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

by
Ronald Eisler

Patuxent Wildlife Research Center
U.S. Fish and Wildlife Service
Laurel, MD 20708

SUMMARY

Carbofuran (2, 3-dihydro-2, 2-dimethyl-7-benzofuranyl methyl carbamate) and other carbamate compounds, together with organophosphorus compounds, have virtually replaced the more persistent and hazardous organochlorine systemic pesticides used in agriculture. In general, carbofuran effectively controls insects through an anticholinesterase mode of action. Compared with chlorinated hydrocarbon insecticides, it has a relatively short residual life in the environment, degrades rapidly, and is almost completely excreted by nontarget organisms. Carbofuran degradation is complex and demonstrably modified by numerous biological and physicochemical factors; little is known of the biological properties of the degradation products, especially nitrosated metabolites, in relation to chronic toxicity, teratogenicity, mutagenicity, or carcinogenicity.

At currently recommended application rates and in present formulations, carbofuran has caused sporadic, and sometimes extensive, field kills of fish, wildlife, and invertebrates. In short-term laboratory tests, significant death rates were observed at concentrations of about 200 ppb carbofuran (in water) for sensitive species of aquatic biota, 238 ppb (acute oral, µg/kg body weight) and 190,000 ppb (dietary, µg/kg diet) for birds, and 2000 ppb (acute oral) and 100,000 ppb (dietary) for mammals. Among representative indicator species, harmful and sometimes life-threatening effects of carbofuran have been recorded for fish at nominal water concentrations of >15 ppb and for aquatic invertebrates at >2.5 ppb. For birds and mammals, harmful effects were observed at 10 to 50 ppb in the diet and 1000 ppb in drinking water. For comparison, the "safe" level of carbofuran in meat products for human consumption is 50 ppb. Current maximum permissible aerosol levels of 0.05 ppb (50.0 $\mu g/m^3$) carbofuran appear sufficient to protect wildlife; however, evidence suggests that aerosol concentrations should never exceed 2 ppb. Plants are significantly more resistant to carbofuran than are invertebrates and higher organisms. Carbofuran hazards to migratory waterfowl may be reduced by prohibiting granular formulations containing more than 3% active ingredients. In rice culture, carbofuran should be applied before the fields are flooded and after the peak of bird migration. Since annual domestic use of carbofuran exceeds 3.2 million kg (7 million pounds), it appears that additional research is merited on the biotic effects of various formulations of carbofuran, especially flowable formulations, and on applications to crops other than rice, such as corn, alfalfa, and hay.

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TABLES

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INTRODUCTION

Carbofuran is a broad spectrum systemic insecticide that is currently registered for use on agricultural crops such as alfalfa (*Medicago sativa*), peanuts (*Arachis hypogaea*), rice (*Oryza sativa*), sugar cane (*Saccharum officinarum*), and especially corn (*Zea mays*) Anon. 1971; Dorough 1973; Palmer and Schlinke 1973; EPA 1976; Finlayson et al. 1979; Flickinger et al. 1980). Carbofuran, together with other carbamate compounds, organophosphorus insecticides, and pyrethroids, are the major substitutes for the more persistent pesticides such as DDT, chlordane, and heptachlor. In 1974, domestic carbofuran use was slightly over 3.2 million kg (7 million pounds) active ingredients, most of which was applied to control corn pests (EPA 1976). As a group, the carbamates, including carbofuran, have controlled insects effectively: their residual life in the environment is relatively short; excretion from the animal body is comparatively rapid and almost quantitative; and the terminal residues produced are polar and formed by chemical processes normally considered as steps in metabolic detoxication.

However, at recommended application rates, which range from 0.28 to 10.9 kg active ingredients/ha (0.25 to 9.7 lb/acre), and in a variety of formulations, carbofuran has been held responsible for sporadic kills of fish, wildlife, beneficial insects, and terrestrial and aquatic invertebrates. For example, among birds that only occasionally consume domestic crops, carbofuran applied to vegetables reportedly killed about 1400 ducks, largely green-winged teal (*Anas carolinensis*), pintail (*A. acuta*), and American wigeon (*A. americana*) in British Columbia between 1973 and 1975 (Flickinger et al. 1980). Carbofuran applied to alfalfa killed 2450 American wigeons at one California location in 1974 (Stickel 1975), 500 Canada geese (*Branta canadensis*) in southern Oklahoma in 1976, 1000 wigeons in Kansas in 1976, and more than 1063 wigeons in California in 1976-1977 (Flickinger et al. 1980). Secondary poisoning of red-shouldered hawks (*Buteo lineatus*) was reported after the application of carbofuran to Maryland cornfields (Balcomb 1983). Aerial application to flooded rice fields in various portions of Texas between 1970 and 1975 at the rate of 0.56 kg/ha resulted in deaths of three species of sandpipers (*Erolia* spp.) and red-winged blackbirds (*Agelaius phoeniceus*), as well as frogs, crayfish, leeches, earthworms, and four species of fish; however, no carbofuran residues were detectable among survivors 2 to 11 days postexposure (Flickinger et al. 1980).

In this account, I summarize the technical literature documenting environmental hazards associated with carbofuran, with special emphasis on game fish and migratory birds, and provide recommendations for protection of sensitive species of fish and wildlife. It is part of a continuing series of synoptic reviews prepared in response to requests for information from environmental specialists of the U.S. Fish and Wildlife Service.

CHEMICAL PROPERTIES AND PERSISTENCE

Carbofuran (2, 3-dihydro-2, 2-dimethyl-7-benzofuranyl methyl carbamate) is also known as Furadan, Bay 70142, CAS 1563-66-2, Curaterr, D-1221, ENT-27164, and FMC 10242 (Johnson and Finley 1980) and Niagara NIA-10242 (Leuck et al. 1968). Carbofuran has a molecular weight of 221.25 and a melting point of 150-152°C; it is comparatively stable under neutral or acidic conditions, but degrades rapidly in alkaline media (Anon. 1971). This white, crystalline, solid of empirical formula $C_{12}H_{l5}NO_3$ is soluble at concentrations up to 700 ppm (mg/l) in water, but at < 30 ppm in various organic solvents. It degrades at >130° and supports combustion if ignited (FMC 1979). The compound is available as a wettable powder, a granular formulation, and in solution as a flowable formulation (Anon. 1971; EPA 1976).

Pharmacologically, carbofuran inhibits cholinesterase, resulting in stimulation of the central, parasympathetic, and somatic motor systems. Acute toxic clinical effects resulting from carbofuran exposure in animals and humans appear to be completely reversible and have been successfully treated with atropine sulfate. However, treatment should occur as soon as possible after exposure because acute carbofuran toxicosis can be fatal; younger age groups of various species are more susceptible than adults (Finlayson et al. 1979). Carbofuran labels indicate that application is forbidden to streams, lakes, or ponds. In addition, manufacturers have stated that carbofuran is poisonous if swallowed, inhaled, or absorbed through the skin; users are cautioned not to breathe carbofuran dust, fumes, or spray mist; and treated areas should be avoided for at least 2 days (Anon. 1971). Three points are emphasized at this juncture. First, some carbofuran degradation products have not been identified. Second, toxicologic, mutagenic, carcinogenic, and teratogenic properties of most carbofuran degradation products have not been satisfactorily evaluated. And third, numerous physical, chemical, and biological vectors modify carbofuran degradation processes, as well as biological uptake, retention, and translocation. Each of these points is developed in greater detail later.

Carbofuran is metabolized by hydroxylation and hydrolysis in plants, insects, and mammals (Metcalf et al. 1968). The primary transformation product in most plants appears to be 3-hydroxycarbofuran. However, levels of 3-hydroxycarbofuran and other degradation products in plants are influenced by numerous factors, including plant age, soil type, pesticide formulation, application method and rate, and weather conditions, as is shown later. Oxidation of unconjugated 3-hydroxycarbofuran yields 3-ketocarbofuran, which is, in turn, rapidly hydrolyzed to the much less toxic 3-ketocarbofuran phenol. Accordingly, 3-ketocarbofuran is not likely to be detected as a terminal residue in plants above trace levels. Residue analyses indicated that carbofuran and 3-hydroxycarbofuran are the compounds that occur most often in plant tissues after treatment (Finlayson et al. 1979). In measurement of carbofuran and its degradation products in corn at 117 and 149 days after carbofuran application (Table 1), the decrease of 62% in the total carbamate residues detected between silage and harvest was attributed to cessation of root uptake, volatilization from drying plant surfaces, and further degradation to phenolic compounds. No losses of carbofuran or 3-hydroxycarbofuran were detected in fortified corn silage after storage at minus 18°C for 1 year (Finlayson et al. 1979).

Variation in content of carbofuran and its degradation products was evident among crop species (Finlayson et al. 1979). Strawberries (*Fragaria vesca*), for example, contained higher residues of phenol than of' either carbamate or hydroxy products. Carbofuran can persist in Mugho pine needles for at least 2 years at insecticidally-active concentrations. This unequal distribution of carbofuran in different parts of a plant has also been observed for tobacco (*Nicotiana tabacum*), in which more of the compound was in large leaves than in the tops of plants, suggesting that carbofuran moved in the plant fluids to the point of greatest transpiration in the leaves.

Carbofuran in animals may also be hydrolyzed to produce carbofuran-7-phenol; hydrolysis of the 3-hydroxyderivitive leads to formation of 3-hydrocarbofuran-7-phenol. Other degradation products include N-hydroxymethyl carbofuran and, as in plants, 3-hydroxy and 3-ketoderivitives. All of these compounds may become conjugated and excreted by animals in urine and, presumably, bile (Metcalf et al. 1968; Finlayson et al. 1979). At least 10 metabolites of carbofuran are known at present; their interrelations are shown in detail by Menzie (1978).

In water, the carbofuran degradation rate is strongly influenced by pH (Chapman and Cole 1982). The time to 50% degradation of carbofuran in water was 3.2 years at pH 4.5, 13.3 years at pH 5.0 and 6.0, 1.9 months at pH 7.0, and only 1 week at pH 8.0. The rate of carbofuran loss is also influenced by sunlight, trace impurities, and temperature, but not as dramatically as by pH (Seiber et al. 1978).

Persistence of carbofuran in soils is a function of many factors, including pesticide formulation, rate and method of application, soil type, pH, rainfall, temperature, moisture content, and microbial populations (Ahmad et al. 1979; Deuel et al. 1979; Finlayson et al. 1979; Fuhremann and Lichtenstein 1980; Gorder et al. 1982). Results of several studies indicate that loss from soil samples also takes place at low temperatures when air drying is used; this loss may present a problem to chemists who are unable to conduct analyses immediately after samples are collected (Finlayson et al. 1979). Soil pH is one of the more extensively documented variables affecting degradation; it may become increasingly important as acidic precipitation (acid rain) increases. Carbofuran decomposes rapidly at pH levels >7.0, but becomes increasingly stable as pH decreases. The hydrolysis half-life is about 16 years at a soil pH of 5.5; the half-lives are about 35, 6, and 0.25 days at pH levels of 7.0, 8.0, and 9.0, respectively (Finlayson et al. 1979). Similar results were reported by Getzin (1973), Caro et al. (1976), Seiber et al. (1978), and, in Table 2, by Chapman and Cole (1982). Temperature and moisture content of soils were both positively related to degradation of carbofuran to 3-hydroxycarbofuran, 3-ketocarbofuran, carbofuran phenol, and 3-ketocarbofuran phenol. In general, an increase in temperature from 15° to 27°C had a greater influence on degradation than did an increase from 27° to 35°C, although 27° to 35° was the range in which maximum degradation rates were observed (Ou et al. 1982). Similar results were recorded by Caro et al. (1976), Seiber et al. (1978), and Gorder et al. (1982).

The role of soil bacteria in carbofuran degradation is unclear. Most investigators agree that carbofuran is hydrolyzed to its phenol, which is immediately bound to soil constituents and then metabolized by microorganisms, either slowly (Getzin 1973; Siddaramappa et al. 1978) or rapidly, especially when associated with a *Pseudomonas* sp. isolate (Felsot et al. 1981). Others believe that carbofuran is degraded primarily by chemical hydrolysis, in which bacterial processes assume a negligible role (Venkateswarlu and Sethunathan 1978; Finlayson et al. 1979). Evidence exists demonstrating that soil microbial populations increased by up to 3

times following application of carbofuran (Mathur et al. 1976, 1980); that prior treatment with carbofuran produced rapid degradation attributed to acclimatized soil bacteria (Felsot et al. 1981); that estuarine bacteria are comparatively resistant to carbofuran (Brown et al. 1975); that sterilized soils did not show evidence of carbofuran degradation (Felsot et al. 1981); and that degradation to carbofuran phenol was most rapid under anaerobic conditions (Venkateswarlu and Sethunathan 1978). It appears that additional research is required on bacterial degradation of carbofuran, with special emphasis on acid-resistant strains.

LETHAL EFFECTS

GENERAL

In acute toxicity tests with aquatic organisms, LC-50 (96h) values, with only one exception, exceeded 130 ppb. The exception was the larva of a marine crab with an LC-50 (96h) value of 2.5 ppb. In tests of longer duration with fish, safe concentrations were estimated to range between 15 and 23 ppb. Among the most sensitive species of birds tested, the acute oral LD-50 was 238 ppb (µg/kg body weight), the dietary carbofuran LD-50 value was 190 ppm, dermal LD-50's exceeded 100 ppm, and the LC-100 value in drinking water was 2 ppm. Mammals were comparatively resistant, having LD-50 acute oral toxicities >2 ppm, a dietary LD-38 of 100 ppm after 8 months, and dermal LD-50's >120 ppm. However, only 2 ppb as an aerosol killed 50% of rhesus monkeys in 6 hours, and 40 ppb killed all pheasants within 5 minutes. Bees and earthworms were relatively sensitive to carbofuran, but test conditions were sufficiently different to preclude a strict comparison with vertebrate species. Among photosynthetic species, concentrations of 200 ppm carbofuran partly inhibited germination of rice seeds, but not other species tested, after exposure for 24 hours. Effects of carbofuran on plants are considered negligible when contrasted to faunal damage effects.

AQUATIC ORGANISMS

Among freshwater organisms, LC-50 values for carbofuran ranged from 130 to 14,000 ppb in tests of 72 to 96 hours; fish were the most sensitive and worms the most resistant (Table 3). A relatively narrow toxic range for carbofuran in the climbing perch (*Anabas testudineus*) was indicated by the LC-0 (120 hour) value of 560 ppb and the LC-100 (24 hour) value of 1560 ppb (Bakthavasalam and Reddy 1981). It is noteworthy that carbofuran was not as toxic to aquatic biota as were various cyclodiene chlorinated hydrocarbon insecticides, almost all of which were subsequently withdrawn from commercial use and replaced by carbofuran and other carbamates, and organophosphorus and other compounds.

In flow-through toxicity tests with the marine sheepshead minnow (*Cyprinodon variegatus*), LC-50 values had stabilized by day 60 of exposure with no significant mortality afterwards; however, the LC-50 value was 386 ppb at 96 hours, or 7.8 times greater than that (49 ppb) at 131 days (Table 3). At concentrations up to 49 ppb, carbofuran did not significantly affect the growth of parent fish nor the number of eggs produced. But mortality of fry from fish exposed to 23 and 49 ppb was measurably greater than that of controls (Parrish et al. 1977). On the basis of these and other observations that indicate that growth of surviving fry in all concentrations was not affected and that carbofuran was degraded rapidly in seawater and in sheepshead minnows, it was concluded that the MATC (maximum allowable toxicant concentration) for carbofuran and sheepshead minnow lies between 15 and 23 ppb (Parrish et al. 1977). This observation is similar to that of Caldwell (1977), who demonstrated that adult Dungeness crabs (*Cancer magister*) showed no deleterious effects on growth, survival, or reproduction during exposure to 25 ppb of carbofuran for 69 days. Larvae of Dungeness crabs were substantially more sensitive than adults in 96-hour tests (Table 3). In addition, Caldwell (1977) indicated that 1.5 ppb of carbofuran inhabited swimming ability in zoeal stages of Dungeness crabs and 1.0 ppb inhibited molting and prevented metamorphosis to more advanced larval stages. These observations require verification because mortality in control groups was high, a typical problem in bioassays with larvae of marine invertebrates.

BIRDS AND MAMMALS

Acute oral toxicities of carbofuran to birds ranged from 238 ppb (µg/kg body weight) for fulvous whistling-ducks (*Dendrocygna bicolor*) to 38,900 ppb for domestic chickens (Table 4). The fulvous whistling-duck has been listed as endangered since 1972 by the Texas Organization for Endangered Species (Flickinger et al. 1980). Concentrations of 1 ppm of carbofuran in drinking water of the ducks caused symptoms of intoxication in 7 days, and 2 ppm was lethal in the same period (Tucker and Crabtree 1970). Acute symptoms of carbofuran poisoning in birds, which may persist for up to 7 days, include a loss in muscular coordination, wings crossed high over the back, head nodding, vocal sounds, salivation, tears, diarrhea, immobility with wings spread,

labored breathing, eye pupil constriction, arching of back, and arching of neck over back; death may occur within 5 minutes after ingestion (Tucker and Crabtree 1970). Among mallards (*Anas platyrhynchos*), sensitivity to carbofuran was greater in ducklings than in older birds (Table 4); this relation appears to hold true for other birds for which data are available. Acute oral toxicities of carbofuran to various species of mammals ranged from 2000 ppb in mice to 34,500 ppb in rats (Table 4). Mammals were generally more resistant than birds to acute biocidal properties of carbofuran.

Carbofuran administered to birds in the diet for 5 days, plus 3 days postexposure on an untreated diet, produced 50% kill values of 140 to 1459 ppm dietary carbofuran; younger birds were more sensitive than older ones (Table 5). Food consumption in groups of Japanese quail (*Coturnix japonica*) with high carbofuran-induced mortality was markedly depressed during the first 3 days of treatment (Hill and Camardese 1982). Red-winged blackbirds, the most sensitive bird species tested in food repellency tests, consumed a normal ration of food contaminated with carbofuran (Schafer et al. 1983). As a result, carbofuran has a high potential for causing acute poisoning episodes in birds (Schafer et al. 1983). Field application of carbofuran granules to corn, at planting, in Maryland during 1980 was presumed to be responsible for deaths of songbirds (order Passeriformes) and white-footed mice (*Peromyscus leucopus*); all organisms contained high levels of carbofuran in the gastrointestinal tract and liver, suggesting extensive feeding in treated fields (Balcomb et al. 1984a). A similar situation occurred in Perry, Florida, after treatment of pine seed orchards (Overgaard et al. 1983). Laboratory studies with house sparrows (*Passer domesticus*) and red-winged blackbirds demonstrated that ingestion of a single carbofuran granule is fatal to either species (Balcomb et al. 1984a,b). In groups of old-field mice (*Peromyscus polionotus*) fed 100 ppm dietary carbofuran for 8 months, mortality was 38%; however, growth, development and behavior was normal among survivors from this group and their offspring (Wolfe and Esher 1980). In a preliminary study with rats and old-field mice fed 100 ppm of dietary carbofuran, parents lost weight (but none died), and the survival of young was reduced (Wolfe and Esher 1980).

Aerosol toxicity of carbofuran to warm-blooded animals ranged from about 2 ppb for rhesus monkeys to 110 ppb for rats (Table 6). These values substantially exceed the established Threshold Limit Value (TLV) of 0.05 ppb (50.0 ug/m^3) for protection of human health (Draper et al. 1981). The TLV is a time-weighted concentration for a 40-hour work week that nearly all workers can withstand without adverse effects, including eye and skin irritations and other minor irritations. Inhalation doses to humans were estimated during and immediately after aerial spraying of Furadan 4-Flowable at the rate of 446 g active ingredients carbofuran/ha, a concentration that generally controls most pests. During aerial sprayings of this level, the concentration of carbofuran in ambient air did not exceed 0.0033 ppb at any location (Draper et al. 1981), suggesting that most birds and wildlife are afforded a high degree of protection during aerial spraying at current recommended dosages. Studies with rats subjected to 1.2 ppb carbofuran aerosols for 50-70 minutes showed a substantial (55%) decrease in red blood cell cholinesterase 10 minutes posttreatment and a return to normal levels in 2 hours (Ferguson et al. 1982). After 8 hours, a maximum of 55% of the carbofuran was excreted by respiration (38%) or in the urine (12%) or feces (5%); the remainder was located primarily in the liver and gastrointestinal tract. Plasma half-lives in rats for carbofuran (36 minutes) and 3-hydroxycarbofuran (62 minutes) were similar to those previously determined after oral and intravenous exposures (Ferguson et al. 1982).

Dermal toxicity of carbofuran to birds and mammals is comparatively low. The LD-50 dermal values ranged from about 1000 ppm in cattle (Palmer et al. 1981) down to 100 ppm in birds; i.e., house sparrows and queleas (Schafer et al. 1983); rats and rabbits were intermediate in sensitivity at 120 and 885 ppm, respectively (Draper et al. 1981). Birds contaminated by carbofuran spray could possibly ingest significant amounts while preening (Finlayson et al. 1979), but such ingestion has not been demonstrated. For humans, the maximum potential dermal exposure based on exposed face, hands, forearms, back and front of the neck, and "V" of the chest is 3.1 mg (Draper et al. 1981) or 0.04 ppm for a person weighing 70 kg. This relationship suggests that human populations would be at greater risk than wildlife populations under current carbofuran spray application protocols.

Secondary poisoning of avian raptors with carbofuran is a recent and disturbing event (Balcomb 1983). Consider the case of a female red-shouldered hawk in adult plumage weighing 683 g, found in a cornfield near Beltsville, Maryland, in May, 1981. The field had been treated the previous day with Furadan 10 granules (10% carbofuran), applied at 1.12 kg/ha active ingredients. The bird was entirely paralyzed except for some head and neck movement, salivating a brown fluid, and respiring in rapid pants. These signs are consistent with those

observed in birds dosed in the laboratory with carbofuran. Stomach contents contained remains of a northern short-tailed shrew (*Blarina brevicauda*) and a common grackle (*Quiscalus quiscula*). A total of 96.6 ug of carbofuran was extracted from the gastrointestinal tract and stomach contents and tissues. Judging by the body weight of the hawk, and an LD-50 range of 0.26 to 5.6 mg/kg in various nondomesticated birds (Table 4), this amount of carbofuran would constitute between 2.5 and 59% of the known LD-50 values; however, carbofuran in birds is readily absorbed from the gut and widely transported in the body. Accordingly, the amount of toxicant extracted from the digestive tract was probably only a portion of that ingested by the hawk. In the same cornfield, at the same time, a smaller adult red-shouldered hawk (possibly the female's mate) was found that showed similar, but less severe, signs. Within 24 hours, it appeared to have recovered completely and was released. As judged by carbofuran residues in small mammals and birds at this site, the residues present in the digestive tract of the female hawk, and the nature of the toxic symptoms observed, the two red-shouldered hawks were probably poisoned by carbofuran acquired from small vertebrate prey or scavenged from the treated areas (Balcomb 1983).

TERRESTRIAL INVERTEBRATES

At recommended field application rates of granular carbofuran formulations, some losses of earthworms, springtails, and other soil-inhabiting organisms should be expected; spray and dust formulations adversely affect honeybees and other airborne crop pollinators (Finlayson et al. 1979). Bees are extremely susceptible to carbofuran. In one study with honeybees (*Apis* spp.), subjected to high levels of carbofuran, some young adults in the contaminated hive were unable to emerge from their cells, and those that did emerge remained weak and unfed. Eventually, the hive became vulnerable to invasion by the greater wax moth (*Galleria melonella*), an insect that subsequently destroyed the entire hive (Keener and Pless 1974). The LD-50 dose for honeybees was estimated at 0.16 ug/bee. If 1.12 kg carbofuran/ha were uniformly distributed at a height of 10 m, flying bees could encounter a lethal dose in only 2 seconds (Atkins et al. 1976).

Studies with susceptible and selectively bred carbofuran-resistant houseflies (*Musca domestica*) indicated that LD-50 values for susceptible and resistant strains were 0.1 and 1.3 ug/insect, respectively (Dorough 1973). Resistant flies contained up to 34% more cholinesterase than susceptible strains and could excrete carbofuran almost twice as fast (Dorough 1973). Carbofuran resistance among pestiferous insects is not yet widely known or adequately documented.

Among earthworms, the characteristic symptoms of carbofuran poisoning were rigidity, immobility, lesions, and segmental swelling, as well as cholinesterase inhibition (Stenerson et al. 1973). Worms maintained in soils to which commercial applications of carbofuran had been applied developed two types of lesions within 72 hours: multisegmental swelling that often ulcerated, causing death of the worm, and a discrete nodular mass protruding from the surface of the worm (Sileo and Gilman 1975). The LC-50 values were 0.5 ppm at 5 hours for *Lumbricus herculeus* (Lebrun et al. 1981), but 2.4 and 13.0 ppm at 5 days for *Lumbricus terrestris* and *Eisenia foetida*, respectively; the differences in sensitivity were attributed to a greater excretion rate of carbofuran by Eisenia (Gilman and Vardanis 1974). When applications of carbofuran to soils was 9.1 kg/ha, 50% of the *Lumbricus* died in 72 hours (Ruppel and Laughlin. 1977). At lower application rates of 2 kg/ha, populations of two species of Australian earthworms were reduced; juvenile stages were most severely affected (Martin 1980). The loss of earthworms could result in reduced food for many wildlife species.

Finlayson et al. (1979) indicated that some species of birds might absorb a lethal dose of carbofuran through foraging on poisoned invertebrates. For example, the woodcock (*Philohela minor*) which preys intensively on earthworms, may consume up to 50% of its body weight (about 125 g of food) per day. If each worm contained 1.3 ppm carbofuran, a woodcock would then ingest 0.16 mg of carbofuran or the equivalent of 0.65 mg/kg body weight (Finlayson et al. 1979), an oral dose lethal to many bird species. To date, secondary poisoning of woodcocks has not been verified under controlled conditions.

PLANTS

Carbofuran was more toxic to blue-green alga (*Nostoc muscorum*) at pH 5 to 6 than at pH 7.5 to 10; toxicity was lessened under conditions of reduced illumination and low population density (Kar and Singh 1978). All effects were observed at comparatively high carbofuran concentrations of 25 to 100 PPM.

Seeds of okra (*Abelmoschus esculentus*) treated with carbofuran, at 1, 3, or 5% active ingredient carbofuran by weight of seed, germinated normally after 90 days of storage (Gaikwad and Pawar 1979). After 6 months of storage, however, germination was measurably reduced at all carbofuran treatments. Okra plants developed normally except for a reduction in plumule length, but this effect was also observed among okra seeds tested with a wide variety of agricultural chemicals.

The effect of carbofuran on the germination of seeds of cotton (*Gossypium hirsutum*), rice, and groundnut (*Arachis hypogea*) were investigated by Arunachalam and Lakshmanan (1982), in rice seeds exposed for 24 hours to 100 or 200 ppm carbofuran, germination decreased 8 and 23%, respectively; seeds of the other two species were not affected at these exposure rates. Treated rice seedlings that germinated grew two to three times faster than controls, especially in the roots and leaves; no reasons were offered to account for these differences in rice plants. Carbofuran residues in seeds of the three test species exposed for 24 hours to 100 ppm carbofuran ranged from 17.5 to 28.1 ppm; at 200 ppm carbofuran these values ranged from 24.0 to 30.4 ppm. At 72 hours posttreatment, residues had declined markedly to 0.1 to 4.7 ppm in the groups treated with 100 ppm carbofuran and 2.3 to 3.8 ppm in the groups treated with 200 ppm. Observed growth promotion effects in certain plants by carbofuran and some of its metabolites may be due to effects on plant oxidase systems, rather than on insecticidal or nematocidal properties of the compound; however, the source of the effects has not been demonstrated conclusively (Finlayson et al. 1979).

SUBLETHAL EFFECTS

GENERAL

Most investigators agree that carbofuran degrades or is biotransformed rapidly, with negligible accumulations in biota. Numerous studies have demonstrated that carbofuran, at high sublethal concentrations, was capable of disrupting enzyme and lipid metabolism, but that effects were reversible with no observable permanent damage. Three major data gaps appear still to exist. First, latent biochemical and physiological effects that appear at substantial intervals posttreatment have not been explained. Second, interaction of carbofuran with other environmental compounds, especially other agricultural chemicals, are largely unknown, and the effects may cause more than additive damage. Third, and most important, data are scarce or lacking on chronic toxicity, teratogenicity, mutagenicity, and carcinogenicity of the degradation products of carbofuran, especially degradation products that may also form nitroso compounds; nitrosated carbofuran metabolites, for example, are demonstrably mutagenic.

AQUATIC ORGANISMS

Carbofuran reportedly disrupts enzyme and lipid metabolism in fishes and may not degrade as rapidly under field conditions as suggested by laboratory studies. However, most investigators argue that carbofuran, under current application rates, does not accumulate to a significant extent in aquatic systems and rapidly degrades under field and model microcosm study conditions.

In studies with the African catfish (*Mystus vittatus*) exposed to 31 or 62 ppb of carbofuran for 30 days, serum transaminases were significantly elevated (Verma et al. 1981a). In comparison with catfish exposed to concentrations of 21 ppb or less during the same period, there were also significant depressions in alkaline phosphatase activity in the liver; acid phosphatase activity in the liver, kidneys, and gills; and gulucose-6-phosphatase in the liver and kidneys (Verma et al. 1981b). In climbing perch, mean lipid levels in muscle and liver were elevated after exposure to an LC-0 (120 hour) dose of 560 ppb carbofuran for 120 hours; a similar pattern was observed following exposure to an LC-100 (24 hour) concentration of 1560 ppb for 6 hours (Bakthavasalam and Reddy 1981). Carbofuran-induced alterations have also been documented in serum chemistry of the African catfish during immersion in 21 ppb for 30 days (Verma et al. 1982b); in brain acetylcholinesterase activity of climbing perch and milkfish (*Channa punctatus*) 30 days after exposure to high sublethal levels for 48 hours (Jash and Bhattacharya 1983); and in blood and tissue enzyme and ammonia levels in the air-breathing catfish (*Clarias batrachus*) 1 month after exposure for 30 days to 500 ppb carbofuran (Mukhopadhyay et al. 1982). In field studies with *Trichogaster pectoralis*, a fish extensively cultured in flooded Malaysian rice paddies, (Gill (1980) found that the degradation of carbofuran in the liver was slower than that reported for laboratory animals and suggested that caution be exercised in the extrapolation of rates of carbofuran oxidative hydroxylation activity from laboratory organisms to fishes cultured in rice fields.

On the other hand, negligible accumulations of carbofuran were observed in egg masses of the caddisfly (*Triaenodus tardus*) during immersion for 120 hours in water containing 8 ppb of carbofuran . ; the low uptake was apparently related to the low partition coefficient of carbofuran (Bellick and Fels 1981). Rapid equilibrium and low accumulation was also reported for the sheepshead minnow (*Cyprinodon variegatus*); in a 28-day flow-through study, maximum tissue concentrations were measured between days 3 to 10 when upper concentration factors of 5 to 20X were recorded (Parrish et al. 1977). Field applications of carbofuran in farm ponds in Arkansas and Kansas were associated with low. mortality in fish (Davey et al. 1976) or negligible effects on fish and plankton (Klaassen and Kadoum 1979). Kansas farm ponds subjected to 25 ppb of carbofuran contained 10.6 ppb in surface waters 1 day later, but nondetectable residues thereafter; residues were <0.4 ppb at 1 day in mud, zooplankton, and fish (Klaassen and Kadoum 1979). Farm ponds treated with 50 ppb of carbofuran after 3 days contained 15 ppb carbofuran in surface water and 26 to 46 ppb in mud, but nondetectable residues in biota; no measurable residues were found in any sample after 25 days (Klaassen and Kadoum 1979). When atrazine at 300 ppb was applied in combination with 50 ppb carbofuran, carbofuran was detectable in surface water at 23 days posttreatment at 1.5 ppb, but not in the soil, biota, or any other compartment (Klaassen and Kadoum 1979).

Koeppe and Lichtenstein (1982), in a well-designed agromicrocosm study, evaluated the influence of percolating water on soils containing 3.6 ppm of radiolabeled (C-14) carbofuran. After 3 weeks, 49% of the carbofuran had been removed with percolating water from soils, and 37% was later recovered from soils and corn. In nonpercolated soils, 80% of the carbofuran was still associated with soils and corn. The aquatic components, including water, lake mud, plants (*Elodea*), and fish (the guppy *Poecilia*), contained 25% of the soil-applied carbofuran, although 49% had been- initially added to the aquariums by way of percolated water. This loss of 24% was attributed partly to the degradation of carbofuran to $C0_2$. About 75% of all the radiocarbon was in lake mud, most of it unextractable. Carbofuran was the major compound recovered from control and percolated soils, accounting for 39 and 15%, respectively; 3-ketocarbofuran and 3-hydroxycarbofuran were identified as the major metabolites. The addition of captafol, a fungicide, to carbofuran-treated soils resulted in a more rapid disappearance of the insecticide from terrestrial soils and reduced uptake by corn. The addition of EPTC, a herbicide, had no measurable effect on terrestrial components, but EPTC and captafol both caused increased recoveries of C-14 labeled carbofuran residues from lake bottom mud. In another study, radiolabeled carbofuran was applied at 1.12 kg/ha to a model ecosystem containing seedling sorghum plants (*Sorghum halopense*) saltmarsh caterpillar larvae (*Estigmene acrea*), the alga *Oedogonium cardiacum*, freshwater clams (*Corbicula manilensis*), crabs (*Uca minax*), a cladoceran (*Daphnia* sp.), mosquito larvae, unidentified species of frogs and snails, and the freshwater macrophyte *Elodea canadensis* (Yu et al. 1979). Carbofuran was rapidly, but not completely, degraded in water to carbofuranphenol, 3-ketocarbofuran, 3-hydroxycarbofuranphenol, N - hydroxy - methyl carbofuran, 3-hydroxycarbofuran, and several unknown compounds. Carbofuran was highly toxic to crabs, clams, and *Daphnia* immediately after application to the model ecosystem, but all animals, except one crab, survived restocking 20 days later.

The freshwater bivalve molluscs *Glebula rotundata* and *Rangia cuneata* absorbed waterborne carbofuran but did not appear to concentrate it (Zakour 1980). Both species of clams were very tolerant, even though symptoms of poisoning, such as shell gaping, foot extension, and incoordination, were evident when carbofuran exposures were high (75 ppm). *Glebula* converted injected radiolabeled carbofuran to a variety of free metabolites, primarily hydrolysis products, and also polar carbofuran metabolites that were not degraded by conditions known to hydrolyze glycosidic conjugates. These polar metabolites may contain some type of amino acid moiety. The rate of carbofuran metabolism by *Glebula* was slower than that reported for most other animals, but was more rapid than that of plants and microorganisms (Table 7). Bacterial metabolism of carbofuran was negligible in both *in vivo* and *in vitro* studies with bivalve molluscs (Zakour 1980).

BIRDS

Birds may encounter carbofuran through respiratory, dermal, and oral routes. Depending on the dietary requirements of particular species, ingestion of contaminated vegetables and poisoned invertebrates may be important exposure routes (Finlayson et al. 1979). Carbofuran may prove harmful alone or in combination with other substances. For example, male Japanese quail fed 0.5 ppm dietary carbofuran for 18 weeks exhibited a 79% inhibition of plasma cholinesterase activity (Dieter and Ludke 1978). The reduction was slightly greater (84%) when carbofuran was fed in combination with 0.05 ppm dietary morsodren, a methyl mercury compound, although morsodren had no measurable effect on cholinesterase activity when fed alone at that dosage. Since

many species of fish-eating birds frequently contain 0.05 ppm of mercury in various tissues, interaction effects of mercury with carbofuran and other cholinesterase-inhibiting compounds may produce synergistic, deleterious effects (Dieter and Ludke 1978).

Low oral dosages or high dietary levels of carbofuran produced no permanent damage effects in northern bobwhites. A single oral dose of 2 mg carbofuran/kg body weight did not affect brain cholinesterase levels at 48 hours, or growth, metabolic efficiency, or metabolized energy at 8 days (Solomon and Robel 1980). The activities of bobwhites fed 131 ppm dietary carbofuran for 14 days was reduced, but this effect was temporary and recovery was complete within 14 days on a carbofuran-free diet. The temporarily reduced activity was attributed to the rapid metabolic breakdown of carbofuran (Robel et al. 1983).

Among laying white leghorn hens, 80% of a single oral dose of 2.7 mg carbofuran/kg body weight was eliminated in feces within 10 days (Hicks et al. 1970). All eggs contained detectable carbofuran; the highest concentration of 0.13 ppm developed on day 4. Residues in liver and kidney were about 2.6 ppm at 6 hours but declined to 0.2 ppm in 24 hours. Muscle and fat contained about 0.3 ppm at 6 hours and <0.l ppm at 24 hours. Hicks et al. (1970) indicated that hydroxylation of carbofuran and hydrolysis of the carbamate ester were the predominant pathways in the metabolism of carbofuran by laying hens; similar results were obtained at single oral doses of 2.7 or 0.3 mg carbofuran/kg body weight.

MAMMALS

Among larger mammals, carbofuran is associated with a variety of stress symptoms, including increased salivation, muscle tremors, prostration, labored breathing, loss of appetite and (in rare cases) death. These symptoms were observed in 1- to 2-week old calves given single doses of carbofuran at 0.25-5.0 mg/kg body weight orally or 0.05-0.1% dermally, in cattle yearlings at 1.0-5.0 mg/kg orally or 0.1% dermally, and in sheep at 2.5-5.0 mg/kg orally (Palmer and Schlinke 1973). All survivors had completely recovered at 5 days posttreatment. Lactating cows fed corn silage containing 1.4-3.9 ppm carbofuran for 8 weeks, or about 74 mg carbofuran daily, showed no decrease in blood cholinesterase; furthermore, no carbofuran residues were detected in the milk (Leuck et al. 1968). Other studies with lactating cows dosed orally with carbofuran (Dorough and Ivie 1968; Ivie and Dorough 1968) showed almost complete excretion in 10 days, mostly through urine (94%), feces (0.7%) and milk (0.2%). Carbofuran metabolites in urine, feces, and excreted milk included the 3-hydroxy-, 3-keto-, and 3-hydroxy-N-hydroxymethyl derivatives, both conjugated and free, and unknown constituents, perhaps carbon dioxide formed by carbofuran hydrolysis.

In investigations of the effects of carbofuran or its metabolites on mice and rats, pregnant mice receiving 0.01 or 0.5 mg dietary carbofuran/kg daily throughout gestation gave birth to viable, overtly normal offspring at term (Barnett et al. 1980). Significant elevation of serum immunoglobins was measured in 101-day old male offspring of female parents receiving 0.5 mg/kg dietary carbofuran. This effect was not observed at day 400 or 800. In female offspring from the group receiving 0.01 mg/kg carbofuran, serum immunoglobins were significantly depressed at day 101, but not thereafter (Barnett et al. 1980). Disturbances in immunoglobulin contents may decrease immunocompetence and, thus, indirectly contribute to morbidity and premature mortality. In rats fed comparatively high dietary levels of 30 ppm carbofuran for 90 days, with mean daily intake of 1.97 mg carbofuran/kg body weight, growth was significantly reduced and ventral prostate gland metabolism of RNA, DNA, and protein was altered (Shain et al. 1977). Prenatal exposure of mice to 0.01 mg carbofuran/kg body weight daily, administered orally during gestation, resulted in persistent postnatal endocrine dysfunction in adults; specifically, the impairment of hepatic metabolism and elevation of plasma corticosterone (Crammer et al. 1978). Unexpectedly, however, at a higher dose of 0.05 mg/kg, there were no significant differences from controls, and the endocrine function of tested mice was normal. In female rats given a single dose of 0.05 mg carbofuran per kg body weight orally on the 18th day of gestation, acetylcholinesterase (AchE) activity decreased significantly in maternal and fetal blood and in the maternal liver within 1 hour (Cambon et al. 1979). At higher dosages of 0.25 and 2.5 mg/kg, AchE was also depressed in the fetal liver and in the maternal and fetal brains; the effects were not measurable 24 hours postadministration.

Carbamate pesticides can easily be converted to N-nitroso derivatives in the presence of sodium nitrite under acidic conditions. The N-nitroso form of carbofuran could possibly be formed in the human stomach (Nelson et al. 1981). Since carbofuran is used routinely on a variety of crops and nitrite is a common component of the human diet and is present in human saliva, nitrosation of carbamates under conditions simulating those in the human stomach is possible. Lijinsky and Schmal (1978) tested nitrosocarbofuran and

five other nitrosated carbamate pesticides for carcinogenicity in rats. Nitrosocarbofuran, at 16.5 mg/kg body weight administered orally once weekly for 23 weeks, was the most toxic compound tested and caused the death of several animals by liver damage early in the experiment. Among survivors, nitrosocarbofuran was the most carcinogenic, as judged by the numbers of carcinomas and tumors that developed. Nitrosation rates of carbofuran in the environment are not now adequately documented, but conceivably could represent an environmental risk to wildlife. Surprisingly, nitrosocarbofuran was among the least mutagenic compounds tested in rats; no obvious explanation is available of the differences in carcinogenic and mutagenic properties (Lijinsky and Schmal 1978). It is noteworthy that data on chronic toxicity, teratogenicity, mutagenicity, and carcinogenicity of degradation products of carbofuran, especially carbofuran-7-phenol, and 3-hydroxycarbofuran-7-phenol are either scarce or lacking (Finlayson et al. 1979); a similar case is made for nitrosocarbofuran and other degradation products of carbofuran which may also form nitroso compounds. More recent work (Nelson et al. 1981) indicated that nitrosated 3-hydroxycarbofuran and 3-ketocarbofuran produced mutagenic responses in bacterial strains of *Salmonella typhimurium* and chromosome aberrations in ovary cells of Chinese hamsters. Nitrosocarbofuran and 3-hydroxynitrosocarbofuran also induced large numbers of sister chromatid exchanges in the same cells. Furthermore, nitroso derivatives of carbofuran were considerably more active than nitroso forms of other carbamate pesticides in producing mutagenicity in *Salmonella* (Nelson et al. 1981). On the other hand, technical formulations of the parent carbofuran were neither genotoxic nor mutagenic to bacteria, yeast, or corn (Gentile et al. 1982).

TERRESTRIAL INVERTEBRATES

In decomposing the dead organic matter in a deciduous forest ecosystem, the detritus food chain may account for more than half the energy flowing through the ecosystem. Carbofuran can significantly disturb decomposition rates of litter communities, with profound consequences for nutrient recycling and incorporation of organic matter into the soils. For example, application of 0.29 kg/ha of carbofuran to a red maple (*Acer rubrum*) litter community near Ottawa, Canada, reduced daily decomposition rates by about 40%; all of the groups of macrodecomposers present, including Collembola, Acarina, Lepidoptera, Coleoptera, Diplopoda, and Annelida, have been shown to be susceptible to carbofuran and may have been affected by the treatment (Weary and Merriam 1978).

CURRENT RECOMMENDATIONS

In Canada, for regulatory purposes, the tolerance level for carbofuran in animal tissues or food, feed, and fiber crops is based on the total carbamate content of the sample, as indicated by total carbofuran, 3-hydroxycarbofuran, 3-ketocarbofuran, and their conjugates (Finlayson et al. 1979), presumably carbofuran phenol, 3-ketocarbofuran phenol , and 3-hydroxycarbofuran phenol. In the United States, the tolerance level is based on carbofuran and four metabolites: 3-hydroxycarbofuran; carbofuran phenol; 3-hydroxycarbofuran phenol; and 3-ketocarbofuran phenol (EPA 1976). Carbofuran levels considered safe range from 0.05 ppm (including 0.02 ppm carbofuran metabolites) in meat, fat, and meat by-products to 40.0 ppm (including 20.0 ppm carbofuran metabolites) in alfalfa hay; intermediate values are 0.1 ppm in milk, 0.2 ppm in corn grain, and 25.0 ppm in corn fodder and forage (EPA 1976). No recommended carbofuran level is currently being promulgated by any regulatory agency for the protection of sensitive species of aquatic biota and wildlife.

On the basis of evidence presented herein, I conservatively estimate that, in terms of total carbofuran in water, damage is possible to aquatic invertebrates at >2.5 ppb and to teleosts at >15 ppb. These levels could he attained during a heavy rainfall shortly after carbofuran treatment of adjacent fields. Among sensitive species of warm-blooded animals, dietary concentrations as low as 10 ppb have demonstrable effects, which were measurable only after extended periods postingestion; for comparison, this level is about 1/5 that allowed in meat by-products for human consumption. Current maximum permissible aerosol levels of 0.05 ppb (50 ug/m^3) appear sufficient to protect wildlife with the proviso that concentrations not exceed 2.0 ppb at any time.

Sporadic kills of migratory birds were associated with carbofuran formulations containing 3% active ingredients (a.i.). For example, migratory sandpipers died after eating Furadan 3 G granules (3% a.i.) applied to rice crops in Texas (Flickinger et al. 1980). The granules probably were ingested while the sandpipers were probing and skimming the surface of wet soil for insects and crustaceans. Other species of migratory waterfowl may have mistaken the small size and density of Furadan granules for seed, particularly in areas where concentrations of granules were abundant after misuse and careless applications. It appears that granular carbofuran formulations need to be developed that contain less than 3% a.i. in order to protect waterfowl, yet

still maintain their effectiveness against target organisms. In rice field pest control, carbofuran should be applied before the fields are flooded and delayed to avoid peak bird migration. Research also appears warranted on the effects on fish and wildlife of the numerous carbofuran formulations used, especially liquid spray formulations (flowables), and on applications to crops other than rice, such as corn, alfalfa, and hay.

Additional long-term research is urgently needed on potential impacts of degradation products of carbofuran on sensitive species of aquatic organisms and wildlife, with special attention to nitrosated carbofuran metabolites. Such data are now scarce or lacking. Research is also needed on chemical and biological interactions of carbofuran with other agricultural chemicals applied to the same locations, which are imperfectly understood. Finally, researchers must elucidate the significance of metabolic upset recorded in various species of laboratory mammals at considerable periods after carbofuran insult.

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Table 1. Carbofuran and its degradation products, in parts per million dry weight, in corn (*Zea mays*) at silage stage (117 days) and at harvest (149 days) following application of carbofuran (10%) granules at 5.41 kg/ha (after Turner and Caro 1973).

	Carbamates			
Plant stage and part	Carbofuran	3-ketocarbofuran	3-hydroxy-carbofuran	Total carbamates
Silage				
Leaves	0.43	0.40	4.67	5.50
Stalks	0.24	0.00	0.04	0.28
Cobs	0.04	<0.02	<0.02	0.05
Kernels	0.00	<0.01	0.00	<0.01
Harvest				
Leaves	0.21	0.34	1.51	2.06
Stalks	0.03	0.00	0.05	0.08
Cobs	0.06	0.00	0.00	0.06
Kernels	<0.01	<0.01	0.00	<0.01

Table 2. Effect of pH, soil type, and application rate on carbofuran degradation in soils (from Chapman and Cole 1982).

Soil type	pH	Initial application rate of carbofuran (ppm)	Carbofuran remaining after 3 weeks (%)
Alumina soils			
Acid	5.4-6.1	1	76
Acid	5.4-6.1	20	82
Neutral	6.9-7.1	1	85
Neutral	6.9-7.1	20	79
Basic	8.3-8.5	1	55
Basic	8.3-8.5	20	72
Natural soils			
Mineral	8.0	1	95
Mineral	8.0	20	92
Mineral	6.8	1	100
Mineral	6.8	20	100
Organic	6.1	1	58
Organic	6.1	20	73
Organic	5.2	1	47
Organic	5.2	20	73
Sandy	6.6	1	8[a]
Sandy	8.0	1	28[a]

[a]Carbofuran remaining after 8 weeks (rather than 3 weeks as indicated in boxheading).

Table 3. Acute toxicities of carbofuran to aquatic organisms. Concentrations shown are in micrograms of carbofuran per liter of medium (ppb) fatal to 50% of test organisms in the designated time period.

Type of water and species tested	Time (h)	LC-50 (ppb)	Reference[a]
Freshwater			
Yellow perch, *Perca flavescens*	96	147	1
Green sunfish, *Lepomis cyanellus*	72	160	2
Lake trout, *Salvelinus namaycush*	96	164	1
Bluegill, *Lepomis macrochirus*	96	240	1
Channel catfish, *Ictalurus punctatus*	96	248	1
Static test, tapwater	96	1,420	3
Partial media replacement	96	510	3
Rice paddy water[b]			
With history	96	130	3
With no prior history	96	370	3
African catfish, *Mystus vittatus*	96	310	4
Rainbow trout, *Salmo gairdneri*	96	380	1
Crayfish, *Procambarus acutus acutus*	96	500	5
Mosquitofish, *Gambusia affinis*	72	520	6
Coho salmon, *Oncorhynchus kisutch*	96	530	1
Indian carp, *Saccobranchus fossilis*	96	547	7
Brown trout, *Salmo trutta*	96	560	1
Fathead minnow, *Pimephales promelas*	96	872	1
Annelid worm, *Limnodrilus hoffmeisteri*	96	11,000	8
Annelid worm, *Tubifex tubifex*	96	14,000	8
Marine			
Dungeness crab, *Cancer magister*			
Larva	96	2.5	9
Adult	96	190	9
Sheepshead minnow			
Cyprinodon variegatus	96	386	10
C. variegatus	3,144	49	10
Bivalve molluscs			
Cockle, *Clinocardium nuttali*	96	3,750	11
Clam, *Macoma nasuta*	96	17,000	11
Mussel, *Mytilus edulis*	96	22,000	11
Clam, *Rangia cuneata*	96	125,000	11

[a]References: 1, Johnson and Finley 1980; 2, Brungs et al. 1978; 3, Brown et al. 1979; 4, Verma et al. 1980; 5, Cheah et al. 1980; 6, Davey et al. 1976; 7, Verma et al. 1982a; 8, Dad et al. 1982; 9, Caldwell 1977; 10, Parrish et al. 1977; 11, Zakour 1980.

[b]Rice paddy water from rice paddies with and without a history of pesticide application, as shown.

Table 4. Acute oral toxicities of carbofuran to birds and mammals. Concentrations shown are in micrograms carbofuran administered per kilogram body weight (ppb) in a single dose fatal to 50% within 14 days.

Taxonomic groupand species tested	LD-50 (ppb)	Reference[a.]
Birds		
Fulvous whistling-duck,		
Dendrocygna bicolor	238	1
Mallard, *Anas platyrhynchos*		
Age 36 h	280-480	2
Age 7 days	530-740	2
Age 30 days	410-640	2
Age 3-4 months	320-500	1
Age 6 months	330-520	2
Red-winged blackbirds,		
Agelaius phoeniceus	422	3
Quelea, *Quelea quelea*	422-562	3
House finch,		
Carpodacus mexicanus	750	3
Japanese quail,		
Coturnix japonica	1,300-2,100	4
House sparrow,		
Passer domesticus	1,330	3
Common grackle,		
Quiscalus quiscula	1,300-3,160	3
Rock dove,		
Columba livia	1,330	3
Brown-headed cowbird,		
Molothrus ater	1,330	3
Ring-necked pheasant,		
Phasianus colchicus	2,380-7,220	1
Northern bobwhite,		
Colinus virginianus	3,640-6,990	1
European starling,		
Sturnus vulgaris	5,620	3
Domestic chicken,		
Gallus gallus	25,00-38,900	5
Mammals		
Mouse, *Mus musculus*	2,000	4
Cat, *Felis domesticus*	2,500-3,500	5
Rat, *Rattus* sp.	3,800-34,500	5

Old-field mouse,		
Peromyscus polionotus	4,000	6
Beagle dog,		
Canis familiaris	7,500-18,900	5
Sheep, *Ovis aries*	8,000	7
Guinea pig,		
Cavia cobaya	9,200	5

[a]References: 1, Tucker and Crabtree 1970; 2, Hudson et al. 1972; 3, Schafer et al. 1983; 4, Sherman and Ross 1969; 5, Finlayson et al. 1979; 6, Wolfe and Esher 1980; 7, Palmer et al. 1973.

Table 5. Toxicity of dietary carbofuran to birds and mammals.

Organism	Concentration[a]	Exposure interval (days)		Mortality (%)	Reference[b]
		Exposure	Postexposure		
Mallard					
Anas platyrhynchos	190	5	3	50	1
Ring-necked pheasant,					
Phasianus colchicus	573	5	3	50	1
Japanese quail,					
Coturnix japonica					
Age 1 day	140-471	5	3	50	2
Age 7 days	436-1,103	5	3	50	2
Age 14 days	586-1,004	5	3	50	2
Age 21 days	779-1,459	5	3	50	2
Old-field mouse,					
Peromyscus polionotus	500	4		100	3
Old-field mouse	250	4		20	3
Old-field mouse	100	240		38	3

[a]Concentration of carbofuran in diet, in mg/kg (ppm) fresh weight.

[b]References: 1, Hill et al. 1975; 2, Hill and Camardese 1983; 3, Wolfe and Esher 1980.

Table 6. Acute aerosol inhalation toxicity of carbofuran to warm-blooded animals.

Organism	Exposure time (min)	Concentration (ppb)	Effect	Reference[a]
Rhesus mondey, *Macaca* sp.	360	2	LC-50	1
Guinea pig, *Cavia* sp.	240	10-70	LC-50	1
Rat, *Rattus* sp.	50-70	26	LC-50	2
Pheasant, *Phasianus* sp.	5[b]	40	LC-100	1
Guinea pig	—	43-53	LC-50	3
Dog, *Canis* sp.	60	50	LC-50	1
Pheasant	5[c]	80	LC-10	1
Rat	60	90-100	LC-50	1
Human	2,400[d]	0.05	[d]	4

[a]References: 1, Finlayson et al. 1979; 2, Ferguson et al. 1982; 3, Anon. 1971; 4, Draper et al. 1981.
[b]Air delivery rate of 8 1/min.
[c]Air delivery rate of 10 1/min.
[d]Exposure = 40 h work week; threshold limit value.

Table 7. Rates of carbofuran metabolism by various organisms (from Zakour 1980).

Organism	Parent carbofuran converted (%)	Time (d = day; h = hour)
Rat, *Rattus* sp.	100	24 h
Chicken, *Gallus* sp.	100	24 h
Saltmarsh caterpillar, *Estigmene* sp.	100	7 h
Intact cotton plant, *Gossypium* sp.	100	5 d
Alfalfa, roots, *Medicago* sp.	95	30 d
Land snail, *Helix aspersa*	90	1 h
Bean plants, *Phaseolus* sp.	68	3 d
House fly, *Musca* sp.	58	1 h
Bivalve, *Glebula rotundata*	54-62	24-48 h
Tobacco, leaves, *Nicotiana* sp.	50	4 d
Microorganisms, unidentified	40-70	7-56 d
Cotton, leaves	20	2 d
Mugho pine, needles, *Pinus* sp.	17	14 d