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CARBOFURAN AFFECTS WILDLIFE ON VIRGINIA CORN FIELDS

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Carbofuran (2,3-dihydro-2,2-dimethyl-7-benzofuranyl methylcarbamate) is a commonly used carbamate insecticide-nematicide that is formulated as a granular and flowable product sold commercially as Furadan®. Carbofuran has caused wildlife die-offs across the United States and Canada (Stone 1979, Flickinger et al. 1980, Balcomb 1983, Balcomb et al. 1984, Flickinger et al. 1986, Smith 1987, Littrell 1988, Mineau 1988, Stinson 1990, Lyon 1991). In Virginia, carbofuran was implicated in at least 12 incidents of wildlife mortality from 1985-1990 (Va. Dep. Game and Inland Fish., unpubl. data). Documented deaths/incident varied from 1 to >200 animals. Species included bald eagle (*Haliaeetus leucocephalus*), red-tailed hawk (*Buteo jamaicensis*), blue jay (*Cyanocitta cristata*), eastern bluebird (*Sialia sialis*), American robin (*Turdus migratorius*), European starling (*Sturnus vulgaris*), chipping sparrow (*Spizella passerina*), red-winged blackbird (*Agelaius phoeniceus*), common grackle (*Quiscalus quiscula*), and American goldfinch (*Carduelis tristis*).

Following 2 bird die-offs caused by Furadan 15G® in Spring 1990, the Virginia Pesticide

Control Board (VPCB) held hearings on the importance of Furadan 15G® (the only granular carbofuran formulation registered in this state) to Virginia agriculture and its effect on wildlife. As a result, FMC Corporation (sole registrant of Furadan 15G® in Virginia) developed a Virginia-specific Furadan 15G® Avian Risk Reduction Plan. The VPCB adopted the plan in an effort to reduce avian risk while allowing continued use of the pesticide in Virginia. Elements of the Avian Risk Reduction Plan included: (1) reduction of the maximum allowable application rate in corn (at planting) to 0.23 kg/305 m (8 oz/1,000 linear feet); (2) restriction of Furadan 15G® use in corn to in-furrow applications only; (3) initiation of an intensive grower education campaign stressing safe granular insecticide application to avoid wildlife exposure; (4) labels indicating the requirement that farmers in a 32-county area east of Interstate 95 leave an 18-23 m zone free of Furadan 15G® at the ends of rows in no-till corn plantings; (5) a pilot program to equip some corn planters with cut-off devices to decrease surface spillage of granules at the ends of turnrows; and (6) a program to monitor Furadan 15G® applications in no-till corn in a 32-county area east of Interstate 95 during the 1991 growing season. The objective of our study was to measure effects of operational Furadan 15G® applications on vertebrate wildlife.

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STUDY AREA AND METHODS

Site Selection and Composition

The 1991 Virginia-specific Furadan 15G® label required farmers using Furadan 15G® on no-till corn in a 32-county area east of Interstate 95 to notify the Virginia Department of Agriculture and Consumer Services (VDACS) ≥ 5 days before Furadan 15G® application. All farms for which pre-use notification was received were selected as study sites by the Virginia Department of Game and Inland Fisheries (VDGIF). The VDGIF learned that some farmers intended to use Furadan 15G® but were not using no-till practices or were not located east of Interstate 95. These farmers were contacted for permission to include their farms in the study. All sites where monitoring was permitted were included in the study.

Nine farms in 7 counties east of Interstate 95 and 2 farms in 2 counties west of Interstate 95 were studied. Seven farms used no-till practices and 4 farms used conventional tillage practices for corn production. Conventional-till fields had little or no crop residue on the soil surface at the time of corn planting and Furadan 15G® application. No-till fields had a variety of soil cover characteristics. Nineteen no-till fields had wheat or rye cover crops that varied from 0.3–1.0 m in height. Fifteen no-till fields had soybean or corn stubble mixed with annual weeds covering the soil surface. Habitat types bordering no-till and conventional-till fields included deciduous woods, pasture, lawns, grass strips, and roadside ditches. All farms used in-furrow application of Furadan 15G®.

Field Searches

Field work was conducted from 4 April–29 May 1991. Thirty-four no-till corn fields and 10 conventional-till corn fields totaling approximately 365 ha were searched. The 44 study fields on 11 farms ranged from 2–24 ha. Initial corn field searches usually were made 24–48 hours after Furadan 15G® application. However, rain or late notification sometimes delayed initial searches up to 10 days after application. Fields were searched on several days (range = 1–11; median = 2) when possible. Evidence of dead or debilitated wildlife was categorized as: (1) carcasses, (2) debilitated animals exhibiting signs consistent with carbofuran poisoning (e.g., ataxia, immobility with wings spread; Hudson et al. 1984), and (3) feather or fur spots, suggesting dead or debilitated animals that were scavenged or predated.

Searches were conducted between 0800 and 1900 along the field perimeter and diagonally across the field center. Searchers walked 18 transects along turnrow borders of the field (borders perpendicular to corn rows) and 6 transects along non-turnrow borders of each field (borders parallel to corn rows). Two transects were searched across the field diagonal. Transects were spaced approximately 3 m apart. On 28 fields, adequate time and personnel were available to search additional transects over other areas of the field.

Wildlife carcasses were collected, labelled, and held on ice until they could be frozen (usually ≤ 8 hr). Approximate locations of dead or debilitated wildlife were recorded for most specimens. Systematic off-field searches were not made. However, carcasses seen from fields or found when searchers walked to and from fields were collected.

The VDGIF received early notification of intent to use Furadan 15G® for 10 no-till fields on 3 farms. Pre-application searches were made on these fields to determine whether there was detectable evidence of dead or debilitated wildlife before Furadan 15G® use. Two of the 10 fields were searched twice, 13 days and 1 day before Furadan 15G® application, for a total of 12 pre-application searches.

Laboratory Analyses

Frozen carcasses were shipped to the Southeastern Cooperative Wildlife Disease Study (SCWDS; Athens, Ga.) for immediate necropsy. Gross lesions were noted and sagittal half-brains and upper gastrointestinal (GI) tracts were removed and stored at -20 C for ≤ 6 days before preparation for residue analysis and cholinesterase (ChE) assay.

Brain ChE activity was determined on specimens for which adequate tissue was available if normal baseline values (control) for the species could be obtained. Control specimens were collected by mist netting and shotgun from areas that were not known to have received recent applications of anticholinesterase pesticides (e.g., organophosphorus or carbamate) and handled the same way as test specimens. Partially thawed half-brains were excised and assayed for whole-brain ChE activity following Ellman et al. (1961) as described by Hill and Fleming (1982). ChE activities for control and experimental specimens were determined the same day on a spectrophotometer at a wavelength of 405 nm and a reaction temperature of 25 C.

Gastrointestinal tracts and contents were homogenized with 125 ml ethyl acetate and 50 g sodium sulfate for 3–5 minutes. Each homogenate was filtered, concentrated using a rotary evaporator, and made to a final volume of 5 ml with ethyl acetate. Each sample was screened for organophosphorus pesticides with a Tracor Model 222 gas chromatograph (GC) equipped with a dual flame photometric detector operated in the phosphorus and sulfur mode simultaneously on a 3% OV-1 column.

Carbofuran concentrations were determined using a Tracor Model 222 GC equipped with a Ni⁶³ electron capture detector with a 3% OV-1 column. Carbofuran residue concentrations recovered from samples were confirmed on a Tracor Model 560 GC equipped with a Tracor Model 702 nitrogen/phosphorus detector.

Residue concentrations were quantified by comparison of sample peak height with known analytical standards obtained from the U.S. Environmental Protection Agency, Research Triangle Park, North Carolina. A reagent blank and spiked sample were included with each set of samples for analysis.

Some frozen bird carcasses were submitted to Consolidated Laboratories in Richmond, Virginia for residue analysis only. Gizzards and contents were sonicated with 10 ml of deionized water adjusted to pH 3 and filtered. Carbofuran concentrations were determined on a High Performance Liquid Chromatograph with a carbamate column (Waters Associates, Milford, Mass.). Detection was by fluorescence (excitation wavelength 339 nm; emission wavelength 445 nm). Carbofuran concentrations were confirmed using a Hewlett Packard Model 5840 GC/mass spectrometer.

RESULTS

Evidence of dead or debilitated wildlife was found on 10 of the 11 farms following Furadan 15G® application. The farm with no evidence of dead or debilitated wildlife was unique because only 1 field was treated with Furadan 15G® and that field was searched 10 days after Furadan 15G® application.

Sixty-six carcasses, 10 debilitated animals, and 48 feather and fur spots were found on or near Furadan 15G®-treated fields (Table 1). Thirty species (25 bird, 4 mammal, and 1 reptile) were recovered as carcasses or observed debilitated. Evidence of dead or debilitated wildlife was found on or near 8 of the 10 conventional-till fields and 25 (including 3 fields where only featherspots were found) of the 34 no-till fields searched.

Location of Wildlife

Evidence of dead or debilitated wildlife was found at various locations within Furadan 15G®-treated fields and in adjacent habitat. Locations of 115 carcasses, debilitated wildlife, feather spots, and fur spots were documented. Of these, we found 84 specimens within corn fields, with 49 found <18 m from the field boundary and 35 found >18 m from the field boundary. Of the specimens found <18 m from the field boundary, 21 were located in turnrow areas and 28 were found in non-turnrow areas. Thirty-one specimens were located outside corn fields. Of these, 21 were <18 m from the field boundary and 10 were found >18 m away. Investigators searched within fields more fre-

quently than outside fields. Also, the in-field area <18 m from the boundary was searched more frequently than the in-field area >18 m from the boundary. These differences in search intensity were not quantified.

Days to First Search

Thirty-one of the 44 Furadan 15G®-treated fields were first searched within 2 days of Furadan 15G® application and 22 fields were first searched within 1 day. Conventional-till fields were first searched 1 (6 fields), 6 (1 field), 7 (2 fields) or 10 (1 field) days after Furadan 15G® application. Evidence of dead or debilitated wildlife was found on the first day of search for 5, 1, 2, and 0 of these fields, respectively. No-till fields were first searched 1 (16 fields), 2 (9 fields), 3 (6 fields), or 6 (3 fields) days after Furadan 15G® application. Evidence of dead or debilitated wildlife was found on the first day of search for 10, 5, 2, and 1 of the no-till fields, respectively. Overall, 53% of no-till fields examined had evidence of affected wildlife on the first day of search compared to 80% of conventional-till fields.

Number of Searches

Thirteen Furadan 15G®-treated fields were searched only once. Evidence of dead or debilitated wildlife was found on 2 of the 7 no-till fields and 5 of the 6 conventional-till fields searched once. No conventional-till field was searched more than twice. No-till fields were searched as many as 11 times and up to 24 days post-application.

Evidence of dead or debilitated wildlife was found during the first search for 26 of the 33 fields on which some evidence was eventually found, and by the fifth search for all of these fields. Eleven of 44 treated fields lacked evidence of dead or debilitated wildlife. These fields were searched ≤3 times.

During searches at 1 field, investigators documented Furadan 15G® effects on a group of kestrels. On 20 April 1991, 5 American kestrels

were observed perching on and hunting from an electrical transmission wire bisecting a no-till corn field. Half of the field had been treated with Furadan 15G® on 19 April and the remaining half was treated on 23 April. On 25 April, 1 dead kestrel was found and 1 was observed to have severely impaired flight, landing, and perching abilities. On 27 April and 29 April, 2 more dead kestrels were found. No live kestrels were observed on the 2 searches made after 29 April.

Searches Before and After Application

Ten of the 44 fields included in this study were searched before and after Furadan 15G® application. No evidence of dead or debilitated wildlife was found during preapplication searches of 8 fields. A scavenged raccoon (*Procyon lotor*) was located near the center of 1 field. On another field, an eastern cottontail fur spot was found within 18 m of the field boundary. No bird carcasses or debilitated birds were found on any of the fields before Furadan 15G® application. After Furadan 15G® application, we found a total of 24 carcasses, 16 featherspots, and 1 debilitated bird on 8 of the 10 fields that received pre-application searches. On 2 fields, we discovered no evidence of dead or debilitated wildlife before or after Furadan 15G® application.

Laboratory Results

Of the 66 carcasses recovered, 52 were necropsied, 10 were analyzed for pesticide residues only, and 4 were not examined due to decomposition. Of the 52 carcasses necropsied, 51 exhibited no gross lesions that would indicate death due to causes other than acute anticholinesterase poisoning. The exception was an eastern cottontail collected with puncture wounds in its neck, indicating possible bite trauma.

Normal brain ChE values were determined for birds only. Brain ChE was inhibited by 14–

90% in 24 of the 32 bird specimens assayed and 21 of those inhibited had detectable carbofuran residues in the GI tract (Table 1). Of the 8 non-inhibited specimens, 5 contained carbofuran residues.

Carbofuran residues were found in the upper GI tracts of 81% of the 58 bird specimens analyzed. Fresh bird carcasses containing carbofuran residues were found up to 15 days postapplication. One eastern cottontail and 1 hairy-tailed mole carcass were found on the same farm 19 days postapplication, but carbofuran was not detected in either carcass. Carbofuran also was not detected in the other mammals or the reptile analyzed and brain ChE activity could not be evaluated because baseline normal values were not available for comparison. The only anticholinesterase residues detected other than carbofuran were phorate in an American robin and carbaryl in the hairy-tailed mole. The exceptional robin and mole were found on different farms. Presence of phorate and carbaryl in these animals may have resulted from agricultural applications of the compounds, both of which were registered for use in Virginia during 1991.

DISCUSSION

Establishing Cause of Mortality

Anticholinesterase poisoning was confirmed or suspected as the cause of wildlife deaths on Virginia corn fields treated with Furadan 15G®. In 2 animals, death was attributed to anticholinesterase pesticides other than carbofuran. Diagnoses of pesticide poisoning were based on 4 lines of evidence: (1) circumstances surrounding kills, (2) necropsies of carcasses, (3) residue analyses, and (4) brain ChE assays. The first 2 lines of evidence are relatively unequivocal. The latter 2 are equivocal in 12 cases where neither brain ChE depression nor carbofuran residues were detected by the analytical methods employed and in 2 cases where brain ChE was depressed but carbofuran residues were not detected. However, when all

Table 1. Wildlife found dead or debilitated in 1991 on 10 Virginia farms treated with Furadan 15C® (carbofuran), with results of carbofuran residue and brain cholinesterase (ChE) analyses of specimens submitted for testing.

Species	No. found		Carbofuran residue (ppm wet wt) ^a	Percent ChE inhibition ^b
	Debilitated	Dead		
Birds				
American kestrel (<i>Falco sparverius</i>)	1	3	2.2 7.4 1.7	0 39 NA
Mourning dove (<i>Zenaidura macroura</i>)		2	9.9 ND	NA 19
American crow (<i>Corvus brachyrhynchos</i>)	1			
Carolina wren (<i>Thryothorus ludovicianus</i>)		1	ND	NA
Eastern bluebird (<i>Sialia sialis</i>)	1	3	ND 35.7 12.5	NA NA NA
American robin (<i>Turdus migratorius</i>)		6	0.9 14.3 7.9 <3.0 ^c ND ND ^d	NA 14 NA 50 0 36
Northern mockingbird (<i>Mimus polyglottos</i>)	1			
Water pipit (<i>Anthus spinoletta</i>)		2	2.0 9.8	NA NA
Cedar waxwing (<i>Bombycilla cedrorum</i>)		1	ND	NA
European starling (<i>Sturnus vulgaris</i>)	2	3	ND 4.3	0 NA
Summer tanager (<i>Piranga rubra</i>)		1	15.9	NA
Northern cardinal (<i>Cardinalis cardinalis</i>)		2	76.8	24
Blue grosbeak (<i>Guiraca caerulea</i>)		1	32.8	NA
Chipping sparrow (<i>Spizella passerina</i>)		4	3.7 17.3 ND ND	80 90 0 47
Savannah sparrow (<i>Passerculus sandwichensis</i>)	4	16	<3.0 4.7 23.6 6.9 32.0 <3.0 <3.0 24.0 22.1 38.7 17.1 18.1 6.8 8.6 0.6	44 25 0 31 46 0 45 86 0 31 41 72 NA NA NA

Table 1. Continued.

Species	No. found		Carbofuran residue (ppm wet wt) ^a	Percent ChE inhibition ^b
	Debilitated	Dead		
Grasshopper sparrow (<i>Ammodramus savannarum</i>)		1	ND	NA
Swamp sparrow (<i>Melospiza georgiana</i>)		1	6.3	NA
White-throated sparrow (<i>Zonotrichia albicollis</i>)		3	208.0 31.4 35.5	78 38 25
Red-winged blackbird (<i>Agelaius phoeniceus</i>)		2	22.0 ND	NA NA
Eastern meadowlark (<i>Sturnella magna</i>)		1	<3.0	NA
Rusty blackbird (<i>Euphagus carolinus</i>)		1	15.3	NA
Boat-tailed grackle (<i>Quiscalus major</i>)		2	18.2 0.9	NA NA
Common grackle (<i>Quiscalus quiscula</i>)		2	<3.0 50.1	NA 43
Brown-headed cowbird (<i>Molothrus ater</i>)		2	1.8	0
House sparrow (<i>Passer domesticus</i>)		1	<3.0	32
Mammals				
Eastern harvest mouse (<i>Reithrodontomys humulidis</i>)		1	ND	NA
Hairy-tailed mole (<i>Parascalops breweri</i>)		1	ND ^c	NA
Eastern cottontail (<i>Sylvilagus floridanus</i>)		1	ND	NA
Striped skunk (<i>Mephitis mephitis</i>)		1	NA	NA
Reptiles				
Eastern garter snake (<i>Thamnophis sirtalis</i>)		1	ND	NA

^a ND = no carbofuran residues detected.

^b NA = assay not performed.

^c Discrete peak with retention time of carbofuran observed but not quantified.

^d Death attributed to phorate poisoning based on 7.9 ppm phorate in GI tract contents.

^e Carbaryl toxicosis suspected based on a residue of 6.0 ppm carbaryl found in the viscera.

lines of evidence are considered together, the case for wildlife mortality due to carbofuran poisoning is compelling.

Circumstances surrounding kills were consistent with acute pesticide poisoning (Schafer 1972, Balcomb et al. 1984, Hudson et al. 1984). Dead animals or their remains were found on or near corn fields soon after application of Furadan 15G®. Searches of 10 fields before Furadan 15G® application indicated that evidence of dead wildlife was seldom present on

untreated fields. Carcass necropsies indicated that animals were in generally good body condition and all but 1 demonstrated signs consistent with pesticide poisoning.

Evidence for wildlife mortality based on residue analyses is somewhat problematic because absence of anticholinesterase residues is expected in some poisoned specimens. Carbofuran residues were found in 47 of the 62 upper GI tracts analyzed. Other cases of pesticide-related wildlife poisoning suggest that pesti-

cide residues may not be found in the GI tracts of all animals (Flickinger et al. 1986, Littrell 1988, E. F. Hill, U.S. Fish and Wildl. Serv., Laurel, Md., unpubl. data). It is also possible for animals to absorb pesticides dermally and by inhalation (Schafer 1972, Driver et al. 1991, Hill 1992). Birds with emetic responses (e.g., doves and hawks) may regurgitate food when poisoned. Small amounts of pesticide in the GI tract may be quickly absorbed. In these cases, laboratory analyses of GI tract tissues and contents may not detect pesticides.

In some instances during this study, carbofuran was not detected in animals found near other animals that contained residues. For example, on 29 April 1991, a male and female eastern bluebird were found dead within 1 m of each other on a no-till field treated with Furadan 15G® on 28 April. Carbofuran residue was detected in the female (35.7 ppm) but not the male. Likewise, 2 red-winged blackbirds were found dead within 1 m of each other 8 days after Furadan 15G® was applied to a no-till field; carbofuran was detected (22 ppm) in 1 but not the other. In another example, an eastern harvest mouse was found dead on 11 April in the furrow of a no-till field treated on 10 April. The mouse was in good overall condition and pregnant with 5 fetuses but carbofuran residues were not detected. On the same day, carcasses of 2 common grackles and 1 savannah sparrow were found on the field and all contained carbofuran.

Evidence for wildlife mortality based on brain ChE assays also may be problematic. Of the 32 birds assayed for brain ChE activity, 47% had between 20–50% brain ChE depression and 19% had $\geq 50\%$ depression. Brain ChE inhibition of $\geq 20\%$ is considered indicative of anti-ChE exposure and $\geq 50\%$ inhibition may be considered diagnostic of death from such exposure (Ludke et al. 1975). Enzyme activity was apparently normal or $< 20\%$ inhibited for 11 specimens. However, recovery of ChE to apparently normal levels following poisoning with carbamate compounds is not uncommon

(Hill and Fleming 1982, Flickinger et al. 1986). Carbamylated brain enzymes can undergo spontaneous post-mortem reactivation, with ChE activity returning to normal or near normal in a matter of hours (Hill and Fleming 1982, Hill 1989). Deterioration of brain tissue, which would proceed rapidly in small song birds under the warm temperatures encountered in Virginia in April and May, may also elevate post-mortem brain enzyme activity (Hill 1989). Westlake et al. (1981) proposed that animals exposed to carbamates may die from disruption of general nerve function before brain enzymes are affected. Depressed brain ChE activity would not be apparent in such cases.

Factors in Evaluating Impact

Wildlife deaths attributable to Furadan 15G® in this study are likely only a portion of the actual number of wildlife affected. The fields searched during this study were not searched daily nor in their entirety. Searched fields exhibited a wide variety of cover conditions, from no vegetative cover on conventional-till fields to dense stands of 1-m tall wheat or rye cover on no-till fields. The difficult search conditions imposed by dense cover types likely reduced detection of affected wildlife. Observers encountered evidence of affected wildlife more frequently on the first day of search for conventional than no-till fields. Also, conventional fields searched once yielded more evidence of dead and debilitated wildlife and feather spots than did no-till fields searched once. These data suggest that cover type may have influenced carcass recovery and searching efficiency. This is consistent with past carcass searching studies. In general, studies where carcasses were placed in areas of low vegetative density and height achieved higher recovery rates than studies where carcasses were concealed by vegetation (Heinz et al. 1979, Stutzenbaker et al. 1984, Tobin and Dolbeer 1990). The influence of cover density on wildlife exposure to granules

cannot be evaluated from these data. However, reduced availability of granules due to increased vegetative cover may be another explanation for differences between recovery of affected wildlife on conventional and no-till sites, and warrants further study.

Recovery of bird carcasses at off-field sites indicated that some wildlife left or were carried from the field site. Presence of feather spots indicated that predators and scavengers were active on most fields, further reducing the probability of recovering affected wildlife. The rate of complete removal of debilitated or dead animals from fields in this study is unknown. However, other studies have demonstrated that carcasses placed in corn fields quickly disappear. Balcomb (1986) monitored the rate of disappearance of songbird carcasses on newly planted corn fields and found that 62–92% of carcasses disappeared within 24 hours of being placed on fields. Woronecki et al. (1979) observed that 28, 34, and 72% of house sparrow carcasses placed in mature corn fields were completely removed by scavengers after 1, 2, and 3 days, respectively. Studies conducted in other habitat types also have shown that complete disappearance of carcasses occurs rapidly (Rosene and Lay 1963, Tobin and Dolbeer 1990, White et al. 1990).

Searchers recovered a diversity of bird species on Furadan 15G®-treated fields, including songbird, gamebird, and raptor species. This study contains the first reports of carbofuran related mortality for 6 bird species, including water pipit, summer tanager, blue grosbeak, swamp sparrow, rusty blackbird, and boat-tailed grackle (Lyon 1991).

There has been some discussion of whether carcass-searching in large, open areas (e.g., crop field centers) is worthwhile when documenting wildlife effects of pesticide applications (Heinz et al. 1979). The relevance of searching field centers is dependent in part on the species that may be expected in the area, time of year, type of pesticide, and the location and nature of field sites. This study took place largely in

Virginia coastal plain counties during April and May. At this time, many bird species are migrating through Virginia on their way to northern breeding areas. Our discovery of water pipits and white-throated sparrows, rare breeders in Virginia, on treated fields illustrated this point.

In this study, we discovered carcasses both in field centers and along field edges. In cases where migrating and nonbreeding birds are potentially present on treated sites, carcass searches that include field centers may yield more information than searches confined to field perimeters. Physical features within fields (e.g., power transmission lines, trees, and grassed waterways) also may act as habitat features that attract birds to mid-field areas.

Only a small fraction of Furadan 15G®-treated land in Virginia was examined in 1991. Gianessi and Puffer (1988) estimated that in the mid-1980's, 96,441 ha of corn in Virginia were treated annually with approximately 186,880 kg of active ingredient carbofuran (including granular and flowable formulations). A pesticide use document by the Virginia Department of Agriculture and Consumer Services (1990) estimated that a maximum of 317,647 kg of active ingredient of carbofuran (including granular and flowable formulations) was used on 111,918 ha of 12 major agricultural crops in Virginia in 1990. These use figures suggest that wildlife exposure to carbofuran in Virginia may have been substantial during 1991 and previous years.

CONSERVATION IMPLICATIONS

Repeated involvement of a particular pesticide in wildlife die-offs may warrant modification of the compound's use to better protect wildlife. Systematic monitoring of operational pesticide applications for potential adverse effects on nontarget wildlife can provide important documentation to support policy or regulatory reform. Considerations in developing systematic monitoring methods include

pesticide use patterns (e.g., primary crop use, formulation, application dates), modes of pesticide action, routes of exposure, action time of toxic effects on nontarget species, and life history and behavioral traits of potentially affected species. In the case of Furadan 15G®, an extremely toxic and relatively fast-acting compound, we demonstrated that carcass searching is an effective method for gathering information about effects on nontarget wildlife; location and date of Furadan 15G® applications were critical in establishing a link between wildlife mortality and use of the pesticide. Because application locations were remote and most specimens recovered were small in size, it is unlikely that the mortality we documented would have been detected without systematic monitoring.

On 10 May 1991, the VPCB voted to enact an emergency cancellation of the sale and use of Furadan 15G® in Virginia and to deny future requests for reregistration of the product in the state. These decisions were based, in part, on historic evidence of wildlife mortality related to carbofuran poisoning and supporting evidence provided by this study. We encourage state wildlife and agricultural agencies to cooperate in actively investigating and documenting pesticide-related wildlife die-offs as they occur and to use this information to prioritize monitoring effects and effect regulatory change for protecting wildlife. We also urge farmers to use recommended chemicals that effectively control target pests but are less toxic to wildlife.

SUMMARY

Forty-four Virginia corn fields on 11 farms were searched for evidence of dead or debilitated wildlife following in-furrow application of granular carbofuran (Furadan 15G®) during April and May 1991. Evidence of pesticide poisoned wildlife, including dead animals, debilitated animals, feather spots, and fur spots was found on 33 fields on 10 farms. Carcasses

of 61 birds, 4 mammals, and 1 reptile were recovered. Anticholinesterase poisoning was confirmed or suspected as the cause of most wildlife deaths based on the circumstances surrounding kills, necropsies of carcasses, residue analyses, and brain ChE assays.

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LITERATURE CITED

- BALCOMB, R. 1983. Secondary poisoning of red-shouldered hawks with carbofuran. *J. Wildl. Manage.* 47:1,129-1,132.
- . 1986. Songbird carcasses disappear rapidly from agricultural fields. *Auk* 103:817-820.
- , C. A. BOWEN, D. WRIGHT, AND M. LAW. 1984. Effects on wildlife of at-planting corn applications of granular carbofuran. *J. Wildl. Manage.* 48:1,353-1,359.
- DRIVER, C. J., M. W. LIGOTKE, P. VAN VORIS, B. D. McVEETY, B. J. GREENSPAN, AND D. B. DROWN. 1991. Routes of uptake and their relative contribution to the toxicologic response of northern bobwhite (*Colinus virginianus*) to an organophosphate pesticide. *Environ. Toxicol. Chem.* 10:21-33.
- ELLMAN, G. L., D. COURTNEY, V. ANDRES, JR., AND R.

- M. FEATHERSTONE. 1961. A new and rapid colorimetric determination of acetylcholinesterase activity. *Biochem. Pharmacol.* 7:88-95.
- FLICKINGER, E. L., K. A. KING, W. F. STOUT, AND M. M. MOHN. 1980. Wildlife hazards from Furadan 3G applications to rice in Texas. *J. Wildl. Manage.* 44:190-197.
- , C. A. MITCHELL, D. H. WHITE, AND E. J. KOLBE. 1986. Bird poisonings from misuse of the carbamate Furadan in a Texas rice field. *Wildl. Soc. Bull.* 14:59-62.
- GIANESSI, L. P., AND C. A. PUFFER. 1988. Use of selected pesticides in agricultural crop production by state. Use coefficients listed by state. *Quality of the Environ. Div. Rep., Resour. for the Future*, Washington, D.C. 80pp.
- HEINZ, G. H., E. F. HILL, W. H. STICKEL, AND L. F. STICKEL. 1979. Environmental contaminant studies by the Patuxent Wildlife Research Center. Pages 9-35 in E. E. Kenaga, ed. *Avian and mammalian wildlife toxicology*. Am. Soc. Testing and Materials Spec. Tech. Publ. 693.
- HILL, E. F. 1989. Divergent effects of postmortem ambient temperature on organophosphorus- and carbamate-inhibited brain cholinesterase activity in birds. *Pestic. Biochem. Physiol.* 33:264-275.
- . 1992. Avian toxicology of anticholinesterases. Pages 272-294 in B. Ballantyne and T. C. Marrs, eds. *Clinical and experimental toxicology of organophosphates and carbamates*. Butterworth-Heinemann, Oxford, U.K.
- , AND W. J. FLEMING. 1982. Anticholinesterase poisoning of birds: field monitoring and diagnosis of acute poisoning. *Environ. Toxicol. Chem.* 1:27-38.
- HUDSON, R. H., R. K. TUCKER, AND M. A. HAEGELE. 1984. Handbook of toxicity of pesticides to wildlife. U.S. Fish and Wildl. Res. Publ. 153. 90pp.
- LITRELL, E. E. 1988. Waterfowl mortality in rice fields treated with the carbamate, carbofuran. *Calif. Fish and Game* 74:226-231.
- LUDKE, J. L., E. F. HILL, AND M. P. DIETER. 1975. Cholinesterase (ChE) response and related mortality among birds fed ChE inhibitors. *Arch. Environ. Contam. Toxicol.* 3:1-21.
- LYON, L. A. 1991. Hazards to birds associated with granular formulations of carbofuran: Analysis prepared for special review—final decision. U.S. Environ. Prot. Agency, Washington, D.C. 168pp.
- MINEAU, P. 1988. Avian mortality in agro-ecosystems. 1. The case against granular insecticides in Canada. Pages 3-12 in *Environmental effects of pesticides*. British Crop Prot. Council. Monogr. No. 40.
- ROSENE, W., JR., AND D. W. LAY. 1963. Disappearance and visibility of quail remains. *J. Wildl. Manage.* 27:139-142.
- SCHAFFER, E. W. 1972. The acute oral toxicity of 369 pesticidal, pharmaceutical and other chemicals to wild birds. *J. Toxicol. Appl. Pharmacol.* 21:315-330.
- SMITH, G. J. 1987. Pesticide use and toxicology in relation to wildlife: Organophosphorus and carbamate compounds. U.S. Fish and Wildl. Serv. Res. Publ. 170. 171pp.
- STINSON, E. R. 1990. Pesticide-related wildlife mortality in Virginia: 1980-1990. Pages 406-414 in D. L. Weigmann, ed. *Proc. Third Natl. Res. Conf., Va. Water Resour. Res. Center, Blacksburg*. 832pp.
- STONE, W. B. 1979. Poisoning of wild birds by organophosphate and carbamate pesticides. *N.Y. Fish and Game J.* 26:37-47.
- STUTZENBAKER, C. D., K. BROWN, AND D. LOBPRIES. 1984. Special report: an assessment of the accuracy of documenting waterfowl die-offs in a Texas coastal marsh. Pages 88-95 in J. S. Feierabend and A. B. Russel, eds. *Lead poisoning in wild waterfowl*. Natl. Wildl. Fed., Washington, D.C. 139pp.
- TOBIN, M. E., AND R. A. DOLBEER. 1990. Disappearance and recoverability of songbird carcasses in fruit orchards. *J. Field Ornithol.* 61:237-242.
- VIRGINIA DEPARTMENT OF AGRICULTURE AND CONSUMER SERVICES. 1990. Commonwealth of Virginia pesticide use estimate 1990. Va. Dep. Agric. and Consumer Serv. Off. Pest. Manage., Richmond. 26pp.
- WESTLAKE, G. E., P. J. BUNYAN, A. D. MARTIN, P. I. STANLEY, AND L. C. STEED. 1981. Carbamate poisoning. Effects of selected pesticides on plasma enzymes and brain esterases of Japanese quail (*Coturnix coturnix japonica*). *J. Agric. Food Chem.* 29:779-785.
- WHITE, D. H., J. T. SEGINKAK, AND R. C. SIMPSON. 1990. Survival of northern bobwhites in Georgia: crop-land use and pesticides. *Bull. Environ. Contam. Toxicol.* 44:73-80.
- WORONECKI, P. P., R. A. DOLBEER, C. R. INGRAM, AND A. R. STICKLEY, JR. 1979. 4-Aminopyridine effectiveness reevaluated for reducing blackbird damage to corn. *J. Wildl. Manage.* 43:184-191.

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