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# Northern Alabama Colonies of the Endangered Grey Bat *Myotis grisescens*: Organochlorine Contamination and Mortality

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*ABSTRACT*

*From 1976 to 1986, dead and dying grey bats* Myotis grisescens *and grey bat guano were collected from caves along the Tennessee River in northern Alabama to determine the possible role of organochlorine chemicals—in particular wastes from a former DDT manufacturing plant near Huntsville—in the mortalities. Concentrations of chemical residues in brains were less than known lethal levels; certain observations and analyses did indicate the possibility of past organochlorine-induced bat deaths. Levels of contaminants in bats declined slowly during the 10-year sampling period, but heavy residue burdens persist. The high ratio of DDD to DDE in residues from the former DDT plant made them identifiable as far as 140 km downriver. Grey bats concentrated chemical residues to higher levels and demonstrated the presence of these residues over much greater distances than did red-winged blackbirds* Agelaius phoenicus. *Grey bats may be the most sensitive indicator available for monitoring the contamination from this former DDT manufacturing site.*

## INTRODUCTION

Tuttle (1979) documented direct human disturbance as a major cause of decline among numerous grey bat *Myotis grisescens* colonies in Alabama and Tennessee. He went on to list harmful effects of water pollution and siltation on aquatic insect life, pesticide contamination, and local deforestation as other possible causes of grey bat declines worthy of study. Mortality and probable population decline of grey bats resulting from routine insecticide usage did occur in Missouri (Clark *et al.*, 1978*b*).

Our attention was focused on the Tennessee River area of northern Alabama where greater than normal grey bat mortalities have continued to occur, where a major portion of the grey bat species uses the caves and waterways of this area (Tuttle, 1976*a*), and where, since 1947, massive amounts of DDTR (DDT and metabolites, DDD and DDE) have flowed, via Huntsville Spring Branch–Indian Creek, into the Tennessee River from a DDT manufacturing site located on the Redstone Arsenal near Huntsville (Fleming & Atkeson, 1980). In 1980, the amount of DDTR estimated to lie in sediments of a 3·8 km stretch of Huntsville Spring Branch was $7{\cdot}3 \times 10^5$ kg (Fleming & Atkeson, 1980). The manufacture of DDT ceased in 1970, but heavy contamination of the local biota has persisted (O'Shea *et al.*, 1980; Fleming & Cromartie, 1981; Fleming *et al.*, 1984; Reich *et al.*, 1986). O'Shea *et al.* (1980) pointed out the occurrence of the grey bat on the Wheeler National Wildlife Refuge (Cave Springs Cave) (Fig. 1), which lies 20 km from the plant site.

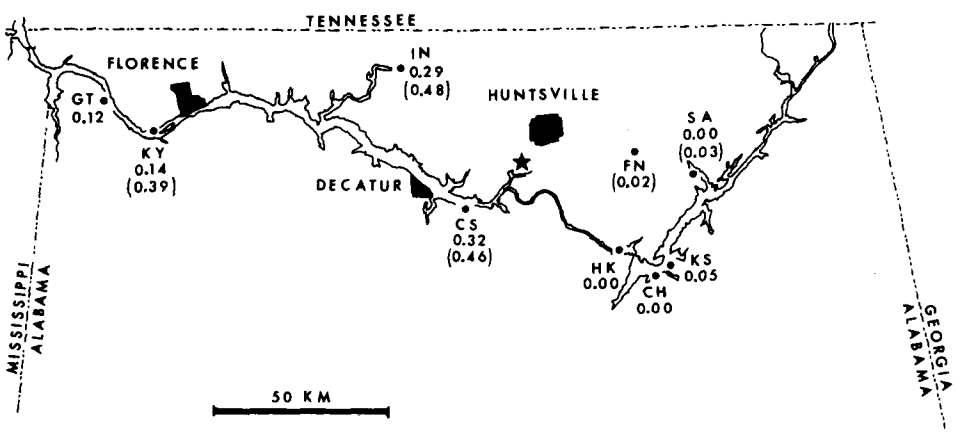


**Fig. 1.** Ratios of DDD to DDE in grey bat guano and in samples of dead and dying grey bats (ratios for bats are in parentheses) at caves near the Tennessee River. River flows east to west. Former DDT manufacturing site is indicated by a star. Abbreviations of cave names: GT, Georgetown; KY, Key; IN, Indian; CS, Cave Springs; HK, Hambrick; CH, Chandler's; KS, King's School; SA, Sauta; and FN, Fern.

Our objectives were to evaluate whether past mortalities had been caused by organochlorine chemical residues and to describe the present contamination status of northern Alabama grey bat colonies by studying residue data from cave colony sites along the Tennessee River.

## METHODS

Dead and dying grey bats and guano were collected as available in nine caves from 1976 to 1986 during routine censuses of colonies (Table 1). Bats were collected in June or early July, and the 1976 guano collections were made in early August. Bats were frozen at collection and shipped to the Patuxent Wildlife Research Center where they were prepared for analysis. Wings, feet, and skin were removed. The head was severed at the base of the skull, and the brain was removed. The gastrointestinal tract was removed from the remaining body portion, which was then analysed as the carcass. Guano was taken from the most recently deposited surface layer of piles beneath roosts, shipped unfrozen, then dried with a desiccant at ambient indoor temperatures before analysis. Milk samples were taken by dissection from stomachs of juvenile bats. Age (adult or juvenile) was estimated based on closure of epiphyses in wing bones, forearm length, and wear on the canine teeth. Collections of bats were not systematic or complete, but most of the

**TABLE 1**
Collection Locations and Years for Grey Bat Samples from Northern Alabama

| *Cave, county* | *Year of sample* | |
|---|---|---|
| | *Bats (N)* | *Guano* |
| Georgetown, Lauderdale | — | 1976 |
| Key, Lauderdale | 1979 (3) | 1976 |
| | | 1985 |
| Indian, Limestone | 1979 (1) | 1976 |
| Cave Springs, Morgan | 1978 (4) | 1976 |
| | 1979 (9) | 1985 |
| | 1980 (2) | |
| | 1986 (10) | |
| Hambrick, Marshall | — | 1976 |
| Chandler's, Marshall | — | 1976 |
| King's School, Marshall | — | 1976 |
| Sauta, Jackson | 1979 (1) | 1976 |
| Fern, Jackson | 1979 (1) | — |

dead and dying bats were juveniles and, unless stated otherwise, the data presented apply to this age group. These sampling procedures have been used previously to identify pesticide-induced bat mortalities in Missouri (Clark *et al.*, 1978*b*; 1983) and New Hampshire (Clark *et al.*, 1978*a*).

Samples were analysed for *p,p'*-DDT, *p,p'*-DDD, *p,p'*-DDE, dieldrin, heptachlor epoxide, oxychlordane, *trans*-nonachlor, *cis*-nonachlor, endrin, hexachlorobenzene (HCB), toxaphene, mirex, and polychlorinated biphenyls (PCBs). The recovered PCBs were quantified as Aroclor 1260. Certain samples discussed below were also analysed for 12-ketoendrin. Analyses were performed on a Hewlett-Packard Model 5753, 5751 or 5840 gas–liquid chromatograph as described by Prouty *et al.* (1977) and Kaiser *et al.* (1980). Chemical contaminants in 10% of the samples were confirmed by gas chromatography–mass spectrometry. Average percentage recoveries from spiked bald eagle *Haliaeetus leucocephalus* or mallard *Anas platyrhynchos* tissues ranged from 80 to 104%. Samples were analysed at sensitivities of $0{\cdot}5\,\mu g\,g^{-1}$ for pesticides and $2{\cdot}5\,\mu g\,g^{-1}$ for PCBs or at sensitivities of $0{\cdot}1\,\mu g\,g^{-1}$ for pesticides and $0{\cdot}5\,\mu g\,g^{-1}$ for PCBs. Samples analysed at the former sensitivities were brains and milk of 1978 bats and brains of 1986 bats. All other samples were analysed at the latter sensitivities. Methods of measuring brain acetylcholinesterase activity were those of Ellman *et al.* (1961) as modified by Hill & Fleming (1982).

Chemical concentrations in tissues are given as $\mu g\,g^{-1}$ fresh weight and in guano as $\mu g\,g^{-1}$ dry weight. Because chemical concentrations were positively skewed, they were log transformed and expressed as geometric means. Means were computed only if one-half or more of the samples contained measurable residues. Chemical levels reported as 'not detected' (ND) entered computations as $0{\cdot}1\,\mu g\,g^{-1}$.

## RESULTS AND DISCUSSION

### Possible causes of bat mortalities

Accurate estimates of mortality were not made, but the collectors at Cave Springs Cave in 1978, 1985, and 1986, and at Indian Cave in 1979 judged mortality to be far above normal. These judgements were based on many years of observation of these colonies. Our observations indicated that more than 1000 grey bats, perhaps several times more, died in Cave Springs Cave in 1986. Again, long experience indicated that this level of mortality was not normal.

Did organochlorine chemicals contribute to the observed mortality of grey bats? Both laboratory and field studies have shown that measurements

of chemical concentrations in brain tissues best separate birds killed by an organochlorine compound from exposed survivors. The range of concentrations associated with chemical-induced death is similar for most species of birds and, once measured, can be used to diagnose whether a bird has been killed by that chemical (Stickel, 1973). Bats also follow this pattern, and they show sensitivities to organochlorines generally similar to those of birds (Clark, 1981). Chemical concentrations in brains that were diagnostic of DDE-induced mortality in adults of the closely related little brown bat *Myotis lucifugus* averaged 603 $\mu g\,g^{-1}$ (range 540–670 $\mu g\,g^{-1}$) (Clark & Stafford, 1981). The comparable value for DDT in juvenile little brown bats was 16·3 $\mu g\,g^{-1}$ (range 11–25 $\mu g\,g^{-1}$) (Clark *et al.*, 1978*a*). Lethal brain concentrations of DDT in adult little brown bats were about 1·5 times greater than in juveniles (Clark *et al.*, 1978*a*). Even if a similar adult–juvenile adjustment was applied to the diagnostic DDE level, the maximum brain concentration found in the present study (36 $\mu g\,g^{-1}$ DDE, Cave Springs Cave, 1986) is still an order of magnitude below that thought to be lethal.

DDD residues may have been more of a threat to the grey bats. Data for birds and other mammals indicate that DDD is one-third to one-half as toxic as DDT (Stickel *et al.*, 1970). This suggests that brain concentrations of as little as 22 $\mu g\,g^{-1}$ DDD (twice the lower limit of toxic concentrations of DDT found in little brown bats by Clark *et al.*, 1978*a*) could be lethal. One grey bat brain (Cave Springs Cave, 1978) contained 29 $\mu g\,g^{-1}$ DDD, but all other values were less than 12 $\mu g\,g^{-1}$.

Tuttle (1976*b*) reported that Georgetown Cave once contained 150 000 grey bats, but that by 1969 this number had declined to 10 000. The remaining bats abandoned the cave when it was gated in 1970. Tuttle (1976*b*) stated that human disturbance caused the decline prior to gating. While this may be true, note that the 1970 guano (collected in 1976) contained more DDE and DDD than did any other 1976-collected sample (Table 2). If the difference (2·4 times) between DDE levels in guano from Georgetown Cave and Cave Springs Cave is applied to the maximum recorded brain levels from Cave Springs Cave (36 $\mu g\,g^{-1}$ DDE, 29 $\mu g\,g^{-1}$ DDD), the resultant DDE concentration (86 $\mu g\,g^{-1}$) is far below a lethal concentration, but the DDD concentration (70 $\mu g\,g^{-1}$) probably would kill grey bats.

In 1978, endrin, the most toxic of the organochlorine insecticides, was found at 4·8 $\mu g\,g^{-1}$ in a milk sample from Cave Springs Cave (Table 3). A diet with 5 $\mu g\,g^{-1}$ endrin fed to laboratory mice *Mus musculus* caused mortality of adults, and probably also of foetuses (Good & Ware, 1969). Endrin was also found in 2 of 4 carcasses (0·49, 0·11 $\mu g\,g^{-1}$) from Cave Springs Cave in 1978 and in 6 of 9 carcasses (ND–0·77 $\mu g\,g^{-1}$) from Cave Springs Cave in 1979, but it was not found in any brains. Because endrin was found in 1978 samples, brains for 1979 were pooled (to allow a more

**TABLE 2**

Organochlorine Residues ($\mu g\,g^{-1}$ dry weight) in Grey Bat Guano Samples Collected in 1976

| *Chemical* | *Cave* | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | *George-town*[a,b] | *Key*[a] | *Indian*[c] | *Cave Springs*[a] | *Hambrick*[a] | *Chandler's*[a] | *King's School*[d,e] | *Sauta*[a] |
| DDE | 8·2 | 1·8 | 2·0 | 3·4 | 0·64 | 0·86 | 3·6 | 1·2 |
| DDD | 0·98 | 0·26 | 0·58 | 1·1 | — | — | 0·17 | — |
| Dieldrin | 0·38 | 0·10 | 0·17 | 0·14 | — | 0·12 | 0·31 | — |
| Heptachlor epoxide | — | 0·15 | 0·16 | 0·41 | — | — | — | — |
| Oxychlordane | — | — | — | 0·06 | — | — | — | — |
| *Trans*-nonachlor | — | 0·11 | 0·19 | 0·17 | — | — | — | — |
| *Cis*-nonachlor | — | — | 0·06 | 0·05 | — | — | — | — |
| PCBs | 4·3 | 1·4 | 1·9 | 2·0 | 1·4 | 1·8 | 6·1 | 1·8 |

[a] Value is average of analyses of two subsamples.
[b] Guano deposited in 1970.
[c] Value is average of two subsamples from each of three samples.
[d] Value is analysis of one subsample.
[e] Guano deposited in 1974 or 1975.

**TABLE 3**

Organochlorine Residues in Guano ($\mu g\,g^{-1}$ dry weight) and Milk ($\mu g\,g^{-1}$ wet weight) Samples of Grey Bats from Cave Springs Cave, Morgan County, Alabama

| *Chemical* | *Guano*[a] | | *Milk*[b] | |
|---|---|---|---|---|
| | *1976*[c] | *1985*[c] | *1978*[d] | *1986*[c] |
| DDE | 3·4 | 2·0 | 25 | 16 |
| DDD | 1·1 | 0·06 | 4·9 | 2·5 |
| DDT | — | — | — | 0·99 |
| Dieldrin | 0·14 | — | — | — |
| Heptachlor epoxide | 0·41 | — | — | — |
| Oxychlordane | 0·06 | — | — | — |
| *Trans*-nonachlor | 0·17 | — | — | — |
| *Cis*-nonachlor | 0·05 | — | — | — |
| Endrin | — | — | 4·8 | — |
| Toxaphene | — | — | 1·1 | — |
| PCBs | 2·0 | — | 3·4 | — |

[a] Each value is the average of analyses of two subsamples.
[b] The 1978 sample was from one bat; the 1986 sample was a composite from seven bats.
[c] Lower limits of analytical sensitivity 0·1 $\mu g\,g^{-1}$ for pesticides and 0·5 $\mu g\,g^{-1}$ for PCBs.
[d] Lower limits of analytical sensitivity 0·5 $\mu g\,g^{-1}$ for pesticides and 2·5 $\mu g\,g^{-1}$ for PCBs.

sensitive analysis) and all 1979 samples plus the two 1980 carcasses from Cave Springs Cave were analysed for endrin's more toxic metabolite, 12-ketoendrin. Twelve-ketoendrin was found in only one of the 1979 carcasses (0·12 $\mu g\,g^{-1}$), but because it can kill at brain concentrations at or below the detection limit (0·1 $\mu g\,g^{-1}$) of these analyses (Stickel *et al.*, 1979), it could conceivably have killed grey bats at Cave Springs Cave in 1978 and 1979. Presumably the source of endrin was agricultural and had nothing to do with the former manufacture of DDT.

In summary, the residue data provide no unequivocal evidence that organochlorine chemicals were important in the observed mortalities. At the same time they suggest that DDD could have killed bats at Cave Springs Cave and Georgetown Cave and that 12-ketoendrin could have been an unmeasured mortality agent at Cave Springs Cave.

Other possible mortality factors were considered with the 1986 sample of dead and dying bats from Cave Springs Cave. Three bats were tested for depression of brain acetylcholinesterase activity. Such depression would occur if the bats had been poisoned by an organophosphate or carbamate pesticide. With untreated little brown bats as controls, these grey bats showed no evidence of cholinesterase depression.

Six other bats from the 1986 sample of dead and dying bats from Cave Springs Cave were necropsied by the National Wildlife Health Center of the US Fish and Wildlife Service in Madison, Wisconsin. Bats were examined for encephalitis, histoplasmosis, and rabies. Bacteriological tests were run on heart, intestine and liver samples. Virology tests were run on brain, liver, lung and spleen. Samples of brain, heart, intestine, kidney, liver, lung, lymph nodes, pancreas, spleen, stomach and thymus were examined histopathologically. The cause of death was not identified by any of these tests.

Finally, human disturbance seems an unlikely cause of the 1986 mortality at Cave Springs Cave because there was no sign of previous human entry. Such entry would have been detected easily in the soft sand at the cave entrance.

## Declining organochlorine residues in bats

The data from brains and carcasses (Table 4) do not lend themselves to an evaluation of whether residue levels have declined because sample sizes are small, and because the total time-span covered is relatively short. Guano samples have the advantage of representing thousands of individual bats, and, in this case, longer time-spans. Guano from Cave Springs Cave (Table 3) indicated a decline of 41% in DDE between 1976 and 1985. Guano from Key Cave contained 1·8 $\mu g\,g^{-1}$ DDE in 1976 (Table 2) and 0·60 $\mu g\,g^{-1}$ DDE in 1985, a decline of 67%. Also, 1985 and 1986 samples contained only DDT

**TABLE 4**

Means and Ranges for Organochlorine Residues ($\mu g\,g^{-1}$ wet weight) in Brains and Carcasses of Juvenile Grey Bats Found Dead or Dying in Cave Springs Cave, Morgan County, Alabama

| *Chemical* | *Brains* | | | | *Carcasses* | | |
|---|---|---|---|---|---|---|---|
| | *1978*[a] *(N = 4)* | *1979*[b] *(N = 9)*[c] | *1980*[b] *(N = 2)* | *1986*[a] *(N = 10)* | *1978*[b] *(N = 4)* | *1979*[b] *(N = 9)* | *1980*[b] *(N = 2)* |
| DDE | 9·1 (3·4–27) | 7·5 | 30 (30, 31) | 8·8 (3·5–36) | 24 (19–35) | 34 (20–63) | 62 (56, 69) |
| DDD | 3·8 (1·1–29) | 3·5 | 7·2 (6·1, 8·5) | — | 9·9 (4·0–21) | 12 (3·8–47) | 15 (20, 11) |
| DDT | 0·28 (ND–0·81) | — | 0·75 (0·47, 1·2) | — | 0·36 (0·23–0·53) | 0·34 (0·11–1·1) | 1·1 (1·1, 1·1) |
| Dieldrin | 0·26 (ND–0·68) | — | 0·47 (0·36, 0·61) | — | 0·45 (0·24–0·84) | 0·64 (0·34–1·4) | 0·86 (0·80, 0·92) |
| Heptachlor epoxide | — | — | 0·38 (0·15, 0·98) | — | 0·38 (0·19–0·62) | 0·46 (0·20–0·85) | 0·42 (0·32, 0·55) |
| Oxychlordane | 0·29 (ND–0·88) | 0·17 | 0·67 (0·30, 1·5) | — | 0·80 (0·53–1·5) | 0·91 (0·48–1·9) | 0·99 (0·76, 1·8) |
| *Trans*-nonachlor | — | — | 0·24 (0·09, 0·60) | — | 0·47 (0·32–0·86) | — | 0·40 (0·23, 0·70) |
| *Cis*-nonachlor | — | — | 0·11 (ND, 0·12) | — | — | — | 0·13 (ND, 0·16) |
| Endrin | — | — | — | — | 0·15 (ND–0·49) | 0·24 (ND–0·77) | — |
| Toxaphene | — | — | 0·19 (ND, 0·37) | — | 0·16 (ND–0·29) | — | 0·62 (1·9, 0·20) |
| Mirex | — | — | 0·11 (0·10, 0·12) | NA[d] | 0·11 (ND–0·17) | 0·20 (ND–0·36) | 0·40 (0·63, 0·25) |
| PCBs | 1·8 (ND–6·9) | 1·6 | 3·1 (2·7, 3·5) | — | 9·8 (8·0–17) | 7·5 (4·4–14) | 12 (9·7, 14) |

[a] Lower limits of analytical sensitivity $0{\cdot}5\,\mu g\,g^{-1}$ for pesticides and $2{\cdot}5\,\mu g\,g^{-1}$ for PCBs.
[b] Lower limits of analytical sensitivity $0{\cdot}1\,\mu g\,g^{-1}$ for pesticides and $0{\cdot}5\,\mu g\,g^{-1}$ for PCBs.
[c] Analysed as a single composite sample.
[d] No analysis for Mirex.

compounds. Thus residue levels do seem to have declined, but the question of when they will reach concentrations that no longer cause concern for wildlife remains open.

The overall decline in residues in grey bats was accompanied by a change in the ratio of DDD to DDE. Under most environmental conditions, DDT breaks down to DDE with the intermediate DDD persisting only briefly. But under anaerobic conditions, DDD is the principal breakdown product (Guenzi & Beard, 1967). Apparently, because most of the DDT wastes were deposited in sediments at the bottom of Huntsville Spring Branch-Indian Creek (Reich *et al.*, 1986), the escaping residues have contained considerable DDD. Grey bats from Cave Springs Cave show abundant DDD residues (Tables 3 and 4), but within the time span of our sampling, the amounts of DDD relative to DDE have declined (Table 5). We assume this is because as time passed more and more of the DDD in the entire contaminated creek-river system was converted to DDE.

## Contamination downriver

The effects of direction (upriver, downriver) and distance from the former DDT plant on residue concentrations can be seen in data from guano collected in 1976 and bats collected in 1979. Consistently higher DDD levels were found in colonies downriver from the source (Cave Springs, Indian, Key, and Georgetown) than in colonies upriver (Tables 2 and 6). Because all colonies above the former DDT plant contained DDE and some also contained DDD and DDT, the DDT plant probably was not the only source of DDT residues in the area. The uniqueness of the residues from the plant (high proportion of DDD) appears when we examine the residues as the ratio of DDD to DDE (Fig. 1). Note that the ratio declined with distance downriver (Fig. 1) as it declined through time (Table 5). In guano, total amounts of DDD and DDE, as well as their ratio, might have been lower at Georgetown and King's School Caves than they were if the guano had not been deposited in years before 1976.

The principal point is that contamination from the DDT plant reached elevated concentrations in grey bat colonies as far as 140 km downriver. Because the bats feed extensively on insects with aquatic larval stages (principally mayflies, Ephemeridae, Tuttle, 1976*c*), our residue data suggest that much contamination has moved far from Huntsville Spring Branch-Indian Creek, where it was originally deposited. This 'escaped' contamination will not be alleviated by the extensive remedial action currently in progress to contain DDTR at Huntsville Spring Branch-Indian Creek.

**TABLE 5**
Ratios of DDD to DDE ($\mu g\, g^{-1}$) in Juvenile Grey Bats Through Time

| *Year* | *Sample* | | | |
|---|---|---|---|---|
| | *Brains* | *Carcasses* | *Guano* | *Milk* |
| *Cave Springs Cave* | | | | |
| 1976 | — | — | 0·32 | — |
| 1978 | 0·42 | 0·41 | — | 0·20 |
| 1979 | 0·47 | 0·35 | — | — |
| 1980 | 0·24 | 0·24 | — | — |
| 1985 | — | — | 0·03 | — |
| 1986 | 0·00 | — | — | 0·16 |
| *Key Cave* | | | | |
| 1976 | — | — | 0·14 | — |
| 1985 | — | — | 0·00 | — |

**TABLE 6**
Organochlorine Residues ($\mu g\,g^{-1}$ wet weight) in Carcasses of Juvenile Grey Bats Found Dead or Dying in 1979

| *Chemical* | *Cave* | | | | |
|---|---|---|---|---|---|
| | *Key (N = 3)* | *Indian (N = 1)* | *Cave Springs (N = 9)* | *Sauta (N = 1)* | *Fern (N = 1)*[a] |
| DDE | 21 | 12 | 34 | 21 | 4·9 |
| DDD | 8·0 | 5·8 | 12 | 0·62 | 0·10 |
| DDT | 0·26 | — | 0·34 | 0·20 | — |
| Dieldrin | 0·35 | — | 0·64 | 0·44 | — |
| Heptachlor epoxide | 0·15 | — | 0·46 | 0·14 | — |
| Oxychlordane | 0·60 | 0·14 | 0·91 | 1·1 | 0·30 |
| *Trans*-nonachlor | — | 0·15 | — | — | — |
| Endrin | — | — | 0·24 | — | — |
| Mirex | — | — | 0·20 | — | — |
| PCBs | 9·0 | 6·7 | 7·5 | 11 | 4·9 |

[a] Bat was adult.

However, the remedial action should remove this site as a continuing source of contaminants farther downstream.

The observed geographic distribution of residues might also have resulted from interchanges of bats among caves, but other considerations argue against much influence from this source. First, only the colonies downriver show high DDD concentrations (Tables 2, 6 and Fig. 1). Also, the bats were (with one exception) juveniles. Twenty of the 30 juvenile grey bats that we analysed had forearm lengths (Table 7) less than the minimum (39·5 mm)

**TABLE 7**
Means and Ranges of Forearm Length (mm) among Samples of Dead and Dying Grey Bats Collected from Caves in Northern Alabama

| *Cave* | *Year* | | | |
|---|---|---|---|---|
| | *1978* | *1979* | *1980* | *1986* |
| Key (*N*) | 39·7 (3) (38·6–40·5) | — | — | — |
| Indian (*N*) | — | 33·0 (1) | — | — |
| Cave Springs (*N*) | 38·2 (4) (35·3–41·5) | 37·9 (9) (34·6–41·8) | 36·9 (2) (36·6–37·3) | 38·2 (10) (33·7–41·1) |
| Sauta (*N*) | — | 34·3 (1) | — | — |
| Fern (*N*) | — | 43·6 (1)[a] | — | — |

[a] Adult bat.

that Tuttle (1975) found necessary for flight. Therefore, two-thirds of these bats had probably not flown, and the other third had been flying only a few days. Young bats show little movement between caves during their first 20–30 days of flight, and most maternity colonies are located within 1 km of feeding areas (Tuttle, 1976*c*). Therefore, residues in young bats probably reflect contamination within a few kilometres of the cave where they were found. Because the guano was from maternity colonies (except King's School, a bachelor colony), the residues in guano also probably reflect local contamination.

**Organochlorine residues in grey bats versus red-winged blackbirds**

Carcasses of five 6–9 day old nestling red-winged blackbirds collected on Huntsville Spring Branch in 1985 at 3·3 km from the DDT plant outfall averaged 7·7 $\mu g\,g^{-1}$ DDE, 0·82 $\mu g\,g^{-1}$ DDD, with no DDT detected. However, at 20 km downstream (about the same distance as Cave Springs Cave), concentrations in five other red-winged blackbird nestlings averaged 0·28 $\mu g\,g^{-1}$ DDE, with no DDD or DDT detected. These data contrast sharply with the bats which, in 1986, were one or two orders of magnitude higher in DDE (Table 4) at this distance from the source.

The grey bat appears to be a sensitive indicator of both the level and geographic extent of Tennessee River contamination from the former DDT plant near Huntsville, Alabama. We believe that monitoring of what may be a long-term dissipation of this contamination might be done best with dead and dying juvenile grey bats found during routine annual population censuses.

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