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BATS AND ENVIRONMENTAL CONTAMINANTS: A REVIEW

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Bats and Environmental Contaminants: A Review

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Abstract

People have applied organochlorine insecticides to bats or their roosts, or both, to study effects on the bats, to kill them, or to kill pest insects. Studies of effects have shown that DDT and dieldrin are more toxic to bats than other organochlorines. They have also shown that DDT usually reduces colony size temporarily but that it rarely exterminates a colony. Application of DDT usually results in dead or dying bats lying outside the roost site, thus increasing the likelihood that people or pets will be bitten. Insecticides used against wood-boring beetles in buildings have caused many bat deaths in England and Europe.

Bats have been fed various organochlorines in laboratory studies. Early research suggested high sensitivity to DDT in big brown bats (*Eptesicus fuscus*) as measured by LD50 (lethal dose in mg/kg for 50% of the sample). However, subsequent work revealed that the LD50 was a function of fat levels in the bats tested. More recent studies, using lethal brain levels or inhibition of brain ATPases as measures of sensitivity, have shown that bats respond to organochlorines similarly to other small mammals and birds. Even though bats are not unusually sensitive, several of their life history characteristics may make them vulnerable to organochlorines.

Surveys of organochlorines have included bats of 25 species from five countries. Residues of the DDT group, dieldrin, and PCB's predominate with levels in most instances far below those known or thought to be harmful. DDT group residues and dieldrin are usually associated with agricultural areas, whereas PCB's are common in suburban sites. Lethal or potentially lethal residues at the population level have been found in two instances. Young Mexican free-tailed bats (*Tadarida brasiliensis*) at Carlsbad Caverns, New Mexico, contain sufficient stored DDE so that mobilization of residues may cause death when the fat is consumed during the bats' first southward migration. Dieldrin caused mortality in two colonies of gray bats (*Myotis grisescens*) in east central Missouri; the more heavily contaminated colony of this endangered species disappeared in 1979.

Japanese bats of five species contained elevated levels of mercury as a result of agricultural use of mercurial fungicides. Similarly high mercury levels were found in eastern pipistrelles (*Pipistrellus subflavus*) collected along the North Fork of the Holston River in western Virginia; the source of the metal was waste water from a now defunct chlor-alkali factory. Data from other mammals indicate that lethal levels of mercury are at least 10 times higher than those found in these bats, hence outright mortality seems unlikely. Elevated lead levels occurred in bodies and stomach contents of big brown and little brown (*Myotis lucifugus*) bats from a roost near a major highway in Maryland. Estimated dosages of lead ingested by little brown bats equalled or exceeded dosages that caused mortality or reproductive impairment in domestic mammals. Average lead concentrations in bodies of both big brown and little brown bats equalled or exceeded those reported for small rodents with lead-induced renal abnormalities.

Organochlorine chemicals are presently a serious threat to certain bat populations of major importance and may have played a role in past declines of many other such populations. It is doubtful, however, that the combined adverse effects of all the various chemical and metal pollutants have been as serious as the total impact of disturbance, vandalism, and habitat destruction.

To date, only outright mortality caused by pollutants has been identified as a threat to bats, and that threat has been due only to organochlorines. Subtle but equally devastating effects are possible from both organochlorines and metals on such aspects as reproduction, acoustic behavior, and hibernation metabolism. Bats have also been widely exposed to organophosphate and carbamate insecticides and such exposures are probably increasing; nevertheless, there has been almost no research into possible harmful effects.

The first bat deaths directly attributable to chemical contaminants in the environment occurred in 1949 and 1950 (Benton 1951; Dalquest 1953) when DDT was applied to bats or their roosts. About this same time, it was first suggested that insecticides might be a cause of observed population declines in bats (Mohr 1953).

Sixteen mass die-offs of bats due to unknown causes have been described in published accounts. The first took place in 1954 (Villa R. 1955). There were three more in the 1950's (Raun 1960; Constantine 1967) followed by 11 during the 1960's (Booth 1965; Davis 1965; Dew 1965; Constantine 1967; Hamilton-Smith 1967; Cockrum 1970; Gosnell 1977). Greenhall and Stell (1960) demonstrated that organochlorine insecticides were effective in killing bats occupying roosts in buildings, and a laboratory feeding experiment at the University of Kentucky suggested that bats were extraordinarily sensitive to DDT (Luckens and Davis 1964). Probably as a result of these reports describing die-offs and sensitivity, an international outpouring of concern occurred in the early 1970's over the possible harm being done to bats by insecticides (Cockrum 1970; Gould 1970; Punt 1970; Stebbings 1970, 1971; Braaksma and van der Drift 1972; Mohr 1972*b*; Findley 1973; Greenhall 1973).

Laboratory experiments in which organochlorines were fed to bats continued throughout the 1970's (Jefferies 1972; Luckens 1973; Dunsmore et al. 1974; Clark and Kroll 1977; Clark and Prouty 1977; Clark 1978). However, most bat-pollution studies in the 1970's were surveys that quantitatively measured residues of organochlorines (or metals) in tissues of free-living bats (Jefferies 1972; Reidinger 1972; Best 1973; Dunsmore et al. 1974; Clark et al. 1975; Clark and Lamont 1976*a*; Clark and Prouty 1976; Miura et al. 1978; Clark 1979). This survey approach led to compelling evidence that bat populations could experience major mortality due to organochlorine insecticides applied agriculturally and carried through food chains (Geluso et al. 1976; Clark et al. 1978*b*).

The purpose of the present report is to review the published research to evaluate the impact of pollutants on bats and render more apparent the potentially important areas for research.

Organochlorines

Characteristics of Organochlorine Chemicals and Residue Data

The most common organochlorine found in bats is DDE, the long-lived breakdown product of DDT. Polychlorinated biphenyls (PCB's) are industrial pollutants that are often as common as DDE, especially in urban and industrial areas. Less common but frequently recovered residues include dieldrin, DDT and its other principal metabolite DDD (sometimes designated as TDE), heptachlor epoxide, oxychlordane, and various fractions of chlordane such as *trans*-nonachlor, *cis*-nonachlor, and *cis*-chlordane. Compounds rarely found in bats include endrin, toxaphene, hexachlorobenzene (HCB), lindane, and mirex. The designations "total DDT's" or "DDT's" refer to the summation of DDT, DDD, and DDE.

All of these compounds are highly soluble in fat and are quickly taken up by this material after they enter the bloodstream of an animal. When fat reserves are large, they may absorb residues rapidly enough to prevent death if the rate of intake is not too high. When fat is metabolized and decreases in total amount, residues concentrate in the remaining fat. The principal site of action is the brain and the percentage (by weight) of the brain that is fat remains nearly constant regardless of the level of fat reserves in the rest of the animal. Thus organochlorines are capable of killing when fat reserves are metabolized because this causes residues to concentrate in the brain. The affinity for fat also causes heavy excretion of residues in the milk of lactating mammals. Even though these organochlorine chemicals share these features, they differ widely in toxicity, which in turn varies with the species or group of species being considered. Also, some of these chemicals are quickly metabolized and excreted, whereas others remain unchanged in the animal almost indefinitely. These compounds also differ in other ways. For example, PCB's can cause death by either the typical neurotoxic mode or by a hemorrhagic mode depending on the dosage rate (Stickel 1975). As another example, in certain species of birds DDE causes thinning of eggshells whereas most other organochlorines do not. The kinetics of organochlorines were reviewed in detail by Stickel (1973).

Since the 1960's most organochlorine analyses have been made by gas-liquid chromatography. This technology generally works well and results are highly accurate. However, certain inadequacies in the measurements have occurred and continue to occur, and therefore should be mentioned. When the methodology does not separate the multiple-peaked PCB's from pesticides, the PCB and pesticide peaks are overlain and intermixed resulting in peaks that are misidentified or PCB's that are read as additional amounts of certain pesticides, or both. In such circumstances only readings of DDE are accurate and then only when amounts of this residue markedly exceed those of the other chemicals. A second major problem involves quantification of PCB's and tox-

aphene; both are actually mixtures of numerous compounds and thus they appear on the chromatograph as multiple peaks. Because identification and quantification of peaks are difficult, many different methods are used and quantifications are much less accurate than for other organochlorines and comparisons of data from different laboratories are likely to be misleading.

Residue data (including metals) from field-collected samples usually show positively skewed distributions. Therefore, such data are often log-transformed before statistical testing and geometric means (retransformed arithmetic mean of log-transformed data) are calculated as the measure of central tendency.

Residue data are usually reported as parts per million (ppm) of wet (fresh) weight or of extractable lipid. Sometimes total micrograms (μg) may be reported. In the present paper, the designation "ppm" means ppm of wet weight, unless otherwise indicated. The abbreviation "ND" indicates that a residue was not detected at the lower limit of analytical quantification.

Major Unexplained Bat Die-offs

During and since the development of insecticide-related concern for bat populations, there have been reports of mass mortalities of bats for which the cause or causes have never been determined (Table 1). Just as the role of these die-offs in population declines is unknown, so too is the role of insecticides in these die-offs. Nevertheless, these large-scale mortalities have contributed to the belief that insecticides have caused significantly increased bat mortality. Davis (1965), Hamilton-Smith (1967), and Cockrum (1970) postulated that insecticides might have caused the die-offs they reported. Davis (1965) analyzed two bats and found insecticide residues, but his results were inconclusive. Cockrum (1970) and Reidinger and Cockrum (1978) analyzed bats collected alive 1 and 2 years after the die-off, at which time pesticide residues were far below lethal levels. Even though there might have been numerous causes for these die-offs (Table 1), they remain of interest because insecticides were not effectively ruled out for any of them, and because the knowledge and technology now exist to evaluate the role of organochlorines if specimens (or parts of specimens) could still be found.

Of the 16 known mass mortalities (Table 1) 11 occurred in Texas, New Mexico, or Mexico within 370 km of the United States-Mexico border; one was in Missouri, and four were in southwestern Victoria, Australia. Nine die-offs involved the Mexican free-tailed bat (*Tadarida brasiliensis*). The apparent geographic localization in southwestern North America probably merely reflects the presence of numerous large bat colonies and intensive research on bats in this area.

Three die-offs involved free-tailed bats at Carlsbad Caverns (Table 1). A fourth die-off at Carlsbad (in August 1955) was probably caused by rabies (and therefore is omitted from Table 1), and pesticides were ruled out by tissue analyses and bioassays (Constantine et al. 1968).

Constantine (1967) noted that the dead free-tailed bats at Carlsbad Caverns were almost entirely adult bats, and mostly males. In contrast, Altenbach et al. (1979) reported that most of the dead they observed were only a few days old. The findings of Geluso et al. (1976) indicated that DDE-induced mortality in the Carlsbad colony would involve newly volant young on their first southward migration. Gray bats (*Myotis grisescens*) that died in Missouri of dieldrin were large nursing young and adult females (Clark et al. 1978*b*, 1980). The mortality at Carlsbad has had varing characteristics and probably several causes.

The Missouri die-off (Table 1) is unique among those recorded in North America because of its location and because it occurred in spring. Among the possibilities: if organochlorines had been involved, they could have been concentrated in brain tissue during hibernation as fat reserves were consumed, causing bats to die in much the same manner as those dosed by Luckens (1973), or the organochlorines could have accelerated metabolic rates of the hibernating bats, causing premature depletion of fat reserves and death (a hypothesis based on a mechanism suggested by the experimental data discussed in a later section).

Of four mass mortalities of bent-winged bats (*Miniopterus schreibersii*) in southeastern Australia (Table 1), one occurred over a broad area (Hamilton-Smith 1967). Three die-offs involved juvenile bats soon after birth in late December-early January (Hamilton-Smith 1967), but the other occurred in winter (July and earlier) and may have involved adult bats (Dew 1965).

In summary, these unexplained die-offs have involved bats of seven species belonging to three families. They have included hibernating and non-hibernating species, both sexes, and various ages, and they have occurred at different times of the year. With respect to the possible role of organochlorines, these mortalities point to the need for preservation (by freezing if possible, otherwise by formalin or by drying) of specimens for future analysis whenever a die-off is discovered.

Direct Applications of Insecticides to Bats or Bat Roosts

Bats have died after they or their roosts (or both)

Table 1. *Major bat die-offs of unknown cause.*

Species, dates, and reference (in parentheses)[a]	Locality (Co. = County)	Condition[b]	Number	Postulated cause
Mormoops megalophylla				
July or Aug. 1954[c] (11)	Cueva de Diablo and Jesus Maria Mine, Nuevo Leon, Mexico	D	Hundreds of thousands at Cueva, thousands at Mine	Epidemic disease
Tadarida brasiliensis				
Aug.-Sept. 1956 (4)	Carlsbad Caverns, New Mexico	D, Dy	Thousands	Migratory stress and rapid cooling
Aug. 1956, July 1962[d] (4)	Frio Cave, Uvalde Co., Texas	D, Dy	Hundreds	Migratory stress and rapid cooling
July 1962 (4)	Railroad tunnel, Kendall Co., Texas	D, Dy	Hundreds	Migratory stress and rapid cooling
27 Dec. 1963 (2)	Cave 16 km S Ciudad Mante, Tamaulipas, Mexico	D, Dy	Hundreds of thousands	None
Summer 1967 (7)	Carlsbad Caverns	D, Dy	"couldn't see the surface of the guano for the carcasses"	Insect shortage due to drought, and the Rio Bravo virus
Late summer 1968 (3)	Cave southeast of Carbo, Sonora, Mexico	D, Dy	Enough to "cover" a hillside	Insecticide
Summer 1968 (3, 10)	Fields near Eagle Creek Cave, Greenlee Co., Arizona	D, Dy	"large numbers"	DDT and its metabolites
Summer 1971 (1)	Carlsbad Caverns	D	Several thousand	None
Myotis velifer				
Summer 1958 (9)	Valdina Farms Sinkhole, Medina Co., Texas	D	Several thousand	Disease, rabies
Myotis grisescens[e]				
May 1965[c] (5)	Carroll and Coffin Caves, Laclede Co., Missouri	D, Dy	Over 100	Insecticide
Miniopterus schreibersii				
Jan. 1964, Jan. 1965[d] (8)	Lake Gillear Guano Cave, Victoria, Australia	D, Dy	"a large number"	Epizootic disease or insecticides

Table 1. (continued)

Species, dates, and reference (in parentheses)[a]	Locality (Co. = County)	Condition[b]	Number	Postulated cause
July 1965 (6)	Fig Tree Cave at Wombeyan Caves, southeastern Australia	D	At least 200	Vandalism or predation
Jan 1967[c] (8)	Warrnambool area, Portland Cave, Lake Gillear Guano Cave, Victoria, and Naracoorte Cave, South Australia	D, Dy	"a large number"	Epizootic disease or insecticides

[a] References: (1) Altenbach et al. 1979; (2) Booth 1965; (3) Cockrum 1970; (4) Constantine 1967; (5) Davis 1965; (6) Dew 1965; (7) Gosnell 1977; (8) Hamilton-Smith 1967; (9) Raun 1960; (10) Reidinger and Cockrum 1978; (11) Villa R. 1955.
[b] D = dead; Dy = dying.
[c] Considered a single die-off because localities were proximate and the date and species were the same.
[d] Considered two die-offs because major mortality occurred in two different years.
[e] Also included a few *Myotis lucifugus* and one *Pipistrellus subflavus*.

were sprayed with organochlorine insecticides in 13 instances (Table 2): in 8 of these, the chemicals were applied to kill bats or to learn how much mortality would result (Greenhall and Stell 1960; Mohr 1972*a*; Humphrey and Cope 1976; Kunz et al. 1977; Clark et al. 1978*a*; Fenton and Hurley 1979; Barclay et al. 1980); in 4, the chemicals were applied to kill insects (Benton 1951; Stebbings 1971; Braaksma and van der Drift 1972; Voûte 1980); the intended target of the 13th trial was not stated (Dalquest 1953).

Greenhall and Stell (1960) and Barclay et al. (1980) applied insecticides to bats and to their roosts in buildings to measure the effectiveness of these chemicals in reducing numbers. Between March 1958 and March 1959, Greenhall and Stell (1960) applied DDT, dieldrin, benzene hexachloride (BHC, a component of which is lindane), and chlordane to roosts of little free-tailed bats (*Molossus major*) and greater free-tailed bats (*Molossus rufus*) beneath roofs of 13 houses in Trinidad (Table 2). Mortality was greatest when power-driven high-volume sprayers applied chemicals as wettable powders or wettable pastes at 2.72 kg of active ingredient per house. DDT and dieldrin were the most toxic to bats. Total numbers of bats were reduced by only one-third, and the reduction was only temporary. Chlordane killed one-fifth and BHC one-sixth of the bats. The most effective repellent was BHC, which caused maximum numbers of bats to fly from houses when it was applied.

On 27 June 1978, Barclay et al. (1980) treated each of two colonies of big brown bats (*Eptesicus fuscus*) with 4.7 kg of DDT (as 50% dust) in separate attics of the same building. Eight dead or dying bats were reported during the 5 days following treatment. After 36 days, the smaller colony (11 bats) was gone, but the larger colony (61 bats), which had initially declined to 8 bats, contained 21 bats.

Barclay et al. (1980) studied two other colonies that had been treated with DDT by property owners. One colony persisted despite three treatments totaling 11.8 kg of DDT over several years. The other colony survived, but at reduced numbers, after treatment with 47 kg of DDT, and contacts between bats and humans increased. Two other treatments, application of sticky deterrents to bat entrances and exits, and closure of access holes, were also tested (Barclay et al. 1980). Closure of holes or "bat proofing" was clearly the most effective method; not only was DDT relatively ineffective, its use increased the likelihood of encounters between bats and people and the likelihood of bites.

Fenton and Hurley (1979; later published as Hurley and Fenton 1980), using little brown bats (*Myotis lucifugus*) confined in small boxes with internal

Table 2. *Organochlorine insecticides applied to bats or their roosts, or both.*

Species, dates of observed mortality, and reference (in parentheses)[a]	Locality	Number of dead bats counted	Chemicals	Number of roosts treated	Purpose of treatment
Lasiurus borealis Summer 1949 (2)	Princeton, New Jersey	1	DDT	Many trees sprayed	Kill bark beetles that carry Dutch elm disease
Myotis yumanensis Early 1950 (5)	Hacienda Bledos, San Luis Potosi, Mexico	Hundreds	DDT	1 house	Not stated
Rhinolophus ferrumequinum 1952 (11)	Near Wareham, Dorset, England	Over 100	Not stated[b]	1 house	Kill woodworm
Molossus major and *Molossus rufus* March 1958–March 1959 (7)	Trinidad	1,931	DDT, BHC, dieldrin, chlordane	13 houses	Measure effects on bat colonies
Eptesicus serotinus, Myotis dasycneme, Plecotus auritus 1964 (3)	Netherlands	78	Lindane, dieldrin, pentachlorophenol, chlorinated naphthalenes	6 churches	Kill woodworm (*Anobium punctatum*) and house longhorn beetle *Hylotrupes bajulus)*
Myotis lucifugus Summer 1964 (8)	Tipton, Indiana	Not recorded	DDT	1 house	Kill bats
Summer 1968 (8)	Brookville, Indiana	Not recorded	DDT	2–4 buildings	Kill bats
Summer 1969 (8)	Shoals, Indiana	Not recorded	DDT	1 building	Kill bats
Sept. 1973 through Summer 1979[c] (4,9)	Near Amherst, New Hampshire	Hundreds	DDT and chlordane	1 barn	Kill bats
July–Aug. 1979 (6)	Ottawa, Canada	4[d]	DDT	6 artificial roosts[e]	Measure potential effects on bat colonies
Eptesicus fuscus Fall 1972 (10)	Blades, Delaware	Not recorded	DDT	1 building	Kill bats

Table 2. (continued)

Species, dates of observed mortality, and reference (in parentheses) [a]	Locality	Number of dead bats counted	Chemicals	Number of roosts treated	Purpose of treatment
Myotis dasycneme Summer 1973 and 1977 (12)	Berlikum, Netherlands	137	Lindane and DDT	1 church	Wood preservation
Myotis lucifugus, Eptesicus fuscus Apr.-Aug. 1978 (1)	Vicinity of Ottawa, Canada	Not recorded	DDT	2 in a single building [f]	Measure effects on bat colonies

[a] References: (1) Barclay et al. 1980; (2) Benton 1951; (3) Braaksma and van der Drift 1972; (4) Clark et al. 1978*a*; (5) Dalquest 1953; (6) Fenton and Hurley 1979; (7) Greenhall and Stell 1960; (8) Humphrey and Cope 1976; (9) Kunz et al. 1977; (10) Mohr 1972*a*; (11) Stebbings 1971; (12) Voûte 1980.
[b] Perhaps as in Braaksma and van der Drift (1972).
[c] Data after 1977 provided by T.H. Kunz (personal communication).
[d] Four other bats died after exposure to zinc phosphide.
[e] Roosts were kept outside under natural weather conditions.
[f] Two were treated by Barclay et al. (1980); another 10 roosts treated by property owners were included in the study.

surface areas of 480 cm^2 or in larger artificial roosts with internal surface areas of 3,963 cm^2, evaluated fenthion (an organophosphate insecticide), zinc phosphide, and DDT as contact poisons or repellents. The chemicals were applied to bats or their roosts and the effects were evaluated after exposures of 24 h. Three of six bats were killed by DDT when it was applied to roosting surfaces in the boxes at 15 or 20 mg/kg. At lesser rates (2, 5, and 10 mg/kg) no bats died, but all showed symptoms and only the control bats were capable of sustained flight at the end of the test. When DDT was dusted directly on bats at 15 mg/kg in the larger roost, it produced symptoms in 5 bats and killed only 1 of 20; all 19 survivors could still fly after 24 h.

Zinc phosphide applied to roosting surfaces at 750, 1,130, or 1,500 mg/kg killed one bat and disabled the eight survivors so they could not fly. Applied at lesser rates (10 and 370 mg/kg) it produced no apparent effect. Zinc phosphide dusted directly on the bats at 750 mg/kg killed 3, rendered 3 incapable of flight, but left 14 unaffected. Reactions to liquid fenthion, when it was sprayed directly on the bats, were limited to an immediate but short-term (less than 5 min) repellent effect. Fenton and Hurley (1979) concluded that none of these chemicals (in the forms in which they were tested) would be effective in eliminating bats from buildings. They also reported that both DDT and zinc phosphide might cause bats to fall and die outside their roosts, thereby becoming a potential health hazard.

In separate incidents in Indiana, Delaware, and New Hampshire, homeowners or professional exterminators attempted to eliminate bat colonies by spraying them and their roosts with DDT or DDT plus chlordane (Mohr 1972*a*; Humphrey and Cope 1976; Kunz et al. 1977; Clark et al. 1978*a*; Table 2). In Indiana during the 1960's, DDT was applied to three maternity colonies of little brown bats (Humphrey and Cope 1976). A medium-sized colony at Tipton was exterminated after it was sprayed with a DDT solution; a colony estimated at 650 females and young at Brookville contained no bats 1 year after treatment with DDT dust; and a colony at Shoals, estimated at 3,000 females and young, was reduced to an estimated 875 bats 1 year after one of four buildings that it used was treated with DDT dust and automobile exhaust (Humphrey and Cope 1976). The fate of these three colonies in later years was not recorded.

In Delaware, concern about rabies was the apparent justification for using DDT against big brown bats (Mohr 1972*a*). The effects of the chemical were not measured.

In New Hampshire, a professional exterminator applied DDT and chlordane to the walls and rafters of a barn to kill little brown bats in three separate sprayings during August and September 1973 (Kunz et al. 1977). Kunz et al. (1977) estimated that 90% of

this maternity colony of about 500 adult bats had already left the barn for hibernation sites when the sprays were applied, but heavy annual mortality, among both adults and newly produced young, began after the bats returned the following spring. Nearly nine times more young than adults (202:23) died in 1974, and twice as many died in 1975 (76:45). Concentrations of DDT in brains of dead or convulsing bats showed that young bats (16.3 ppm DDT) were one and one-half times more sensitive than adults (24.5 ppm DDT); also, milk from stomachs of dead young contained three times as much DDT as did insects from the stomachs of adults (Clark et al. 1978*a*). The increasing numbers of dead adults with time may reflect a slower buildup of DDT in their bodies and lesser sensitivity among adults than among juveniles. Juvenile mortality was concentrated soon after birth or when young approached adult size (Kunz et al. 1977). This mortality pattern suggests that the young died either on their initial exposure to residues in the milk or when their fat reserves were mobilized for flight.

Concentrations of nine organochlorines in carcasses of young bats increased significantly throughout the nursing period in the New Hampshire colony (Clark et al. 1978*a*). For many young this increase set the stage for mortality when fat reserves were consumed. Even though chlordane residue products clearly indicated the recent use of this chemical, deaths were most closely correlated with DDT levels (Clark et al. 1978*a*). Hundreds of bats have died in the summers after spraying, and T. H. Kunz (personal communication) stated that in August of 1979 bats were still dying and the colony numbered less than 100. The chemical approach seems ineffective because, although diminished, the colony survives 6 years after spraying; furthermore, in any barn sprayed as this one was livestock or crops would soon become contaminated and therefore unmarketable.

Insecticides used against wood-boring beetles in old buildings probably caused bat deaths in England (Stebbings 1971) and the Netherlands (Braaksma and van der Drift 1972; Voûte 1980; Table 2). Stebbings (1971) reported a single instance in which over 100 greater horseshoe bats (*Rhinolophus ferrumequinum*) were found dead in 1952. Such occurrences probably are numerous because he also reports that over 35,000 houses are fumigated yearly in England. Braaksma and van der Drift (1972) reported deaths of the serotine bat (*Eptesicus serotinus*), the pond bat (*Myotis dasycneme*), and the gray long-eared bat (*Plecotus auritus*) in churches that had been treated. Analyses of "entire bodies" of one serotine bat and one pond bat showed lindane at 267 and 463 μg and dieldrin at 0.68 and 0.64 μg. The dieldrin was probably not responsible for these deaths because a lethal brain concentration of dieldrin (at least 5 ppm; Clark et al. 1978*b*) would require 0.5 to 1.0 μg in the brain alone, assuming a brain weight of 0.1 to 0.2 g. However, at present there are no data by which the levels of lindane can be evaluated.

In two groups of dead pond bats recovered from the same church in different years, each after the empty winter roost had been sprayed, Voûte (1980) found up to 74 ppm of lindane in carcasses, 28 ppm of lindane in brains, and 60–70 ppm of total DDT's in desiccated and decomposed bodies that were analyzed whole. Based on data from little brown bats that died of DDT (Clark et al. 1978*a*), these total DDT levels are not high enough to have caused mortality; levels in brains would be needed for certainty. Lindane at 28 ppm in brain tissue may represent the diagnostic lethal level. Feeding studies are needed to evaluate and quantify the effects of lindane on bats.

DDT sprayed on elm trees (*Ulmus americana*) at 1.36 kg per tree apparently caused the first recorded case of organochlorine poisoning of a bat (Benton 1951). The chemical was applied to kill bark beetles (family Scolytidae) which carry Dutch elm disease (a fungus, *Ceratostomella ulmi*). The bat (a red bat, *Lasiurus borealis*) was found on its back in tremors. Actual circumstances can only be surmised, but red bats roost solitarily in trees; therefore, this individual may have been sprayed directly.

Dalquest (1953) observed hundreds of dead yuma myotis (*Myotis yumanensis*) after the rooms of a hacienda were painted with DDT. Bats were able to reoccupy the building about 9 months later. It is not clear whether the bats were the target of the chemical treatment.

Experimental Feeding Studies

Investigators have fed five organochlorine compounds to bats of six species in a total of nine studies (Table 3). The principal objectives of this work were to determine (1) sensitivity of bats to these chemicals and how their sensitivity compares with that of other mammals and birds, (2) lethal diagnostic levels in tissues in order to interpret levels found in free-living bats, and (3) whether PCB residues found in bats cause young to be stillborn.

Luckens and Davis (1964) found what appeared to be great sensitivity to DDT in big brown bats. All bats fed single doses of 40 mg/kg or more died within 11 days, and one of three died at the lowest dosage of 20 mg/kg. Comparisons with data for laboratory rats (*Rattus norvegicus*) and mice (*Mus musculus*) suggested that these bats were 5 to 6 times more sensitive than rats and 15 times more sensitive than mice. In a second study, Luckens and Davis (1965) fed dieldrin or endrin to big brown bats. This time the

Table 3. *Organochlorines fed to bats.*

Species, source of bats, and reference (in parentheses)[a]	Purpose	Toxicants	Method of dosage	Dosage level	Tissues analyzed
Eptesicus fuscus					
Georgetown and Lexington, Kentucky (8)	To measure LD50's[b]	DDT	Injected into mealworms[c]	Single doses 20–800 mg/kg	None
Georgetown and St. Helens, Kentucky (9)	To measure LD50's	Dieldrin, endrin	Applied to "glop"[d]	Single doses 10–200 mg/kg dieldrin, 1–50 mg/kg endrin	None
Lexington, Kentucky (7)	To measure LD50's of fat bats in relation to hibernation	DDT	Applied to "glop"	Two doses totalling 60–1,488 mg/kg	Single pooled samples of brains, livers, and carcasses
Montgomery and Prince Georges counties, Maryland (3)	To measure brain levels diagnostic of death	DDE and PCB (Aroclor 1254)	Mealworms reared in contaminated wheat bran	DDE 166 ppm for 54 days, PCB 9.4 ppm for 37 days	Individual brains and carcasses
Laurel, Maryland (1)	To determine whether stillbirths were caused	PCB (Aroclor 1260)	Mealworms reared in contaminated wheat bran	6.4 ppm for 17–27 days	Individual female carcasses and entire neonates
Pipistrellus pipistrellus[e] Eastern England (6)	To measure LD50 and compare tissue levels to those of wild bats	DDT	Dripped onto tongue	Single doses 33–277 mg/kg	Individual livers and whole bodies[f]
Miniopterus schreibersii Cleatmore and Marble Arch Caves, New South Wales, Australia (5)	To compare carcass levels to those of wild bats	DDT	Injected into mealworms	15 μg/bat per day for 20 days or until death	Individual carcasses
Tadarida brasiliensis Bracken Cave, Texas (2)	To measure brain levels diagnostic of death	DDE	Mealworms reared in contaminated wheat bran	107 ppm for 40 days	Individual brains and carcasses
Myotis lucifugus North East, Maryland (4)	To measure brain levels diagnostic of death	DDE and PCB (Aroclor 1260)	Mealworms reared in contaminated wheat bran	DDE 480 or 150 ppm, PCB 1,000 or 15 ppm for 40 days	Individual brains and carcasses

[a] References: (1) Clark 1978; (2) Clark and Kroll 1977; (3) Clark and Prouty 1977; (4) Clark and Stafford 1981; (5) Dunsmore et al. 1974; (6) Jefferies 1972; (7) Luckens 1973; (8) Luckens and Davis 1964; (9) Luckens and Davis 1965.
[b] Lethal dose for 50% of bats treated, measured in mg/kg of bat.
[c] Larvae of the beetle *Tenebrio molitor*.
[d] A blend of equal parts of banana, cream cheese, dog food, and mealworms.
[e] Also included one *Myotis mystacinus*.
[f] Perhaps carcass, author was not specific. "Carcass" designates what remains after at least the skin is removed; in most studies some or all of the head, gastrointestinal tract, wings, feet, and tail were also removed.

apparent sensitivities (LD50 20-40 mg/kg for dieldrin, 5-8 mg/kg for endrin) were similar to laboratory rats. Even though the bats were the same species and from essentially the same source in both studies, those used in the DDT experiment were caught in May when their fat reserves would have been low, whereas those used in the dieldrin-endrin experiment were caught in August when fat reserves would have been moderate.

Continuation of this work led to speculation that the fat level was controlling the observed sensitivity (Davis 1966, 1967). Finally, using big brown bats caught in late September and containing peak amounts of fat, Luckens (1973) demonstrated both tolerance (before fat loss) of dosages up to 1,488 mg/kg and subsequent mortality due to mobilization of stored DDT when fat reserves were consumed during laboratory hibernation. Several bats survived dosages up to 306 mg/kg and were released after hibernation, but above this dosage level all bats died. Tests indicate bats with large fat reserves can tolerate heavier mg/kg dosages than can thin bats, even after fat loss, if the rate of residue release from the fat, which depends on the amount stored and the speed with which the fat is diminished, does not exceed a certain threshold level. Even though this work added greatly to our understanding of how organochlorines behave in bodies of bats, it left open the question of relative sensitivity.

Jefferies (1972) extended the work of Luckens and Davis (1964, 1965) by the addition of chemical analyses. However, the chemical methodology may have been inadequate because no PCB's were recovered even though they had been commonly found in British birds. Jefferies (1972) first surveyed residues in wild bats from several localities in England. Then he fed DDT at dosages of 33 to 277 mg/kg to captive pipistrelles (*Pipistrellus pipistrellus*) and one whiskered bat (*Myotis mystacinus*) and measured residues in livers and whole bodies of those that died and those that survived. Results of the various dosages indicated an LD50 of 63 mg/kg and, therefore, great sensitivity in comparison to laboratory rats and mice. These bats had been in captivity for several months and may have been fatter than comparable wild bats. As Jefferies (1972) suggested, even lower doses might have been fatal to wild bats. The comparison with laboratory rodents is difficult because nothing is known of relative fat levels.

Results from tissue analyses of dosed bats suggested that residue levels in livers were more closely correlated with death than were levels in whole bodies. Jefferies (1972) pointed out that mean liver residues of DDT plus DDE (values given by Jefferies as "DDT" are DDT plus measured DDD multiplied by 1.11) in wild bats collected in 1968 were 18 ppm; those from 1969 wild bats were 16 ppm, and the highest liver residue was 65 ppm. The lowest level found among bats that died on dosage was 44 ppm. Similar findings were obtained from whole body residues but most whole body data for wild bats were estimated from liver levels by use of the relation between residues in livers and bodies of the laboratory-dosed bats.

Jefferies (1972) concluded that "the bats of the area studied were carrying one-third of a lethal level of insecticide material (mainly DDT + DDE) as 'background' contamination throughout the year rising to just under the lethal level after hibernation." Present evidence suggests two major drawbacks to this conclusion. First, the residues recovered from wild bats were principally DDE; in March, residues in livers were 66.5% DDE, and they rose to 91.2% DDE in November (Jefferies 1972). On the other hand, residues recovered from livers of the eight bats that died while on DDT dosage were mostly DDT+DDD; DDE constituted an average of only 8.4 ± 1.9% (range 3.3-16.4%). This is a serious bias because the relative toxicities of DDT, DDD, and DDE are about 1 to 5 to 15 ppm, measured as residues in brains of birds (Stickel et al. 1970) and substantiated by the data from bats (Table 4). When the comparison is limited to DDT (that is, Jefferies' DDT + DDD × 1.11) in livers, the 24 bats collected in 1968 and 1969 averaged 5.23 ± 1.55 ppm (range ND-28.6 ppm, geometric mean 1.36 ppm), whereas concentrations for the eight that died of DDT averaged 243 ± 87 ppm (range 38.85-747.7 ppm, geometric mean 160 ppm). By this interpretation, the status of wild British bats appears less problematical.

The second drawback concerning Jefferies' (1972) conclusion stems from studies showing that whereas brain concentrations can be used successfully to diagnose death from organochlorine toxicants, residues in livers and carcasses cannot (Dale et al. 1963; Stickel et al. 1966, 1969, 1970; Stickel and Stickel 1970).

Working with the bent-winged bat, Dunsmore et al. (1974) used an approach similar to that of Jefferies (1972) to evaluate residues in wild bats. In this study, however, residues were measured only as total DDT's and only as total μg per bat carcass. Furthermore, PCB's were not separated. Among eight bats that were fed DDT, some died, some were killed while showing signs of poisoning, and some showed no signs and were killed after 20 days of dosage. These eight averaged 680 μg (estimated to equal 50 ppm). One individual killed while in tremors contained 255 μg. The largest amount of DDT's sampled among wild bent-winged bats was 56 μg. The free-living bats contained low residues relative to those in bats killed

Table 4. *Lethal brain concentrations of organochlorines in bats compared with other small mammals and birds.*

Organochlorine, species, and reference (in parentheses)[a]	N	Sex	Lethal concentration (ppm wet weight) Mean[b]	Range	Type of data[c]
DDT					
Myotis lucifugus (4)	11	F	24.5[d] (18.1–33.1)	12–47	F
Rattus norvegicus (7,9)	4	M	47 ± 5[e,f]	35–52	L
Rattus norvegicus (11)	10	M,F	44 ± 2[e]	—[g]	L
Mus musculus (8)	3	M	49 ± 4.5	—	L
	3	F	58 ± 14		
Blarina brevicauda (1)	12	M	34.2 (15.9–73.5)	2.5–96.4	L
	10	F	17.2 (7.4–40.4)	2.1–85.2	
Molothrus ater (13)	16	M	39.2 (21.3–72.2)	27.3–89.8	L
	6	F	40.1 (19.7–81.7)	27.0–76.6	
DDE					
Tadarida brasiliensis (2)	8	F	519 (483–558)	458–564	L
Myotis lucifugus (3)	4	F	603 (519–701)	540–670	L
Molothrus ater (15)	15	M	499 (442–572)	250–660	L
PCB					
Myotis lucifugus (3)	2	F	1,400[h]	1,300; 1,500	L
Molothrus ater (12)	5	M	484[i] (428–549)	439–556	L
Dieldrin					
Myotis grisescens (5,6)	18	M,F	7.8 (6.7–9.2)	4.6–13	F
Rattus norvegicus (10)	—	M	—	2.1–10.8	L
Sigmodon hispidus (14)	5	M,F	7.9 (5.5–11.4)	5.6–11.1	F
Blarina brevicauda (1)	6	M	6.8 (4.5–10.4)	3.7–12.6	L
	8	F	6.7 (4.8–9.3)	4.3–11.2	
Sturnella magna (14)	5	M	9.3 (7.7–11.2)	8.4–12.1	F
Turdus migratorius (14)	7	—	9.6 (6.7–13.9)	5.0–17.0	F
Coturnix coturnix (14)	10	M	14.8 (10.0–21.9)	6.2–32.0	L
	7	F	21.8 (15.5–30.6)	11.0–32.9	

[a] References: (1) Blus 1978; (2) Clark and Kroll 1977; (3) Clark and Stafford 1981; (4) Clark et al. 1978*a*; (5) Clark et al. 1978*b*; (6) Clark et al. 1980; (7) Dale et al. 1963; (8) Gingell and Wallcave 1974; (9) Hayes 1965; (10) Hayes 1974; (11) Henderson and Woolley 1969; (12) W.H. Stickel, personal communication; (13) Stickel and Stickel 1970; (14) Stickel et al. 1969; (15) Stickel et al. 1970.

[b] Geometric means are given with 95% confidence intervals in parentheses, arithmetic means are given ±1 standard error.

[c] F = field; L = laboratory.

[d] Brains of eight juveniles killed by DDT contained significantly lower concentrations; geometric mean 16.3 ppm, range 11–25 ppm.

[e] Based on extrapolation from original source.

[f] Chemical methodology did not distinguish DDT from DDD.

[g] Information not published.

[h] Aroclor 1260.

[i] Aroclor 1254.

by DDT. Again in this study (Dunsmore et al. 1974), most of the residue in wild bats was probably DDE, whereas most of it in those that were dosed was DDT, so the risk to free-living bats is even less than it appears.

Two of three experimental feeding studies (Clark and Kroll 1977; Clark and Prouty 1977; Clark and Stafford 1981) that attempted to measure lethal brain concentrations of organochlorines in bats were successful. By using the results of these studies plus

brain residue measurements from bats killed by organochlorines in the field, it is possible to evaluate the relative sensitivity of bats with a parameter that is independent of fat (Table 4). Little brown bats seem about twice as sensitive to DDT as laboratory rats and mice, slightly more sensitive than cowbirds (*Molothrus ater*), and similar to short-tailed shrews, *Blarina brevicauda* (Table 4). Brains of juvenile little brown bats that died of DDT contained significantly less than adult females (Table 4); thus juveniles appear to be about 1.5 times more sensitive (Clark et al. 1978*a*). No such difference was found between adult and juvenile gray bats in respect to dieldrin (Clark et al. 1980), but larger samples might have revealed a difference. Luckens (1973) reported 130 ppm DDT in a pooled sample of 37 brains from big brown bats that died during hibernation after being dosed. Either this value represents an analytical error, or big brown bats are much less sensitive to DDT than any mammal or bird tested thus far.

Toxicity of DDE appears similar in two species of bats and one bird (Table 4). Brain concentrations of 260 and 330 ppm DDE were measured by Geluso et al. (1976) in two Mexican free-tailed bats from Carlsbad Caverns, New Mexico, that died after exhibiting what appeared to be symptoms of DDE poisoning. These bats mobilized DDE stored in their fat when they were exercised daily and deprived of food. These values are well below the lethal range measured in a laboratory feeding experiment with bats of this species (Table 4). However, the bats used by Geluso et al. (1976) were juveniles, whereas those in the feeding experiment were adults. If juvenile Mexican free-tailed bats are about 1.5 times more sensitive to DDE than adults, as was true of DDT in little brown bats, 260 and 330 ppm would convert to 390 and 495 ppm; these values resemble those determined experimentally.

In an unsuccessful attempt to determine lethal brain concentrations of DDE in brains of big brown bats, 17 bats were fed mealworms (larval beetles *Tenebrio molitor*) that contained 166 ppm DDE for 54 days (Clark and Prouty 1977). No big brown bats died, even though this dietary level was higher and the dosage period longer than the dietary level and dosage period used with Mexican free-tailed bats (Clark and Kroll 1977), because they had been overfed and their carcasses averaged 41.6 ± 3.9% fat (range 29.0–52.3% fat) at the end of dosage. Brain levels of DDE rose in response to the loss of fat when these bats were deprived of food. However, all bats died of starvation-related physiological causes before fat levels declined enough to result in poisoning by DDE. The highest brain concentration (132 ppm) was found in the bat with the least carcass fat (8.5%).

From the few data available, it appears that little brown bats can tolerate three times more PCB in their brains than can cowbirds (Table 4). However, because quantification of PCB is difficult and two different laboratories performed these analyses, this comparison is risky. Furthermore, the PCB's were different. Aroclor 1260 was fed to bats and Aroclor 1254 was fed to birds. The latter may be more toxic than the former (Fishbein 1974). Aroclor 1254 was fed to 16 big brown bats (Clark and Prouty 1977); however, the dosage rate was low (9.4 ppm in the diet) and, as occurred in the big brown bats to which DDE was fed, these bats were allowed to become fat and no toxic effects were seen. The sensitivity of gray bats to dieldrin appears similar to that of the majority of other species for which there are data (Table 4).

The data presently available on lethal brain levels vary greatly in how they were obtained and also in sex and age categories tested. Also, relatively few species and chemicals have been tested. Nevertheless, bats appear generally similar to other small mammals and birds in their sensitivity to organochlorines.

Additional information on the relative sensitivity of bats was provided by another approach that is also independent of fat levels. By adding DDT or DDE to in vitro preparations of brain tissue from eight eastern pipistrelles (*Pipistrellus subflavus*) and eight evening bats (*Nycticeius humeralis*), Esher et al. (1980) determined that both chemicals were inhibitors of brain ATPases. Actual amounts of DDT and DDE in the preparations were unknown because background levels in the brains were not measured; however, the results indicated sensitivity similar to that of laboratory mice.

Because bats do not seem unusually sensitive to organochlorines, it is probably more important to evaluate their relative vulnerability to these materials. The bat species whose survival is most in doubt are insectivorous, temperate region microchiropterans. These species possess characteristics that increase their likelihood of being harmed. Like other insectivorous mammals and birds, these bats encounter more organochlorines in their diets than do herbivores because of food chain buildup. The higher metabolic rates of these bats associated with small size and the activity of flight demand higher rates of food consumption than found in larger, less active birds and mammals. Greater food intake causes greater amounts of organochlorines to be available for concentration in the fat of bats. Bats are similar to small insectivorous birds but more at risk than other small mammals because of their pronounced fat cycles with rapid depletion during migration (or, unlike birds, with extreme but slow depletion during hibernation), and

because of their high mobility which may increase their chances of encountering a lethal dose. Mobility may also assure movement to less contaminated food sources and thereby reduce the danger.

Bats resemble other small insectivorous mammals but differ from small insectivorous birds in that large doses of organochlorines from their female parent probably are administered more rapidly by the milk than by the stored fat of the egg. Even though this question has not been studied directly, it is known that organochlorines are concentrated in the milk secreted at the beginning of lactation (in domestic cows and laboratory rats; Laben et al. 1965; Ottoboni and Ferguson 1969). Jefferies (1972) argued that bats may be physiologically less able than birds to break down DDT. However, questions leave this conclusion in doubt (i.e., concerning the relative amounts of fat in the birds and bats that he dosed with possible effects on excretion rates, and whether PCB's were identified and separated in the various sets of data that were compared).

Three unique characteristics of bats increase the likelihood of their being harmed by organochlorines. First, many bat species form large roosting aggregations (maternity or hibernation) and thereby become vulnerable to direct applications of chemicals. The probability of this is increased for some species because their maternity aggregations often occupy human dwellings. Second, because bats fly and are active at night, they may be directly exposed to chemicals that are sprayed in the evening. Third, the long life-spans of most bats allow more time for contact with dangerous levels of organochlorines, and the low reproductive rates associated with these long life-spans may cause slow recovery of decimated populations.

The precise effects of any of these characteristics on bat-pesticide interactions are not known, and some are undoubtedly more important than others. Nevertheless, collectively they suggest that insectivorous, temperate-region bats are vulnerable to organochlorine pollutants.

If diagnoses of death by organochlorine poisoning in wild bats are to be reliable, more species and compounds must be tested. Brain concentrations are the most accurate measurements for such diagnoses. However, when residues exceed certain concentrations in the carcass lipids (about 60–90 ppm for DDE), they begin to appear in the brain. From this point on, brain and carcass lipid concentrations are significantly correlated (Clark and Prouty 1976, 1977; Clark and Kroll 1977; Clark et al. 1978*a*,1978*b*; Clark and Stafford 1981). Thus it may be possible to estimate brain concentrations of organochlorines at time of death in bats from carcass lipids (Clark 1981), even from small amounts of lipid that remain in bats now kept as dry museum study skins. Peakall's (1974) study of eggs of peregrine falcons (*Falco peregrinus*) clearly suggests that ppm lipid values for carcass fat of bats could be obtained by analysis either of small pieces of skins or of solvents used to rinse the inside of such skins. This is a noteworthy prospect in situations where large numbers of bats died of unknown causes (e.g., Villa R. 1955).

Possible reproductive effects of organochlorines to bats have been the subject of a single feeding study (Clark 1978; Table 3). PCB (Aroclor 1260) was fed to 18 of 36 pregnant big brown bats collected 21–23 May 1975 in Laurel, Maryland. This study was conducted because earlier data (Clark and Lamont 1976*b*) showed an association between stillbirths and higher PCB levels in neonate big brown bats. The experimental feeding raised PCB levels in both females and neonates nearly 10 times above levels in controls, but no additional stillbirths (or other effects) occurred (Clark 1978). Apparently the stillbirths resulted from unknown difficulties in young, reproductively inexperienced female bats. Because PCB levels were highest in this age group, there appeared to be a cause-and-effect relationship between stillbirths and PCB's (Clark 1978). The issue of possible reproductive effects of PCB's (or other organochlorines) on bats is certainly not closed. Recent data from little brown bats (Clark and Krynitsky 1978) indicated an association between high PCB levels and abnormally small or stillborn young that was not fully attributable to young, inexperienced females.

Possible metabolic effects of organochlorines on bats were suggested by significant differences in weight between dosed and control bats at the end of dosage periods during which approximately equal weights of food were fed to all bats (Clark and Kroll 1977; Clark and Prouty 1977; Clark and Stafford 1981). DDE enhanced weight gain in big brown bats (Clark and Prouty 1977), depressed it in Mexican free-tailed bats (Clark and Kroll 1977), and had no effect in little brown bats (Clark and Stafford 1981). PCB (Aroclor 1254) depressed weight gain in big brown bats (Clark and Prouty 1977), enhanced it (Aroclor 1260) in little brown bats (Clark and Stafford 1981), but had no effect on weights (Aroclor 1260) of pregnant big brown bats (Clark 1978). These results are contradictory; however, the studies differed in the amount of fat the bats gained, the level of chemical fed, and the type of PCB used. Thus consistent trends are probably not to be expected.

A potentially more troublesome effect of organochlorines occurred in relation to loss of weight after dosage. When dosage periods ended, surviving bats were starved to mobilize residues stored in fat (Clark and Kroll 1977; Clark and Prouty 1977; Clark

and Stafford 1981). Weights were recorded at the beginning and end of starvation. Rates of weight loss were calculated by using the total weight lost and the number of days required for the bat to starve. Comparisons of the rates between dosed and control bats suggested significant differences (Clark and Kroll 1977; Clark and Prouty 1977), but they were artificial and the result of differences in survival times without food. However, when the differences in survival times were accounted for by use of covariance analysis, little brown bats dosed with DDE lost weight significantly faster than controls (Clark and Stafford 1981). Measurements of oxygen consumption in fasted short-tailed shrews showed rates 20% higher in animals that had received dietary DDE than in controls (Braham and Neal 1974). These few data suggest that stored DDE in fat of hibernating bats may increase the rate at which the fat is metabolized and thereby cause premature depletion of fat reserves. The large numbers of dead and dying gray bats found in May in two Missouri hibernation caves (Davis 1965) are suggestive of mortality from premature fat depletion. Also, studies in which metabolic rates are measured (e.g., Riedesel and Williams 1976; Studier and O'Farrell 1976) could be affected by stored residues.

Tooth wear in captive big brown bats was not affected by low dietary levels of PCB (Clark 1978); thus there was no suggestion that the chemical affected behavior relative to chewing on the wire mesh of the cages. However, studies with rodents have shown that organochlorines may have several kinds of behavioral effects. Laboratory rats fed dietary DDT appeared normal except for a decreased ability to swim in cold water (Durham 1967). A single dose of DDT (2.5 mg/kg) given to pregnant laboratory mice depressed the acquisition of a conditioned avoidance response in their young (Al-Hachim and Fink 1968). DDT in the diet (7.0 ppm) of laboratory mice reduced their normal aggressiveness toward other mice (Peterle and Peterle 1971). Dietary dieldrin (10 ppm) slowed the onset of maternal nursing behavior in female laboratory mice (Virgo and Bellward 1977), and caused deer mice (*Peromyscus leucopus*) to be more active than controls and thereby behave in ways that made them more susceptible to avian predators (Bildstein and Forsyth 1979). There have been no studies devoted to effects of organochlorines on bat behavior, but these data on rodents suggest that such effects are possible.

Residue Surveys

Investigators have measured organochlorine residues in bats of 25 species from five countries: United States, Australia, Nigeria, England, and Mexico (Table 5). Three more studies (Braaksma and van der Drift 1972; Clark et al. 1978*a;* Voûte 1980) reported residues that would bring the totals to 27 species and six countries (adding the Netherlands), but because these studies involved roosts to which organochlorines were applied directly, they are discussed in an earlier section.

Eight reports (Constantine et al. 1968; Cockrum 1970; Reidinger 1972, 1976; Clark et al. 1975; Geluso et al. 1976, 1981; Reidinger and Cockrum 1978) dealt with residues in bats from the southwestern United States and adjacent Mexico. Residues generally were low with the principal pollutant, DDE, averaging less than 10 ppm in carcasses. However, in three instances residues appeared to have been lethal or were high enough to be potentially lethal. Five Mexican free-tailed bats found dead on the University of Arizona campus in March–April 1970 contained 2.4, 8.6, 220, 350, and 550 ppm of DDT as the principal residue when analyzed as whole animals (Reidinger 1972, 1976). The last three values are high enough to indicate death from DDT. The residue data indicate exposure to recently applied DDT because of a high DDT to DDE ratio and because the o,p'-DDT isomer was detected. These bats may have been sprayed directly, occupied a recently sprayed roost, or fed on recently sprayed insects.

Five big brown bats from a Tucson house contained a median DDE level of 120 ppm in their carcasses and had a maximum of 160 ppm (Reidinger 1972, 1976). A pooled sample of brains from four of these contained only 9 ppm DDE; thus, the bats were in no immediate danger. However, Geluso et al. (1976) showed that when young Mexican free-tailed bats from a population with similar DDE levels (N = 8 carcasses, median 92 ppm, range 50–300 ppm; brains, median 3.7 ppm, range 1.5–17 ppm) were forced by food deprivation and exercise to metabolize stored fat, brain levels reached lethal or near lethal levels (N = 5, median 160 ppm, range 66–330 ppm). Thus the DDE in the big brown bats from Tucson may have been potentially lethal if sensitivity to DDE is similar in these two groups of bats.

The bats studied by Geluso et al. (1976) were collected at Carlsbad Caverns in August 1974, and the results of this experiment clearly suggest that the stress of fall southward migration may cause stored DDE to reach lethal levels in brains of young Carlsbad bats. Furthermore, these data suggest that DDE may have played a role in the decline of the Mexican free-tailed bat colony at Carlsbad from 8.7 million in 1936 to 200,000 in 1973 (Geluso et al. 1976).

Food chain buildup usually causes predatory animals to contain higher levels of organochlorines than herbivorous animals where both occur in the

Table 5. *Surveys reporting organochlorine residues in bats.*[a]

Locality, reference (in parentheses)[b], and species	Materials analyzed	Principal residues
Carlsbad Caverns, New Mexico (10)		
Tadarida brasiliensis	Not reported	DDE, DDT[c]
Eagle Creek Cave, Arizona (9)		
Tadarida brasiliensis	Whole bat[d]	DDE, DDT[e]
Northeastern Nigeria (16)		
Hipposideros commersoni	Liver, fat	DDE, dieldrin[f]
Scotophilus nigrita	Liver	DDE, dieldrin[f]
Southeastern Arizona and northern Sonora, Mexico (17)[g]		
Antrozous pallidus	Carcass	DDE, DDT, dieldrin[e,f]
Eptesicus fuscus	Carcass, embryo, brain, liver, GI tract	DDE, DDT, dieldrin[e,f]
Tadarida brasiliensis	Carcass, whole bat, embryo, brain, liver, GI tract, mammary, guano	DDT, DDE, DDD[e,f]
Leptonycteris sanborni	Carcass, embryo, brain, liver, GI tract	DDE, DDT, DDD[e,f]
Macrotus waterhousii	Carcass, GI tract	DDE, DDT, dieldrin[e,f]
Pipistrellus hesperus	Carcass, GI tract	DDE, DDT, dieldrin[e,f]
Eastern England (15)		
Pipistrellus pipistrellus	Liver, whole bat[d], fat	DDE, DDT, dieldrin[f,h]
Myotis nattereri	Liver, whole bat[d]	DDE, DDT, dieldrin[f,h]
Myotis daubentoni	Liver, whole bat[d]	DDE, DDT[f,h]
Plecotus auritus	Liver, whole bat[d]	DDE, DDT, dieldrin[f,h]
North and central Australia (1)		
Pteropus alecto	Fat	DDT, lindane, DDE[f]
Eptesicus pumilis	Whole bat (petroleum extract)	Dieldrin, DDE, lindane[f]
Taphazous georgianus	Fat	DDE, dieldrin[f]
Southeastern Australia (11)		
Miniopterus schreibersii	Carcass	Only total DDT's were measured[h]
Bracken Cave, Texas (6)		
Tadarida brasiliensis	Carcass, brain, embryo, milk, guano, masticated insects	DDE, dieldrin, DDT[f,h]
Gaithersburg, Maryland (3)		
Eptesicus fuscus	Carcass, brain, milk, masticated insects	PCB, DDE, DDT[f,h]
Laurel, Maryland (4)		
Eptesicus fuscus	Carcass, whole bat (neonates)	PCB, DDE, DDT[f,h]
Maryland and West Virginia (5)		
Myotis lucifugus	Carcass, brain, guano, masticated insects	DDE, PCB, DDT[f,h]
Eptesicus fuscus	Carcass, brain, masticated insects	DDE, PCB, DDT[f,h]
Pipistrellus subflavus	Carcass, brain	DDE, PCB, DDT[f,h]
Carlsbad Caverns, New Mexico (12)		
Tadarida brasiliensis	Carcass, brain	DDE, DDD, DDT[f]
Northeastern Florida (18)		
Nycticeius humeralis	Whole bat[d]	Only mirex was measured[f]
Lasiurus borealis	Whole bat[d]	Only mirex was measured[f]
Franklin County, Missouri (7)		
Myotis grisescens	Carcass, brain, milk	Dieldrin, DDE, heptaclor epoxide[e,f]

Table 5. (continued).

Locality, reference (in parentheses)[b], and species	Materials analyzed	Principal residues
Laurel, Maryland (2)		
Myotis lucifugus	Carcass, whole bat (neonates)	PCB, DDE, oxychlordane[f]
Franklin County, Missouri (8)		
Myotis grisescens	Carcass, brain	Dieldrin, heptachlor epoxide, oxychlordane[e,f]
Eastern Oregon (14)		
Myotis californicus	Carcass, brain	DDE, DDT, DDD[f]
Myotis volans	Carcass, brain	DDE, DDT, DDD[f]
Myotis evotis	Carcass, brain	DDE[f]
Eptesicus fuscus	Carcass, brain	DDE, DDT[f]
Lasionycteris noctivagans	Carcass, brain	DDE, DDT, DDD[f]
Carlsbad Caverns; Eagle Creek Cave; Bracken Cave; and Newman Bridge, California (13)		
Tadarida brasiliensis	Carcass, brain	DDE, DDT, dieldrin[f,h]

[a] Three other studies reported organochlorine residues in bats (Braaksma and van der Drift 1972; Clark et al. 1978*a*; Voûte 1980) but these involved roosts to which organochlorines were applied directly and are, therefore, discussed in an earlier section.

[b] References: (1) Best 1973; (2) Clark and Krynitsky 1978; (3) Clark and Lamont 1976*a*; (4) Clark and Lamont 1976*b*; (5) Clark and Prouty 1976; (6) Clark et al. 1975; (7) Clark et al. 1978*b*; (8) Clark et al. 1980; (9) Cockrum 1970; (10) Constantine et al. 1968; (11) Dunsmore et al. 1974; (12) Geluso et al. 1976; (13) Geluso et al. 1981; (14) Henny et al., unpublished data; (15) Jefferies 1972; (16) Koeman et al. 1971; (17) Reidinger 1972; (18) Wheeler et al. 1977.

[c] Not stated whether residues in ppm wet weight or ppm lipid weight.

[d] Perhaps carcass, author was not specific. "Carcass" designates what remains after at least the skin is removed; in most studies some or all of the head, gastrointestinal tract, wings, feet, and tail were also removed.

[e] Residues in ppm lipid weight.

[f] Residues in ppm wet weight.

[g] The data for adult carcasses (Reidinger 1976) and for free-tailed bats from Eagle Creek Cave, Arizona (Reidinger and Cockrum 1978) were published separately.

[h] Residues in total micrograms.

same habitat. Reidinger (1972, 1976) confirmed this in bats when he found that the nectar-pollen feeding long-tongued bat (*Leptonycteris sanborni*) contained lower levels than the four insectivorous species that he studied.

Cockrum (1969, 1970) and Reidinger and Cockrum (1978) documented the decline of a maternity colony of the Mexican free-tailed bat at Eagle Creek Cave in eastern Arizona from 50–100 million bats in 1964 to about 30,000 bats in 1969 and suggested that DDT's may have been the causative agent. Reidinger and Cockrum (1978) noted that DDT sales in Arizona jumped to a high of 2.52 million pounds (1,143 metric tons) in 1967, and that farmers in Safford Valley, where bats from Eagle Creek Cave are known to feed, encountered their first severe attacks on cotton by the pink bollworm (Coleoptera, *Pectinophora gossypiella*) in 1966–67. Therefore, Reidinger and Cockrum (1978) believe that this bat population probably received peak exposure to DDT in 1967–68. They point out that the timing of these events corresponds with reports by farmers living near Eagle Creek Cave of unusually high numbers of dead and dying bats in their fields in the summer of 1968 (Table 1). These authors also note that Arizona placed a moratorium on DDT use in 1969 and that bats increased in Eagle Creek Cave to 250,000–325,000 in 1970.

These observations suggest that DDT caused the 1968 die-off and the population decline, but because none of the 1968 bats found dead or dying was analyzed for pesticides, conclusive data are not available. However, analyses were performed on bats and guano collected in 1969 and 1970 (Cockrum 1970; Reidinger 1972; Reidinger and Cockrum 1978). Suckling young free-tailed bats often fail to cling to the ceilings and walls of the caves where they are born, and many fall to the floor where they die. Seventeen such fallen young collected in Eagle Creek Cave in June 1969 and analyzed as a single pooled

sample contained only 6 ppm DDE in their fat (Cockrum 1970; Reidinger and Cockrum 1978). If DDT had caused a major die-off in this population in 1968, DDT, not DDE, probably would have been the principal residue in these young. Geluso et al. (1976) found flying young free-tailed bats (N = 8) at Carlsbad with 92 ppm DDE wet weight (range 50–300 ppm) in their carcasses, but these authors did not report unusually large numbers of fallen young relative to the colonies at Eagle Creek Cave, Bracken Cave in Texas, or Newman Bridge in California (Geluso et al. 1981).

Eight pregnant female free-tailed bats collected in Eagle Creek Cave in June 1970 contained 4.3 ppm DDE (range 2.8–27 ppm) and 0.2 ppm DDT (range 0.1–0.4 ppm) in their carcasses, and seven fallen young, which were analyzed whole except for removal of the lower jaw, contained 1.9 ppm DDE (range 1.0–6.5 ppm) and less than 0.1 ppm DDT (Reidinger and Cockrum 1978). Levels associated with death are much higher than these. Six pregnant little brown bats killed by DDT contained 12.4 ppm DDT (range 2.2–30 ppm) in their carcasses and eight young contained 24.0 ppm (range 8.6–54 ppm; Clark et al. 1978*a*). Eight adult free-tailed bats killed by DDE contained 870 ppm DDE (range 620–1,200 ppm) in their carcasses (Clark and Kroll 1977). Considering the persistence of these chemicals, it seems improbable that levels could have decreased to those found in 1970 at Eagle Creek Cave, if the population had suffered major DDT mortality only two summers earlier.

C. J. Henny et al. (unpublished data) reported residues of DDT and its metabolites in five bat species in Oregon at 1, 2, and 3 years after their forest habitat was sprayed once with 0.84 kg/ha of DDT. DDT residues in four species 2 years after the spray were 0.34, 0.12, 0.35, and 0.09 ppm (a mean for the fifth species was not computed because DDT was present in only two of six bats). This application, which is not known to have killed any bats and which is well below rates used on cotton, resulted in residues of DDT in bats similar to those found in free-tailed bats at Eagle Creek in 1970 (Reidinger and Cockrum 1978). Residues reported by C. J. Henny et al. (unpublished data) show that the ratio of DDT to DDE, in the four species where both chemicals could be measured, averaged 0.13 at 2 years postspray. The same ratio for pregnant females from Eagle Creek was 0.05. The relatively greater amount of the metabolite DDE in Eagle Creek bats indicates that exposure to DDT occurred much longer ago than in the Oregon bats.

Levels of organochlorines in bat guano correspond to levels in the bats themselves (Clark and Prouty 1976). Mortality in gray bats caused by dieldrin (Clark et al. 1978*b*) was first suspected because of high dieldrin residues found in guano from the roost caves. Reidinger (1972) removed a 76.2-cm column core of guano at Eagle Creek Cave in 1970 and analyzed 15 samples from it at 5.1-cm intervals. The results showed only low levels of DDE, with amounts declining significantly with increasing depth (Reidinger 1972). The span of years represented by these results was not stated but periods of high residue levels were not evident.

This evaluation of the residue data from the Eagle Creek population offers no support for the hypothesis that DDT caused the observed population decline. Nevertheless, DDT was often applied to cotton in combination with methyl parathion (an organophosphate that leaves no long-lived residues), and DDE has been shown to act synergistically with parathion to increase mortality in Japanese quail, *Coturnix coturnix* (Ludke 1977). Thus methyl parathion, by itself or in combination with low DDE-DDT levels, might have caused mortality that corresponded with increased spraying activity but that left only the observed residues.

Reidinger (1972) and Reidinger and Cockrum (1978) suggested that residues of DDT and its metabolites might cause more suckling free-tailed bats to lose their grip and fall. Clark et al. (1975) compared organochlorine residues in young free-tailed bats that had fallen with those still attached to the ceiling at Bracken Cave in south central Texas and found no evidence that residues increased the likelihood of falling. However, residues were low (6.67 ppm DDE in carcasses of 12 fallen young, 5.26 ppm DDE in 15 young from the ceiling), and it is undoubtedly true that if residues increased sufficiently, more young would fall. The residue data of Geluso et al. (1976, 1981) for Carlsbad free-tailed bats show that levels of DDE in a population would have to be very high before such an effect became apparent. Comparisons of residues in fallen young and young collected from the ceiling in the Carlsbad population might document such an effect, but large samples, perhaps hundreds or thousands of bats, might be required because several factors (e.g., starvation, disease, clumsiness) can cause young to fall.

Among female Mexican free-tailed bats at Bracken Cave it appeared that young of the year nearing the end of their nursing period contained the highest DDE levels (Clark et al. 1975). Levels appeared slightly lower when they returned to Bracken Cave as yearlings (as indicated by tooth wear), but then dropped sharply with their first lactation. From the latter low point DDE increased with age but amounts generally did not exceed those in yearlings. Residues were measured in milk taken from nursing young (Table 6).

Geluso et al. (1981) surveyed residues in flying

Table 6. *Organochlorine residues (ppm wet weight) in milk recovered from stomachs of young bats.*

Species, locality, and reference (in parentheses) [a]	DDE	DDD	DDT	Dieldrin	Hepta-chlor epoxide	Oxy-chlor-dane	Misc. chlor-danes	PCB (Aroclor 1254)
Tadarida brasiliensis Bracken Cave, Texas (2)	2.72 0.83–10.1[b]	ND	ND	0.45, 0.87[c]	ND	ND	ND	ND
Eptesicus fuscus[d] Gaithersburg, Maryland (1)	2.3	ND	0.58	ND	ND	ND	ND	8.8
Myotis grisescens[e] Franklin County, Missouri (4)	ND	ND	ND	36	4.1	0.58	2.3	ND
Myotis lucifugus[f] Near Amherst, New Hampshire (3)	17	7.7	21	ND	0.96	1.9	15.8	11

[a] References: (1) Clark and Lamont 1976*b*; (2) Clark et al. 1975; (3) Clark et al. 1978*a*; (4) Clark et al. 1978*b*.
[b] Geometric mean and range of 11 samples.
[c] Concentrations in 2 of 11 samples; ND in other 9 samples.
[d] Milk from four bats pooled and analyzed as a single sample.
[e] Milk from two bats pooled and analyzed as a single sample.
[f] Milk from five bats pooled and analyzed as a single sample.

young-of-the-year Mexican free-tailed bats collected 1973–76 at Carlsbad Caverns (New Mexico), Eagle Creek Cave (Arizona), Bracken Cave (Texas), and Newman Bridge in California. Their results emphasized the dangerously high levels of DDE at Carlsbad (geometric means for carcasses reaching 101 ppm in 1974, DDT at 0.46 ppm) contrasted to low levels (geometric means reaching only 13 ppm, DDT at 2.09 ppm) at the other sites. Seventeen free-tailed bats taken from Carlsbad Caverns in 1955 showed 5 ppm of DDT and 11 ppm of DDE (Constantine et al. 1968). If these concentrations are on a wet weight basis for carcasses, the DDT value of 5 is within the range (2.2–30 ppm) found in adult little brown bats that died of DDT poisoning (Clark et al. 1978*a*). However, if these concentrations are on a lipid weight basis, or are from analyses of fat bodies, they are too low to suggest mortality, which seems more likely because these residues were not associated with mortality in the population. In either alternative, the high ratio of DDT to DDE, in contrast to the 1973–76 data, indicates recent exposure to DDT.

Evidence from the most recent surveys (1976–77) of organochlorines in birds (White 1979*a*, 1979*b*) suggested that eastern New Mexico was the region of the United States most heavily contaminated with DDE. Thus the residue problem in the Carlsbad colony may originate in New Mexico rather than at some point, or points, along these bats' extensive southern migration route. Residue data for this 1973–76 period are suggestive of a decline in DDE levels at Carlsbad (Geluso et al. 1981), but data for more years are required for a definitive conclusion.

Dieldrin caused mortality in two maternity colonies of the endangered gray bat in Franklin County, Missouri, in 1976 and 1977 (Clark et al. 1978*b*, 1980). These were the first reports to link field mortality of bats directly to insecticide residues acquired through the food chain. In this instance aldrin, dieldrin's parent compound, was applied to cornfields to control cutworms (larval moths, family Noctuidae). Residues were transferred to young from the milk (Table 6). Brain levels of dieldrin from 4.6 to 13 ppm appeared to be lethal because they resembled experimentally determined lethal levels in other species of mammals (Clark et al. 1978*b*, 1980). The range of carcass levels associated with these lethal brain levels was 4.3 to 100 ppm.

Between 1976 and 1977 heptachlor epoxide, oxychlordane, *cis*-chlordane, and *trans*-nonachlor increased significantly in brains and carcasses of dead bats from the more severely affected colony, leading to the conclusion that heptachlor was being substituted for aldrin in this area (Clark et al. 1980). Maximum brain levels of heptachlor epoxide (3.7 ppm) and oxychlordane (2.3 ppm) were at the low end of the known lethal range for birds (Stickel et al. 1979). Additional data show that this mortality continued in 1978; the more strongly affected colony could not be located in 1979.

In England, Jefferies (1972) measured residues from 30 bats of four species collected between 1963

and 1970. Twenty-four of these bats were pipistrelles and 18 were collected in 1969. Although nine bats were dead or in poor health when collected, the highest residues were in four male pipistrelles collected alive in Ramsey, Huntingdonshire, on 31 March 1969. DDE in livers of these four averaged 38.5 ppm (geometric mean) and reached as high as 53.7 ppm. When compared with the value of 90 ppm DDE in four pooled livers of big brown bats from Tucson (Reidinger 1972), the residues in England suggest a lesser danger to the bats. However, Jefferies (1972) also found the highest levels of DDT in the livers of the four bats from England (geometric mean 17.9 ppm, highest value 28.6 ppm). DDT was only 0.6 ppm in the pooled livers of the big brown bats from Tucson (Reidinger 1972) but the comparison is not absolute because Jefferies (1972) added results for DDT and DDD and his analyses did not separate or quantify PCB's. Both factors would increase the apparent DDT.

Jefferies (1972) fed DDT to 19 pipistrelles and one whiskered bat and recorded DDT as low as 38.85 ppm (geometric mean 160.1 ppm, maximum value 747.7 ppm) in livers of eight bats that died of DDT. There was no overlap between this range and the DDT range of those from the field. The mean for the eight that died is nearly nine times that for the four most heavily contaminated field bats. Thus the risk of lethality to English bats seems small.

In Maryland and West Virginia, residues of organochlorines from 119 bats of three species collected at four localities in 1973 showed several patterns (Clark and Prouty 1976). Residues of DDE, DDD, and DDT were highest in carcasses from Round Top Mountain, Maryland, a locality surrounded by apple orchards. DDE averaged (geometric mean) as high as 38.5 ppm (N = 3, maximum 87 ppm) and DDT averaged as high as 2.45 ppm (N = 3, maximum 5.0 ppm) in the same group of little brown bats. Residues were lowest in bats from Trout Cave, West Virginia, a locality surrounded almost entirely by forest and devoid of agriculture or industry. PCB's (Aroclor 1260) and oxychlordane were highest at Montpelier Barn in Laurel, Maryland, the most urbanized locality. A sample of five little brown bat carcasses averaged (geometric mean) 11.6 ppm PCB (maximum 21 ppm); oxychlordane had a geometric mean of 1.52 ppm (maximum 2.7 ppm) in the same group. The little brown bat usually had the highest residue concentrations, the big brown bat had the lowest, and the eastern pipistrelle was intermediate.

Age (as indicated by tooth wear), sex, and time of year (spring vs. fall) affected the residue results to various degrees (Clark and Prouty 1976). In reference to age, one of four possible tests produced a significant regression in which DDE declined with increased tooth wear among 12 male eastern pipistrelles collected at Trout Cave. In evaluating the difference between sexes, 6 of 47 possible comparisons based on total μg in bat carcasses were significant and all 6 showed more residue in males. Five of the six tests were for a sample of seven (four males, three females) big brown bats collected at Montpelier Barn in spring; residues were PCB, DDE, DDT, DDD, and oxychlordane. The sixth was for nine (six males, three females) big brown bats collected at Round Top Mountain in spring, and the residue was DDD. This same bat sample from Montpelier Barn also showed significantly more residue in males for the same five chemicals when ppm were compared. The ppm concentration of DDT was also significantly greater in male big brown bats in the fall sample (two males, three females) collected at Montpelier Barn. Clark and Prouty (1976) speculated that lactation played a role in producing these differences between males and females.

Seasonal differences in μg of residue in carcasses showed no consistent trend (Clark and Prouty 1976). However, when comparisons were based on ppm concentrations, 12 of 58 possible tests showed significant differences; residue concentrations were always higher in spring. The 12 significant tests included five chemicals (PCB, DDE, DDD, DDT, and oxychlordane), both male and female bats, all three species (big brown bat, little brown bat, eastern pipistrelle), and all four localities. The higher levels occur in early spring when the residues are concentrated in the greatly reduced fat reserves present at that time. In brains, DDE reached as high as 3.5 ppm in little brown bats, 1.0 ppm in eastern pipistrelles, and 0.82 ppm in big brown bats. These residue levels appeared to pose no threat to the populations.

Organochlorine residues were measured in 18 big brown bats collected from a house attic in Gaithersburg, Maryland, in June 1973 (Clark and Lamont 1976*a*). The bats included 11 recently parturient females, 6 of which had a total of 7 nursing young, ranging from 1 to 13 days old. Geometric means in carcasses of adult females were 0.70 and 0.54 ppm for PCB (Aroclor 1260) and DDE; comparable means for the young were 1.43 and 1.85 ppm. A pooled milk sample from stomachs of nursing young contained PCB, DDE, and DDT (Table 6). Large amounts of residues were probably transferred by lactation to the young. Amounts of PCB and DDE in young increased with the weight of the young. Although lactation transferred DDE more readily than PCB (Clark and Lamont 1976*a*), another study of big brown bats (Clark and Lamont 1976*b*) demonstrated that PCB crossed the placenta more readily than DDE.

PCB in females and their young declined with increased age (as indicated by tooth wear; Clark and Lamont 1976*b*). The cause of smaller PCB residues among older females is unknown. This same trend was observed in two other series of big brown bats from Maryland (Clark and Lamont 1976*a*; Clark 1978).

Among the 18 brains of bats collected in Gaithersburg (Clark and Lamont 1976*a*), only those of three young contained detectable residues. One had 8.2 ppm DDE, another 4.8 ppm PCB (Aroclor 1260), and the third 11 ppm PCB (Aroclor 1248). Aroclor 1248 is rarely found in bat tissues and may have been accidentally introduced during the dissection and analysis process.

In the forests of eastern Oregon, organochlorine residues were measured in bats of five vespertilionid species (Table 5) from sprayed and control areas 1, 2, and 3 years after a 1974 aerial spraying of DDT (0.84 kg/ha) for control of tussock moths, *Orgyia pseudotsugata* (C. J. Henny et al. unpublished data). Residues of DDE, DDT, and DDD were higher in bats from the sprayed area. DDE, the principal residue, ranged from a maximum geometric mean of 5.42 ppm (N = 19) in carcasses of the long-legged myotis (*Myotis volans*) 1 year after spraying to a minimum of 0.16 ppm (N = 8) in the long-eared myotis (*Myotis evotis*) 3 years after spraying. Means for DDE exceeded those for DDT by 8 times (range 1.3 to 30.5 times), and levels of DDD were still lower with all means below 0.48 ppm. DDE was found in brains of some bats but amounts were less than 2.5 ppm.

Residues peaked at 1 year postspray in the long-legged myotis, the California myotis (*Myotis californianus*), and the big brown bat, and at 2 years postspray in the silver-haired bat (*Lasionycteris noctivagans*) and the long-eared myotis (C. J. Henny et al. unpublished data). Residues had almost returned to control levels at 3 years postspray. These residues posed no threat to the bats; however, this single spray produced residue levels that remained elevated for at least 3 years. It is unknown whether higher residues immediately after spraying caused harm to the bats, but no mortality was observed.

Organochlorine residues in Australian bats have been reported by two studies, neither of which separated PCB's. Best (1973) collected mammals, birds, reptiles, and fish from May 1970 to December 1971 in two areas of the Northern Territory. Near Darwin on the tropical north central coast, she collected 54 black flying foxes (*Pteropus alecto*). Thirty-one of these were from developed areas within 90 km of Darwin; the other 23 were from undeveloped areas farther from the city. Carcass fat was pooled and three pools from developed areas and two from undeveloped areas were analyzed. In arid central Australia, Best (1973) collected 18 little brown bats (*Eptesicus pumilis*) and four free-tail bats (*Taphazous georgianus*) from developed areas within 20 km of Alice Springs. The little brown bats were analyzed as two pooled samples of light petroleum extract of the whole animal. Fat samples from the four free-tail bats were pooled and analyzed as a single sample.

The samples from black flying foxes collected in undeveloped tropical areas contained no detectable residues (Best 1973). In contrast, two of three pools of black flying foxes from developed tropical areas contained measurable residues of DDT, DDE, and lindane, but amounts reached only 0.10 ppm. From the arid area the single sample of free-tail bats contained only 0.06 ppm DDE and 0.05 ppm dieldrin, whereas pools from little brown bats contained as much as 0.25 ppm DDT, 1.82 ppm DDE, 4.03 ppm dieldrin, 0.31 ppm lindane, 0.06 ppm HCB, and 0.12 ppm endrin. Among these various samples, the light petroleum extract was used only for the little brown bats. Whether this extraction method altered the residues measured is not known. Overall, levels were low and those of bats were comparable to or less than those of other mammals, birds, reptiles, and fish sampled.

Dunsmore et al. (1974) collected 116 bent-winged bats in 1971 and 1972 from four localities in New South Wales. Carcasses were analyzed only for total DDT's and results were presented as arithmetic means of total μg representing different localities, age groups (adults, juveniles), and collection dates. Means for body weight were also given, so I was able to calculate mean ppm. Levels were higher in adults than in juveniles but were very low in both; the highest average concentration was about 1.41 ppm.

In northeastern Nigeria, 17 birds of seven species (six insectivores, one insectivore-herbivore) and two bats of two species (both insectivores) were collected in November 1970 (Koeman et al. 1971). Portions of the area sampled had been treated with dieldrin or DDT in 1969 and 1970 to control tsetse flies *(Glossina).* Analyses of livers from individual birds measured dieldrin from 0.003 to 2.4 ppm (or 0.007 to 2.4 ppm if the one insectivore-herbivore is not considered) and DDE from 0.018 to 1.2 ppm. Levels in the two bats were contained within these ranges but were near the low extremes (i.e., leaf-nosed bat, *Hipposideros commersoni,* dieldrin 0.073 ppm, DDE 0.08 ppm; yellow house bat, *Scotophilus nigrita,* dieldrin <0.020 ppm, DDE 0.17 ppm).

In northeastern Florida mirex was applied in July 1972 and residues were measured in birds and bats collected immediately before, 1 year after, and 2 years after application to the study area (Wheeler et al. 1977). Pools of two red bats and two evening bats collected before application showed no mirex. A

single evening bat collected 1 year postspray also contained no mirex, and a pool of two evening bats collected 2 years postspray contained only 0.09 ppm. These levels resembled those for insectivorous birds.

Clark and Lamont (1976*b*) and Clark and Krynitsky (1978) attempted to determine whether the birth of occasional stillborn young in nursery colonies of big and little brown bats was related to organochlorine levels. In the first study 26 pregnant big brown bats caught May–June 1974 in Montpelier Barn, Laurel, Maryland, were kept in individual cages. Females and litters (two young per litter) were frozen at parturition and later analyzed for organochlorines. In females PCB (Aroclor 1260) and DDE measured as much as 3.6 and 3.5 ppm; in litters they reached 3.3 and 0.84 ppm. Seven young in 5 litters were born dead; 21 litters contained only living young. Concentrations of only PCB were significantly higher in litters with dead young than in litters where both young were born alive; thus it appeared that PCB might have been responsible for the deaths. However, Clark and Lamont (1976*b*) and Clark (1978) demonstrated a significant relation between age (tooth wear) and PCB in which the youngest adult females had the highest residues. Therefore when experimental feeding of PCB to pregnant big brown bats increased levels in both females and young nearly 10 times over controls without inducing additional stillbirths (Clark 1978), it was concluded that higher PCB levels were merely associated with stillbirths because both were most common among young, reproductively inexperienced females.

The second study (Clark and Krynitsky 1978) began in June 1976 when 45 pregnant little brown bats were collected at Montpelier Barn. Procedures were identical to those with big brown bats. PCB averaged (geometric means) 11.4 ppm in adult females and 4.16 ppm in young (one young per litter); maximum levels were 24 and 25 ppm. DDE averaged (geometric means) 1.65 ppm in adults and 0.50 ppm in young. Among 43 females that gave birth, 12 produced dead young that contained more than twice as much PCB as did live young (6.68 ppm vs. 3.04 ppm) but the difference was not significant. Levels of DDE were almost identical in dead and live young. Also, 11 of 12 dead young weighed less than live young and there was a significant overall negative relation in newborn bats between weight (as a percentage of female weight) and PCB concentration. In spite of the overall relation, 7 of the 11 small dead neonates contained only low (≤ 7 ppm) levels of PCB. Even though there was no correlation in the adult females between age (tooth wear) and PCB level, five of the seven female parents of these small dead young with low PCB levels showed no tooth wear and were probably yearlings producing their first offspring. This ratio (5 of 7) was significantly greater than the corresponding ratio (9 of 30) among females that produced young of normal weight. Most of the small dead neonates were attributable to unknown reproductive difficulties associated with first pregnancies. However, the female parents of the four small dead young that contained the highest residues showed tooth wear. In conclusion, PCB cannot be completely ruled out as a cause of stillbirths until feeding studies are conducted.

Metals

Studies of metal pollutants and bats have been, with three exceptions, field surveys of residues. Mercury has been the metal most often investigated (Table 7). Levels in breast muscle from five species collected in southeastern Arizona and adjacent Mexico were low; the averages were 0.218 ppm for five big brown bats, 0.130 ppm for two western pipistrelles (*Pipistrellus hesperus*), 0.074 ppm for five Mexican free-tailed bats, 0.03 ppm for five desert pallid bats (*Antrozous pallidus*), and 0.0065 ppm for four long-tongued bats (Reidinger 1972). Among the five big brown bats, two males contained higher levels (0.32 and 0.31 ppm) than three females (0.15, 0.15, and 0.16 ppm; Reidinger 1972).

Petit and Altenbach (1973) measured mercury in a core of Mexican free-tailed guano taken from the same locality, Eagle Creek Cave in Arizona, where Reidinger (1972) collected his free-tailed bats. The guano was stratified by year and mercury was measured for the 16 years from 1956 through 1971. Mercury levels ranged from a high of about 0.159 ppm in 1971 to a low of about 0.071 ppm in 1968 and were correlated with copper production 1 year earlier at a nearby copper smelter (Petit and Altenbach 1973). Mercury in guano was about 0.133 ppm in 1970 when Reidinger (1972) collected his bats. Petit and Altenbach (1973) believed the mercury entered the bats' food chain when moth larvae fed on vegetation and that this caused the 1-year lag. Reidinger (1972) proposed that most of the mercury measured in his bats came from their drinking water and not their food. Either route of contamination seems possible and the balance between them could vary with the bat population being sampled. The varying mercury levels in the guano (Petit and Altenbach 1973) showed no correlation with the precipitous decline in the free-tailed bat population that was recorded by Cockrum (1970) and Reidinger and Cockrum (1978) at Eagle Creek Cave between 1964 and 1969.

Japanese farmers used mercurial fungicides extensively from about 1953 to 1968, and the highest mercury levels thus far recorded in bats were from

Table 7. *Surveys reporting metal residues in bats.*[a]

Locality, reference (in parentheses)[b], and species	Materials analyzed	Metal reported
New York State (6)		
"Bat"	Heart, lung, liver, kidney, feet + wings, skin + fur, muscle	Selenium
Southeastern Arizona and Carbo, Mexico (5)		
Eptesicus fuscus	Breast muscle	Mercury
Pipistrellus hesperus	Breast muscle	Mercury
Tadarida brasiliensis	Breast muscle	Mercury
Antrozous pallidus	Breast muscle	Mercury
Leptonycteris sanborni	Breast muscle	Mercury
Eagle Creek Cave, Arizona (3)		
Tadarida brasiliensis	Guano	Mercury
Japan (2)		
Rhinolophus cornutus	Kidney, liver, brain, muscle, hair	Mercury
Rhinolophus ferrumequinum	Kidney, liver, brain, muscle, hair	Mercury
Miniopterus schreibersii	Kidney, liver, brain, muscle, hair	Mercury
Pipistrellus abrams	Liver, brain, muscle, hair	Mercury
Vespertilio superans	Liver, brain, muscle, hair	Mercury
Western Virginia (4)		
Pipistrellus subflavus	Liver, muscle	Mercury
Laurel, Maryland (1)		
Eptesicus fuscus	Whole bat[c], embryo, guano, masticated insects	Lead
Myotis lucifugus	Whole bat[c], embryo, guano, masticated insects	Lead

[a] All studies reported residues as ppm wet weight.
[b] References: (1) Clark 1979; (2) Miura et al. 1978; (3) Petit and Altenbach 1973; (4) G.V.N. Powell, unpublished data; (5) Reidinger 1972; (6) Schroeder et al. 1970.
[c] Minus gastrointestinal tract and large embryos; results also given as ppm dry weight.

individuals collected in Japan during these years (Miura et al. 1978). Using bats collected in 1890, 1965–67, and 1970–75, Miura et al. (1978) compared mercury levels before, during, and after the use of organomercuries. These comparisons utilized 66 bats of three species from eight localities in mountainous areas near farms. The bats included 36 lesser horseshoe bats (*Rhinolophus cornutus*), 19 greater horseshoe bats, and 11 bent-winged bats.

Among the lesser horseshoe bats were six preserved in alcohol since 1890; all other bats had been frozen or refrigerated until analysis (Miura et al. 1978). The six from 1890 contained the smallest amounts of mercury; levels in kidney were 0.11; liver, 0.19; muscle, 0.25; and hair, 2.63 ppm (Miura et al. 1978). The other 60 bats contained mercury in greater amounts but showed no difference between the period during usage of organomercuries and the period after. Average mercury levels reached as high as 0.99 ppm in kidney (N = 9, greater horseshoe bats), 1.15 ppm in liver (N = 11, lesser horseshoe bats), 0.35 ppm in brain (N = 9, greater horseshoe bats), 0.64 ppm in muscle (N = 9, greater horseshoe bats), and 10.5 ppm in hair (N = 5, bent-winged bats; Miura et al. 1978).

Bats of two other species collected during the usage period contained significantly higher residues apparently because they inhabited human dwellings in towns in the midst of farms (Miura et al. 1978). These species, the yellow-headed bat (*Pipistrellus abrams*, N = 2) and the frosted bat (*Vespertilio superans*, N = 2) contained average mercury levels that exceeded the maximum levels found in the other three species in nearly every instance. Even though levels were similar in the yellow-headed and frosted bats, average amounts in the frosted bat were higher in all four tissues: liver, 2.77; brain, 0.59; muscle, 1.06; and hair, 33.7 ppm (Miura et al. 1978). The authors postulated that mercury levels had not diminished significantly after use because of persistently high residues in fields or because the bats are long-lived and do not readily excrete stored mercury. The authors could not exclude the possibility that the 1890

specimens contained less mercury because it had eluted into the alcohol and was discarded as the preservative was replaced during the long storage period.

From 1954 to 1972 the North Fork of the Holston River in western Virginia received large amounts of mercury in waste water from a chlor-alkali factory. Six eastern pipistrelles collected in 1978 within 10 km downstream of the factory site evidenced this pollution (G.V.N. Powell, unpublished data). Average levels of 0.86 ppm in muscle and 1.92 ppm in liver resembled levels in birds of five insectivorous species collected along the same portion of the river (G.V.N. Powell, unpublished data) and were similar to the maximum averages reported by Miura et al. (1978).

There are no data available to evaluate the possible effects of these mercury residues on bats. However, none of these studies indicated that the bats had suffered any ill effects. Average residue levels in liver, kidney, muscle, and brain associated with death in three other mammalian species (mink, *Mustela vison;* ferret, *Mustela putorius;* and house cat, *Felis domesticus*) (Wobeser 1976) were all 10 times or more greater than the highest Miura et al. (1978) reported in bats.

Lead residues were measured in 18 big brown bats (8 males, 10 females) and 12 little brown bats (all females) captured at Montpelier Barn located 0.61 km from the Baltimore-Washington Parkway in Laurel, Maryland (Clark 1979). At the same time, series of meadow voles (*Microtus pennsylvanicus*), white-footed mice, and short-tailed shrews were collected both at the Barn and within 18 m of the Parkway. Geometric means for lead measured 46.55 ppm in male big brown bats, 31.49 ppm in female big brown bats, and 16.97 ppm in female little brown bats (Clark 1979). These values were significantly greater than comparable means from the terrestrial species except that lead in shrews caught at the Parkway averaged 26.20 ppm and this value did not differ significantly from any of these for bats (Clark 1979).

Reasons why bats accumulated more lead than white-footed mice and meadow voles are unknown. Possible explanations are (1) that the bats' diets coupled with their high rates of food consumption caused their total lead intake to exceed that of the other two species, (2) that the longer life-spans of bats allowed accumulation of more lead, or (3) that bats eliminate lead from their bodies at much slower rates than do the other two species. Evidence supporting any of these hypotheses is either inconclusive or nonexistent.

Lead was measured in embryos of both bat species but levels were not correlated either with amounts in the female parent or with weight of the litter (Clark 1979). There was no relation between age (tooth wear) and amount of lead. The average lead level was significantly higher in male than in female big brown bats. Big brown bats contained significantly more lead than little brown bats, but whether the lead exposure experienced by these bats had any ill effects is uncertain.

Previous observations at Montpelier Barn showed that stillborn young were occasionally produced by both species. Other studies (Clark 1978; Clark and Krynitsky 1978) showed that most stillbirths were produced by young females and were, therefore, attributable to unknown reproductive difficulties associated with first pregnancies rather than the result of a toxicant. Nevertheless, certain evidence suggests that the amounts of lead in these bats' diets and in their bodies were high enough to cause reproductive problems, sickness, or death.

Pooled samples of masticated insects from stomachs of 17 big brown bats and 12 little brown bats contained 3.8 and 26 ppm of lead (Clark 1979). When these values were combined with estimates of daily food intake, big brown bats appeared to consume about 1.0 mg/kg per day of lead, and little brown bats about 7.4 mg/kg per day (Clark 1979). Literature reports for domestic mammals indicate that pregnant sheep aborted while receiving 1 mg/kg per day of lead, and on dosages equal to or less than 7 mg/kg per day horses and cattle died, dogs suffered anorexia and convulsions, and mice showed reduced frequency of pregnancy or reduced frequency of ova implantation (Clark 1979). Authors in Wales reported renal inclusions and renal edema in field voles (*Microtus agrestis*) that contained body burdens of lead averaging 42.8 to 45.3 ppm and renal edema in field mice (*Apodemus sylvaticus*) that averaged 8.60 ppm (Roberts et al. 1978).

Lead poisoning caused the deaths of three Australian fruit bats (*Pteropus poliocephalus*) at the National Zoological Park, Washington, D.C. (Zook et al. 1970). The investigators concluded that the lead was ingested as paint chips. The dead bats contained both renal and hepatic inclusion bodies and analyses of liver tissue from two bats (tissues were not analyzed from the third bat) showed over 500 ppm (wet weight) of lead. Because this concentration is 5 to 10 times higher than concentrations reported in other animals killed by lead, Zook et al. (1970) speculated that bats of this species are relatively resistant to lead poisoning.

Selenium levels were measured in heart, lung, liver, kidney, feet plus wings, skin plus fur, and muscle of three bats of undetermined species from New York State (Schroeder et al. 1970). Levels in these tissues averaged 2.86, 0.77, 1.07, 1.17, 3.87, 4.0, and 0.49 ppm. One bat liver contained 3.34 ppm. The significance of these residues is unknown.

In one of two laboratory studies involving bats and metals, single subcutaneous injections of cadmium chloride at 0.04 Mol/kg given to 30 male rat-tailed bats (*Rhinopoma kinneari*) captured near Jaipur, India, caused necrosis of the testes with shrinkage of the seminiferous tubules but without loss of testicular weight (Dixit and Lohiya 1974). Laboratory mice given similar injections showed greater destruction of the seminiferous epithelium and significant loss of testicular weight. The rat-tailed bat was thus shown to be an exception to the generality that nonscrotal mammalian species are insensitive to cadmium salts (Dixit and Lohiya 1974).

In the other laboratory study, zinc phosphide was applied to little brown bats or to their artificial roosts (Fenton and Hurley 1979). This study also included experiments with DDT and was discussed in an earlier section.

Conclusions and Recommendations

Recent studies demonstrated that certain organochlorine insecticides received through the food chain caused, and appear to be continuing to cause, major populational mortality among gray bats in Missouri (Clark et al. 1978*b*, 1980) and probably among Mexican free-tailed bats in New Mexico (Geluso et al. 1976). In the United States, the organochlorines (DDT, dieldrin, endrin, and heptachlor) now causing, or likely to have caused, mortality were used much more extensively in the past than they are now. Thus it is probable that mortality, too, was much more extensive when greater quantities of these chemicals were being used. Unfortunately, opportunities for documentation of major mortalities are largely gone, even though museum specimens from die-offs and column cores of guano could, and should, be investigated.

To date only outright mortality on a local population level has been identified as a threat to bats from these pollutants. Subtle but equally devastating effects on such aspects as reproduction, acoustic behavior, and hibernation metabolism are certainly possible and should be studied. However, there is no evidence that any bat species suffers from a mammalian equivalent of the DDE-induced thin eggshell syndrome, which adversely affected populations of such birds as the peregrine falcon and brown pelican, *Pelecanus occidentalis* (Stickel 1973), or from severe reproductive impairment such as that caused by PCB in mink (Ringer et al. 1972; Platonow and Karstad 1973).

Corrective actions are needed where bats are threatened. Heptachlor is being substituted agriculturally for aldrin (dieldrin's parent compound) in Missouri, and residues of heptachlor are already accumulating to dangerous levels in gray bats (Clark et al. 1980). In this limited area of Missouri, other chemicals such as toxaphene, which is also recommended as a substitute for aldrin, or diazinon should be used for control of cutworms in corn. Cooperation among local, State, and Federal agricultural and wildlife agencies is needed to implement a program to protect colonies of the endangered gray bat in this region. The DDE problem at Carlsbad Caverns is more difficult to solve because the source, or sources, of the chemical is unknown. The limited information now available suggests that contamination is associated with the Pecos River. A sampling program that analyzes suitable animals collected at intervals along the river might reveal the source, or sources, of the DDE, and spread of the chemical might be stopped.

In the United States, DDT is currently approved for application to bat roosts in human dwellings. This use, however, is tightly controlled by regulatory agencies. Data summarized in this review clearly indicate that unless combined with permanent closure of roost entrances, the application of organochlorine chemicals has only limited, short-term effectiveness in reducing colony size in dwellings (of course, when roost entrances are closed, the chemical is unnecessary).

Public discussion of this use of DDT is usually emotional. Nevertheless, relative to other threats faced by bats, the continuance or discontinuance of this use is probably unimportant for several reasons. First, DDT is ineffective and only a small percentage of the treated colonies are exterminated. Second, the species to which it is applied are the big brown and little brown bats, both of which are extremely abundant and tenacious in the face of efforts to displace them. Finally, the total amount of DDT applied in this way is inconsequential in comparison to amounts applied agriculturally in years past. It appears that considering the low probability of eliminating a bat colony from a house, any benefit is more than offset by the unknown long-term health risks posed by the chemical to the human occupants.

In summary, organochlorine chemicals are presently a serious threat to certain bat populations of major importance and may have played a role in past declines of many other such populations. It is doubtful, however, that chemical pollutants have had the total adverse impact that disturbance, vandalism, and habitat destruction have had. Examples of these destructive factors in gray bat ecology were discussed by Tuttle (1979).

The idea that bats are extraordinarily sensitive to insecticides has repeatedly appeared in scientific and popular articles since the report by Luckens and

Davis (1964). This idea persists even though further work by these authors (Luckens and Davis 1965; Davis 1966, 1967; Luckens 1973) demonstrated that fat level controlled the mg/kg LD50 values they obtained. These authors' results showed that by the LD50 criterion big brown bats could be either several times more or several times less sensitive to DDT than laboratory rats and mice depending on how fat the bats were when tested. Data for other criteria (lethal brain concentrations and inhibition of brain ATPases) unaffected by fat levels, and summarized in this review, indicate that bats are neither more nor less sensitive to organochlorines than other small mammals and birds.

Mink exhibit extreme reproductive sensitivity to PCB. Platonow and Karstad (1973) reported that only 1 of 12 mink receiving a diet with 0.64 ppm of Aroclor 1254 reproduced, and her 3 kits all died the day after birth. Similarly extreme sensitivities may exist in certain bat species, but none has been demonstrated by the limited research accomplished thus far.

Research is needed on the effects of organophosphate and carbamate insecticides on bats. The effects of such chemicals have not been studied even though bats are probably often exposed to such chemicals (e.g., free-tailed bats in cotton-growing regions of the southwestern United States where methyl parathion is commonly applied in the evening; other bat species where malathion is used as an adulticide against mosquitoes and sprayed after dark). Exposures of bats to organophosphate and carbamate insecticides are probably increasing as they replace the older organochlorines for agricultural use.

Dieldrin-contaminated colonies of gray bats in Missouri (Clark et al. 1978*b*, 1980) were first noticed because analyses of guano samples revealed dieldrin levels much above those of other colonies. This sampling technique is recommended for surveying levels of organochlorines in colonial bats because it allows sampling without killing bats and because it produces results that may be more indicative than analyses of bats themselves. A single guano sample can represent several hundreds or thousands of bats across a complete feeding season. When questions arise from guano analyses, they can be answered by analyses of bats themselves.

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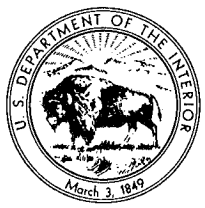

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