water toxaphene levels were 0.63 ug/l. Trout grew rapidly, although whole body burdens up to 24 mg/kg were recorded. Miller Lake, a deep, biologically sparse lake, was treated with 40 ug/l toxaphene; trout could not be restocked for 6 years until water levels had dropped to 0.8 ug/l toxaphene. Toxaphene at 50 ug/l was used to eradicate fish from Clayton Lake, New Mexico (Kallman et al. 1962). Water concentrations of 1.0 ug/l were measured 250 days post-treatment, but the lake remained toxic for 9 months, with restocking possible only after 12 months. Residues in fish surviving treatment were 3.5 mg/kg whole body wet weight shortly after exposure and 0.3 mg/kg about 5 months postapplication. Some lakes treated with toxaphene to kill fish have remained toxic for 3 to 4 years (Webb 1980). In another study (Johnson et al. 1966), lake water that contained 1.0 ug/l toxaphene (9 years after toxaphene treatment) supported healthy fish populations. In this lake, particulate matter contained 70 ug of toxaphene/kg, and plankton contained 15,000 ug/kg. However, there were changes in gas chromatographic profiles of toxaphene residues taken from the lakes, suggesting that the parent toxaphene had been altered or degraded into compounds with lower environmental hazards to biota. Clearly, this subject area merits additional research effort.

In soils, toxaphene can persist for lengthy periods, with microbial degradation occuring under aerobic and anaerobic conditions (Cohen et al. 1982). Pimentel (1971) reported that toxaphene, applied at 140 mg/kg of soil persisted for more than 6 years; when applied at 50 mg/kg, half the toxaphene was measurable after 11 years. Further, in sandy loam soils, 45% of the toxaphene remained 14 years after initial application of 100 mg/kg. Some investigators suggest that toxaphene degradation is more rapid under anaerobic conditions (Pollock and Kilgore 1978). Thus, toxaphene in anaerobic salt marsh sediments generally degraded within a few days to shorter-lived components (Williams and Bidleman 1978). Toxaphene accumulated only slightly in anaerobic marsh soils not flooded daily by tides (Gallagher et al. 1979), and the highest pesticide concentrations were associated with roots of dead plants.

Degradation of toxaphene in plant, air, and soil samples was evident following toxaphene application of 9

kg/ha to a San Joaquin Valley, California, cotton field (Seiber et al. 1979). Cotton leaves contained 661 mg toxaphene/kg immediately after application and 135 mg toxaphene/kg after 58 days, with the greatest loss attributed to components of highest volatility. Air samples were essentially the same at 2 and 14 days postapplication (1.8-1.9 ug/m^3); this was attributed to a corresponding enrichment of volatile components. Top soil samples immediately after application and 58 days later contained 13.1 and 6.4 mg/kg, respectively; loss was primarily via vaporization, but at least one component was significantly degraded. One year later, soil cores and irrigation ditch samples showed extensive toxaphene degradation resulting in a selective decline of some components; anaerobic reduction occurred in these environmental compartments.

In rats, the half-time of toxaphene (time to 50% excretion) was 1 to 3 days. If the trend persisted, virtually all toxaphene would be eliminated in five half-lives. Elevated blood toxaphene levels in a human subject who had eaten catfish fillets containing 52 mg of toxaphene/kg dropped 67% in 11 days. By 14 days after the initial measurement, toxaphene blood levels were below analytical detection limits (EPA 1980a).

RESIDUES IN FIELD POPULATIONS

National contaminant monitoring surveys, conducted in the period 1974-76, show that toxaphene was detected in about 6% of all fish sampled; this is a higher percentage than recorded in fruits, vegetables, poultry, and meat (Ludke and Schmitt 1980). Fish collected Nationwide at 109 stations between 1976 and 1979 had measurable toxaphene residues at about 60% of all stations sampled; concentrations in fish from the Great Lakes stations exceeded those in fish from most of the rest of the United States, including locations within the cotton-growing areas (Schmitt et al. 1983). Lake trout (*Salvelinus namaycush*) from Lake Michigan typically contained 5 to 10 mg of toxaphene/kg whole body on a wet weight basis; lake trout from Lake Huron contained 9 mg/kg. These residues are considered harmful to various sensitive species of freshwater teleosts (Schmitt et al. 1983). Since relatively little toxaphene has been used in the Great Lakes region when compared to cotton-growing areas in the mid-South, Northeast, and Southeast, it is postulated that atmospheric transport from areas to the south and southwest are the sources of toxaphene contamination in the Great Lakes (Schmitt et al. 1983).

Freshwater fishes of the Arroyo Colorado, a major waterway traversing the lower Rio Grande Valley in Southern Texas, were highly contaminated with toxaphene and DDE residues when compared to fish collected elsewhere in the Valley; toxaphene concentrations ranged up to 31.5 mg/kg wet weight in whole fish composite samples (Table 1). These values were within or above the range producing adverse effects in sensitive species of fish. In addition, toxaphene residues in carcasses of fish-eating birds contained up to 3 mg/kg toxaphene (Table 1). Unlike fishes, avian species readily metabolize and excrete toxaphene, so that little accumulation occurs in tissues; in any event, these levels of toxaphene in carcasses of piscivorous birds are probably biologically insignificant (White et al. 1983). In the Arroyo Colorado area, toxaphene was being used, to some extent, on crops such as cotton, not only as an insecticide, but as a carrier for more effective chemicals. Another possible source of contamination is a former pesticide plant at Mission, Texas, near the headwaters of the Arroyo Colorado. Soil at this site contained high concentrations of various pesticides, including toxaphene. Contaminant laden runoff from this site could eventually reach the Arroyo from storm sewers and other water diversion facilities. The contaminated Arroyo Colorado, in turn, empties into the Laguna Madre, one of the more important breeding and nursery grounds for fish and wildlife in the United States. The Texas Department of Health, in an advisory to consumers, has stated that consumption of fishes from the Arroyo Colorado, especially blue catfish *(Ictalurus furcatus*) and gizzard shad (*Dorosoma cepedianum*), is not advised (White et al. 1983)

Birds, unlike fish, generally contained low or nondetectable levels of toxaphene, and the frequency of occurrence was relatively low when compared with that of other organochlorine pesticides. This generalization held for eggs of the osprey (*Pandion haliaetus*) collected in southern New Jersey in 1974 (Wiemeyer et al. 1978); carcasses of 103 skinned shorebirds from Corpus Christi, Texas, during winter 1976-77 (White et al. 1980); eggs of the brown pelican (*Pelecanus occidentalis*) from 1971-76 in Louisiana (Blus (et al. 1979); and eggs of clapper rail (*Rallus longirostris*), purple gallinules (*Porphyrula martinica*), and limpkins (*Aramus guarauna*) from the Southeast in 1972-74 (Klass et al. 1980). Among 105 herons found dead Nationwide since 1976, only nine contained measurable quantities of toxaphene; for DDE, PCB'S, dieldrin, and DDD, these frequencies were 96, 90, 37 and 35, respectively (Ohlendorf et al. 1981). Levels of toxaphene and other organochlorines in canvasbacks (*Aythya valisineria*) from Chesapeake Bay, Maryland, during 1973-76 were below the levels known to cause problems in other species (White et al. 1979). However, adipose tissues from 55 male wild turkeys (*Meleagris gallopavo*) killed during the 1974 hunting season in southern Illinois contained 0.2 to 0.9 mg/kg of toxaphene (Bridges and Andrews 1977), suggesting that certain species of birds may

selectively accumulate low concentrations of toxaphene.

Two bird kills reported in California have been attributed to toxaphene poisoning (Pollock and Kilgore 1978). In one case, the apparent route of exposure was from contaminated fish, with bird poisoning the result of toxaphene biomagnification in the food chain. In that case, algae contained 0.1 to 0.3 mg toxaphene/kg wet weight, snails and daphnids 0.2, fish 3 to 8, and fish-eating birds 39 mg/kg. The latter value is substantially in excess of 3 mg/kg, a concentration considered biologically insignificant to fish-eating birds (White et al. 1983). The second incident involved some birds that were apparently killed by toxaphene when it was used to control grasshoppers on a shortgrass range. At 2 to 3 weeks postspray, bird carcasses contained 0.1 to 9.6 mg toxaphene/kg.

Biomagnification of toxaphene through food webs was clearly demonstrated in 16 species of organisms collected from oxbow lakes in northeastern Louisiana during 1980 (Neithammer et al. 1984). Without exception, residues were highest (3.6 mg/kg whole body wet weight, range 1.7 to 5.5) in tertiary consumers, such as green-backed heron (*Butorides striatus*), various species of snakes, spotted gar (*Lepisosteus oculatus*), and largemouth bass (*Micropterus salmoides*). Secondary consumers, such as bluegill (*Lepomis macrochirus*) blacktail shiner (*Notropis venustus*), and yellow-crowned night-heron (*Nycticorax violaceus*), contained lower residues (0.9 mg/kg wet weight, range 0.7 to 1.2). Primary consumers, including crayfish (*Procambarus* spp.) and threadfin shad (*Dorosoma petenense*), contained the lowest levels (0.8 mg/kg wet weight, range 0.6 to 1.0) of all consumer groups. Toxaphene levels were not detectable in water and sediments from these oxbow lakes.

LETHAL EFFECTS

Toxaphene is extremely toxic to freshwater and marine biota. In laboratory tests of 96 hours duration, 50% mortality was recorded for the most sensitive species of freshwater and marine teleosts, marine crustaceans, and freshwater insects at nominal water concentrations of less than 10 ug/l of toxaphene, and, in several cases, less than 1 ug/l (Table 2). Bioassays of longer duration, based on exposure of aquatic organisms for the entire or most of the life cycle, produced significant adverse effects on growth, survival, and reproduction at toxaphene concentrations between 0.025 and 1.0 ug/l (Table 3). Based on its high toxicity and extensive use, it is not suprising that toxaphene was considered a major cause of Nationwide fish kills in 1977 (EPA 1980b).

Warm-blooded organisms are relatively resistant to toxaphene, as determined from results of short-term tests involving oral, dermal, and dietary routes of administration. In acute oral toxicity tests with birds and mammals, LD-50 values ranged between 10 and 160 mg/kg body weight (Table 4). The acute oral toxicities of toxaphene to rats, mice, dogs, guinea pigs, cats, rabbits, cattle, goats, and sheep extended from 25 to 270 mg/kg body weight (Pollock and Kilgore 1978; EPA 1980a); these values are in good agreement with those shown in Table 4. Dermal toxicities of toxaphene ranged from 250 mg/kg body weight for rabbits and 930 mg/kg for rats to 25,000 mg/kg for cattle (Pollock and Kilgore 1978). As was true for acute oral and dermal toxicity data, comparatively high levels of dietary toxaphene were required, i.e., 538 to 828 mg/kg diet, to produce significant death rates in various species of birds (Heath et al. 1972). In their study on four species of gamebirds, each aged 2 weeks, Heath et al. (1972) fed them diets containing graded concentrations of toxaphene for 5 days, followed by 3 days of untreated food. LD-50 values at the end of day 8 were 828 mg toxaphene/kg diet for northern bobwhite, 686 for Japanese quail (*Coturnix coturnix japonica*), 542 for ring-necked pheasant (*Phasianus colchicus*), and 538 for mallard (*Anas platyrhynchos*). It appears that toxaphene is not a major hazard to bird survival at previously recommended field application rates (Hoffman and Albers 1984). However, at toxaphene levels not considered life-threatening to birds and mammals, fetotoxic effects have been recorded. For example, ring-necked pheasants fed 100 mg/kg dietary toxaphene produced eggs with significantly reduced hatch over controls; similarly, toxaphene administered orally to pregnant rats and mice during organogenesis caused fetal toxicity at 15 mg/kg body weight (Pollock and Kilgore 1978).

Some human deaths, especially those of children, have been reported following the ingestion of toxaphene-contaminated foods (EPA 1980a). Known toxaphene residues in food items of victims ranged from 9.7 to 47 mg/kg; a total dose of 2 to 7 g of toxaphene is considered acutely toxic to a 70 kg adult. For comparison purposes, a 4.5 kg bird would probably die after consumption of 45 to 450 mg of toxaphene.

SUBLETHAL EFFECTS

Among sensitive species of marine and freshwater fish and invertebrates, water concentrations of 0.054 to 0.299 ug/l of toxaphene were associated with growth inhibition, reduced reproduction, backbone abnormalities, or histopathology (Table 5). Aquatic biota are capable of spectacular accumulations of toxaphene from the medium; factors ranged between 1,270 and 52,000X those of water under laboratory conditions (Table 6). A similar pattern was observed in Big Bear Lake, California, where toxaphene was applied at 200 ug/l to eradicate goldfish (Pimentel 1971). Biomagnification factors of 365 were calculated for plankton, 1,000 for goldfish, and 8,500 in pelican fat, representing residues of 73 mg/kg toxaphene in phytoplankton, 200 in goldfish, and 1,700 in pelican fat. Accumulation of toxaphene by various species of fish food organisms is dependent on exposure time and concentration. For example, insect nymphs subjected to 20 ug/l of toxaphene for <24 hours did not accumulate doses lethal to fish; however, algae, diatoms, and protozoan ciliates held for 24 hours in 20 ug/l toxaphene solutions, and *Daphnia magna* held 120 hours in 10 ug/l, were lethal when fed to fish (Schoettger and Olive 1961).

Fish accumulated part-per-million toxaphene concentrations in various tissues within a few days when placed in toxaphene-treated lakes that contained less than 1.0 ug/l (Cohen et al. 1982). Freshwater teleosts experienced acute and chronic effects when whole body levels were in excess of 0.4 mg/kg but less than 5 mg/kg (this latter value being the Food and Drug Administration "action level" for human consumption; Cohen et al. 1982). Thus, groups of brook trout eggs containing 900 ug toxaphene/kg had drastically reduced survival when compared to controls (Cohen et al. 1982), and brook trout tissue residues exceeding 400 ug toxaphene/kg were associated with reductions in growth, abnormal bone development, and reduced fecundity (Mayer and Mehrle 1977). Fathead minnows containing more than 400 ug toxaphene/kg grew more slowly than controls (Mayer and Mehrle 1977); similar results were reported in channel catfish fry containing 600 to 3,400 ug toxaphene/kg (Mayer and Mehrle 1977). Toxaphene retention by aquatic organisms is relatively lengthy when compared to mammals. In one case, eastern oysters (*Crassostrea virginica*) held for 24 weeks in 10 ug/l toxaphene solutions contained 32.4 mg/kg in soft tissues; after 16 weeks in noncontaminated seawater, oysters still contained 3.0 mg/kg (Pollock and Kilgore 1978).

Sublethal effects of toxaphene observed in mammals, small laboratory animals, and birds were similar to those recorded for aquatic organisms; however, there was general agreement that effects were induced at much higher concentrations. In domestic white leghorn chickens, for example, toxaphene at 100 mg/kg in the diet for 30 weeks did not significantly alter egg production, hatchability, or fertility, although some bone deformation and kidney lesions were recorded (Bush et al. 1977). The highest dietary dose of toxaphene fed to chickens in life-time exposure studies, which produced no effect on any parameter measured, ranged between 3.8 and 5 mg/kg (Bush et al. 1977). Several studies with the American black duck (*Anas rubripes*) produced effects similar to those recorded in chickens. In one study, ducklings that were fed diets containing 10 or 50 mg of toxaphene/kg for 90 days had reduced growth and impaired backbone development (Mehrle et al. 1979). Collagen, the organic matrix of bone, was significantly decreased in cervical vertebrae of ducklings fed the 50 ppm toxaphene diet. Calcium concentrations increased in vertebrae of ducklings fed either 10 or 50 mg/kg dietary toxaphene; effects were observed only in female ducklings. In a long-term feeding study lasting 19 months, which included two breeding seasons, American black ducks, fed 10 or 50 mg toxaphene/kg in a dry mash diet, showed no significant differences when compared to control birds in survival, egg production, fertility, hatchability, eggshell thickness, or growth and survival of young (Haseltine et al. 1980). The only negative effects recorded included weight loss among treated males during summer and a slight delay in the number of days required to complete a clutch. Carcass toxaphene residues, which seldom exceeded 0.5 mg/kg, were found in only one duck fed the 50 mg/kg diet (Haseltine et al. 1980), suggesting low body accumulations in American black duck. However, toxaphene residues were present in the liver of all birds fed toxaphene. At dietary concentrations of 10 or 50 mg/kg, there was no change in avoidance behavior of young American black ducks (Heinz and Finley 1978), which, if interrupted, is considered life-threatening.

Ring-necked pheasants (*Phasianus colchicus*) fed diets containing 300 mg toxaphene/kg showed decreases in egg deposition, egg hatch, food intake, and weight gain; at 100 mg/kg, all of these parameters, except reduced hatch, were the same as controls (Pollock and Kilgore 1978). In a field study, aerial applications of a DDT-toxaphene mixture in southwestern Idaho during 1970, at recommended concentrations to control pests, had no major impact on penned or feral ring-necked pheasants (Messick et al. 1974), suggesting that conformance with recommended application rates should be endorsed whenever possible. However, we emphasize that recommended toxaphene application rates, until recently, varied widely and depended, in part,

on the pest species to be controlled, the number and type of other pesticides applied jointly, and climatic conditions. Laboratory studies with mallard eggs suggest that recommended toxaphene application rates in excess of 1.12 kg/ha, which is generally exceeded in most cases, may produce severe embryotoxic effects, including dislocated joints and poor growth (Hoffman and Eastin 1982).

Northern bobwhite fed 5 mg/kg dietary toxaphene for 4 months showed thyroid hypertrophy (Pollock and Kilgore 1978) and interference with the ability of bobwhites to discriminate patterns (Kreitzer 1980). In the latter investigation, Kreitzer fed 10 or 50 mg/kg dietary toxaphene to 3-day old bobwhites for 20 weeks and found that toxaphene-treated birds made 50% more errors than controls on initial testing. These effects appeared as early as 30 days after toxaphene exposure. In a second test, there was no difference between experimentals and controls, indicating that the ability to learn these tasks was not permanently impaired.

Rats, mice, dogs, deer, sheep, and cattle are all relatively resistant to toxaphene. No-effect levels of 20 to 25 mg/kg dietary toxaphene were documented during multigeneration exposure of rats and during 2-year feeding studies with mice and dogs (EPA 1980a). No effects were observed in monkeys over a 2-year period during which they were fed diets containing 0.7 ppm toxaphene (Pollock and Kilgore 1978). However, carcinogenic responses have been induced in mice and rats by toxaphene when residues in the diet exceeded 50 mg/kg during lifetime exposure (EPA 1980a). "These results, together with the positive mutagenic response (to *Salmonella* bacteria) constitute substantial evidence that toxaphene is likely to be a human carcinogen" (EPA 1980a). Penned and wild deer fed toxaphene at 1,000 mg/kg appeared normal but showed a decreased digestion rate, which was attributed to a decrease in rumen bacteria (Schwartz and Nagy 1974). Steers fed alfalfa hay containing 306 mg toxaphene/kg for 19 weeks stored 772 mg/kg in abdominal fat and 27 mg/kg in lean meat without apparent ill effects, demonstrating the lipophilicity of toxaphene and the relatively low accumulation rates. For sheep under an identical regimen, these values were 317 mg/kg in fat and 36 mg/kg in meat (Pollock and Kilgore 1978).

RECOMMENDATIONS

In November, 1982 the U.S. Environmental Protection Agency cancelled the registration of toxaphene for most uses and, thus, joined a growing number of Nations in Western Europe, Scandinavia, North America, and North Africa that previously initiated similar actions. With some restrictions, toxaphene presently may be used domestically for treatment of scabies in cattle and sheep; controlling sporadic infestations of armyworms, cutworms, and grasshoppers on cotton, corn, and small grains; and, in Puerto Rico and the Virgin Islands, to control mealy bugs, pineapple gummosis moths, and banana weevils. Existing stocks of toxaphene may be used through 1986 for control of sicklepod in soybeans and peanuts, for insects in corn cultivated without tillage, and for pests of dry and southern peas (EPA 1982).

Although toxaphene is not markedly hazardous to most wildlife species for which data were available, the decision to withdraw or curtail agricultural uses of toxaphene was popular with most natural resource managers. Their concerns, apparently shared by others, were based, in part, on the following observations. First, toxaphene causes death and deleterious effects to nontarget aquatic biota at extremely low concentrations, i.e., <1.0 ug/l. Second, toxaphene is persistent in soils, water, and other environmental compartments, with residence times measured in years. Third, toxaphene accumulates in aquatic organisms and biomagnifies through food chains. Fourth, toxaphene is widely-distributed, even when the initial application point is hundreds of kilometers distant; transport is presumably by atmospheric and other vectors. Fifth, technical difficulties continue to exist in the chemical analysis of toxaphene, a 177-isomer compound. Sixth, there is an imperfect understanding of the fate and effects of individual toxaphene components. Seventh, there is inadequate knowledge of interaction effects of toxaphene with other agricultural chemicals (especially when mixtures are applied simultaneously) and with other persistent compounds in aquatic ecosystems, such as PCB'S, DDT and its isomers, and petroleum. Finally, there is the perception that suitable alternative pesticidal chemicals are available, including some carbamates, organophosphorus compounds, and synthetic pyrethroids.

At present, available stocks of toxaphene may be used throughout 1986. However, large but unknown quantities of toxaphene that were discharged into the environment over the past several decades remain undegraded and potentially bioavailable. Also, knowledge of toxaphene ecotoxicology is incomplete or inadequate. Accordingly, we recommend to fish and wildlife managers that they review all current and proposed uses of this compound in their jurisdictional areas. Specifically, we recommend that toxaphene use should not be permitted if there is a history of extensive prior treatment with toxaphene, if alternative control methods are available, or if there is no clear threat to crop existence or to health of livestock and humans. Current limits for

toxaphene residues in air, water, biota, and other environmental compartments for the protection of fish, livestock, and human health are summarized in Table 7. The concentration of toxaphene in seawater considered safe for marine life protection is 0.07 ug/l; for sensitive freshwater species this lies between 0.008 and 0.013 ug/l. This contrasts sharply with the current recommended drinking water criterion for human health protection of 5.0 to 8.8 ug/l. Other existing criteria for human health protection, which range in various foods from 0.1 to 7.0 mg/kg, appear adequate at this time to protect sensitive species of wildlife. We emphasize that these values, and others shown in Table 7, are considered criteria and not administratively-legislated standards.

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Table 1. Toxaphene residues in whole composite samples of freshwater fish and fish-eating birds collected from the Arroyo Colorado, Texas, in 1978 and 1979 (White et al. 1983).

Taxonomic group, year of collection, species	Residue, in ppm wet weight
Fish	
1978	
Blue catfish, *Ictalurus furcatus*	9.7-31.5
Gizzard shad, *Dorosoma cepedianum*	11.2-29.6
Sea catfish, *Arius felis*	ND[a]-0.4
Spotted seatrout, *Cynoscion nebulosus*	ND
1979	
Blue catfish	19.5-24.8
Gizzard shad	5.4
Channel catfish, *Ictalurus punctatus*	0.8-19.5
Striped mullet, *Mugil cephalus*	4.4
Birds	
1978	
Laughing gull, *Larus atricilla*	ND-3.0
Ringed-billed gull, *L. delawarensis*	ND-3.0
Franklin's gull, *L. pipixcan*	ND-2.0
Herring gull, *L. argentatus*	ND
Pied-billed grebe, *Podilymbus podiceps*	ND
Forster's tern, *Sterna forsteri*	1.7
Great-tailed grackle, *Quiscalus mexicanus*	ND
Red-winged blackbird, *Agelaius phoeniceus*	ND
1979	
Laughing gull	ND-0.4

[a]ND = not detectable.

Table 2. Acute toxicity of toxaphene to aquatic organisms. Concentrations shown are in micrograms of toxaphene per liter (ppb) of medium fatal to 50% of the test organisms in 96 hours.

Type of water, taxonomic group, species	LC-50 (96 h)	Reference[a]
Freshwater		
Insects		
Stonefly, *Claassenia* sp.	1.3	1
Stonefly, *Pteronarcys* sp.	2.3	1
Cranefly, *Tipula* sp.	18.0	1
Midge, *Chironomus* sp.	30.0[b]	1
Snipefly, *Atherix* sp.	40.0	1
Amphibians		
Leopard Frog, *Rana sphenocephala*	32.0-54.0	2
Crustaceans		
Daphnid, *Daphnia magna*	10.0[b]	1
Daphnid, *Daphnia pulex*	14.2[b]	1
Daphnid, *Simocephalus* sp.	19.0[b]	1
Amphipod, *Gammarus fasciatus*	26.0	1
Glass shrimp, *Palaemonetes kadiakensis*	28.0	3
Fish		
Largemouth bass, *Micropterus salmoides*	2.0	1
Bluegill, *Lepomis macrochirus*	2.4-29.0	1,3,4
Brown trout, *Salmo trutta*	3.1	1
Common carp, *Cyprinus carpio*	3.7	1
Channel catfish, *Ictalurus punctatus*	4.2-13.1	1,3
Black bullhead, *Ictalurus melas*	5.8	1
Coho salmon, *Oncorhynchus kisutch*	8.0	1
Rainbow trout, *Salmo gairdneri*	10.6	1
Yellow perch, *Perca flavescens*	12.0	1
Green sunfish, *Lepomis cyanellus*	13.0	1
Redear sunfish, *Lepomis microlophus*	13.0	3
Goldfish, *Carassius auratus*	14.0	1
Fathead minnow, *Pimephales promelas*	18.0	1
Guppy, *Poecilia reticulata*	20.0	3
Saltwater		
Molluscs		
Eastern oyster, *Crassostrea virginica*	16.0	3
Quahaug clam, embryo, *Mercenaria mercenaria*	1,120.0	3
Crustaceans		
Drift-line crab, *Sesarma cinereum*	0.05-8.8	3
Copepod, *Acartia tonsa*	0.11	3
Pink shrimp, *Penaeus duorarum*	1.4-2.2	3
Grass shrimp, *Palaemonetes pugio*	4.4	3
Mysid shrimp, *Mysidopsis bahia*	4.5	3
Korean shrimp, *Palaemon macrodactylus*	21.0	3
Mud crab, larva, *Rithropanopeus harrisii*	43.8	3
Blue crab, *Callinectes sapidus*	824.0	3
Fish		
Pinfish, *Lagodon rhomboides*	0.5	3
Sheepshead minnow, *Cyprinodon variegatus*	1.1	3
Striped bass, *Morone saxatilis*	4.4	3
Threespine stickleback, *Gasterosteus aculeatus*	8.2	3

[a]References: 1, Johnson and Finley 1980; 2, Hall and Swineford 1980; 3, EPA 1980a; 4, Isensee et al. 1979.
[b]48-hour value.

Table 3. Maximum acceptable toxicant concentration values (MATC) for toxaphene and aquatic organisms, based on exposure for the entire or most of the life cycle. Concentrations are in micrograms of toxaphene per liter (ppb).

Type of water, organism	MATC (µg/1)	Reference[a]
Freshwater		
Arthropods		
Daphnid, *Daphnia magna*	0.07-0.12	1
Amphipod, *Gammarus pseudolimnaeus*	0.13-0.25	1
Midge, larva, *Chironomus plumosus*	1.0-3.2	1
Fish		
Fathead minnow, *Pimephales promelas*	0.025-0.054	2
Channel catfish, *Ictalurus punctatus*	0.049-0.072	2
Saltwater		
Fish		
Sheepshead minnow, *Cyprinodon variegatus*		
Early life stage	1.1-2.5	3

[a]References: 1, Sanders 1980; 2, Mayer et al. 1977; 3, EPA 1980a.

Table 4. Acute oral toxicity of toxaphene to birds and mammals (Tucker and Crabtree 1970; Hudson et al. 1984). Concentrations shown are in milligrams of toxaphene ingested per kilogram body weight fatal to 50% of test animals. A single dose was administered orally and survival data gathered over a 14-day posttreatment observation period.

Organism	LD-50 (ppm)
Birds	
California quail, *Callipepla californica*	11.9-47.4
Sharp-tailed grouse, *Tympanuchus phasianellus*	14.1-28.2
Gray partridge, *Perdix perdix*	20.0-28.3
Ring-necked pheasant, *Phasianus colchicus*	20.0-80.0
Mallard, *Anas platyrhynchos*	
Duckling	23.3-40.6
Adult	37.6-133.0
Fulvous whistling-duck, *Dendrocygna bicolor*	37.2-264.0
Northern bobwhite, *Colinus virginianus*	59.3-123.0
Lesser sandhill crane, *Grus canadensis canadensis*	100.0-316.0
Horned lark, *Eremophila alpestris*	425.0-794.0
Mammals	
Mule deer, *Odocoileus hemionus hemionus*	139.0-240.0
Domestic goat, *Capra hircus*	>160.0

Table 5. Sublethal effects of toxaphene to aquatic biota.

Type of medium, organism	Toxaphene concentration in medium, in µg/1 (ppb)	Exposure duration, in days	Effect	Reference[a]
Freshwater				
Daphnid, *Daphnia magna*	0.12	14	Reduced reproduction	1
Midge, *Chironomus plumosus*	3.2	20	Delayed emergence	1
Goldfish *Carassius auratus*	0.44-1.8	4	Behavioral disruption	2
Brook trout, *Salvelinus fontinalis*	0.068	161	Reduced reproduction	3
Brook trout	0.288	161	Growth inhibition	4
Fathead minnow, *Pimephales promelas*				
Adult	0.097	30	"	4
Fry	0.054	30	"	4
Channel catfish, *Ictalurus punctatus*				
Adult	0.299	30	"	4
Fry	0.072	15	Backbone abnormalities	4
Largemouth bass, *Micropterus salmoides*				
Larvae	0.2	14	Histopathology of kidney and GI tract	2
Saltwater				
Eastern oyster, *Crassostrea virginica*	100.0	1	Growth inhibition	5
Mysid shrimp, *Mysidopsis bahia*	0.14	28	Reduced reproduction	5
Spot, (teleost) *Leiostomus xanthurus*	0.1	long-term	Histopathology	2

[a]References: 1, Sanders 1980; 2, Pollock and Kilgore 1978; 3, Mayer et al. 1975; 4, Mayer et al. 1977; 5, EPA 1980a.

Table 6. Bioconcentration factors (BCF) for toxaphene and selected species of aquatic biota (modified from EPA 1980a).

Medium, tissue, species, developmental stage	BCF	Exposure duration in days
Freshwater		
Whole body		
Brook trout	10,000	140
Fathead minnow	52,000	98
Channel catfish		
Adults	22,000	100
Fry	40,000	90
Muscle		
Brook trout	3,400	161
Channel catfish	7,800	137
Saltwater		
Whole body		
Eastern oyster	32,800	168
Sheepshead minnow	9,800	28
Longnose killifish, *Fundulus similis*		
Fry	27,900	28
Juvenile	29,400	28
Adult	5,400	32
Egg		
Longnose killifish	1,270	14
Longnose killifish	3,700	52

Table 7. Current recommendations on toxaphene concentrations (EPA 1980a).

Environmental or other factor	Allowable concentration
Freshwater life protection[a]	0.013 µg/1 (24-hour average); 1.6 µg/1 maximum at any time
Saltwater life protection	0.07 µg/1 maximum at any time
Fish tissues	5.0 mg/kg maximum, wet weight basis; 0.4 to 0.6 µg/kg maximum, wet weight basis (Mayer and Mehrle 1977)
Fat of meat from livestock	7.0 mg/kg
Milk and milk products, fat weight basis	0.5 mg/kg
Sunflower seeds	0.1 mg/kg wet weight basis
Citrus fruits	5.0-7.0 mg/kg wet weight basis (Canada); 0.4 mg/kg wet weight (W. Germany, Netherlands)
Drinking water	5.0-8.75 µg/1
Safe daily dose: human	3.4 µg/kg body weight
Acceptable daily intake: human	1.25 µg/kg body weight
Daily intake from air: human	0.00018 µg/kg body weight

[a]The International Joint Commission of the United States and Canada recommended a water standard of 0.008 µg/1 for protection of freshwater aquatic life. This standard is based on the study by Mayer et al. (1975), who found that toxaphene at 0.039 µg/1 in water, caused a significant increase in mortality and a significant decrease in growth of surviving brook trout fry over a 90-day period. The standard of 0.008 µg/1 is obtained by applying an application factor of 5.