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Simulating cholinesterase inhibition in birds caused by dietary insecticide exposure

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Abstract

We describe a stochastic simulation model that simulates avian foraging in an agricultural landscape to evaluate factors affecting dietary insecticide exposure and to predict post-exposure cholinesterase (ChE) inhibition. To evaluate the model, we simulated published field studies and found that model predictions of insecticide decay and ChE inhibition reasonably approximated most observed results. Sensitivity analysis suggested that foraging location usually influenced ChE inhibition more than diet preferences or daily intake rate. Although organophosphorus insecticides usually caused greater inhibition than carbamate insecticides, insecticide toxicity appeared only moderately important. When we simulated impact of heavy insecticide applications during breeding seasons of 15 wild bird species, mean maximum ChE inhibition in most species exceeded 20% at some point. At this level of inhibition, birds may experience nausea and/or may exhibit minor behavioral changes. Simulated risk peaked in April–May and August–September and was lowest in July. ChE inhibition increased with proportion of vegetation in the diet. This model, and ones like it, may help predict insecticide exposure of and sublethal ChE inhibition in grassland animals, thereby reducing dependence of ecological risk assessments on field studies alone. © 1998 Elsevier Science B.V.

Keywords: Cholinesterase inhibition; Dietary insecticide exposure; Ecotoxicology; Bird foraging

1. Introduction

The estimated annual worldwide use of insecticides exceeds 1.1 million metric tons (Morley, 1992). In Texas, USA, farmers apply an average of 2.7 million kg of insecticides per year (Gianessi and Anderson, 1995). Strict regulation of highly persistent organochlorine compounds in the USA leads farmers to apply less persistent, but more toxic, organophosphorus (OP) and carbamate (CA) compounds. Applications of these insecti-

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cides frequently harm non-target organisms and have caused several wildlife kills (Hamilton and Stanley, 1975; Zinkl et al., 1978; Balcomb et al., 1984; Flickinger et al., 1984, 1986; Elliott et al., 1996). Smith (1987) suggested that over 50% of organophosphates and over 90% of carbamates are 'extremely toxic' (i.e. $LD_{50} < 40$ μg/g) to most bird species. However, birds usually receive only sublethal exposure, primarily by ingesting pesticide-contaminated food items (Matsumura, 1975). Previous studies have examined dietary pesticide-exposure in birds (White et al., 1979; Townsend et al., 1981; Balcomb, 1983; Henny et al., 1985), but few include models that simulate transfer of insecticides among organisms in terrestrial systems.

We present a model that simulates bird foraging in insecticide-treated fields in eastern Texas, USA. Because hazard of insecticides to animals is a function of exposure and toxicity (Kjolholt, 1990), we examined foraging behavior, diet and residue concentrations in prey, as well as sensitivity of birds to certain insecticides. The model estimates magnitude of exposure by simulating the degree of cholinesterase (ChE) inhibition in the brain. ChE inhibition often is used as an indicator of insecticide exposure in wild birds (Hill and Fleming, 1982). Signs of sublethal ChE inhibition in birds include nausea, lethargy, nutation, wing-drop, loss of righting-reflex, paralysis, opisthotonos and coma (Somers et al., 1991).

2. Model description

2.1. Model overview

Agricultural land-uses, insect pests, insecticide applications and breeding-season birds present in an 18-county area in eastern Texas, USA, serve as the system-of-interest. The five agricultural land-uses that cover the greatest area in this region include rangeland and fields of maize (*Zea mays*), cotton (*Gossypium* spp.), wheat (*Triticum aestivum*) and sorghum (*Sorghum* spp.). Conceptually, three submodels represent the system-of-interest: (1) insecticide application, deposition, and decay; (2) bird foraging; (3) residue ingestion and ChE inhibition in the bird (Fig. 1). The model simulates insecticide applications on crop fields both at planting and later, when high densities of pest insects provoke them. Insecticide residues settle on soil and on or in vegetation and animals in the fields. Insecticide residues decay over time based upon half-lives of each insecticide's active ingredient. Meanwhile, birds foraging in these fields ingest insecticide residues with their prey. Ingested residues of OP and CA insecticides inhibit ChE production in the brain until the bird excretes or metabolizes them.

We used difference equations to construct the multivariate, stochastic, compartment model with STELLA® II software (High Performance Systems, 1994). The model simulates 6 months (14 March–19 September) of agricultural and bird-foraging activity using an hourly time-step. Although ChE inhibition can occur within an hour of insecticide exposure (Hill, 1995), an hourly time-step provides sufficient temporal resolution to represent effects of ecological significance.

2.2. Insecticide application, deposition and decay submodel

2.2.1. Insecticide selection and application

We obtained data from a survey of Texas agricultural extension agents (Dr Rodney Holloway, Texas A&M University, personal communication) listing acre-treatments of insecticides applied to control insect species in maize, cotton, wheat and sorghum fields during one growing season in 18 Texas counties. From these data, we selected five insect species in each crop whose presence had provoked the greatest insecticide applications (Table 1). Of the insecticides recommended to control each insect species (CPCR, 1990; Meister, 1995; TAEX, 1995b), we chose the one OP and the one CA insecticide that ranked highest in the survey data (Table 1). To detect differences in impact between insecticide classes, an indicator variable directs the model to apply either OP or CA compounds. Similarly, farmers rotate insecticide classes to reduce chances of creating genetically resistant pest insects (Immaraju et al., 1990). For those insect species for which sources recommended no CA insecticide, the model applies the selected organophosphate in CA simulations.

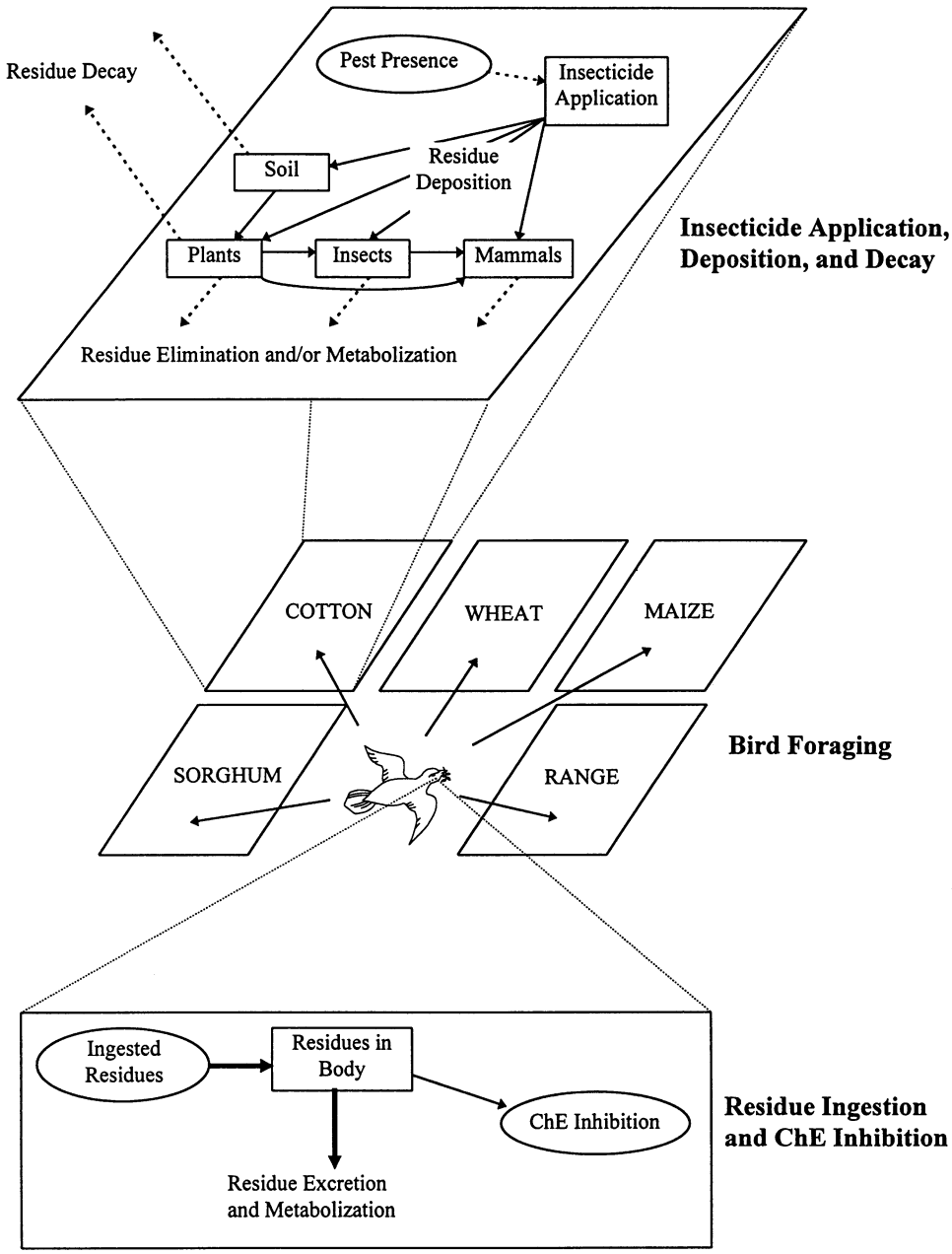

Fig. 1. Conceptual model of ChE inhibition caused by dietary exposure of grassland birds to insecticides.

To explore worse-case scenarios, the model applies insecticides at the highest recommended rates. The model applies three insecticides only at planting and the rest twice during the growing season (Table 1). We simulated at-planting insecticide applications by estimating crop planting dates (Jost, 1993; TASS, 1994). We selected 1 day within the possible infestation period of each insect species on which to perform the first (for some insecticides, the only) post-planting insecti-

Table 1
Planting dates for each crop in the model and the insecticide from each class used for each pest insect species[a]

Crop	Planted (date)	Pest	Pesticide class[b]	Chemical	Form[d]	1st application		2nd application	
						Date	g/ha	Date	g/ha
Cotton	25 April	Boll weevil	OP	Azinphos-methyl	L	24 June	280	29 June	280
			CA	Carbaryl	L	24 June	2242	29 June	2242
		Thrip	OP	Acephate	G	25 April	1121		
			CA	Aldicarb	G	25 April	616	5 May	616
		Bollworm	OP	Acephate	L	4 July	1121	7 July	1121
			CA	Thiodicarb	L	4 July	1009	7 July	1009
		Cotton fleahopper	OP	Dicrotophos	L	30 May	224	4 June	224
			CA	Oxamyl	L	30 May	280	4 June	280
		Cotton aphid	OP	Acephate	L	25 April	1121	25 May	1121
			CA	Methomyl	L	25 May	252	30 May	252
Maize	1 May	Corn rootworm	OP	Terbufos	G	5 April	1463		
			CA	Carbofuran	G	5 April	1463		
		SW corn borer	OP	Chlorpyrifos	L	3 August	1121	13 August	1121
			CA	Carbofuran	L	3 August	1121	13 August	1121
		2-Spotted spider mite	OP	Dimethoate	L	3 August	560	8 August	560
			CA	N/A[c]					
		Banks grass mite	OP	Dimethoate	L	3 August	560	8 August	560
			CA	Carbofuran	L	3 August	1121	13 August	1121
		L. cornstalk borer	OP	Terbufos	G	5 April	1463		
			CA	Carbofuran	G	5 April	4388		
Sorghum	1 April	Sorghum midge	OP	Disulfoton	L	5 June	560	8 June	560
			CA	Carbaryl	L	5 June	2242	8 June	2242
		Greenbug	OP	Chlorpyrifos	L	21 April	1121	26 April	560
			CA	Aldicarb	G	1 April	1121		
		Corn rootworm	OP	Terbufos	G	1 April	1463		
			CA	Carbofuran	G	1 April	1463		
		Chinch bug	OP	Chlorpyrifos	L	21 April	1121	26 April	560
			CA	Aldicarb	G	1 April	1121		
		Cornleaf aphid	OP	Disulfoton	G	1 April	1121	26 April	1121
			CA	Carbofuran	G	1 April	1463		
Wheat	15 October	Greenbug	OP	Dimethoate	L	14 March	420	19 March	420
			CA	N/A					
		Wheat aphid	OP	Dimethoate	L	3 May	420	8 May	420
			CA	Methomyl	L	3 May	504	8 May	504
		Fall armyworm	OP	Methyl parathion	L	2 June	420	7 June	420
			CA	Carbaryl	L	3 May	1681	7 June	1681
		Brown wheat mite	OP	Dimethoate	L	23 May	420	28 May	420
			CA	N/A					
		Winter grain mite	OP	Methyl parathion	L	2 June	420	7 June	420
			CA	N/A					

[a] Includes formulation, date(s) of application and application rates (in g active ingredient a.i./ha).
[b] Pesticide class: OP, organophosphate; CA, carbamate.
[c] No carbamate insecticide recommended (organophosphorous insecticide applied).
[d] Form: L, liquid; G, granular.

Table 2
Maximum concentration (in μg/g) of granular insecticides in plants following application at 1121 g a.i./ha and model half-lives of all insecticides (in days) in soil, on/in vegetation and on/in vertebrates

Insecticide	Maximum concentration (μg/g)	Days to maximum	Half-lives (d)		
			Soil	Vegetation	Vertebrates
Acephate			2.25[a]	3.50[i]	0.26[l]
Acephate (g[p])	13.12[k,q]	21[k,q]	14.00[b,q]	7.50[j]	0.26[l]
Aldicarb (g)	13.12[k]	21[k]	14.00[b]	18.07[k]	0.26[m]
Azinphos-methyl			21.00[c]	4.00[i]	0.43[d]
Carbaryl			7.00[c]	7.40[i]	0.37[c]
Carbofuran			45.00[d]	3.50[i]	0.38[c]
Carbofuran (g)	18.05[o,r]	14[o,r]	16.67[e]	15.16[g,r]	0.38[c]
Chlorpyrifos			90.00[c]	4.00[i]	1.00[c]
Dicrotophos			3.08[f]	2.80[i,s]	0.45[d]
Dimethoate			10.00[c]	4.00[i]	0.50[c]
Disulfoton			10.54[c]	11.34[c]	0.21[c]
Disulfoton (g)	18.05[o]	14[o]	25.82[g]	15.16[g]	0.21[c]
Malathion			6.00[c]	1.50[c]	0.40[c]
Methomyl			38.00[c]	1.70[i]	0.29[c]
Methyl parathion			35.00[c]	1.30[i]	0.15[c]
Monocrotophos			0.43[h]	3.10[i]	0.71[d]
Oxamyl			21.00[c]	4.30[i]	0.58[n]
Terbufos (g)	4.82[v]	22[v]	22.40[v]	9.72[v]	2.42[c]
Thiodicarb			5.50[d]	2.70[i,t]	0.38[c,u]

[a] Smith (1987), [b] Maitlen and Powell (1982), [c] Extoxnet (1993), [d] RSC (1990), [e] Harris et al. (1988), [f] Corey (1965), [g] Szeto et al. (1983), [h] Dureja (1989), [i] Willis and McDowell (1987), [j]Bart (1979), [k] Sites and Cone (1989), [l] Hussain et al. (1985), [m] Matthews (1984), [n] Harvey and Han (1978), [o] Ridgway et al. (1965)[p] g is the granular formulation of insecticide.[q] Data for aldicarb; [r] Data for granular disulfoton; [s] Mean of many OP insecticides in southern US; [t] Mean of many CA insecticides in southern US; [u] Data for carbofuran; [v] Szeto et al. (1986).

cide application (Bottrell, 1979; TAEX, 1979a,b; Matthews, 1989; TAEX, 1995a). Insecticides that require a second post-planting application are applied 3–7 days after the initial application (Meister, 1995). Application dates of OP and CA insecticides used to control an insect species usually coincide. The same insecticide and application date listed for two insect species in the same field indicate only one application (Table 1).

2.2.2. *Insecticide deposition, uptake and decay*

Insecticides applied to model fields contaminate all vegetation, insects and mammals in them. To calculate residues on plant surfaces following application of liquid insecticide formulations, we used a proposed revision to the EPA Kenaga nomogram (Fletcher et al., 1994), which estimates maximum residues expected on plants after pesticide application at 1121 g active ingredient (a.i.)/ha (1 lb/acre). We used its estimates of 135 μg/g on 'broadleaf' plants for cotton and 110 μg/g on 'long grass' plants for maize, sorghum and wheat. We assumed a uniform coating of insecticide on all plants and plant parts. Granular formulations of insecticides differ in that plants must absorb them in solution with soil water. For these we assumed a linear increase in residue concentration in plants that peaks in 14–22 days to a maximum calculated as a proportion of initial application (Table 2). Due to limited data, we used aldicarb uptake data for acephate and disulfoton uptake data for carbofuran. We assumed uniform uptake of insecticide to all plant parts and that simulated birds avoid ingesting insecticide granules.

Insects in the model receive insecticide residues from direct surface coating at application and through ingestion of residues from vegetation. Extrapolating from Forsyth and Westcott (1994), insecticide application at 1121 g a.i./ha in the model leaves 20.91 μg of residue/g of insects.

Because we lacked data to quantify transfer of plant-surface residues to insects, we estimated that insects incorporate each hour, a (residue dose)/(g body weight) equal to a nominal 1% of surface residue concentration. To quantify transfer of granular insecticide residues, we assumed that insects ingest a dose/(g body weight) equal to 50% of granular insecticide residues absorbed each hour by plants. Mammals in the model receive residues either from direct contact with liquid insecticides at application, or through ingestion of granular insecticide residues in plants and insects. We assumed that mammals, on average, would receive 10% of the value reported by Forsyth and Westcott (1994) for liquid insecticide residues left on insects. We also estimated that mammals receive doses/(g body weight) equal to 10% of the amount of granular insecticide residue absorbed by both insects and plants. We assumed an exponential decay rate for all insecticides, calculated with half-lives of each compound in soil, vegetation and vertebrates (Table 2). We assumed that birds and mammals lose residues at the 'vertebrate' rate and that insects lose 2% of their (body burden)/day, equivalent to a half-life of 1.43 days; on average, this rate falls between those for vegetation and vertebrates. In summary, concentrations of each liquid insecticide's residues on vegetation, insects and mammals are calculated with the following equations:

$$\mathrm{VLR}_{t+1} = \mathrm{VLR}_t + \mathrm{APP}_t * \mathrm{VEGPROP} - (\mathrm{VLR}_t * (1 - e^{(\ln 0.5)/T_P})) \quad (1)$$

$$\mathrm{ILR}_{t+1} = \mathrm{ILR}_t + \mathrm{APP}_t * 0.01866 + \mathrm{VLR}_t * 0.01 - (\mathrm{ILR}_t * (1 - e^{(\ln 0.5)/T_i})) \quad (2)$$

$$\mathrm{MLR}_{t+1} = \mathrm{MLR}_t + \mathrm{APP}_t * 0.00187 - (\mathrm{MLR}_t * (1 - e^{(\ln 0.5)/T_v})) \quad (3)$$

where VLR_t, ILR_t and MLR_t represent residue concentrations (in μg/g) remaining at time t on vegetation, insects and mammals, respectively; APP_t equals g of insecticide applied at time t; VEGPROP converts g of insecticide to μg of residue/g of vegetation (equals 0.12043 for cotton and 0.09813 for other crops); e is the base of the natural logarithm; and T_p, T_i and T_v represent half-lives of the compound on plants, in insects and in vertebrates, respectively.

For each granular insecticide, concentrations of residues in plants, insects and mammals are calculated with the following equations:

$$\mathrm{UPTAKE}_t = (\mathrm{APP}_t * \mathrm{CONV})/(\mathrm{TIMETOMAX} - 8), \quad \text{for } t = 0 \text{ to TIMETOMAX} \quad (4)$$

$$\mathrm{VGR}_{t+1} = \mathrm{VGR}_t + \mathrm{UPTAKE}_t - (\mathrm{VGR}_t * (1 - e^{(\ln 0.5)/T_P})) \quad (5)$$

$$\mathrm{IGR}_{t+1} = \mathrm{IGR}_t + \mathrm{UPTAKE}_t * 0.5 - (\mathrm{IGR}_t * (1 - e^{(\ln 0.5)/T_i})) \quad (6)$$

$$\mathrm{MGR}_{t+1} = \mathrm{MGR}_t + \mathrm{UPTAKE}_t * 0.15 - (\mathrm{MGR}_t * (1 - e^{(\ln 0.5)/T_v})) \quad (7)$$

where UPTAKE_t represents residue concentration taken up by plants each h; APP_t equals g of insecticide applied at time t; CONV converts g of insecticide applied to maximum μg of residue/g of vegetation; TIMETOMAX is the number of h needed for concentrations in plants to reach maximum; VGR_t, IGR_t and MGR_t represent residue concentrations (in μg/g) remaining at time t in plants, insects and mammals, respectively; e is the base of the natural logarithm; and T_p, T_i and T_v represent half-lives of the compound in plants, insects and vertebrates, respectively.

2.3. Bird foraging submodel

This submodel randomly determines hourly foraging location and calculates amount of food eaten/h from each of the three prey types. To estimate effects of diet preferences and foraging behavior on dietary exposure, we selected one individual from each of 15 bird species and simulated its response in the model. Each species resides in the study area during the breeding season (ca. March–September) and feeds on the ground in agricultural fields and pastures. We classified species into feeding assemblages according to DeGraaf et al. (1985) and chose 3–4 species from each of four assemblages: granivores, omnivores, insectivores and carnivores (Table 3).

Accurate estimation of bird foraging characteristics presented the greatest challenge. Model di-

Table 3
Bird species from the four feeding assemblages simulated in the model

Assemblage[o]	Species	BW (g)	DFI (g)	Vegetation	Insects	Mammals
Granivores	Mourning dove *Zenaida macroura*	126[a]	15.6[i]	100[m]	0[m]	0[m]
	Common ground dove *Columbina passerina*	35[b]	5.4[j]	100[m]	0[m]	0[m]
	Inca dove *Columbina inca*	50[b]	7.7[j]	100[m]	0[m]	0[m]
Omnivores	Brown-headed cowbird *Molothrus ater*	49[c]	5.4[k]	57[m]	43[m]	0[m]
	Dickcissel *Spiza americana*	28[d]	4.3[j]	29[m]	71[m]	0[m]
	Horned lark *Eremophila alpestris*	32[d]	5.0[j]	68[m]	32[m]	0[m]
	Red-winged blackbird *Agelaius phoeniceus*	65[e]	10.0[j]	55[m]	45[m]	0[m]
Insectivores	Cattle egret *Bubulcus ibis*	383[f]	74.0[l]	0[n]	100[n]	0[n]
	Eastern bluebird *Sialia sialis*	28[b]	4.3[j]	12[m]	88[m]	0[m]
	Eastern meadowlark *Sturnella magna*	108[d]	16.5[j]	10[m]	90[m]	0[m]
	Carolina wren *Thryothorus ludovicianus*	21[b]	3.2[j]	2[m]	98[m]	0[m]
Carnivores	Barn owl *Tyto alba*	474[g]	110.0[g]	0[h]	0[h]	100[h]
	Loggerhead shrike *Lanius ludovicianus*	50[b]	7.7[j]	0[m]	72[m]	28[m]
	Red-tailed hawk *Buteo jamaicensis*	1126[h]	96.8[h]	0[m]	0[m]	100[m]
	American kestrel *Falco sparverius*	114[h]	21.9[h]	0[h]	0[h]	100[h]

Includes estimated body weight (BW) (g), daily food intake (DFI) (g) and percentage of vegetation, insects and mammals in the diet.
[a] Nelson and Martin (1953), [b] Oberholser (1974), [c] Lowther (1993), [d] Wiens and Rotenberry (1979), [e] Yasukawa (1995), [f] Telfair II (1979), [g] Marti (1992), [h] Craighead and Craighead (1956), [i] Lewis (1993), [j] Taber (1928), [k] Belovsky (1990), [l] Siegfried (1969), [m] Martin et al. (1951), [n] Telfair II (1983)
[o] Species classified into feeding assemblages using method of DeGraaf et al. (1985).

urnal birds forage once each hour between sunrise and sunset, except for 1 h beginning at noon. The model nocturnal species forages once each hour between 1 h after sunset and 1 h before sunrise (Marti, 1992). We estimated body weight and daily food intake for each bird (Table 3). When we could not find an estimate of daily food intake, we estimated a rate of 15.4% of body weight (Taber, 1928). We then estimated percentage of diet mass consumed as vegetation, insects and mammals (Table 3) and assumed that each bird would obtain an equal proportion of its daily intake every hour it forages. Thus, a bird eating 140 g/day and foraging 14 h/day with a diet composed of 50% vegetation and 50% insects eats 5 g of vegetation and 5 g of insects per foraging hour. A submodel using equations from Ryan (1977) calculated times of sunrise and sunset each simulated day for Bryan, Texas, a city located in the center of the study area. Although small birds

Table 4
For selected bird species, percent chance of an individual selecting cotton, maize, sorghum, wheat or range in which to forage each hour during different periods of the year[a]

Species	Dates	Range	Wheat	Sorghum	Maize	Cotton
Mourning dove	14 March–2 May	23	23	23	23	8
Inca dove	3 May–10 May	40	6	23	23	8
	11 May–3 June	57	6	6	23	8
	4 June–19 September	80	6	6	6	2
Common ground dove	14 March–2 May	84	4	4	4	4
	3 May–10 May	87	1	4	4	4
	11 May–3 June	90	1	1	4	4
	4 June–19 September	96	1	1	1	1
Brown-headed cowbird	14 March–2 May	24	19	19	19	19
	3 May–10 May	38	5	19	19	19
	11 May–3 June	52	5	5	19	19
	4 June–19 September	80	5	5	5	5
Dickcissel	14 March–3 June	30	20	20	20	10
	4 June–19 September	37	20	20	20	3
Horned lark	14 March–2 May	20	20	20	20	20
Cattle egret	3 May–10 May	35	5	20	20	20
Eastern bluebird	11 May–3 June	50	5	5	20	20
Carolina wren	4 June–19 September	80	5	5	5	5
Red-winged blackbird	14 March–29 June	8	25	25	40	2
	30 June–19 September	0	15	15	70	0
Eastern meadowlark	14 March–2 May	24	25	17	17	17
	3 May–10 May	43	6	17	17	17
	11 May–3 June	56	6	4	17	17
	4 June–19 September	82	6	4	4	4
Barn owl Loggerhead shrike Red-tailed hawk American kestrel	14 March–19 September	20	20	20	20	20

[a] Assumes equal area and availability of field types.

can compose ca. 20% of red-tailed hawk diets (Craighead and Craighead, 1956), we excluded birds as prey items in the model.

To determine in which fields each bird forages, we estimated the probability of foraging in each field type each hour assuming equal availability and equal area of field types (Craighead and Craighead, 1956; Bent, 1965; Telfair II, 1983; Best et al., 1990; Dolbeer, 1990; Lewis, 1993; Davis and Roca, 1995) (Table 4). Each simulated foraging hour, the model randomly determines which field the bird visits. We assumed that probability of foraging in a crop field would decrease by 75% of its initial value when that crop reached a height of 30 cm, increasing probability of foraging in rangeland by that same amount. Only one species in the model, the red-winged blackbird, becomes attracted to a crop (maize) once the crop seeds. We ignored other bird attractors such as flushed insects at planting and spilled grain at harvest and bird repulsors such as bare ground at planting.

2.4. Residue ingestion and ChE inhibition submodel

This submodel estimates ChE inhibition based upon residue concentration in the bird's body. To calculate hourly residue ingestion, the model multiplies residue concentration in each gram of each prey type by grams of each prey type eaten that hour and divides it by body weight. Data indicate that birds excrete 25, 10, 20 and 10% of carbaryl, chlorpyrifos, dimethoate and disulfoton, respectively, unmetabolized (Extoxnet, 1993); these

Table 5
Amount (in μg/g of bird body weight) of each insecticide in the model required to inhibit cholinesterase by 5, 10, 25 and 50%, maximum percent inhibition for each compound and species from which data were obtained

Insecticide	Amount required to inhibit ChE (μg/g)				Maximum % inhibition	Species	
	5%	10%	25%	50%			
Acephate[a]	5.6	11.9	71.7	301.7	52.0	Mallard	*Anas platyrhynchos*
Aldicarb[b,g]	1.5	2.4	4.3	7.4	55.0	Northern bobwhite	*Colinus virginianus*
Azinphos-methy l[a,h]	5.6	11.9	71.7	301.7	52.0	Mallard	
Carbaryl[a,i]	80.0	212.5	473.1	683.3	51.0	Mallard	
Carbofuran[c]	0.8	1.6	4.0	—	46.0	Savannah sparrow	*Passerculus sandwichensis*
Chlorpyrifos[d,j]	0.6	1.1	2.9	13.8	70.0	Coturnix quail	*Coturnix coturnix*
Dicrotophos[a]	0.2	0.5	0.9	1.8	61.0	Mallard	
Dimethoate[d]	0.6	1.1	2.9	13.8	70.0	Coturnix quail	
Disulfoton[d,j]	0.6	1.1	2.9	13.8	70.0	Coturnix quail	
Malathion[a]	80.0	212.5	473.1	683.3	51.0	Mallard	
Methomyl[a,i]	80.0	212.5	473.1	683.3	51.0	Mallard	
Methyl parathion[b]	1.5	2.4	4.3	7.4	55.0	Northern bobwhite	
Monocrotophos[a,k]	0.2	0.5	0.9	1.8	61.0	Mallard	
Oxamyl[a,k]	0.2	0.5	0.9	1.8	61.0	Mallard	
Terbufos[e,f]	0.6	0.8	2.5	6.7	97.8	Northern bobwhite and deer mouse	*Peromyscus maniculatus*
Thiodicarb[a,h]	5.6	11.9	71.7	301.7	52.0	Mallard	

[a] Fleming and Bradbury (1981), [b] Galindo et al. (1985), [c] Stinson et al. (1994), [d] Radvanyi et al. (1986), [e] Kendall (1992), [f] Block et al. (1993).
[g] Using data for methyl parathion; [h] using data for acephate; [i] using data for malathion; [j] using data for dimethoate; [k] using data for dicrotophos.

residues exit the bird's body before they can inhibit ChE. Once residues enter the bloodstream and reach the brain, they tend to inhibit ChE production along a log-linear dose–response curve (Greig-Smith, 1991). We found data on ChE inhibition in birds after ingestion of known insecticide doses for nearly half of the model insecticides. When forced to substitute one compound's data for another, we chose data from a compound with a similar dietary toxicity (LC_{50}, obtained from Hill and Camardese, 1986) to the same bird species. The model calculates ChE inhibition by linearly interpolating between data points obtained from the literature. Except for the upper end of terbufos dosage, we used values for inhibition of brain ChE, as opposed to plasma ChE, because brain ChE indicates the degree of anticholinesterase (antiChE) exposure better (Fleming, 1981). For carbofuran, we used inhibition data from savannah sparrows found dead, with insecticide residue in their guts as a dose. These values may be less reliable than others, because ChE activity inhibited by carbamates may reactivate after death (Hill and Fleming, 1982) and gut residues may not reflect dose received (Greig-Smith, 1991). Most importantly, we assumed that ingested residues of each insecticide would inhibit ChE in all bird species equally (Table 5).

The model calculates ChE inhibition due to each insecticide separately and adds them together to estimate total ChE inhibition each hour. Studies suggest that effects of antiChE compounds taken concurrently usually are additive and that

Table 6
Comparison of percent ChE inhibitions in pheasant predicted by the model with those observed by Martin et al. (1996)

Insecticide	Application rate (g/ha)	Observed mean ± S.E.	Model predictions	
			37 h	48 h
Chlorpyrifos	279	5.5 ± 3.7	36.6	30.7
	1116	13.8 ± 6.1	62.7	56.6
Dimethoate	213	9.2 ± 2.4*	10.0	5.3
	852	29.9 ± 5.5*	24.5	17.3
Carbofuran	132	12.4 ± 3.0*	9.2	3.9
	528	35.9 ± 6.4*	33.1	25.0

* Means significantly ($P<0.05$) different from control means.

any synergism between compounds is rarely more than 3-fold (Hill, 1992). We did not consider cumulative effects of exposure or inhibition, even though recovery to normal ChE levels after exposure to OP compounds often takes longer than 24 h (Grue et al., 1991). Insecticide residues in the bloodstream instantaneously inhibit ChE until lost from the body according to each compound's half-life in vertebrates. Thus, output of interest from the model is percentage of ChE inhibited by insecticide residues in the bird's body.

3. Model evaluation

We evaluated model performance by Eq. (1) simulating field studies of avian ChE inhibition after insecticide application and comparing model predictions with their results, and Eq. (2) determining relative sensitivity of ChE inhibition to foraging location, diet selection and food intake/body weight ratio.

3.1. Simulation of field studies

We simulated a study by Custer and Mitchell (1987), who shot four mourning doves near a field that had been sprayed 1–2 days previously with monocrotophos. They observed mean brain ChE inhibition of 41 ± 20% ($\bar{X}\pm 1$ S.D.) and our model estimated mean maximum inhibition of 26.4 ± 17.5% ($\bar{X}\pm 1$ S.D.) ca. 1 day after application (100 replicate stochastic simulations), which falls within 67% confidence limits of the field data. We also simulated the study of Martin et al. (1996), who sprayed grasshoppers with each of three insecticides (chlorpyrifos, dimethoate or carbofuran), fed them to pheasant chicks and measured brain ChE after 48 h. Although model predictions of dimethoate- and carbofuran-induced ChE inhibition 48 h after application were lower than those observed, predictions of maximum inhibition, which occurred in the model 37 h after application, fell within 67% confidence limits of the field data (Table 6). The model may overestimate impact of chlorpyrifos because we substituted dose-response data of dimethoate. Although mean LC_{50}s for chlorpyrifos and dimethoate in Coturnix quail are similar (293 and 341 ppm, respectively Hill and Camardese (1986)), the dose-response curve for dimethoate taken from Radvanyi et al. (1986) overestimates observed chlorpyrifos-induced inhibition. Insecticide deposition on insects (as a proportion of amount applied) in the study of Martin et al. was 0.9–3.7 times ($\bar{X}=2.6$ times) the value from Forsyth and Westcott (1994) used in the model; however, insects in the former study were sprayed in a laboratory while insects in the latter were caught after field applications. General agreement between results of these two studies and model predictions of them provided good support for the model.

Simulation of the following three studies provided good support for insecticide decay predic-

tions, but only very weak support for ChE inhibition predictions because the studies found no significant differences in inhibition. We simulated a study of Somers et al. (1991) which showed that 5 days after spraying grain with carbofuran, insecticide concentration declined from 3.55 ± 2.77 μg/g ($\bar{X} \pm 1$ S.D.) to 1.39 ± 0.75 μg/g. The model predicted 1.33 μg/g after 5 days, which falls within 67% confidence interval of the field data. Somers et al. found 12.5% ChE inhibition in pheasants (*Phasianus colchicus*) sprayed with carbofuran, which was not significantly ($P > 0.05$) different from that in control pheasants. Likewise, the model predicted no marked increase in inhibition: 0.7% inhibition at the end of 5 days, with a maximum inhibition of 1.4%, 20 h after exposure. In this case, simulated inhibition levels were roughly one order of magnitude lower than mean observed levels. We then simulated the study of Fair et al. (1995), who examined impacts of carbaryl on killdeer (*Charadrius vociferus*). They found no significant ($P > 0.05$) differences in ChE activity between pre-treatment and post-treatment killdeer in the treated field. The model's estimate of maximum inhibition of 0.2% after 110 h of exposure fell within 67% confidence limits of the field data. Substitution of malathion-induced inhibition data for those of carbaryl may have underestimated impact of carbaryl. Finally, we simulated three birds from a study of George et al. (1995): the American kestrel, horned lark and western meadowlark. They found no significant ($P > 0.1$) difference in ChE inhibition between birds of the same species in treatment and non-treatment areas. The model also predicted very low maximum inhibitions of 0.0062% in the American kestrel and 0.65% in the horned lark after 14 h and 0.25% in the western meadowlark after 38 h.

3.2. Sensitivity analysis

To discern relative importance of foraging location, diet selection and food intake/body weight ratio on levels of ChE inhibition, we simulated hypothetical birds from each feeding assemblage eating either 15.4 or 30.8% of their body weight daily. Diet proportions of the birds were as follows: herbivores, 100% vegetation; insectivores, 100% insects; carnivores, 100% mammals; omnivores, 50% vegetation and 50% insects. We simulated each bird foraging in only one of each of the four crop fields for the entire season. These runs were deterministic because foraging location is the only stochastic variable in the model. The model applied only OP insecticides to focus on avian characteristics.

Results of sensitivity analysis show large differences in temporal dynamics of ChE inhibition by field type for all feeding assemblages except carnivores, which had very little inhibition (Figs. 2 and 3). Inhibition peaked at different times in each field, beginning with wheat in March and June, sorghum in April, cotton in May and maize in August and September. ChE inhibition among field types shows the largest differences in April, August and September; during these months, foraging location seems to have a larger effect than diet preferences. During months that potential ChE inhibition among field types is more similar, diet preferences become more important than foraging location. Within each field, granivores experienced the greatest ChE inhibition, followed by omnivores, insectivores and carnivores. Finally, a 100% increase in daily intake rate resulted in 0–200% increases in ChE inhibition, depending upon month, diet and foraging location. Because the model predicts little ChE inhibition in carnivores, it is insensitive to estimates of carnivore foraging location or food intake. For the other assemblages, ChE inhibition usually appears highly sensitive to estimates of foraging location and moderately sensitive to estimates of food intake.

4. Simulation of cholinesterase inhibition in selected birds

To predict sublethal impacts of insecticide applications on selected wild bird species in Texas, and to relate these impacts to diet preferences, foraging location and insecticide class, we ran 25 stochastic simulations of one individual from each of 15 species of birds foraging in a landscape composed of equal areas of rangeland, maize,

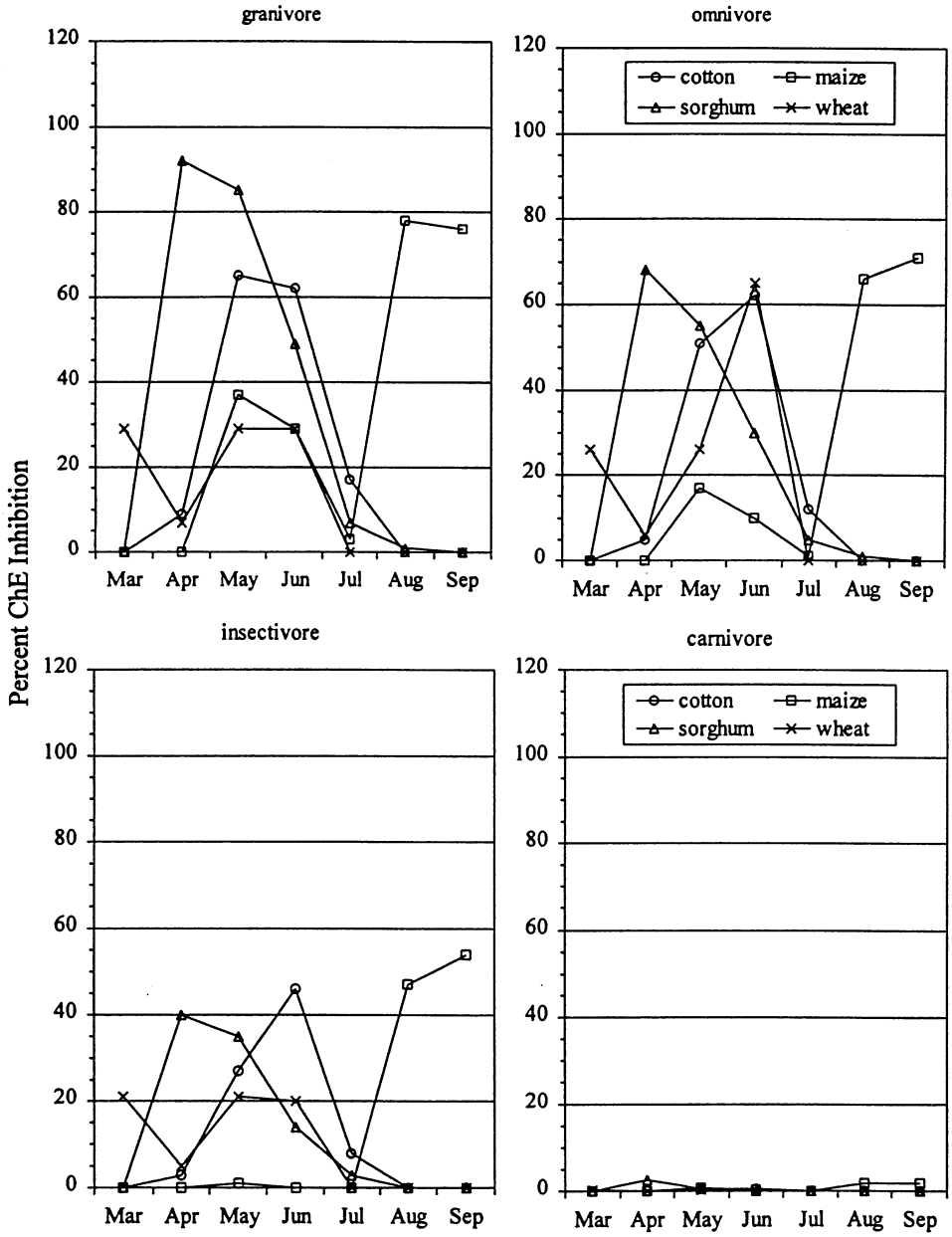

Fig. 2. Maximum percent ChE inhibition each month in a granivore, omnivore, insectivore and carnivore each ingesting food equal to 15.4% of body weight when foraging in only one of each of the four field types.

cotton, sorghum and wheat with up to two applications of OP or CA insecticides (Figs. 4–7). In general, granivores and omnivores experienced similar levels ChE inhibition during the first 3 months of the season, with less inhibition in insectivores and carnivores. By the end of the season we found no trends by feeding assemblage; instead, most species within each assemblage showed much variation. Except for the loggerhead shrike, which ate 28% insects, carnivores barely

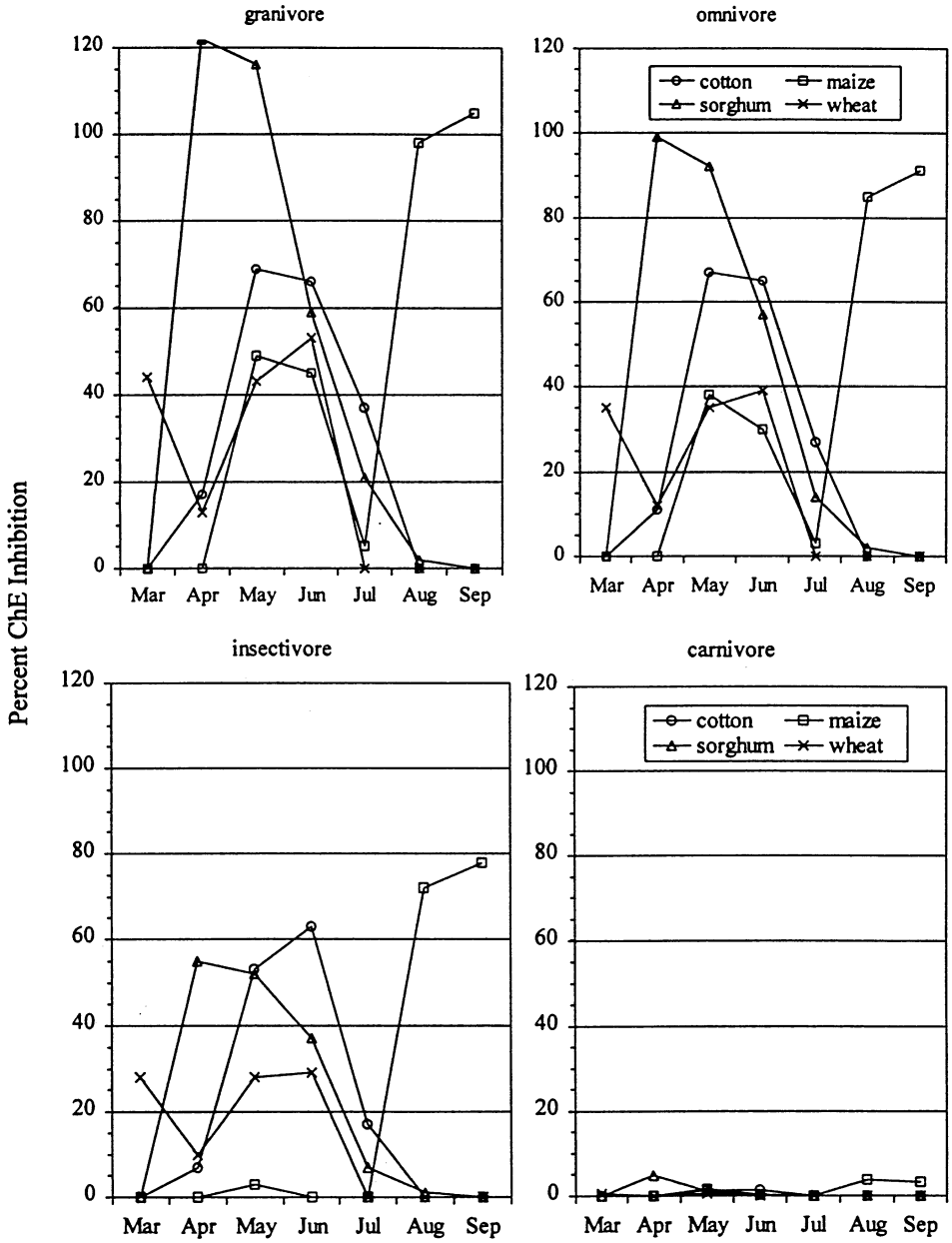

Fig. 3. Maximum percent ChE inhibition each month in a granivore, omnivore, insectivore and carnivore each ingesting food equal to 30.8% of body weight when foraging in only one of each of the four field types.

experienced inhibition. Effects of field selection were considerable; frequent inhibition showed influence from overlapping insecticide applications, while dips in inhibition showed non-application periods or increased ingestion of uncontaminated prey in rangeland.

During many months we observed large differences between mean maximum ChE inhibition caused by organophosphates and carbamates. Inhibition caused by organophosphates peaked in

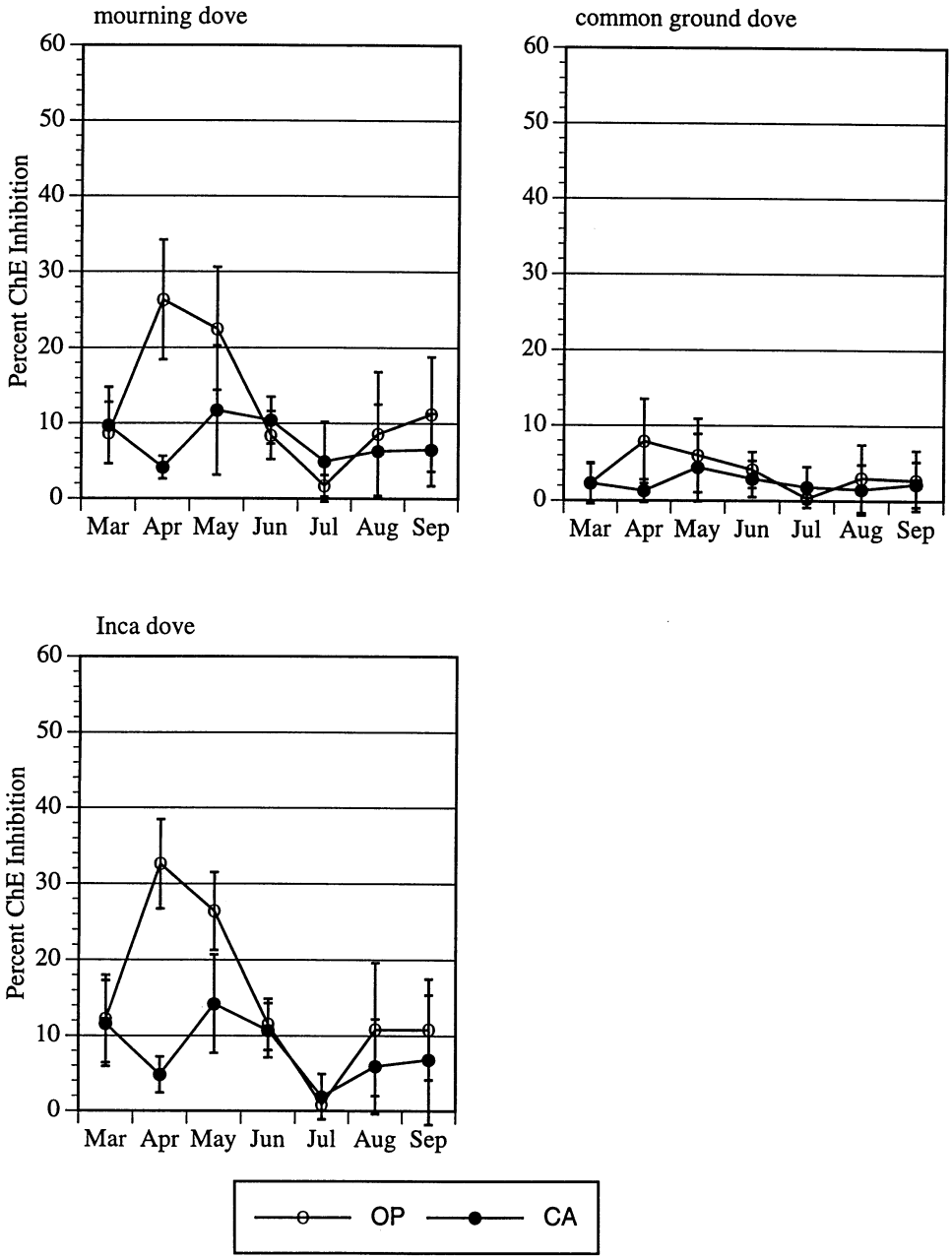

Fig. 4. Mean maximum percent ChE inhibition each month in granivores when exposed to OP or CA. Error bars represent ± 1 S.D.

April for all birds except the horned lark, red-winged blackbird, cattle egret and eastern bluebird, for which it peaked in August. Carbamate-induced inhibition peaked in May for all birds except the red-winged blackbird, for which it also peaked in August. All birds except for the common ground dove, eastern meadowlark, Carolina wren and the carnivores experienced at least 20% mean maximum ChE-inhibition after OP insecticide applications.

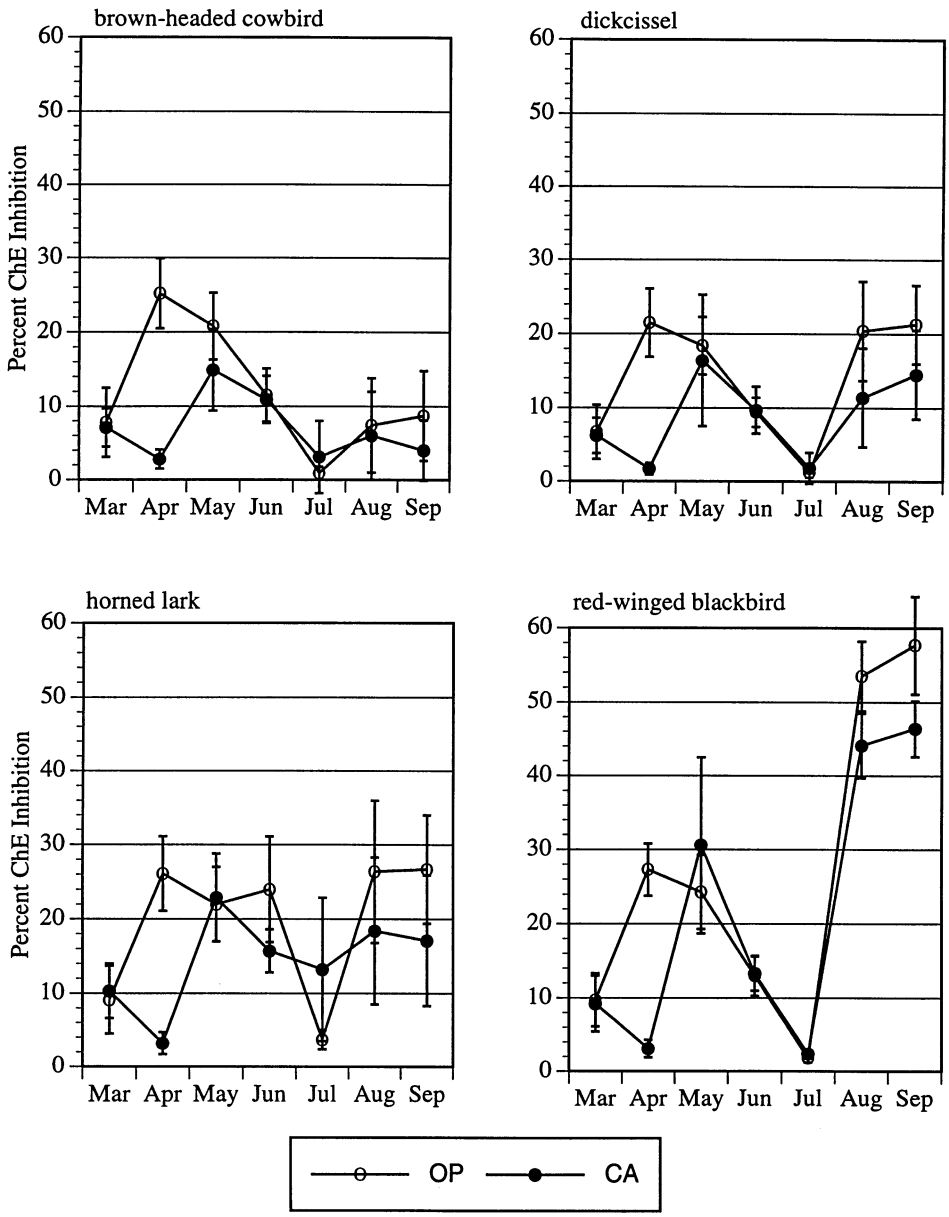

Fig. 5. Mean maximum percent ChE inhibition each month in omnivores when exposed to OP or CA. Error bars represent ± 1 S.D.

Only the horned lark and red-winged blackbird experienced the same mean maximum inhibition after CA insecticide applications. The red-winged blackbird experienced the greatest mean maximum inhibition of all birds, in September, of 57.7% and 46.4% for OP and CA insecticides, respectively.

Finally, we used the model to estimate impacts on birds if farmers over-apply insecticides. We doubled application rates of all insecticides and ran 25 replicate stochastic simulations of one bird from each foraging assemblage: the common ground dove, dickcissel, cattle egret and loggerhead shrike. Increase in mean maximum ChE

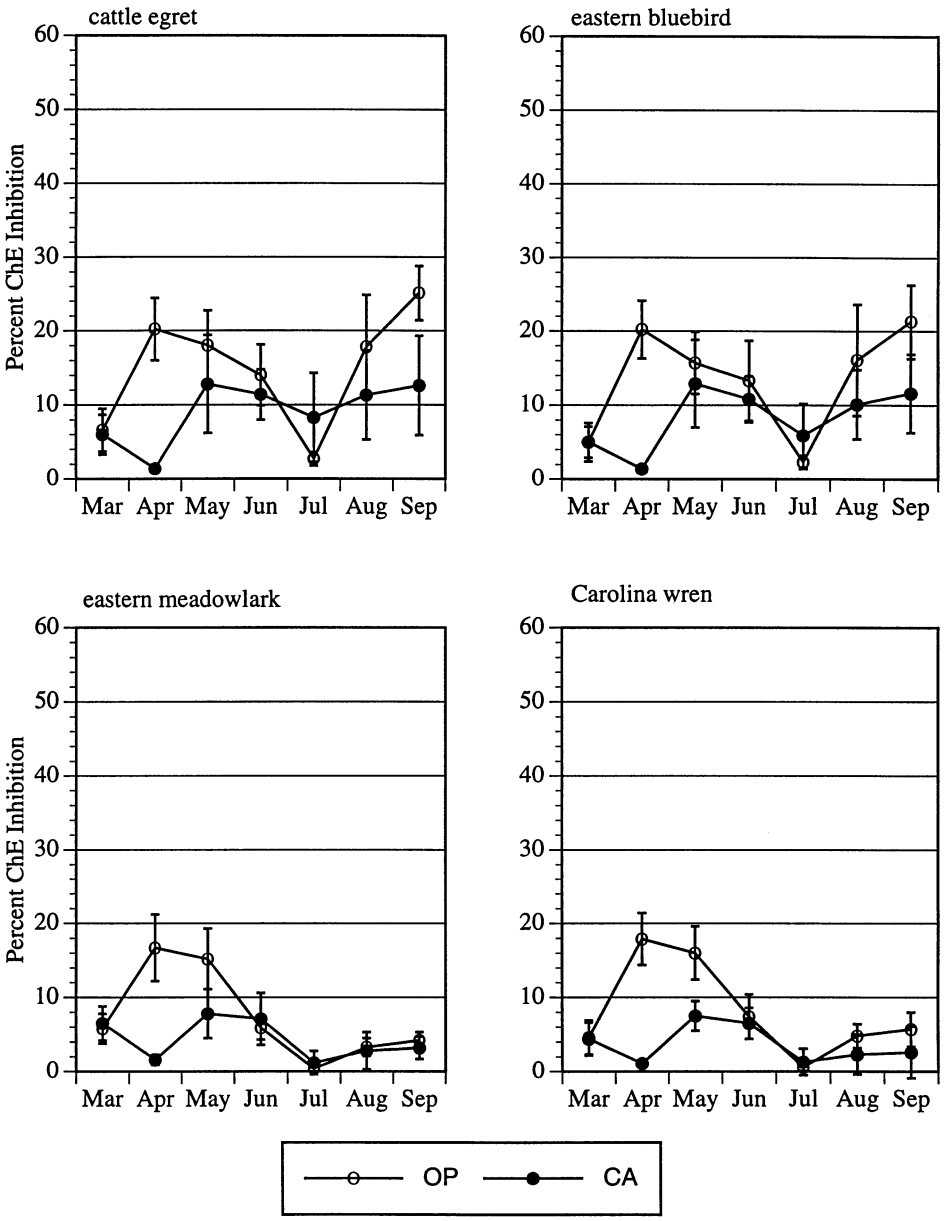

Fig. 6. Mean maximum percent ChE inhibition each month in insectivores when exposed to OP or CA. Error bars represent ± 1 S.D.

inhibition each month ranged from 13.3 to 161.1% among all birds (Figs. 8 and 9). Mean monthly increases averaged over the season ranged from 68.7 to 88.9% for organophosphates and from 98.3 to 115.9% for carbamates. Lumping insecticide classes together, overall mean increases for the common ground dove, dickcissel, cattle egret and loggerhead shrike were 83.6, 88.5, 103.3 and 101.5%, respectively. Total mean increase for all birds, regardless of species and insecticide class, was 94.2%. Thus, prediction of maximum ChE inhibition in the model does not appear overly sensitive to predictions of residue exposure.

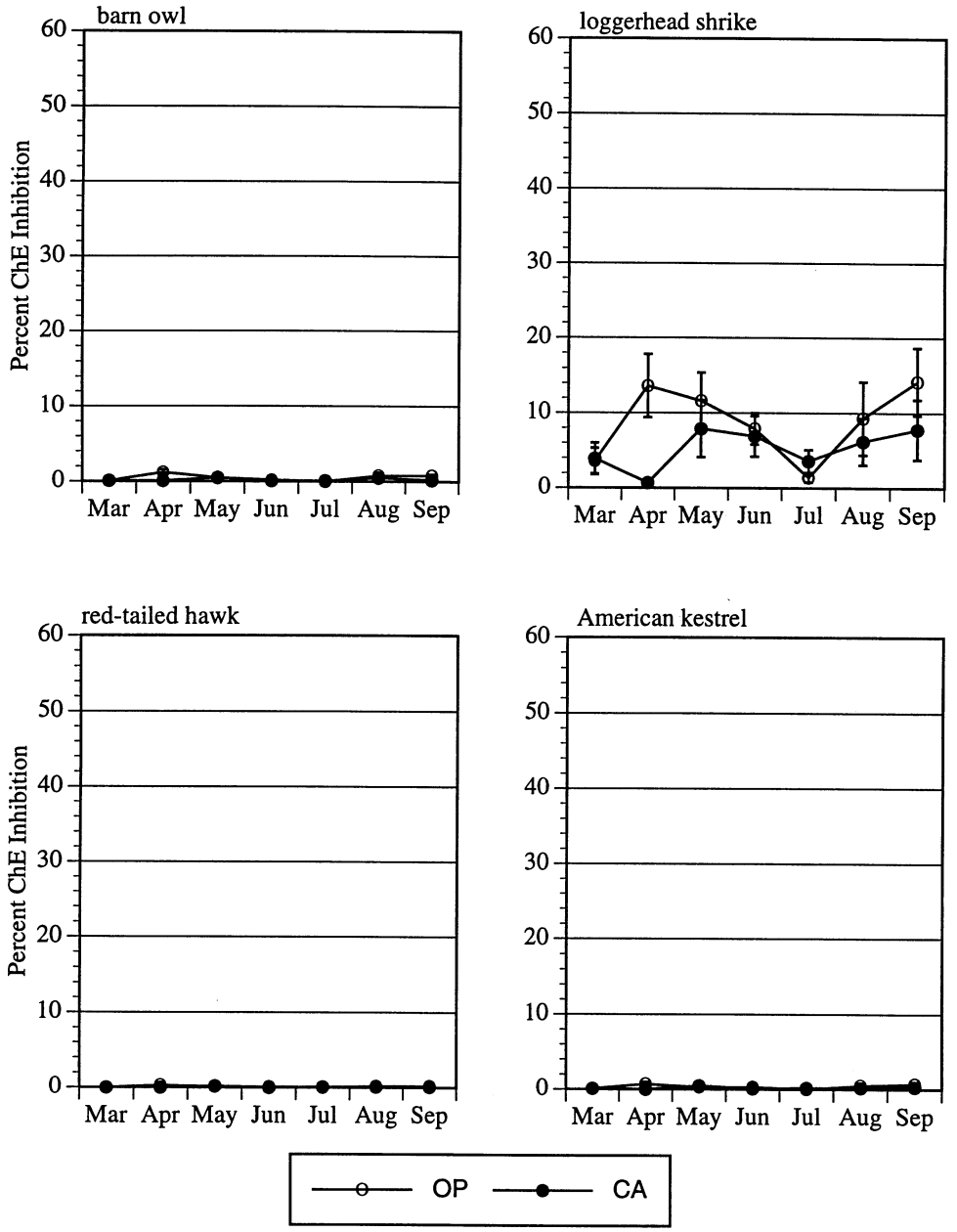

Fig. 7. Mean maximum percent ChE inhibition each month in carnivores when exposed to OP or CA. Error bars represent ± 1 S.D.

5. Discussion

5.1. Model results

Insecticide applications, foraging location, diet composition and daily intake rate directly drive temporal dynamics of ChE inhibition. For most birds, foraging location influences exposure more than diet. Birds that forage more frequently in rangeland experience lower ChE inhibitions because no insecticide applications occur there. Exposure and inhibition decrease as crops grow and

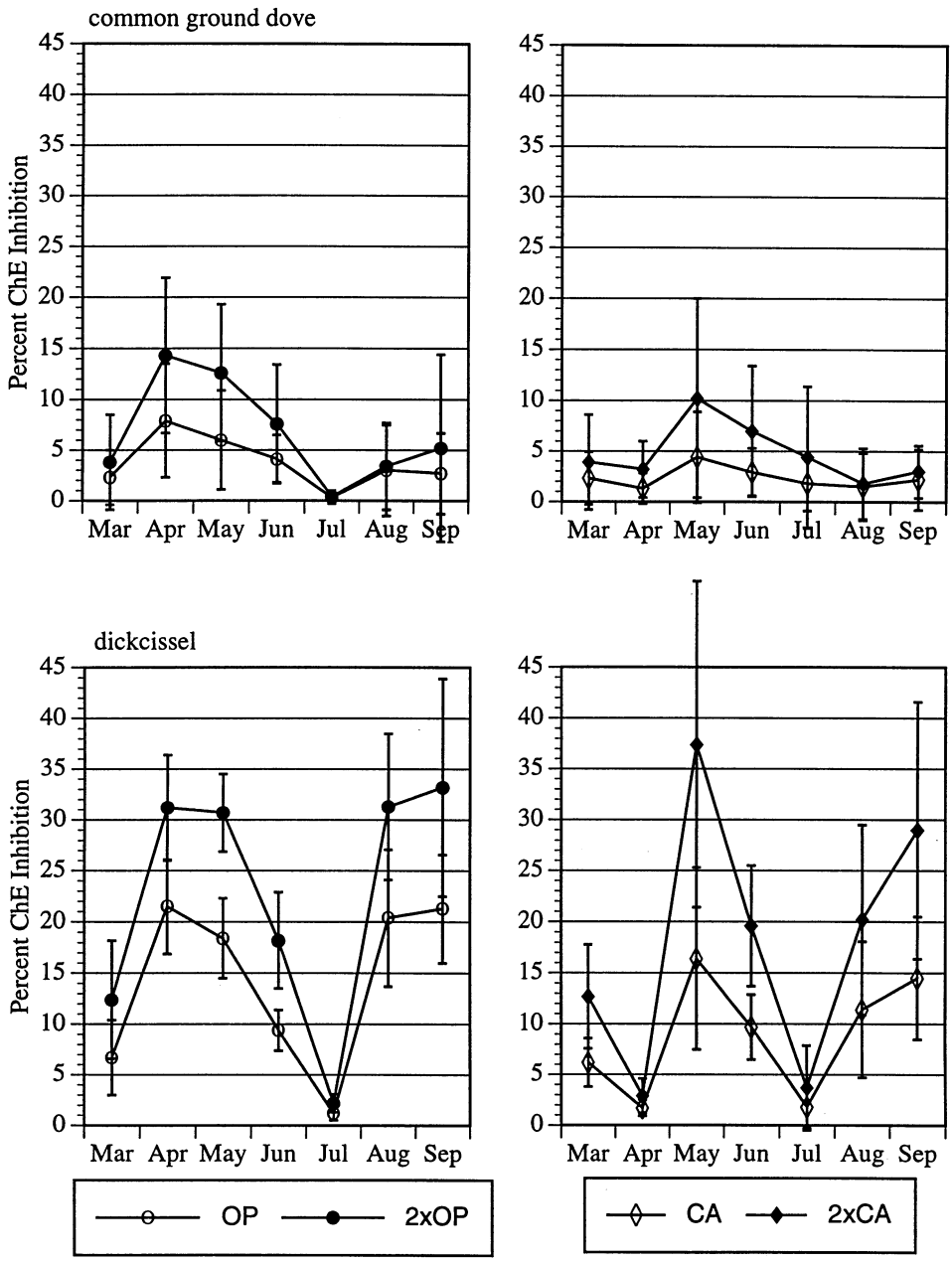

Fig. 8. Mean maximum percent ChE inhibition each month in the common ground dove and dickcissel when exposed to 1- or 2-times the recommended amount of OP or CA. Error bars represent ± 1 S.D.

birds spend less time in crop fields. Because the model red-winged blackbird eventually spends 70% of foraging time in maize, it experiences the highest mean maximum ChE inhibition of all birds due to application of chlorpyrifos or carbofuran. During periods when amounts of residues in fields are more equal, diet preferences and daily intake become more important. For example, in July, when most residues have decayed, daily intake is more important in determining exposure

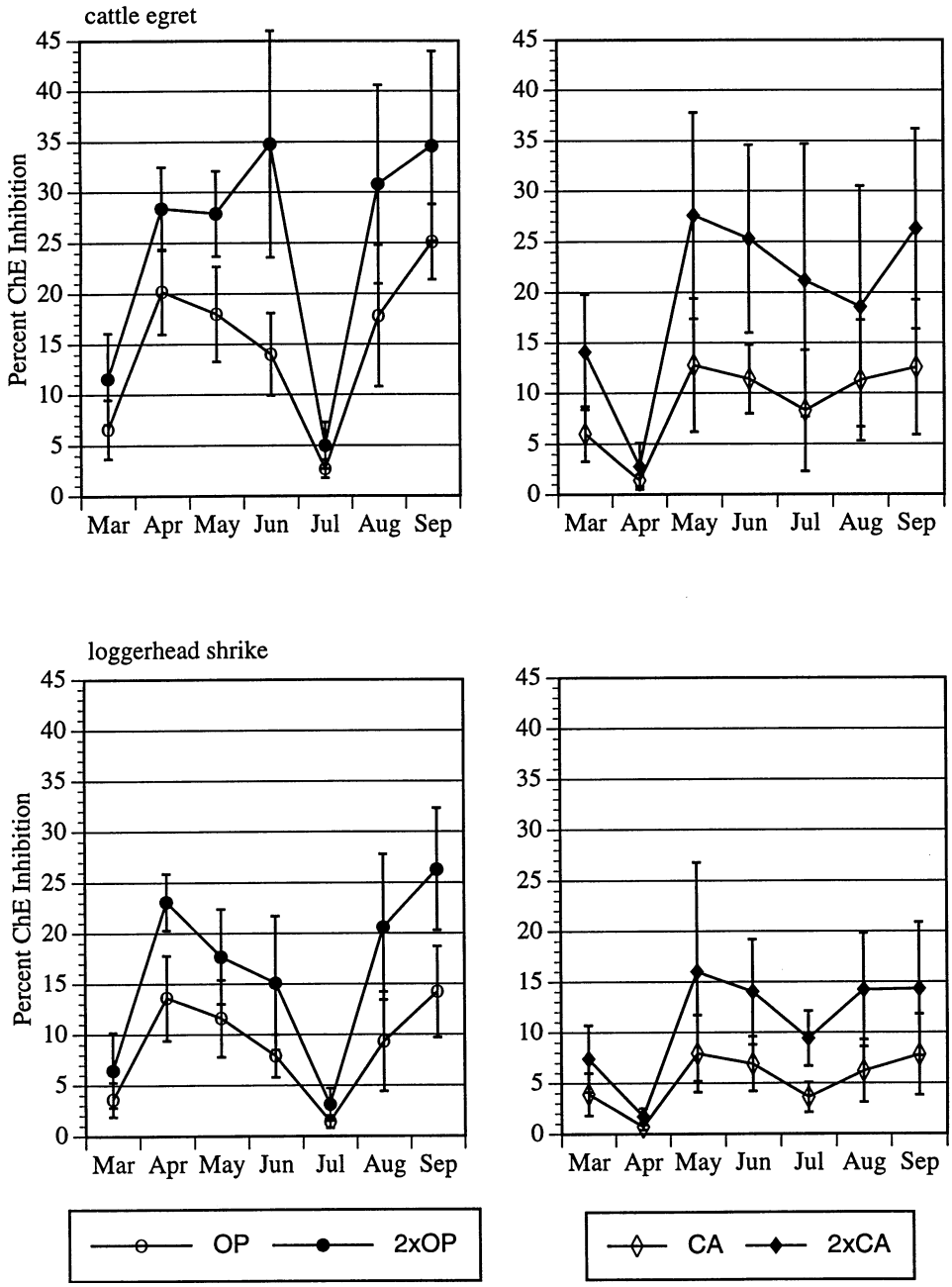

Fig. 9. Mean maximum percent ChE inhibition each month in the cattle egret and loggerhead shrike when exposed to 1- or 2-times the recommended amount of OP or CA. Error bars represent ± 1 S.D.

for most birds than foraging location. Granivores usually experience more inhibition because vegetation contains higher concentrations of residues than insects or mammals. Inhibition tends to increase with proportion of vegetation in the diet. Carnivores experience very little ChE inhibition because their mammal prey rapidly lose the few residues they receive.

Finally, differential sensitivity to insecticides played a smaller role. Organophosphates tended to cause greater ChE inhibition than carbamates in the model. Large inhibition in the red-winged blackbird mentioned previously owes more to the high toxicity of chlorpyrifos (though dimethoate was substituted) than to concentrations of its residues, which were lower than those of most other insecticides. The largest differences in impact between insecticide classes, in May and September, seem due to application of chlorpyrifos in sorghum and maize, respectively. The peak in inhibition under the CA scheme that occurs in May is due to application of dimethoate (an organophosphate, because no carbamate was recommended).

5.2. Effects of sublethal ChE inhibition

Relationships between degree of ChE inhibition and effects on wild birds have not been established because they vary tremendously (Hill, 1995). Ludke et al. (1975) suggested that 50% ChE inhibition in wild birds found dead suggests mortality caused by antiChE chemicals, while 20% inhibition suggests 'significant' exposure. Peakall (1985) stated that 80% ChE inhibition is necessary to kill a bird in a single dose, but that chronic inhibition of 50% also can cause death (though he did not offer a time-frame). Laboratory birds experiencing ChE inhibition greater than 50% often survive, but impact of such inhibition in wild birds is less well known (Grue et al., 1991). Forsyth and Martin (1993) found that inhibitions of 42–59% decreased foraging and hopping time in white-throated sparrows (*Zonotrichia albicollis*). Conversely, ChE inhibition of 25% in American kestrels did not alter predatory vigilance and attack behavior (Rudolph et al., 1984). In European starlings (*Sturnus vulgaris*) 12% ChE inhibition altered body posture, $>27\%$ inhibition decreased foraging activity and $>39\%$ reduction reduced flying and singing; however, 10% inhibition increased flying (Hart, 1993). Galindo et al. (1985) found that 57.2% inhibition made northern bobwhite more susceptible to domestic cat predation. Hill (1995) states that sublethal exposure of 5% of LD_{50} can reduce a bird's core body temperature by 2–6°C and that hazard may be greater for hatchlings, which have underdeveloped thermoregulatory mechanisms. Thus, birds in the model that experienced 20% ChE inhibition might have experienced nausea and/or exhibited behavioral changes such as fatigue, listlessness or restlessness. The birds that experienced 30–57% inhibition would have a much greater chance of more severe behavioral and physiological effects. Three bird species simulated (i.e. dickcissel, loggerhead shrike and American kestrel) have been listed as 'special interest' birds (TPWD, 1996) because their populations have decreased in certain areas of the USA. The model predicts mean maximum ChE inhibitions of 21.5, 13.6 and 0.7% for the dickcissel, loggerhead shrike and American kestrel, respectively. While habitat loss may help explain their declines, insecticide exposure also may play a role.

5.3. Comparing model with reality

The model assumed a worse-case scenario with two applications of several OP or CA insecticides at the highest recommended rates due to five pest insect species causing economic damage in each field. Although such a scenario could occur, birds in this area usually would experience less impact because the proportion of each land type is very unequal. In the study area, each land-use type takes up the following percentage of area: rangeland, 95.7%; maize, 0.3%; cotton, 0.2%; wheat, 0.1%; sorghum, 0.1% (TASS, 1994). Only birds in certain locations might have all five land types in equal proportions in their foraging ranges (TASS, 1994). Even birds showing a preference for croplands (e.g. red-winged blackbirds) would have a much greater opportunity to forage in rangeland than they do in the model. Nonetheless, we chose to use equal land areas in the model to estimate the greatest possible ChE inhibition in a bird that visits all five land-use types. In addition, application of so many insecticides rarely occurs because farmers usually do not need to control so many insect species in a single season. However, some infestations of a species, especially one highly resistant to certain insecticides, can result in repeated applications of the same insecticide (Custer

and Mitchell, 1987). Cotton farmers in the Rio Grande valley of Texas recently (1995) adopted a boll-weevil eradication program that mandates several applications of malathion over large areas (Howe Verhovek, 1996); these many applications increase exposure risk to wildlife. Cotton tends to require more pest control than other crops in this area because its indeterminate flowering habit makes it susceptible to attack over a longer period (Tivy, 1990).

We did not explore factors affecting behavior of insecticide residues. Factors such as temperature, precipitation, solar radiation, wind speed and soil type can change insecticide loss rate dramatically. Loss rate tends to increase with increasing temperature, wind speed and solar radiation, and to decrease with increasing precipitation (Nigg et al., 1983); however, these factors often confound one another. Too much precipitation can dislodge residues and increase loss rates due to runoff (Cessna and Westcott, 1993).

Ingestion of insecticide granules is a problem that poses great risk to some granivores and omnivores. Balcomb et al. (1984) found that ingestion of one granule of carbofuran could cause death in house sparrows (*Passer domesticus*) and red-winged blackbirds. Granules similar in size or shape to grit that birds ingest put some species at greater risk; Best (1992) showed that sizes of Counter® (terbufos) 15G granules overlap grit sizes preferred by American crows (*Corvus brachyrhynchos*) by 79%.

Granivores may be at greater risk not only because vegetation contains greater residue concentrations than insects or mammals, but because systemic insecticide metabolites produced by plants may be more toxic than the parent compound (Hill, 1995). Further, systemic residues inside plants are less readily detected and therefore more easily ingested than surface residues that birds in some studies have rejected (Hill, 1995). On the other hand, Best and Fischer (1992) state that only species that eat developing seedlings risk exposure to systemic insecticides. Assuming in the model that these insecticides would be available in all plant parts beginning the day of application may have overestimated granivore exposure. Carnivores may experience greater insecticide exposure than predicted because prey affected by antiChEs may be more conspicuous and/or easier to catch. Hunt et al. (1992) found that American kestrels preferentially caught fenthion-affected house sparrows over control sparrows. Wild birds also may receive significant insecticide exposure by drinking contaminated water from puddles left in fields after rainstorms (Hill, 1992). Contaminated puddles may persist relatively longer in Texas because most Texas soils contain large amounts of clay (Hallmark, 1986). Birds also receive insecticide exposure through preening of contaminated feathers, dermal contact, and inhalation (Hill, 1992).

5.4. Uncertainties in model parameterization

Variations in normal ChE levels and uncertainties in measuring them may generate some of the model's inaccuracies. Researchers consider wild birds to have received recent exposure to antiChE compounds if brain ChE activity lies below the normal bound ($\bar{X} - 2$ S.D., ca. 20% inhibition) for that species (Hill, 1992). However, normal levels of ChE vary among individuals of the same species depending upon age, sex, season and body condition (Greig-Smith, 1991). Young birds, which produce less ChE, usually are at greater risk from antiChE application because (1) they eat a larger amount of food in proportion to body weight daily than adults, thus increasing exposure, and (2) toxicity of a compound often is greater for juveniles due to their underdeveloped metabolism (Hill, 1995). However, age-specific effects are confounded as well; juveniles are less susceptible to some compounds because their immature nervous systems are less sensitive (Hill, 1992).

Including more accurate ChE-inhibition dose–response curves of each insecticide in every bird in the model would improve accuracy greatly. Although LC_{50} data from Hill and Camardese (1986) provided relative toxicities for substitution purposes, ChE inhibition observed by other researchers in both field and laboratory may not show the same pattern. Tucker and Leitzke (1984) suggest that an LC_{50} dosage of many OP insecticides will produce an average brain ChE inhibition of $80 \pm 10\%$; however, more studies are

necessary to support the relationship. The necessity of using data for mice, ducks, quail and sparrows for all birds in the model reduced its accuracy. Hill (1992) states that although any avian test species can represent adequately acute sensitivity of birds to antiChE compounds, one cannot use response of one species to estimate sensitivity of another. Due to physiological differences, each bird species has a different mean tolerance for the same compound; e.g. red-winged blackbirds are 10-times more acutely sensitive to some insecticides than pheasants (Hill, 1995). Further, brain ChE activity of northern bobwhites rarely decreases by more than 25% from exposure to terbufos, even with lethal exposure (Tank et al., 1993); thus, the model, which uses bobwhite data for terbufos, may underestimate terbufos-induced ChE inhibition. Insecticides also can have very different effects upon ChE of individuals of the same species (Hill, 1992). Effects of OP insecticides tend to be cumulative, while those of carbamate insecticides are more easily reversible (Grue et al., 1991); however, animals acutely poisoned by carbamates may die of respiratory arrest before brain ChE is inhibited (Greig-Smith, 1991).

In addition, responses to the same compound often differ depending upon whether the dose is taken all at once (i.e. acute) or in small amounts in the diet (Hill, 1992). Because we took all ChE-inhibition data, except those for carbofuran, from studies of acute doses, the model may not represent responses to subacute dietary doses accurately. The hazard of sequential sublethal exposure to both OP and CA insecticides may depend upon which compound class the bird encounters first. Studies suggest that initial exposure to CA insecticides may protect against subsequent exposure to OP insecticides (Hill, 1992), but the reverse may not be true: exposure to malathion followed by carbaryl increased ChE inhibition in red-legged partridges (*Alectoris rufa* cross) 2-fold in acute studies (Thompson, 1996). Further, repeated sublethal exposure to antiChE compounds may confer a degree of protection on acute antiChE toxicity (Hill, 1992).

Although our representation of bird foraging may be inaccurate, its utility lies more in exploring relative importance of foraging characteristics that determine dietary insecticide exposure and subsequent ChE inhibition than in making predictions. Birds that forage in landscapes with a higher diversity of crop types or birds with larger foraging ranges, may have greater risk of insecticide exposure due to overlapping applications. Whether this means that these birds will experience greater ChE inhibition depends upon types and amounts of insecticides applied to other crops. More detailed information about foraging behavior of individuals can improve predictions of their exposure to insecticide residues. Smaller birds, which have greater daily intake/body weight ratios, may be at greater risk because they ingest proportionally larger amounts of residue.

5.5. Importance of modeling to risk assessment

In the US and Europe, risk assessment decisions for vertebrate wildlife are based upon estimated daily residue intake/LD_{50} quotients (dose/toxicity (D/T) quotients) for certain pesticides tested on laboratory animals (Thompson, 1996). Low risk is defined for D/T values < 0.1, high risk for values > 1.0, and medium risk for values in between. The use of a model that could estimate D/T quotients for wildlife could be very useful for management decisions, even if they do not take into account sublethal effects that may cause reduced survival of individuals. Improved models could weigh relative advantages and disadvantages of similar insecticides. In addition, accurate models can have advantages over field studies of ecological risk assessment, including needing less time and money and allowing quicker response for managers. Birds are an excellent choice for toxicological monitoring because they are highly visible, are sensitive to many toxic substances, have well-known life histories and occupy most trophic levels in nearly all ecosystems (Kendall and Akerman, 1992). This model performs an important component of risk assessment by predicting maximum possible insecticide exposure and resulting ChE inhibition. Perhaps in the future, simulation models of ecological impacts of insecticides will guide the focus of laboratory bioassays, which currently are the first steps taken in registration of new insecticides.

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