SETAC PRESS

Environmental Toxicology and Chemistry, Vol. 21, No. 7, pp. 1497–1506, 2002
© 2002 SETAC
Printed in the USA
0730-7268/02 $9.00 + .00

ESTIMATING THE PROBABILITY OF BIRD MORTALITY FROM PESTICIDE SPRAYS ON THE BASIS OF THE FIELD STUDY RECORD

PIERRE MINEAU*
National Wildlife Research Centre–Canadian Wildlife Service, 100 Gamelin Boulevard, Hull, Quebec KIA OH3, Canada

(*Received* 11 *September* 2001; *Accepted* 11 *January* 2002)

Abstract—The outcome of avian field studies was examined to model the likelihood of mortality. The data were divided into clusters reflecting the type of pesticide application and bird guilds present on site. Logistic regression was used to model the probability of a bird kill. Four independent variables were tested for their explanatory power: a variable reflecting acute oral toxicity and application rate; a variable reflecting the relative oral to dermal toxicity of the pesticides; Henry's law constant; and a variable reflecting possible avoidance of contaminated food items, the hazard factor (HF). All variables except for HF significantly improved model prediction. The relative dermal to oral toxicity, especially, was shown to have a major influence on field outcome and clearly must be incorporated into future avian risk assessments. The probability of avian mortality could be calculated from a number of current pesticide applications and the conclusion was made that avian mortality occurs regularly and frequently in agricultural fields.

Keywords—Cholinesterase-inhibiting insecticides Avian mortality Modeling Field studies Pesticide impacts

INTRODUCTION

Currently, most of the regulatory effort in pesticide avian risk assessment centers on the development of models incorporating laboratory-determined toxicity as well as estimates of food item residues and consumption rates. These models can be of a deterministic nature but, increasingly, natural variation and model uncertainty are incorporated into the risk assessment process through probabilistic tools such as Monte Carlo simulations. These assessments typically are carried out chemical by chemical or, in some cases, on a cluster of pesticides used for the same purpose. Exposure models are particularly difficult to construct and are very data-intensive. Furthermore, none of the avian models used by pesticide regulatory agencies worldwide have yet been formally validated against actual field outcomes.

Field investigation typically takes one of two forms. The first is active monitoring or directed field studies where the experimental conditions are controlled and the outcome is completely open. The second is passive monitoring, also referred to as incident monitoring, where observers investigate a problem that has arisen during operational pesticide use. The first type is of particular interest here, although the latter has also provided valuable insights on some pesticides under specific use situations.

A single directed field study by itself may not be sufficient to dispel a presumption of high risk that is placed on a pesticide. This is because of the stochastic variability encountered in most field situations, as well as the inability to detect impacts every time they occur (in part because of the difficulty of finding evidence of an impact such as carcasses). Recognizing this, the U.S. Environmental Protection Agency proposed that a large number of fields needed to be monitored to increase the confidence of a finding of no effect [1]. This requirement meant that substantial costs needed to be expended for each pesticide under presumption of causing avian harm (essentially mortality). This was one of the reasons field studies eventually fell out of favor with the U.S. pesticide regulatory system. Yet, the logic was probably sound: Several field studies are needed to uncover avian mortality that is either sporadic or even regular, but with a low frequency of occurrence.

Recognizing this limitation of compound-specific assessment, the aim of this exercise was to analyze the entire body of pesticide avian field research to draw parallels between products and to develop predictive models to assess the likelihood of bird impacts. Analysis of the field studies will also provide data necessary for validation of current risk assessment schemes.

METHODS

The Canadian Wildlife Service conducted an extensive search for all field studies where investigators had looked for impacts to birds after a pesticide application. Studies were found for two groups of pesticides: persistent organochlorines such as DDT and dieldrin, and cholinesterase-inhibiting pesticides, namely organophosphorous and carbamate compounds. Only the latter were retained. The often lengthy delay between exposure and effect in the case of bioaccumulative organochlorine compounds proved difficult to address in this analysis. Furthermore, the intent was to construct predictive models of avian impact for modern (i.e., nonbioaccumulating) pesticides. Three types of studies were obtained: studies published in the open literature; unpublished government reports, mostly from the United States and Canada; and studies submitted by manufacturers in support of registration. The latter were restricted to those available under U.S. freedom of information provisions and, therefore, deemed not to contain confidential business information. Many of these studies have been summarized on the U.S. Environmental Protection Agen-

* To whom correspondence may be addressed (pierre.mineau@ec.gc.ca).

Table 1. Classification of data points into similar logical clusters based on the type of pesticide application and the putative routes of exposure for birds present on site and/or affected. The first three clusters have been sufficiently tested and it is not necessary to include incident data. The last three represent situations where the incident data are needed to better assess the risk

Application site or route of exposure	Principal bird guilds represented in study	Typical insect pest	Ground or aerial[a]	No. study data points used in the analysis	No. incident data points used in the analysis	Total no. data points
Field and pasture crops	Primarily insectivore–granivore species	Aboveground insects (e.g., Lepidoptera, Coleoptera)	Aerial and ground	88	0	88
Forestry	Primarily insectivore–granivore species	Arboreal insects; mostly Lepidoptera	Primarily aerial (primarily ULV)	41	0	41
Orchards	Primarily insectivore–granivore species	Arboreal insects; mostly Lepidoptera	Primarily ground-based air-blast application	16	0	16
Mosquito control	Primarily insectivore–granivore species	Mosquitoes	Ground or aerial (thermal fogger or UULV)	4	4	8
Drinking spray solution[b]	Any guild	Aboveground insects (e.g., Lepidoptera, Coleoptera)	Primarily ground	0	8	8
Crops suitable for grazing or turf	Grazing herbivores	Aboveground or soil insects	Aerial and ground; turf applications often accompanied by heavy irrigation to drive pesticide into thatch and soil	5	15	20

[a] ULV = ultra-low volume; UULV = ultra–ultra-low volume.
[b] Cases where access to spray solution was specifically mentioned as the cause of bird impacts. This could be birds drinking from puddles, irrigation tubes, or from the leaf whorls of plants.

cy website in response to the ongoing organophosphorus and carbamate reevaluation.

Minimum criteria of acceptability

To be accepted into the analysis, studies had to fulfill several criteria. First, the identity, rate of application, and crop had to be given and the pesticide had to be applied as a spray. Second, some evidence had to exist that birds were present on or near the site of application. Third, the methodology had to be determined to be adequate and scientifically credible. Because virtually all the studies attempted to document acute effects such as lethality, this amounted to evaluating the adequacy of the study author's carcass-searching procedures. In arriving at this determination, the experience and track record of the team as well as the description of the methods were used to evaluate the validity of the study. For example, in the absence of a sufficiently detailed description of the methods, a negative (no-effects) finding by a team of researchers with extensive experience and prior demonstrated ability to detect avian mortality was more likely to be accepted than that by another team with no such track record.

In an attempt to confirm and add credence to available field studies, a number of sources of avian incident data were searched, namely the U.S. Environmental Protection Agency EIIS database, yearly incident reports from the United Kingdom and France, and sporadic publications or reports of cases from other countries. This search uncovered exposure routes and situations which had not been formally field-tested or, which had been tested infrequently. Incident data were not considered for situations or routes of exposure already well represented in the field study record. Where similar incidents were uncovered, the information was summarized by a single data point (but see below for caveats associated with incident data). For example, a large number of incidents were found of waterfowl mortality with the insecticide diazinon applied to turf grass at 4 kg active ingredient (a.i.)/ha [2]. All of these incidents made up a single data point in the consideration of the overall data record. In a few cases of repeat mortality incidents with a pesticide at an unspecified rate of application, the maximum labeled rate of application was assumed.

Description of the data set

A total of 181 data points were used in this analysis. Space constraints made it necessary to place this information on the *Environmental Toxicology and Chemistry* web site [*www.etc.allenpress.com*]. These data points represent field investigations on 35 cholinesterase-inhibiting pesticides with between 1 and 14 investigations per pesticide.

Clearly, the different application methods or circumstances under which pesticides are applied can lead to radically different exposure levels for birds. In standard risk assessment, exposure is assessed by measuring or estimating residue levels in foodstuffs after a certain type of application [3], and then by estimating food intake on the basis of feeding preferences and intake rates based either on body weight alone or on a combination of body weight and the caloric value of the food. Based on experience of pesticide application, avian ecology, and a few exploratory analyses of the data set, the data were partitioned into logical clusters reflecting comparable exposure conditions. These are described in Table 1. Ground and aerial applications were not separated, so as not to reduce sample sizes any further, and because exploratory analyses indicated that this was not a significant variable. For example, attempts to introduce a correction factor to account for higher putative deposits resulting from ground applications (deposits measured after aerial application are often 40–60% of equivalent ground applications) failed to improve model prediction (see below).

Evidence of impact

All of the studies were short-term studies and the analysis therefore was restricted to acute effects such as immediate mortality after application. The analysis was unable to address other possible impacts such as delayed mortality resulting from more subtle debilitation, reproductive effects, or indirect effects mediated through pesticide-induced changes in cover or

Table 2. Breakdown of data points by impact score from 1 to 4 (see text)

Data cluster	Impact score of 1	Impact score of 2	Impact score of 3	Impact score of 4
Field and pasture crops	36	15	30	7
Forests	22	4	12	3
Orchards	5	4	5	2
Fogging for mosquito control	3	0	2	3
Access to spray solution	0	0	2	6
Any grazed crop	1	0	9	10

food supply. Because of this, the degradation rates of the different pesticides were ignored assuming that, in most cases, the kills occurred within a few days after application. Because few studies corrected carcass search results to account for searcher efficiency or for scavenging, a semiquantitative four-level scoring system was used to categorize each of the 181 data points. A score of 1 was given to studies with no demonstrated avian impact. A score of 2 was given to studies where avian effects were slight and thought to be of a sublethal nature only. This score was given to studies where some toxic signs or debilitation were seen, although comprehensive carcass searches failed to document mortality. This score also was given to studies that documented substantial (i.e., >50%) brain acetylcholinsterase inhibition but, again, no mortality. A score of 3 was assigned to studies where compound-related mortality was deemed to have occurred. Criteria used to assess whether the mortality was compound-related included the number of deaths or comparison with check plots where available, residue and or cholinesterase data, and signs of intoxication. Finally, a score of 4 was given to cases of mass mortality, either a large number of birds of one or a few species or a few casualties from each of many species. For the purpose of the analyses described here, the data were further combined into two categories: applications that were deemed not to give rise to mortality (1 and 2 combined), and applications that did give rise to mortality (3 and 4 combined).

Table 2 gives the breakdown of data points by impact score. A number of biases are introduced at the outset into such a rating scheme. First of all, confidence is greater about giving a score of 3 or 4 than a score of 1 or 2. A score of 1, especially, is much more vulnerable to the experimenter's failure to detect evidence of impact such as carcasses. Carcasses are difficult to detect and many are scavenged [4,5] This means that any general extrapolation obtained from an analysis of field studies will, by definition, tend to underprotection because a number of 1 and 2 scores likely were in fact detection failures. Also, the distinction between a score of 3 and a score of 4 may be somewhat arbitrary. A score of 4 may have resulted from a higher bird presence (such as a wintering flock) rather than an inherently more hazardous situation. Finally, the necessary use of incident data for three of the six data clusters (mosquito spraying, birds drinking spray solution, or grazing sprayed vegetation) introduces a bias towards overprotection. This is because incident data are a priori classified as scores of 3 or 4. Safe applications not causing mortality would not be similarly reported, seriously limiting what inferences can be drawn from these clusters, for example, defining rates of application that do not kill birds. Incidents may represent situations where application rates were in fact higher than stated, although this is unlikely here. Mosquito control operations are conducted under the close supervision of government agencies, thus reducing the likelihood of overapplication. As for the other two data clusters, the level of risk assessment is believed to be adequate to show that problems can and do occur at labeled rates of application and overapplication need not be invoked to explain bird kills (e.g., see Holmes et al. [6] for spray solutions, and Mineau [7] and Stone and Gradoni [2] for grazers).

Constructing probabilistic models to predict impact scores from several explanatory variables

Logistic regressions [8] were used to construct empirical models to predict the likelihood of a given impact score on the basis of one or several independent variables. Model fit was determined from chi-square statistics. Because of the relatively small sample size available, model fit was not tested against an independent sample of data points. Therefore, calculated classification errors should be considered minimum estimates. Based on knowledge of the ways birds could be exposed to pesticide residues after an application, the following explanatory variables were constructed.

Variable incorporating acute oral toxicity and application rate. Thinking that the toxicity of a pesticide is key to understanding whether birds are likely to die under actual use conditions is logical. Two laboratory-derived measures of pesticide toxicity generally are available for birds: the median lethal dose (LD50) or the 5-d dietary median lethal concentration in feed (LC50). Mineau and colleagues [9] argued in favor of the LD50, at least where a certain degree of food avoidance has been shown, as is the case with many of the organophosphorous and carbamate pesticides that make up the field database. Also, the large number of bird species killed after pesticide applications means that the toxicity of the pesticides to each of the affected species cannot be known. Therefore, a measure of the susceptibility of birds at large to the pesticides of interest is needed. Too few species have been used in dietary tests to allow a good understanding of interspecies sensitivity differences, whereas interspecies variability has been well characterized in the case of acute dosing. The large interspecies variability typically seen means that it is difficult to compare fairly the toxicity of different pesticides to birds, especially when the products to be compared have been tested against different numbers of species. Based on the species distribution work of Aldenberg and Slob [10] modified for the way toxicity scales to body weight in birds [11], a distribution-based method has been developed that seems to provide for fair among-chemical comparisons [12]. In keeping with the work presented by Mineau and colleagues [12], the 5% hazardous dose (HD5) is used here as the measure of a pesticide's inherent oral toxicity to birds (Table 3). These values are the median estimates of the LD50 at the 5% lower tail of the avian species' sensitivity distribution for each pesticide. These values are corrected to be representative of species ranging from 20 to 1,000 g in weight, which accounts for the majority of avian casualties seen in documented bird kills.

The application rate of a pesticide clearly affects the potential for expression of a toxic response. In current risk assessment practices [13], the assumption is made that hazard increases linearly with application rate, because of the linear relationship between application rate and residue levels in putative avian food items. However, a recent reanalysis suggests that this relationship may need to be revisited [14]. A simple index combining toxicity and application rate first was pro-

Table 3. Tabulated chemical-specific variables used in the analysis of avian field studies. See text for definitions and procedures[a]

Pesticide	HD5 (mg/kg)	HLC (unitless—calculated)	Log K_{OW} (calculated)	MW	MV (cm^3/mol)	Rat oral LD50 (mg/kg)	Rat dermal LD50 (mg/kg)	DTI (Eqn. 3)	DTI (Eqn. 4)	HF [28]
Acephate	18.52	1.15×10^{-10}	−0.9025	183.16	135.7	700	2,500	3.33	3.39	
Aminocarb	6.59	2.30×10^{-8}	1.8973	208.26	250	30	275	1.43	1.54	
Azinphosmethyl	2.28	1.17×10^{-8}	2.5315	317.32	270.4	7	88	2.70	2.86	1.54
Bendiocarb	0.72	2.69×10^{-9}	2.5515	223.23	231.7	40	566	1.76	1.99	
Carbaryl	30.10	1.28×10^{-7}	2.3484	201.23	218.7	230	4,000	1.60	1.88	0.29
Carbofuran	0.21	6.66×10^{-8}	2.3007	221.26	240.8	5	120	1.54	1.84	5.11
Chlorpyrifos	3.76	1.03×10^{-4}	4.6582	350.59	298.8	82	202	2.77	2.64	0.48
Cyanophos	0.83	1.68×10^{-5}	2.48	243.22	206.4	215	800	2.71	2.73	
Demeton-*S*-methyl	7.24	1.05×10^{-7}	1.1104	230.28	223.7	30	85	2.50	2.43	
Diazinon	0.59	3.57×10^{-6}	3.8637	304.35	320.2	66	180	1.83	1.66	0.77
Dicrotophos	0.42	4.89×10^{-11}	−1.0957	237.19	255.2	13	42	2.45	2.32	
Dimethoate	5.78	8.63×10^{-10}	0.2781	229.25	205.6	60	353	2.75	2.82	6.55
Disulfoton	0.81	8.57×10^{-5}	3.8578	274.39	282.1	2.6	6	1.95	1.80	
Ethyl parathion	0.40	1.21×10^{-5}	3.7309	291.26	251.9	10	8	2.88	2.59	4.31
Fenamiphos (Nemacur)	0.43	3.95×10^{-9}	3.2932	303.36	315.8	8	80	1.73	1.79	
Fenitrothion	3.37	7.59×10^{-6}	3.296	277.23	229.7	250	1,002	2.76	2.78	0.43
Fenthion	0.87	5.60×10^{-5}	4.0791	278.32	264.6	180	330	2.28	2.12	2.73
Isofenphos	0.44	6.45×10^{-7}	4.6542	345.4	367.6	28	188	1.49	1.43	
Malathion	139.10	3.43×10^{-8}	2.2878	330.35	319.1	290	4,444	2.18	2.30	0.30
Methamidophos	1.70	3.43×10^{-8}	−0.9262	141.13	140.2	7.5	50	2.49	2.64	
Methiocarb	1.06	4.66×10^{-8}	2.8678	225.31	261.4	20	350	1.27	1.49	1.47
Methomyl	8.46	8.27×10^{-8}	0.6095	162.21	179.9	17	1,600	1.52	2.12	
Methyl parathion	2.13	6.87×10^{-6}	2.7487	263.21	207.5	6.01	67	2.77	3.00	2.45
Mevinphos	0.70	1.59×10^{-7}	−0.2376	224.15	221.1	3	4.2	2.78	2.55	
Mexacarbate	1.39	2.54×10^{-8}	2.4446	222.29	224.1	14	1,500	1.51	2.11	
Monocrotophos	0.42	2.23×10^{-11}	−1.3069	223.17	233	8	112	2.33	2.49	4.31
Oxamyl	0.78	1.44×10^{-9}	−1.1991	219.26	212.4	2.5	300	2.16	2.75	
Phosalone	106.27	1.61×10^{-5}	4.2924	367.8	328.6	120	1,530	2.36	2.48	
Phosphamidon	1.08	6.20×10^{-11}	0.3788	299.69	283.5	8	125	2.51	2.66	
Phoxim	1.71	3.02×10^{-3}	4.39	298.3	304.1	300	1,000	1.86	1.75	7.69
Pirimicarb	6.78	1.07×10^{-7}	1.3994	238.29	264	100	500	1.88	1.85	
Propoxur	1.31	1.37×10^{-7}	1.9034	209.25	244.7	41	800	1.39	1.64	0.72
Sulprofos	6.85	6.52×10^{-5}	5.6483	322.44	325.5	65	2,500	1.29	1.62	
Triazophos	1.68	3.17×10^{-9}	2.9226	313.31	306.8	57	1,100	1.96	2.15	
Trichlorfon	13.36	1.08×10^{-10}	−0.277	257.44	194.9	560	2,000	3.52	3.52	

[a] HD = hazardous dose; HLC = Henry's law constant; MW = molecular weight; MV = molecular volume; LD50 = median lethal dose; DTI = dermal toxicity index; HF = hazard factor.

posed by DeWitt [15]. In analogous fashion, toxic potential (TP) of a pesticide application is defined here as

$$\text{TP} = \log \text{HD5 equivalents/kg of avian body weight /m}^2 \text{ of treated area} \quad (1)$$

Adjustment of the TP for the actual body weight of birds found affected in the field studies was considered but was rejected because of the inability to adjust TP for unaffected birds, that is, in the case of studies with impact scores of 1 or even unaffected species in studies where impacts were detected.

Variable to estimate the relative importance of dermal exposure. Although dermal exposure currently is not considered in routine risk assessment, it is known to be important and possibly to exceed other routes of exposure under the right conditions [16,17]. Unlike current mammalian risk assessment procedures, no routine testing of pesticide dermal toxicity is conducted in birds. However, Schafer and colleagues [18] studied both the oral and dermal toxicity of several cholinesterase-inhibiting insecticides in the house sparrow (*Passer domesticus*) and red-billed quelea (*Quelea quelea*). Dermal toxicity was measured by placing graded concentrations of pesticides on the sparsely feathered skin overlying the pectoralis muscle of immobilized birds. Hudson and colleagues [19] compared the acute toxicity of pesticides given orally to ducks to that of a solution of the same pesticide placed on the foot and subsequently covered for a 24-h period. Hudson and colleagues [19] also introduced the concept of a dermal toxicity index (DTI), a ratio of the oral to the dermal toxicity of a pesticide to the same species. Here, the DTI is defined as

$$\text{DTI} = \log(\text{oral LD50/dermal LD50} \cdot 1{,}000) \quad (2)$$

Therefore, the higher the dermal toxicity (the lower the dermal LD50) relative to the oral, the higher the DTI. Hudson and colleagues [19] showed that DTIs of the pesticides varied greatly, suggesting that the relative importance of dermal exposure was likely to vary among pesticides (a finding well known from the mammalian literature). They also showed that the mallard (*Anas platyrhynchos*) DTI could be predicted from a simple log–log regression of the more readily available DTIs measured in the shaved rat. Unfortunately, when avian DTIs were regressed against rat DTIs for the same sample of pesticides, a statistically significant regression was not obtained. Given that mammalian dermal toxicity models typically use a number of physicochemical properties to predict the ability of pesticides and other chemicals to penetrate intact skin, the possibility of predicting avian DTIs from such parameters as well as from rat DTIs was investigated. The more commonly used variables include octanol–water partition coefficient (log K_{OW}), as well as the molecular weight (MW) and molecular

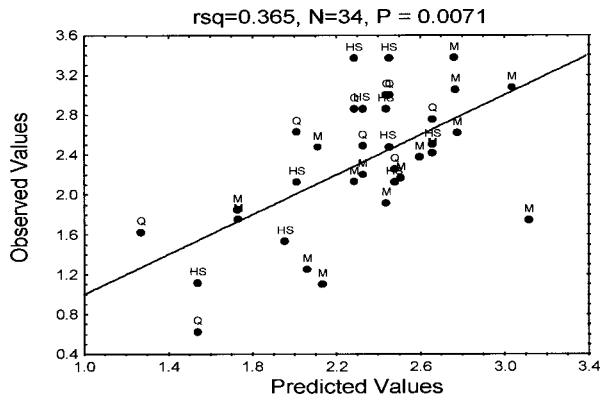

Fig. 1. Plot of predicted versus actual dermal toxicity indices (DTIs) in birds calculated from a step-wise linear regression model that incorporates rat DTI, K_{OW}, molecular weight, and molecular volume (Eqn. 3). The label M refers to DTI values calculated from dermal median lethal dose (LD50) values obtained from the mallard foot [19]. Labels Q and HS refer to DTI values calculated from dermal LD50 values obtained from the breast area of red-billed quelea and house sparrows [18].

volume (MV) of the substance. Henry's law constants also were used to account for the loss of pesticide applied to the skin. Octanol–water partition coefficients were calculated with the KowWin program of the SRC [20] to avoid any potential biases associated with the various methods of measurement. Molecular volumes were estimated by the LeBas method, based on atomic volumes with adjustment for ring formations. The full method was described by Reid and colleagues [21]; however, the volumes and rules summarized by MacKay and colleagues [22] were used here. Rat oral and dermal LD50s were obtained from standard data sources such as *The Pesticide Manual* [23]. Henry's law constants were obtained with Henry software of the SRC [24]. Stepwise multiple regression was then used to develop an empirical model that would predict avian DTIs from those physicochemical constants as well as from the rat DTI. Note that the data of both Hudson and colleagues [19] and Schafer and colleagues [18] were combined, even though their methods differed considerably. However, inspection of Figure 1 does not reveal any apparent separation or clumping of these different data sets. The best predictive equations ($R^2 = 0.36$ and 0.29 for Eqn. 3 and Eqn. 4, respectively) with and without the rat data were

$$\text{avian DTI} = 1.298941 - 0.150307(\log K_{OW}) + 0.015736(\text{MW}) - 0.014838(\text{MV}) + 0.418344(\text{rat DTI}) \quad (3)$$

$$\text{avian DTI} = 2.558388 - 0.135791(\log K_{OW}) + 0.016018(\text{MW}) - 0.016404(\text{MV}) \quad (4)$$

For the 35 pesticides represented in the field-study database, Table 3 presents the physicochemical constants, and the rat toxicity data used in the model as well as avian DTIs calculated from Equations 3 and 4. Unfortunately, formulation-specific toxicity data seldom are available for birds. Clearly, the numerous adjuvants, stickers, and emulsifiers that usually comprise about one half of a pesticide formulation could have a major impact on the relative oral to dermal toxicity of a pesticide active ingredient. The calculated avian DTI values were quite similar whether or not rat data were available (Table 3). No correlation was found between DTI and the avian HD5 values for the sample of 35 pesticides that made up the field sample ($r = 0.12$, $p = 0.49$). The independence of those two variables means that they can both be used in a predictive model without concern for cocorrelation.

Variable to estimate the potential for inhalation exposure. Inhalation is another route of exposure not currently factored into risk assessments. Yet, this route also can be significant [17]. As was the case with other routes of exposure, the various formulation components and final makeup of the spray solution are expected to alter the volatility of the pesticide active ingredient, but these could not be factored in this analysis. In addition, the extent to which inhalation is important as a route of exposure likely depends on environmental conditions such as temperature and relative humidity at the time of application. Unfortunately, these were seldom reported in the available sample of field studies. As the simplest available measure of the potential inhalation exposure, Henry's law constants (HLCs) were used as a crude measure of the volatility of pesticides once they are applied to plant surfaces in an aqueous spray solution. The variable HLC was uncorrelated with either the HD5 ($r = -0.06$, $p = 0.74$) or the DTI ($r = -0.09$, $p = 0.61$).

Variable to estimate the potential for active avoidance of pesticide residues. Birds exposed to cholinesterase-inhibiting pesticides are known to reduce their food intake. This may simply be as a result of the physiological distress caused by chemical ingestion (pesticide-induced anorexia [25]) or through a mechanism of conditioned aversion to the contaminated foodstuffs [26,27]. The most readily available measure of the extent to which different pesticides may be avoided by birds is the hazard factor (HF) designed and measured by Schafer and colleagues [28]. This factor is the median repellency of a pesticide to red-winged blackbirds (*Agelaiius phoeniceus*) when applied to rice grains, a favorite food item, divided by the LD50 for the same species. The HF provides a measure of the proportion of a lethal dose the bird is willing or able to ingest under standardized conditions of hunger and motivation. The higher the HF value, the greater the chance that a bird will ingest a lethal dose. These values were available for only a subset of 15 of the 35 pesticides (Table 3). The variable HF did not correlate with HD5 ($r = -0.31$, $p = 0.26$) or DTI ($r = 0.08$, $p = 0.772$), but it did correlate significantly with HLC ($r = 0.57$, $p = 0.026$), largely as a result of a single data point, the pesticide phoxim, which had both high HF and HLC values.

RESULTS AND DISCUSSION

Presentation of model results

The predictive logistic models developed here all take the following form (assuming here that all of the independent variables are significant):

$$\ln\frac{p}{1-p} = a + b(\text{TP}) + c(\text{DTI}) + d(\text{HLC}) + e(\text{HF}) \quad (5)$$

where p is the probability that a given pesticide application will give rise to bird mortality (scores of 3 and 4 combined) and a to e are model constants. Collapsing the data into two outcome classes (scores of 1 or 2 vs scores of 3 or 4) has the advantage of maximizing the sample size for each outcome as well as allowing graphic depiction of the model. Provided an acceptable model fit was obtained, the probability of bird mor-

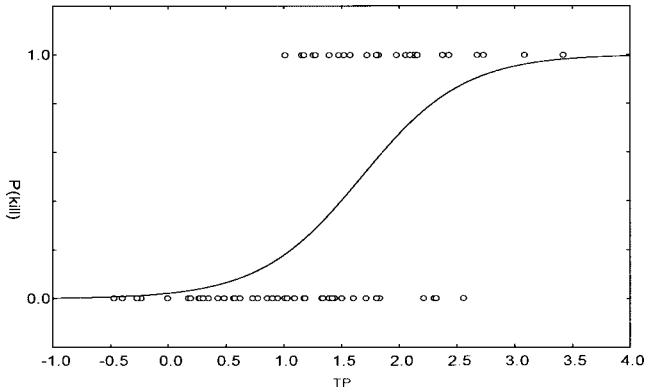

Fig. 2. Logistic regression model for the field crop and pasture data cluster with two outcomes (kills and no kills) and toxic potential (TP; Eqn. 1) as the only independent variable.

tality p could be calculated by solving Equation 5 as follows:

$$p = \frac{e^{a+b(\mathrm{TP})+c(\mathrm{DTI})+d(\mathrm{HLC})+e(\mathrm{HF})}}{1 + e^{a+b(\mathrm{TP})+c(\mathrm{DTI})+d(\mathrm{HLC})+e(\mathrm{HF})}} \quad (6)$$

For example, a plot of p against the single variable TP for the field crop and pasture data cluster is shown in Figure 2. Plotting the data in this fashion draws attention to four outliers, studies with apparently no bird mortality despite TP values of 2 or more (>100 HD5 equivalents/m^2) . Closer inspection revealed that all of these points were associated with the carbamate pesticide methiocarb. Methiocarb was used as a bird repellent rather than as an insecticide. For the product to be efficacious, birds need to be sublethally intoxicated, after which they develop a conditioned aversion for the treated crop. In some cases, birds exhibited temporary paralysis. Because symptom onset is very rapid and recovery (through hydrolysis of bound cholinesterase) also is very fast, avian mortality is thought to be relatively low even at extremely high application rates [29]. Clearly, other products of equivalent toxicity would most certainly have caused extensive mortality at those high rates of application. Nevertheless, from Figure 2, as a rule of thumb, a TP of 1.5 or higher (~30 HD5 equivalents/m^2) should result in some avian mortality occurring on one half of treated fields.

Summary parameters for all significant models as well as their overall goodness of fit (chi-square and proportions of correct data classification) are provided in Table 4. Independent variables were inserted in a stepwise fashion based on their explanatory power. Models were not developed for those data clusters that included incidents, although the data are presented in Figure 3a to c and are discussed below. The variable TP invariably was the most significant independent variable for the field crop–pasture, forest, and orchard data clusters. For all three clusters, addition of the variable DTI resulted in significant model improvement. The importance of DTI was very evident in the forest cluster, where this variable was significant even when entered alone without TP (data not shown).

The HLC contributed significantly (although only marginally compared to the other independent variables) to the models elaborated from the field–pasture cluster and just missed significance in the forest cluster. However, HLC as a single factor produced a marginally significant model in the forest cluster (data not shown). Exploratory analyses showed that the data were highly skewed with a few high values (e.g., phoxim studies) and any attempts to normalize the variable (e.g., through a log transformation) rendered the variable insignificant. This suggests a strong threshold effect, with inhalation exposure being important only in the case of extremely volatile chemicals. However, the HLC was still a significant explanatory variable when the most extreme values (phoxim studies) were omitted. For most chemicals (all but phoxim), the net effect of incorporating HLC in the models was to reduce the probability of mortality associated with any given TP.

Finally, avoidance as measured by Schafer's HF was not helpful in improving model prediction. The explanatory power of the variable HF was lower than that of the other three independent variables although, as noted above, HF correlated with HLC. Realistically, only field–pasture and forest data points provided a sufficient sample to test this variable because it was not available for all pesticides represented in the field studies. The failure of HF suggests that avoidance may not be very important for liquid applications under actual field conditions, although, as described earlier, the carbamate methiocarb is thought to function through conditioned aversion. Avoidance certainly is thought to operate under conditions where birds can recognize the source of the intoxication, for example, treated seed [27]. An alternative explanation for the lack of explanatory power of avoidance is that the pesticides examined here may all be sufficiently avoided (all were organophosphorous or carbamate chemicals) so that an effective avoidance gradient was not available to model.

These results constitute the first clear demonstration that dermal toxicity in particular, and possibly inhalation exposure

Table 4. Regression parameters for the two outcome (kills and no kills) logistic models with either one, two, or three independent variables inserted in stepwise fashion based on significance level. Only those significant models or models showing a significant improvement over less complex ones are shown here[a]

	Constant a	b(TP)	c(DTI)	d(HLC)	Model chi-square	p value for chi-square	% Correct classification: observed no kills	% Correct classification: observed kills	p for chi-square comparison with previous model
	−3.7917	2.2628	—	—	40.70	<0.0000001	82.4	70.3	—
Field crops and pasture	−10.9301	3.5287	2.2645	—	54.20	<0.0000001	82.4	70.3	0.00024
	−14.9667	4.5765	3.1677	1395.1	61.45	<0.0000001	84.3	78.4	0.0071
	−4.0073	3.7224	—	—	17.01	0.00004	88.4	66.7	—
	−10.960	5.0943	2.1971	—	25.50	0.0000029	96.2	73.3	0.0036
Forests	−12.641	5.6924	2.3053	255 179	29.21	0.0000020	84.6	73.3	0.054
	−12.193	5.1785	—	—	12.00	0.00054	100	85.7	—
Orchards	−74.783	25.028	10.092	—	17.57	0.00015	100	85.7	0.018

[a] a–d = model constants; TP = toxic potential; DTI = dermal toxicity index; HLC = Henry's law constant.

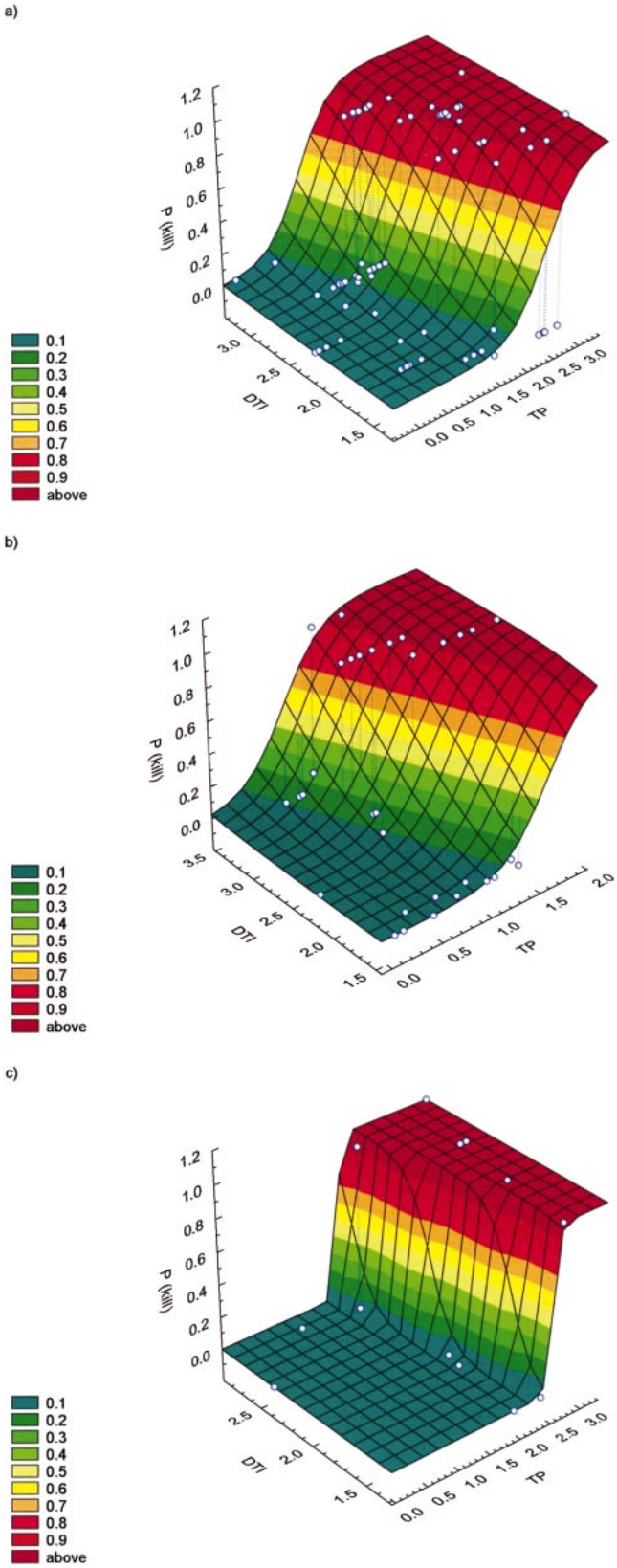

Fig. 3. Three-dimensional representation of logistic regression models for (**a**) field crops and pasture, (**b**) forest, and (**c**) orchard data clusters with two outcomes (kills and no kills) and the two best independent variables, toxic potential (TP) and the calculated avian dermal toxicity index (DTI) from Equation 3.

also, need to be factored into pesticide avian risk assessments. They appear to have a significant effect on field outcomes. Figure 3a to c show the plots of the probability of kill *p* against TP and DTI to provide a visual representation of the importance of DTI. An examination of the probability planes shows clearly that, given two pesticides of equivalent oral toxicity and application rate, the one with the higher DTI will lead to a higher probability of kill. For example, if one was intent on keeping the probability of an avian kill occurring below an arbitrarily chosen 0.10 (1 in 10 field applications) for treatment of a field crop or pasture (Fig. 3a), the application rate of a pesticide with a DTI of 3 would have to be limited to about 10 HD5 equivalents/m^2. On the other hand, for a pesticide with low relative dermal toxicity (DTI of 1.5), the model suggests that the application rate could be increased by approximately 10-fold without increasing the frequency of mortality incidents. The very low DTI of the pesticide methiocarb appears to be yet another reason why studies have failed to document much mortality even at very high application rates. The degree of torsion in the plane of the probability of kills *p* for the forest cluster model (Fig. 3b) suggests that dermal exposure is even more important there than in field crop or pasture applications. Because of the smaller droplet spectrum and higher delivery of the spray in forest insect control operations, it is logical for dermal (and also inhalation) uptake to be more important than in the course of equivalent field crop applications. Another indication that this is the case is the fact that the variables DTI and HLC on their own were significant in the forest but not the field crop cluster. By comparison, dermal toxicity appears to be less important in explaining the outcome of orchard applications (Fig. 3c), although the data are very limited.

Also, for equivalent numbers of HD5 equivalents/m^2, kills apparently are more likely in forest than in field crops and pastures. This could be a result of more birds of more species potentially being exposed in the course of forest spraying. However, another possible reason is that the average forest bird tends to be smaller, so that the actual number of HD5 equivalents (not standardized for a 1-kg bird as done here) would be higher in the forest environment. Conversely, higher TPs appear to be better tolerated by the birds present in orchards, although this needs to be confirmed with more data. In part, this may be because of the overrepresentation of methiocarb data points in the sample. However, other reasons may exist. Because of repeat spraying being normal in orchards, foraging opportunities and, hence, exposure, may be more limited for birds, even those nesting within the orchards proper.

Because data for the other three clusters (mosquito spraying, direct access to spray solutions, and grazing herbivores) were derived in part or totally from incidents rather than field studies, the fitting of these data to logistic models would be improper. The data are simply plotted against TP in Figure 4a to c. Incidents that came to light in the course of mosquito fogging operations (Fig. 4a) or when birds were found to be drinking spray solutions, either from puddles or leaf whorls (Fig. 4b) suggest that, under some exposure conditions, kills can occur at a much lower number of HD5 equivalents/m^2 than the field or forest models would suggest. Mosquito fogging may give rise to particularly high dermal and inhalation exposures because of the very small droplet spectrum generated. The kills seen when birds had access to spray solutions may be indicative of situations that prevail in relatively arid environments, where birds are attracted to fields as a source of water. Finally, grazing herbivores are well acknowledged to

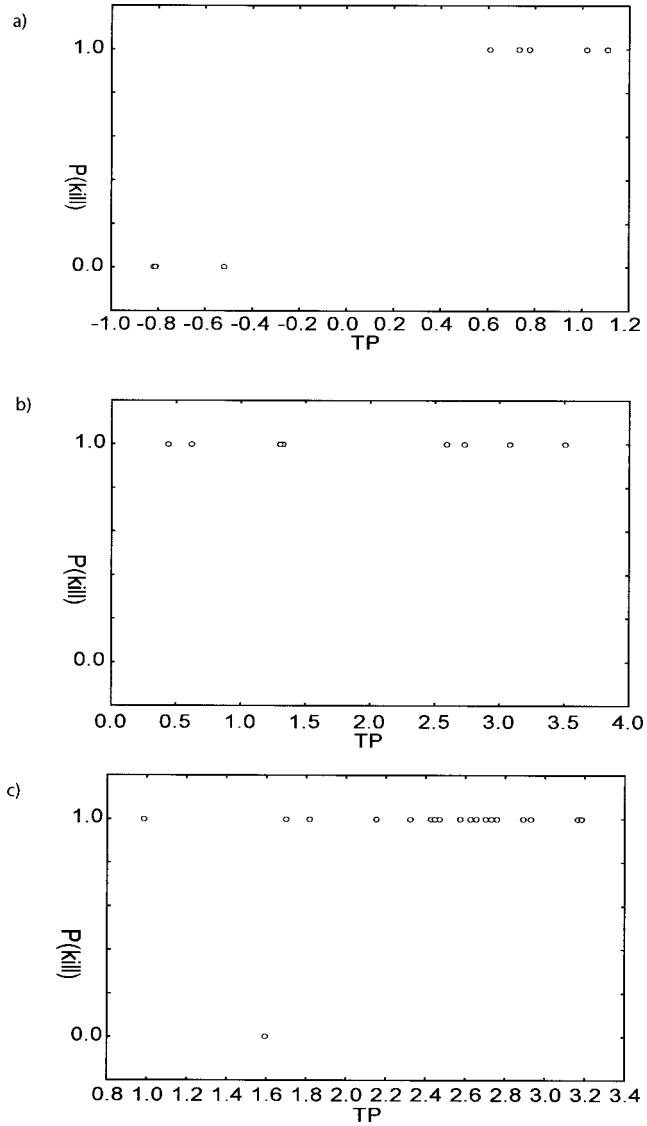

Fig. 4. Plot of data available for (**a**) mosquito spraying, (**b**) drinking of spray solutions, and (**c**) grazing herbivore data clusters. Data represent study determinations of kills or incidents plotted against the variable toxic potential (TP).

be at high risk of pesticide poisoning because of their high food consumption and, hence, exposure relative to that of insectivorous or granivorous birds frequenting the same crops. Unfortunately, too few data were available (Fig. 4c) to test this rigorously. Because relatively high rates of pesticides commonly are applied to turf, many of the incidents involving grazers occurred at TP values similar to those that cause mortality in crop field insectivores or granivores.

Calculation of application rates for given probabilities of kill

One advantage of simple two outcome logistic models is that the equations can be rearranged easily to solve for an unknown, namely the application rate of any given pesticide that will result in conditions conducive to a bird kill in a defined proportion of applications. For example, to determine the application rate that has a probability of killing birds 10% of the time in the course of a field crop–pasture application (model parameters from Table 4), the equation can be rearranged as follows:

$$\mathrm{TP} = \frac{14.9677 - 3.1677(\mathrm{DTI}) - 1395.1(\mathrm{HLC}) + \ln\frac{p}{1-p}}{4.5765} \quad (7)$$

Fixing the probability of kill p at 0.1 and given that TP = log HD5 equivalents/kg/m^2, rearranging the equation gives

$$\text{HD5 equivalents (mg/kg/m}^2) = 10^{[14.9667-3.1677(\mathrm{DPI})-1395.1(\mathrm{HLC})-2.1972]/4.5765} \quad (8)$$

The application rate (in g a.i./ha) predicted to cause avian mortality 10% of the time is then calculated as follows:

$$\text{application rate (g a.i./ha)} = \text{HD5 equivalents (mg/kg/m}^2)\cdot\text{HD5 (mg/kg)}\cdot 10 \quad (9)$$

As an illustration, solutions for 10 and 50% probability of kills for the significant models developed for the field crop–pasture cluster are provided in Table 5. It is important to stress that a 10% (or 50%) probability of kill will only be realized where birds are present at the site (one of the conditions for the field data point to have been accepted as valid). A pesticide bird kill cannot occur where no birds are present. However, very few situations exist in forestry or agriculture where birds and other nontarget species are not present and potentially exposed to a spray application of a pesticide. The choice of 10% as the lower probability of mortality for which application rates were calculated is completely arbitrary. Allowing bird mortality on 10% of treated fields may not be acceptable or defensible when uncertainty exists about which species are likely to be killed, whether local populations can sustain that level of damage, and whether the species in question are beneficial to agriculture [30]. However, because of the shape of the logistic equation, the smallest uncertainty will be associated with the determination of parameters associated with kill probabilities of 50%.

Table 5 shows how calculated rates of application change with increasing model complexity. The inclusion of the parameters DTI and HLC clearly increase the apparent margin of safety associated with pesticides such as carbofuran, methiocarb, and mexacarbate, while having minimal influence on pesticides such as fenitrothion and ethyl and methyl parathion. This explains why the impact of some pesticides (e.g., fenitrothion) has always been considerably higher than predicted on the basis of oral toxicity only [31]. For example, based on the lower application rate of 485 g a.i./ha used for locust control [32], the probability of a bird kill is calculated to be just under 30% (from Eqn. 6 with variables TP, DTI, and HLC). If, on the other hand, fenitrothion had a lower DTI, say equal to that of methiocarb, the application rate could be increased theoretically to a little over 5 kg a.i./ha while maintaining the same probability of a bird kill. Models based solely on the variable TP (HD5 and rate of application) provide the closest indication of how these pesticides would be assessed following current risk assessment procedures, which ignore all but the oral route of exposure. Unfortunately, current risk assessment schemes also rely on a single, arbitrarily chosen LD50 value rather than a more defensible HD5 based on likely species sensitivity distributions. When rates of application calculated in Table 5 are compared to label rates (many of which are represented in the studies summarized on the *Environmental Toxicology and Chemistry* web site [www.etc.allenpress.com]), regardless of the model chosen, several pesticides present a

Table 5. Solving for application rates and arbitrarily chosen probabilities of avian mortality based on dichotomous models of increasing complexity applied to the field crop–pasture data cluster[a]

	Application rates expected to give rise to avian mortality in 1 out of every 10 field crops or pastures where it is applied (10% probability of a kill)			Application rate expected to give rise to avian mortality in 1 out of every 2 field crops or pastures where it is applied (50% probability of a kill)		
	TP only	TP and DTI	TP, DTI, and HLC	TP only	TP and DTI	TP, DTI, and HLC
Acephate	938	405	568	8,776	1,697	1,717
Aminocarb	334	2,362	4,134	3,123	9,907	12,487
Azinphos-methyl	115	127	192	1,080	532	579
Bendiocarb	36	158	267	341	665	806
Carbaryl	1,525	8,396	14,407	14,264	35,216	43,518
Carbofuran	11	64	111	100	270	336
Chlorpyrifos	190	186	259	1,782	781	783
Cyanophos	42	45	68	393	190	204
Demeton-*S*-methyl	367	535	827	3,431	2,245	2,499
Diazinon	30	118	197	280	495	594
Dicrotophos	21	33	52	199	140	157
Dimethoate	293	298	447	2,739	1,248	1,351
Disulfoton	41	135	209	384	564	631
Ethyl parathion	20	17	25	190	71	75
Fenamiphos	22	100	169	204	418	509
Fenitrothion	171	170	254	1,597	713	766
Fenthion	44	89	135	412	372	409
Isofenphos	22	145	253	209	609	763
Malathion	7,047	16,601	26,650	65,917	69,633	80,501
Methamidophos	86	128	198	806	538	599
Methiocarb	54	484	864	502	2,031	2,609
Methomyl	429	2,671	4,628	4,009	11,203	13,979
Methyl parathion	108	107	159	1,009	447	481
Mevinphos	35	35	52	332	145	156
Mexacarbate	70	445	772	659	1,867	2,333
Monocrotophos	21	40	64	199	169	192
Oxamyl	40	95	153	370	399	462
Phosalone	5,383	9,713	15,097	50,359	40,739	45,603
Phosphamidon	55	79	122	512	332	370
Phoxim	87	328	66	810	1,375	198
Pirimicarb	343	1,250	2,076	3,213	5,243	6,272
Propoxur	66	501	881	621	2,102	2,662
Sulprofos	347	3,054	5,193	3,246	12,808	15,688
Triazophos	85	279	459	796	1,169	1,387
Trichlorfon	677	218	300	6,331	916	905

[a] TP = toxic potential; DTI = dermal toxicity index; HLC = Henry's law constant.

clear risk to birds and are expected to be causing some mortality on a frequent and routine basis.

CONCLUSION

Clearly, the toxicity of a pesticide to birds (if measured adequately), as well as its rate of application, go a long way in predicting whether or not mortality is likely to occur after a spray. However, other pesticide-specific factors, such as the relative dermal toxicity and, possibly, volatility of a product clearly also are critical. The analysis presented here illustrates the value of comparative assessments. On a chemical by chemical basis, one would need to generate a vast number of expensive field studies to estimate a 10% probability of mortality as modeled here.

The caveat that the models computed here have not been tested against an independent sample of points is reiterated. Because of the makeup of the sample, model performance in the case of cholinesterase-inhibiting pesticides is likely to be better than for a sample of pesticides with different modes of action. Yet, because of the simplicity and general nature of the models developed, no compelling reason exists to think that they cannot be applied to new chemicals that have not yet had the benefit of field testing. However, the calculation of the avian DTI, in particular, should be verified for new pesticides. In fact, the results presented here suggest that the most gains in pesticide risk assessments would be made by placing more emphasis on dermal toxicity, as well as adopting testing strategies that provide a reliable estimate of the HD5.

If and when new pesticides are taken to the field to look for evidence of direct effects in birds, the task may prove difficult. Because organophosphorous and carbamate insecticides can kill birds relatively quickly, chances of finding carcasses, however poor, are undoubtedly better than for many of the other types of chemicals now coming on to the market.

Finally, that these models only address acute lethal effects resulting from pesticide exposure must be reiterated. They say nothing about reproduction effects, indirect effects, or even delayed mortality. Nevertheless, they finally will allow computation of the direct losses of birds to pesticide use. These calculations are currently underway.

Acknowledgement—Technical support was provided by Jason Duffe, Angela Clarke, and Allison McLaughlin. Brian Collins provided valuable statistical advice and support. Sheryl Tittlemier helped with physicochemical constant calculations. Support for this work was provided by the Canadian Wildlife Service of Environment Canada, as well as by Charles Benbrook and the Consumers Union with a grant from the W. Alton Jones Foundation. Rick Bennett and Doug Urban were instrumental in arranging access to field studies under U.S. Freedom

of Information provisions. Mark Fletcher and Gerhard Joermann provided supplemental information on incidents and U.S. Government Archives helped with the tracking down of old reports. Special thanks to Lowell McEwen, who authored many of these unpublished reports.

REFERENCES

1. Fite EC, Turner LW, Cook NJ, Stunkard C. 1988. Hazard evaluation division, standard evaluation procedure: Guidance document for conducting terrestrial field studies. EPA/540/09-88/109. Washington, DC.
2. Stone WB, Gradoni PB. 1985. Wildlife mortality related to the use of the pesticide diazinon. Wildlife Resources Centre–New York Department of Environmental Conservation, Delmar, New York, NY, USA.
3. Hoerger F, Kenaga EE. 1972. Pesticide residues on plants: Correlation of representative data as a basis for estimation of their magnitude in the environment. In Coulston F, Korte F, eds, *Environmental Quality and Safety* Vol 1—Global Aspects of Chemistry, Toxicology, and Technology as applied to the Environment. Academic, New York, NY, USA, pp 9–29.
4. Rosene WJ, Lay DW. 1963. Disappearance and visibility of quail remains. *J Wildl Manage* 27:139–142.
5. Mineau P, Collins BT. 1988. Avian mortality in agro-ecosystems. 2. Methods of detection. In Greaves MP, Smith BD, Greig-Smith PW, eds, *Field Methods for the Study of Environmental Effects of Pesticides.* British Crop Protection Council, Croydon, UK, pp 13–27.
6. Hommes VM, Büchs W, Joermann G, Siebers J. 1990. Poisoning risk of birds by residues of pesticides in leaf puddles of cole crops (translation from German). *Nachrichtenbl Deutsch Pflanzenschutzdienst* 42:113–117.
7. Mineau P. 1993. The hazard of carbofuran to birds and other vertebrate wildlife. Canadian Wildlife Service Technical Report Series 177. Canadian Wildlife Service, Environment Canada, Ottawa, ON.
8. Statsoft. 2000. *Statistica* (Ver 5.5). Tulsa, OK, USA.
9. Mineau P, Jobin B, Baril A. 1994. A critique of the avian 5-day dietary test (LC50) as the basis of avian risk assessment. Canadian Wildlife Service Technical Report Series 215. Canadian Wildlife Service, Environment Canada, Ottawa, ON.
10. Aldenberg T, Slob W. 1993. Confidence limits for hazardous concentrations based on logistically distributed NOEC toxicity data. *Ecotoxicol Environ Saf* 25:48–63.
11. Mineau P, Collins BT, Baril A. 1996. On the use of scaling factors to improve interspecies extrapolation of acute toxicity in birds. *Regul Toxicol Pharmacol* 24:24–29.
12. Mineau P, Baril A, Collins BT, Duffe JA, Joermann G, Luttik R. 2001. Pesticide acute toxicity reference values for birds. *Rev Environ Contam Toxicol* 170:13–74.
13. Urban DJ, Cook NJ. 1986. Ecological risk assessment. EPA 540/9-85-00l. Washington, DC.
14. Crocker J, Hart ADM, Thompson H. 2001. Estimating avian exposure to pesticides. *Proceedings,* European Workshop on Probablistic Risk Assessment for the Environmental Effects of Plant Protection Products, Leeuwenhorst, The Netherlands, June 5–8, 2001, p 28.
15. DeWitt JB. 1966. Methodology for determining toxicity of pesticides to wild vertebrates. *J Appl Ecol* 3:275–278.
16. Mineau P, Sundaram KMS, Sundaram A, Feng C, Busby DG, Pearce PA. 1990. An improved method to study the impact of pesticide sprays on small song birds. *J Environ Sci Health B* 25: 105–135.
17. Driver CJ, Ligotke MW, Van Voris P, McVeety BD, Greenspan BJ, Drown DB. 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.
18. Schafer EW Jr, Brunton RB, Lockyer NF, DeGrazio JW. 1973. Comparative toxicity of seventeen pesticides to the quelea, house sparrow and red-winged blackbird. *Toxicol Appl Pharmacol* 26: 154–157.
19. Hudson RH, Haegele MA, Tucker RK. 1979. Acute oral and percutaneous toxicity of pesticides to mallards: Correlations with mammalian toxicity data. *Toxicol Appl Pharmacol* 47:451–460.
20. Meylan WM, Howard PH. 1995. Atom/fragment contribution method for estimating octanol–water partition coefficients. *J Pharm Sci* 84:83–92.
21. Reid RC, Prausnitz JM, Polling BE. 1987. *The Properties of Gases and Liquids.* McGraw-Hill, New York, NY, USA.
22. Mackay D, Shiu WY, Ma KC. 1992. *Illustrated Handbook of Physical–Chemical Properties and Environmental Fate for Organic Chemicals.* Lewis, Boca Raton, FL, USA.
23. Tomlin CDS. 1997. *The Pesticide Manual,* 11th ed. British Crop Protection Council, Binfield, Berks, UK.
24. Meylan WM, Howard PH. 1991. Bond contribution method for estimating Henry's law constants. *Environ Toxicol Chem* 10: 1283–1293.
25. Grue CE. 1982. Response of common grackles to dietary concentrations of four organophosphate pesticides. *Arch Environ Contam Toxicol* 11:617–626.
26. Hart ADM. 1993. Relationships between behavior and the inhibition of acetylcholinesterase in birds exposed to organophosphorus pesticides. *Environ Toxicol Chem* 12:321–336.
27. McKay HV, Prosser PJ, Hart ADM, Langton SD, Jones A, McCoy C, Chandler-Morris SA, Pascual JA. 1999. Do wood pigeons avoid pesticide treated cereal seed? *J Appl Ecol* 36:283–296.
28. Schafer EW Jr, Bowles WA Jr, Hurlbut J. 1983. The acute oral toxicity, repellancy and hazard potential of 998 chemicals to one or more species of wild and domestic birds. *Arch Environ Contam Toxicol* 12:355–382.
29. Hardy AR, Westlake GE, Lloyd GA, Brown PM, Greig-Smith PW, Fletcher MR, Tarrant KA, Stanley PI. 1993. An intensive field trial to assess hazards to birds and mammals from the use of methiocarb as a bird repellent on ripening cherries. *Ecotoxicology* 2:1–31.
30. Kirk DA, Evenden MD, Mineau P. 1996. Past and current attempts to evaluate the role of birds as predators of insect pests in temperate agriculture. *Curr Ornithol* 5:175–269.
31. Pauli BD, Holmes SB, Sebastien RJ, Rawn GP. 1993. Fenitrothion risk assessment. Canadian Wildlife Service Technical Report Series 163. Canadian Wildlife Service, Environment Canada, Ottawa, ON.
32. Mullié WC, Keith JO. 1993. The effects of aerially applied fenitrothion and chlorpyrifos on birds in the savannah of northern Senegal. *J Appl Ecol* 30:536–550.