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Simulating the impact of cholinesterase-inhibiting pesticides on non-target wildlife in irrigated crops

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ABSTRACT

We present a simulation model for risk assessment of the impact of insecticide inhibitors of cholinesterase (ChE) applied in irrigated agricultural fields on non-target wildlife. The model, which we developed as a compartment model based on difference equations ($\Delta t = 1$ h), consists of six submodels describing the dynamics of (1) insecticide application, (2) insecticide movement into floodable soil, (3) irrigation and rain, (4) insecticide dissolution in water, (5) foraging and insecticide intake from water, and (6) ChE inhibition and recovery. To demonstrate application of the model, we simulated historical and "worst-case" scenarios of the impact of ChE-inhibiting insecticides on white-winged doves (*Zenaida asiatica*) inhabiting natural brushland adjacent to cotton and sugarcane fields in the Lower Rio Grande Valley of Texas, USA. Only when a rain event occurred just after insecticide application did predicted levels of ChE inhibition surpass the diagnostic level of 20% exposure. The present model should aid in assessing the effect of ChE-inhibiting insecticides on ChE activity of different species that drink contaminated water from irrigated agricultural fields, and in identifying specific situations in which the juxtaposition of environmental conditions and management schemes could result in a high risk to non-target wildlife.

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1. Introduction

Approximately 40% of global food production is supported by irrigated agriculture, which comprises 20% of the world's farmland (FAO, 2003). Compared to rain-fed agricultural areas, irrigated ones support high intensive agriculture which is characterized by an elevated use of agrochemicals such as fertilizers, pesticides, and plant-growth regulators. All these agrochemicals may threaten non-target wildlife; however, pesticides, and especially insecticides, are the most dangerous because they directly affect the survival and reproduction of organisms. Currently, organophosphates (OPs) and carbamates (CAs) are the most commonly used insecticides. For example OPs and CAs represented 54% and 22%, respectively, of all insecticides applied in the USA during 1997 (Gianessi and Silvers, 2000). Although they are assumed to be environmentally safer than organochlorine insecticides due to their short half-lives, they have an elevated toxicity. Several accidental or intentional mortality events attributed to anticholinesterase pesticide poisoning have been reported (Stone et al., 1984; White and Kolbe, 1985; Grue et al., 1991; Flickinger et al., 1991; Smith et al., 1995; Goldstein et al., 1999; Mineau et al., 1999; Wobeser et al., 2004; Fleischli et al., 2004). Animals may incorporate them by ingestion, inhalation, or eye or skin contact. The outcome of exposure to CAs and OPs is the inhibition of acetylcholinesterase (ChE), an enzyme that degrades the

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neurotransmissor acetylcholine. This enzyme is responsible for nervous firing within the peripheral and central nervous system. In addition, CAs and OPs bind to other cholinesterases (e.g. butyrylcholinesterase in liver and plasma) and insecticide detoxifying enzymes. Animals with ChE depression show anorexia, lethargy, and behavioral and physiological disorders (Grue and Shipley, 1981; Grue et al., 1991, 1997; Bishop et al., 2000a,b; Solecki et al., 2001; Burger et al., 2002). All of these may decrease notably their potential for survival and reproduction.

For terrestrial animals dermal exposure and ingestion of insecticide are the principal routes of contamination by OPs and CAs. For instance, frugivorous, granivorous, and insectivorous birds are particularly susceptible because of their capability of moving between and within crops. Most research has been focused on the incorporation of insecticide by intake of contaminated foods, inhalation or skin absorption in nesting adult birds and nestlings during insecticide applications. Less attention has been given to identifying the circumstances under which the intake of insecticide-contaminated drinking water might be dangerous for wildlife: for example, in irrigated areas located within arid and semiarid regions, where flooded fields often are the only source of water for wildlife.

In this paper we present a model that simulates the level of ChE inhibition in animals drinking pesticide-contaminated water from flood-irrigated crop fields. We first present an overview of the entire model and then describe each of the six submodels in detail. Finally, to demonstrate application of the model, we simulate a field study that examined the impact of methyl parathion and Azinphos methyl on white-winged doves (*Zenaida asiatica*) in the Lower Rio Grande Valley of Texas, USA (Custer and Mitchell, 1987), and use the model to search for possible "worst-case" scenarios that might arise from slightly different irrigation and pesticide application schemes.

2. Model description

2.1. *Model overview*

The model simulates an animal that drinks water from agriculturally flooded fields. The amount of insecticide that the animal ingests depends on its water intake rate and concentration of the dissolved insecticide in the water. Insecticide

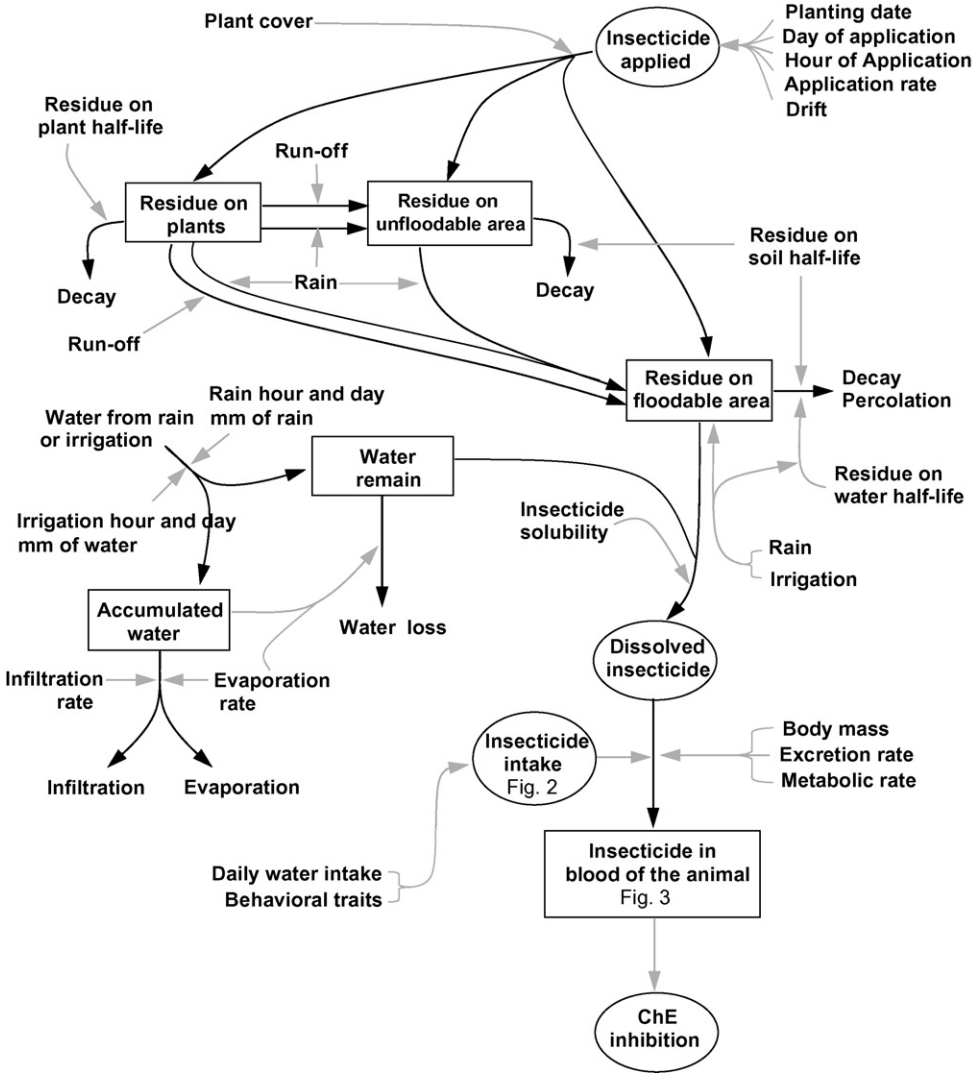

Fig. 1 – General conceptual model. Gray and black arrows represent information and material flows, respectively.

water concentration is a function of the amount of insecticide residue and the volume of water accumulated as a result of either an irrigation or rain event. Insecticide residue is related to the application rate and decay rate of the insecticide. The concentration of insecticide in the body of the animal depends on body mass, amount of insecticide ingested and excretion and metabolic rates of the insecticide. Finally, the model estimates the degree of ChE inhibition as a function of insecticide concentration in the body (Fig. 1).

The model was developed as a compartment model based on difference equations ($\Delta t = 1$ h) and programmed with Stella® VII software (High Performance Systems, Inc., New Hampshire, USA). Conceptually the model is compartmentalized in six submodels: (1) insecticide application, (2) insecticide movement into floodable soil, (3) irrigation and rain, (4) insecticide dissolution in water, (5) foraging and insecticide intake from water, and (6) ChE inhibition and recovery. The structure of the model has been replicated under an array of n insecticides × m crops (see Section 2.2.). It allows up to n applications in each crop at the same time step; n and m are specified by the model user.

2.2. Submodel I—insecticide application

This submodel allows at least one application per hour in each agricultural land use unit (ALU). ALUs may be (1) annual or biannual crops (crop type 0, such as cotton, corn, wheat, sugarcane, sorghum, sunflower); (2) trees, shrubs or vines (crop type 1 = citrus, apples, pears, plums, peaches, pecans, grapevines, etc.); or (3) rangelands. Under the classification of rangelands are considered those areas without pesticide treatments. For each application a certain amount of insecticide is lost from the target ALU due to drift.

Drift is defined as the percentage of insecticide applied that is carried out from the target field crop by wind or another weather variable (Fig. 1).

2.2.1. Quantitative development

Insecticide applied (iap in g of active ingredient $ha^{-1}\,h^{-1}$) can be represented by the equation:

$$iap_t = iar_t \times \left(1 - \frac{d_t}{100}\right) \quad (1)$$

where iar represents the pesticide application rate (g of active ingredient $ha^{-1}\,h^{-1}$) and d represents the percentage of the pesticide that drifts in the air away from the application area.

2.2.2. Input information

The information required for this submodel is the following: (1) planting day, (2) day of application, (3) hour of application, (4) application rate, and (5) drift. For crops of type 0, planting day is entered as the day-of-year when the crop is planted; whereas for crops of type 1, planting day is equal to one. Day of application is entered as number of days after the planting day when the insecticide is applied, and hour of application is entered as a 1–24 h system. The application rate or concentration is entered in grams of insecticide active ingredient per hectare (g a.i. ha^{-1}) (Fig. 1). Drift is entered as a percentage. No pesticides are applied on rangelands; therefore, it is considered an area free of pesticides.

2.3. Submodel II—insecticide into floodable soil

2.3.1. Granulated insecticides

Model predictions are based on the application of liquid insecticides using aerial or ground sprayers. The use of granulated insecticides is not considered in the model. Because granulated insecticides are applied under the ground we assume they will not be dissolved into the free upper water. The rationale is that granulated insecticides would be washed to deep soil profiles and/or they would be adsorbed by the soil organic matter or clay components. However, if the applications are incorrectly performed, some of the granules may remain on the furrow ground surface, and they potentially could be washed off by rain into floodable rows. In this case the model could be run using the amount of free granulated insecticide as an application rate. Also, birds could ingest uncovered granules, which might cause them an acute intoxication (Houseknecht, 1993; Augspurger et al., 1996; Wilson et al., 2002).

2.3.2. Liquid insecticides

A certain amount of the liquid insecticides applied on crops of type = 0 by means of aerial or ground sprayers drops on the plant canopy. The remnant drops directly on the bare soil or is carried out from the crop through drift ((Salyani and Cromwell, 1992; Stover et al., 2002; Siebers et al., 2003). Because plant cover increases throughout the growing season, there is a time when plants start to grow over the floodable rows. From this point, the amount of insecticide that directly reaches this area decreases over time (Himel et al., 1990); yet, runoff from the canopy above the floodable rows begins. Runoff is defined as the insecticide that rolls down from leaves, fruits and branches and falls on the ground; as well as, the insecticide that reaches the ground after crossing through the canopy without being intercepted. The runoff from the portion of the canopy over the non-floodable area is not taken into account in the model. However, if a rain event happens (above 13 mm h^{-1}), the model assumes that insecticide residue accumulated on the non-floodable area plus a portion of the residue on plants will be washed-off to the floodable area (Gunther et al., 1977; McDowell et al., 1984; Willis et al., 1986; Himel et al., 1990; Chen et al., 2003).

Applications on crops of type 1 are commonly carried out with air-carrier ground sprayers, which launch the insecticide directly towards the canopy. Spray droplets generated by nozzles or atomizers are transported by an air flux that is produced by one or more fans. The amount of insecticide that remains on the plant or drops as runoff during the application depends on several factors such as: nozzle arrangement, pesticide type, spray volume, ground speed, canopy size and density, and weather conditions (Salyani and Cromwell, 1992; Cunningham and Harden, 1998; Stover et al., 2002; Salyani, 2004). Small droplets produce better insecticide coverage, but they are prone to drift or evaporation (Salyani and Cromwell, 1992). Also, small droplets cannot penetrate dense canopies or travel too far away because they can be easily deflected by leaves, fruits and branches. On the other hand, larger

droplets can travel long distances; therefore, they penetrate dense canopies; but the probability of these droplets coalescing with other droplets and falling to the ground is greater than small droplets (Cunningham and Harden, 1998; Stover et al., 2002). In the model, the amount of insecticide residue that reaches the floodable area comes almost exclusively from canopy runoff. Unlike crop type = 0, the floodable area in crop type = 1 is relatively greater (commonly 90–95%) compared with the non-floodable area. It is assumed that there is bare soil under the trees or vines.

2.3.3. *Insecticide residue degradation*

While the field is not flooded, we assume the soil is normally not saturated with water; therefore, the insecticide that drops on the ground penetrates no more than 1 mm into the soil. In that way, the residue can be totally dissolved in the irrigation water (Ahuja et al., 1981; Ahuja and Lehman, 1983; Ahuja, 1986). On the other hand, if the soil is saturated, the insecticide might dissolve in the soil water and percolate deeper into the soil profile (Roy et al., 2001).

Insecticides have a first-order degradation curve, $C_t = C_0 \times e^{-kt}$ where C_t is the concentration of the insecticide at time t, C_0 is the insecticide initial concentration, e is the base of the natural logarithm, and k is a rate constant; k is related to the insecticide half-life time by the equation $T_{1/2} = \ln 2/k$. Half-life time ($T_{1/2}$) is the period of time in which the insecticide concentration is reduced to half of the initial concentration (Khan, 1980; Beulke and Brown, 2001; Sakellarides et al., 2003). Based in the above formula, the concentration of insecticide residue in soil is calculated as:

$$C_{t+1} = C_t + A_t - (C_t(1 - e^{-(\ln 2)/T_{1/2}})) \quad (2)$$

where C_t represents concentration of residue in g ha^{-1} remaining at time t, and A is equal to insecticide applied (g ha^{-1}) at time t.

Insecticide half-life depends on several factors such as soil clay component, soil organic matter content, soil microflora and fauna, temperature, time of exposure to sunlight, whether it is dissolved in free water or soil water (Khan, 1980; Hebert and Miller, 1990; Racke, 1992; Suett and Jukes, 1993; Scheunert, 1993; Bhushan et al., 1997; Karpuozas and Walker, 2000; Liu et al., 2000; Rao and Hornsby, 2001; Sakellarides et al., 2003; Sanchez-Martin and Sanchez-Camazano, 2003). The fraction of humic substances within the soil organic matter has strong adsorptive power on organophosphate and carbamate insecticides. For instance for methyl parathion it accounts for 96% of the variance in adsorption, while the remnant variation is due to adsorption to clay soil components (Sanchez-Martin and Sanchez-Camazano, 2003). Because humic substances in the upper few millimeters of ground surface are degraded by photo-oxidation (Hebert and Miller, 1990; USDA, 2001; Sakellarides et al., 2003), and microbial activity in this soil portion is considered unimportant when it is dry (Yaron et al., 1974), we assume that insecticide in this fine layer can be totally dissolved during an irrigation or rain event.

The decay of insecticides starts immediately after their application. Whether they are on the ground, on plants or dissolved in water, the dynamic of degradation is the same; however, their half-lives are different under each condition. Once the water has totally infiltrated into the soil, we assume that the insecticide is carried by mass flow by water through the soil profile (Khan, 1980). Therefore, there is no insecticide that can be re-dissolved in a new irrigation event, except if there has been a new application between two successive irrigations. If the application occurs while the field is flooded, then all the insecticide that drops on the water will be dissolved and will be added to the insecticide, if any, that is already dissolved. The amount of dissolved insecticide cannot be greater than the insecticide solubility in water.

2.3.4. *Quantitative development*

The dynamics of pesticides in the environment are represented by changes in the accumulation of residues on plants (*IRP*), on soil in floodable areas (*IRF*), and on soil in non-floodable areas (*IRN*), all in g of active ingredient/ha (Fig. 1):

$$\mathrm{IRP}_{t+1} = \mathrm{IRP}_t + (\mathrm{ifp} - \mathrm{idp}) \times \Delta t \quad (3)$$

$$\mathrm{IRF}_{t+1} = \mathrm{IRF}_t + (\mathrm{iff} - \mathrm{ids_f}) \times \Delta t \quad \text{if soil is not flooded} \quad (4a)$$

$$\mathrm{IRF}_{t+1} = \mathrm{IRF}_t + (\mathrm{iff} - \mathrm{idw}) \times \Delta t \quad \text{if soil is flooded} \quad (4b)$$

$$\mathrm{IRN}_{t+1} = \mathrm{IRN}_t + (\mathrm{ifn} - \mathrm{ids_n}) \times \Delta t \quad (5)$$

where ifp, iff, and ifn represent the amount of pesticide falling on plants, floodable areas, and non-floodable areas, all in g of active ingredient ha^{-1} h^{-1}, idp, ids and idw are the degradation rates in g h^{-1}of insecticide on plants, soil and water, respectively. Then:

$$\mathrm{ifp} = \mathrm{iap}_t \times \frac{\mathrm{pc}_t}{100} \times \left(1 - \frac{\mathrm{irp}}{100}\right) \quad (\text{see Eq. (1) for } \mathrm{iap}_t) \quad (6)$$

$$\mathrm{iff} = \mathrm{iap}_t \times \frac{\mathrm{fa}}{100} \quad \text{if } \mathrm{pc}_t \le \mathrm{nfa} \quad (7a)$$

$$\mathrm{iff} = \mathrm{iap}_t \times \left(1 - \frac{\mathrm{pc}_t}{100}\right) + \mathrm{iap}_t \times \left(\frac{(\mathrm{pc}_t - \mathrm{nfa})}{100}\right) \times \frac{\mathrm{irp}}{100} \quad \text{if } \mathrm{pc}_t > \mathrm{nfa} \quad (7b)$$

$$\mathrm{ifn} = \mathrm{iap}_t \times \left(\frac{(\mathrm{nfa} - \mathrm{pct})}{100}\right) + \mathrm{iap}_t \times \frac{\mathrm{pct}}{100} \times \frac{\mathrm{irp}}{100} \quad \text{if } \mathrm{pc}_t \le \mathrm{nfa} \quad (8a)$$

$$\mathrm{ifn} = \mathrm{iap}_t \times \frac{\mathrm{nfa}}{100} \times \frac{\mathrm{irp}}{100} \quad \text{if } \mathrm{pc}_t > \mathrm{nfa} \quad (8b)$$

$$\mathrm{nfa} = 100 - \mathrm{fa} \quad (9)$$

$$\mathrm{pc} = f(t) \quad (10)$$

where pc represents percentage of the area of ALU covered by plant canopy; *irp* is the percentage of pesticide that drops from plants to the soil as run-off; fa and nfa are, respectively, the floodable and non-floodable portion percentages of an ALU. The term (1 – (irp/100)) represents the proportion of insecticide that remains on the plants after runoff. Once

plants start to grow above the floodable area, $(1 - pc_t/100)$ represents the proportion of floodable area that is not covered by plants, whereas $(pc_t - nfa)/100$ represents the proportion of plant canopy overlapping the floodable area.Idp, ids and idw can be represented as:

$$idp = IRP_t \times \left(1 - \exp\left(-\left(\frac{LOG\,N(2)}{T_{1/2p}}\right)\right)\right) \quad (11)$$

$$ids_f = IRF_t \times \left(1 - \exp\left(-\left(\frac{LOG\,N(2)}{T_{1/2s}}\right)\right)\right) \quad (12)$$

$$ids_n = IRN_t \times \left(1 - \exp\left(-\left(\frac{LOG\,N(2)}{T_{1/2s}}\right)\right)\right) \quad (13)$$

$$idw = IRF_t \times \left(1 - \exp\left(-\left(\frac{LOG\,N(2)}{T_{1/2w}}\right)\right)\right) \quad (14)$$

where $T_{1/2p}$, $T_{1/2s}$ and $T_{1/2w}$ are the half-lives of insecticide on plants, soil and water; and IRP_t, IRF_t, and IRN_t represent the amount of residues remaining at time t on plants, floodable and non-floodable areas, respectively.

If a rain of 13 mm or over occurs, then:

$$IRF_{t+1} = IRF_t + \left(\frac{IRP_t \times wff}{100}\right) + IRN_t + (iff - idw) \times \Delta t \quad (15)$$

where wff represents the percentage of insecticide that is washed off from plant canopy by rain.

2.3.5. *Input information*

The input information for this submodel is: (1) crop type, 0 or 1; (2) percentage of floodable area; (3) temporal change in the percentage of plant cover; (4) percentage of insecticide applied that drops from the canopy (runoff); (5) half-life (in hours) of the insecticide in soil, dissolved in water, and on plants; and (6) percentage of insecticide accumulated on plants that is washed off by rain.

2.4. *Submodel III—irrigation and rain*

This submodel allows at least one irrigation event per hour in each ALU. Similarly, the submodel allows at least one rain event per hour in each crop.

Once the field is covered by water after an irrigation or rain event, the water starts to disappear due to evaporation and infiltration processes (Fig. 1). Therefore, how fast the water disappears is a function of the amount of water covering the field combined with the evaporation and infiltration rates.

2.4.1. *Quantitative development*

The water accumulated (AW) in the floodable area is represented by the following equation:

$$AW_{t+1} = AW_t + (raw + irw - evw - inw) \times \Delta t \quad (16)$$

where raw and irw are water added by rain and irrigation event; and evw and inw are evaporation and infiltration rates, all in $l\,ha^{-1}\,h^{-1}$.

2.4.2. *Input information*

The input information that the submodel requires is: (1) day of irrigation; (2) hour of irrigation; (3) irrigation rate; (4) day of rain; (5) hour of rain; (6) amount of rain; (7) evaporation rate; and (8) soil infiltration rate. Day of irrigation and day of rain are entered as day-of-year; hour of irrigation and hour of rain are entered based on a 1–24 h system. The irrigation rate and amount of rain are entered as the thickness of a layer of water (mm). The evaporation rate and the infiltration rate are entered as mm per year and millimeters per hour, respectively.

2.5. *Submodel IV—insecticide dissolution in water*

In this submodel it is assumed that the remaining residue in the floodable area is totally dissolved into the irrigation or rain water. The maximum allowed concentration of insecticide is limited by the insecticide solubility. During the time between the irrigation or rain events and water disappearance, two counteracting processes determine the insecticide concentration. Simultaneously, the insecticide concentration increases and decreases due to the evaporation rate and the degradation rate, respectively (Fig. 1).

2.5.1. *Quantitative development*

The concentration of residue in water is represented by IRW in ppm or $\mu g\,g^{-1}$ or $\mu g\,ml^{-1}$.

$$IRW_t = REW_t \times IRF_t \quad \text{if } REW_t \times IRF_t \leq isw \quad (17a)$$

$$IRW_t = isw \quad \text{if } REW_t \times IRF_t > isw \quad (17b)$$

$$REW_{t+1} = REW_t + (raw + irw - evw - inw) \times \Delta t \quad (18a)$$

$$REW_{t+1} = 0 \quad \text{if } AW_t(16) = 0 \quad (18b)$$

$$inw_t(16) = 0 \quad \text{if } IRF_t(16) > 0 \quad (19)$$

REW is equal to AW (16), although here inv (16) equals 0 if IRF (3) >0. Thus, once the residue has been dissolved the concentration is only affected by evw (16), or by raw (16) or irw (16) if more water from rain or irrigation is added. Isw represents the solubility of the insecticide in water measured in ppm or $mg\,l^{-1}$.

2.5.2. *Input information*

The input information that this submodel requires is the insecticide water solubility measured in milligrams per liter, or microgram per gram, or parts per million.

2.6. *Submodel V—animal contamination with insecticide*

Although the model can be used to simulate the level of contamination of individuals of different species inhabiting an environment composed of different ALUs, we will focus on one individual of one bird species. As an example, a hypothetical bird lives in an environment consisting of four ALUs and rangelands.

The places where the bird forages are important because they determine where the animal drinks. Two modes can be

used for simulations: in mode 1, the model user specifies in which ALUs the bird forages, whereas in mode 2, the bird forages according to the bird's foraging rules. According to these rules a bird species spends a proportion of its time foraging in each ALU and range in a particular proportion. For each time step the bird's decision on where to forage is randomly generated, but is constrained by the proportion of time devoted to each ALU with respect to the whole time used for foraging. It is assumed that ALUs are spatially distributed such that there is no effect of distance on foraging choices.

When a crop of type 0 reaches a critical height, the proportion of use of that crop is reduced by a decrement factor (Corson et al., 1998). Then, the proportion of use reduction is divided by the number of ALUs not affected by critical heights and added to each of these ALUs.

It is assumed that if a flooded ALU is chosen, the bird drinks in it. On the other hand, if a non-flooded ALU is chosen, the bird decision on where to drink is determined randomly by a probability distribution generated from the relative amount of water in each ALU with respect to the water of all ALUs pooled. It is also assumed that the previous choice does not affect the choice of the next drinking site.

How much water the bird drinks is a function of the particular daily intake rate of the species and the water intake reduction after drinking contaminated water. After animals consume contaminated water, they may show an aversion to ingest this water, which results in a decrement of daily water intake during the following days (Brust et al., 1971; Provenza, 1995; Small et al., 1998; Burkepile et al., 2002; Mineau, 2002). Water intake reduction is a datum required by the model and represents the percentage of daily water intake reduction as a function of insecticide concentration in the water. The model allows up to two drinking bouts for a bird to satisfy its daily water requirements. It is assumed that the duration of these bouts is equal to, or shorter than 1 h. Starting and ending times of these bouts are data required by the model. The proportion of the daily water intake drunk in the first bout is also a datum required by the model. The bird completes its daily water requirements during the second drinking bout. The bird develops a "pesticide aversion" the first time it drinks contaminated water, and reduces water intake during the following drinking bouts. However, "pesticide aversion" disappears the next time the bird drinks water without pesticide.

Summarizing, the amount of insecticide ingested hourly depends on the particular ALU where the bird drinks, the amount of water that it drinks, and the insecticide concentration in the water (Fig. 2).

2.6.1. *Quantitative development*

Amount of insecticide ingested is represented by IIN in $\mu g\,h^{-1}$.

$$IIN_t = WIN_t \times IRW_t \quad (12) \quad (20)$$

$$WIN_{t+1} = WIN_t + (WIN_1 + WIN_2 - WIR) \times \Delta t \quad (21)$$

where *WIN* corresponds to water intake measured in g or $ml\,h^{-1}$. See equation 12 for IRW. WIN1 and WIN2 in g or $ml\,h^{-1}$ symbolize water intake during period 1 and 2, respectively.

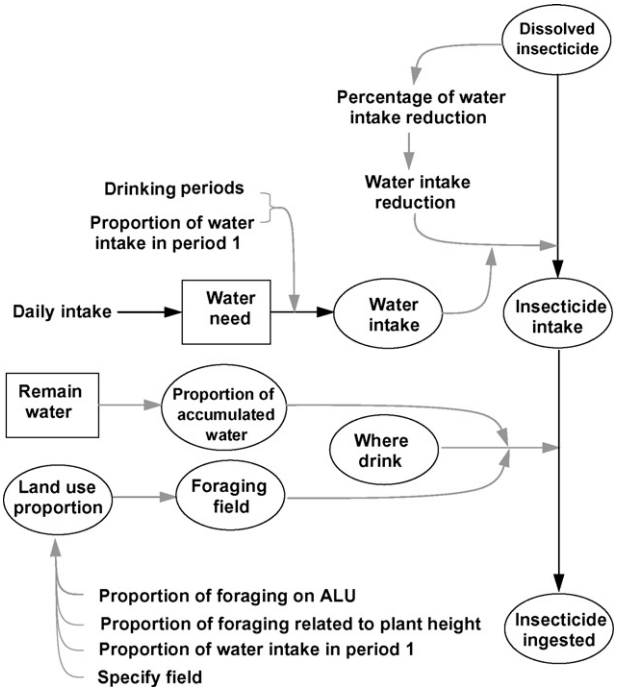

Fig. 2 – Conceptualization of the submodel representing animal contamination with insecticide. Gray and black arrows represent information and mass flows, respectively.

WIR, in g or $ml\,h^{-1}$, represents the reduction of water intake after drinking contaminated water.

2.6.2. *Input information*

The model user has to specify: (1) if the bird will forage in a specific ALU or if it will forage in a random way; (2) the proportion of each ALU the bird will use; (3) time when the crop reaches a critical height; (4) amount of reduction of ALU use proportion once the critical height has been reached; (5) starting time of the first drinking period; (6) ending time of the first drinking period; (7) daily water intake; (8) proportion of the daily water intake drunk in the first drinking period; (9) starting time of the second drinking period; (10) ending time of the second drinking period; and (11) percentage of water intake reduction after drinking contaminated water.

The proportion of each ALU the bird will use is specified as percentage of the total number of ALUs pooled. The time when the critical height has been reached is entered as day-of-year. Starting and ending times of drinking periods are entered based on a 1–24 h system.

2.7. *Submodel VI—cholinesterase inhibition and recovery*

The ChE inhibition in a bird is related to load of the insecticide residue in the animal's bloodstream. Once the bird ingests contaminated water, a portion of the insecticide is liberated intact with feces. The remnant portion is absorbed into the portal bloodstream system and transported to the liver (Fig. 3). A portion of OPs is activated to oxon-form (toxic form of organophosphates) via desulfuration by mono-

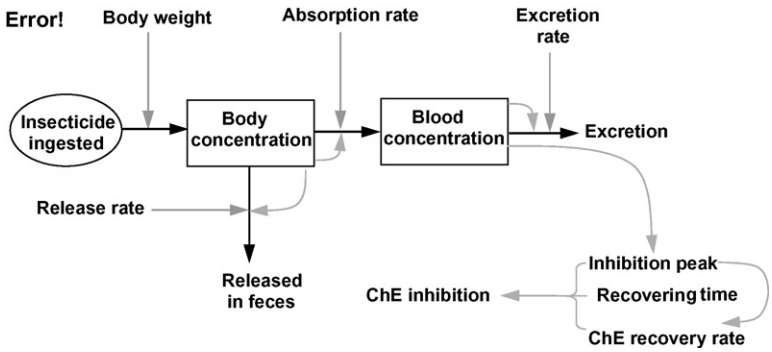

Fig. 3 – Conceptualization of the submodel representing insecticide ingested and ChE inhibition. Gray and black arrows represent information and mass flows, respectively.

oxygenases, P450-dependent or flavin-containing. Part of the oxon form is inactivated or degradated by "B" esterases or "A" esterarses, mono-oxigenases and glutation tranferases, respectively (Sultatos, 1987; Thompson et al., 1991; Parker and Goldstein, 2000). The remaining portions of liver-activated oxon and non-activated OPs are exported to the bloodstream. Here, the same process occurs as in the liver, but only inactivation by "B" esterases is important. Finally, OPs that reach the brain are activated to the oxon form. Depression of ChE activity is the result of the presence of brain-activated oxon and oxon transported by bloodstream.

CBs, on the other hand, are applied in their active form; therefore, they do not need bioactivation. Their ChE inhibiting effect is faster than the effect of OPs (Vandekar et al., 1971), but also the recovery from the CB inhibition is faster due to a spontaneous ChE decarbamylation. After ChE has been exposed to the inhibiting effect of an OP, a rapid recovery of around 50% of the depressed ChE activity is observed, continuing with a slow increment until the normal level is reached. Fleming (1981) described this recovery behavior in mallard ducklings exposed to dicrocrotophos and fenthion. He also found that exposure to these organophosphates followed by recovery of brain ChE did not significantly affect the degree of ChE inhibition or recovery at subsequent exposure. Recovery of brain ChE activity followed a general model $Y = a + b(\log X)$, which is supported by evidence obtained by other authors cited by Fleming (1981). Two processes would be implied in the recovery of inhibited ChE. The first rapid recovery would be based on ChE reactivation, whereas the slower phase would be based on *de novo* synthesis of ChE (Fleming, 1981).

In the model, insecticide blood concentration is the balance between insecticide absorption and insecticide excretion in the bird's body. The disappearance of the activated-form of the insecticide in the animal body follows a first-order degradation curve (see Section 2.3.3. and Table 2 in Corson et al. (1998) for insecticide half-live in vertebrates). Brain ChE inhibition is estimated by linear interpolation in a dose-ChE inhibition curve built from data found in the literature. Brain ChE inhibition was chosen because it is a better predictor of exposure to a ChE inhibitor (Fleming, 1981; Small et al., 1998; Maul and Farris, 2004). The final output of this model is the percentage of ChE inhibition resulting after adding the effects of the different insecticides to which the bird has been exposed. It is assumed that no synergistic effects occurs, although some interactions among effects of insecticides may exist (Gordon et al., 1978; El-Sebae et al., 1978; Janardhan et al., 1979; Johnston et al., 1994; Johnston, 1995; Subramanya et al., 2004; Rendon-von Osten et al., 2005). A 20% inhibition or decrement in ChE activity (about 2 standard deviations below the mean ChE activity of non-exposed animals) is considered a sign that the animal has been exposed to a ChE-inhibiting substance. An inhibition of more than 50% is considered lethal (Ludke et al., 1975; Hill and Fleming, 1982).

We use the equation $Y = a + b(\log X)$ to represent ChE activity recovery, or decrease of ChE inhibition. In the formula, Y is the percentage of ChE activity compared with unexposed animals; X is the time in hours since the last exposure. The constants a and b, which equal to 29 and 48, respectively, were estimated from data in Fleming (1981).

2.7.1. *Quantitative development*

The loads of insecticide in the digestive system, IDS, and in the bloodstream, IBT, are calculated as:

$$\mathrm{IDS}_{t+1} = \mathrm{IDS}_t + \left(\frac{\mathrm{IIN}_t}{\mathrm{bw}} \times \left(1 - \frac{\mathrm{ife}}{100}\right)\right) \times \Delta t \quad (22)$$

$$\mathrm{IBT}_{t+1} = \mathrm{IBT}_t + (\mathrm{IDS}_t - \mathrm{iex}_t) \times \Delta t \quad (23)$$

$$\mathrm{Iex}_t = \mathrm{IBT}_t \times \left(1 - \mathrm{EXP}\left(-\left(\frac{\mathrm{LOGN}(2)}{T_{1/2a}}\right)\right)\right) \quad (24)$$

$$\mathrm{ChE} = f(\mathrm{IBT}_{t+1}) \quad (25)$$

IDS and IBT are measured in μg g body weight^{-1}. Body weight is symbolized by bw. Ife corresponds to the percentage of ingested insecticide that is released in feces. Iex represents the amount of μg of insecticide that is metabolized and excreted per hour. $T_{1/2a}$ is the half-life of the insecticide in the animal body. The percentage of ChE inhibition is a function of IBT. See Eq. (20) for IIN.

2.7.2. *Input information*

For this submodel the following information has to be specified: (1) body weight of the animal; (2) insecticide release rate in feces; (3) insecticide half-life in the animal's body; and (4) insecticide dose–brain ChE inhibition relation curve. Body weight is entered in grams. Insecticide excretion rate

is entered as the percentage of insecticide ingested that is directly released in the feces. Insecticide half-life is entered in hours. The insecticide dose–ChE inhibition relation is entered as the percentage of ChE inhibition related to insecticide dose in micrograms per gram of body weight.

3. Model application

To demonstrate application of the model, we parameterized the model to represent, as closely as possible, part of a field study that examined the effect of exposure to insecticides on ChE activity in several species of wildlife in the Lower Rio Grande Valley (LRGV) of Texas, USA (Custer and Mitchell, 1987). We simulated the effect on ChE activity in white-winged doves (WWDO) of chemical treatment of a particular cotton field (Santa Maria) in which Azinphos methyl (AM) and Methyl parathion (MP) were applied (Custer and Mitchell, 1987). Cypermethrin and Fenvalerate also were applied; these insecticides do not inhibit ChE, therefore were not included in the model.

In the following sections, we first provide pertinent background information on WWDO, irrigated agriculture in the LRGV, and characteristics of AM and MP. We then describe parameterization and use of the model to simulate part of the field experiment of Custer and Mitchell (1987). Finally we use the model to simulate a variety of hypothetical alternative scenarios that could have increased the risk of pesticide-induced inhibition of ChE activity in WWDO, and report results of a "worst case" scenario.

3.1. *White-winged dove*

Due to the incomes generated by hunting licenses and hunter payments to landowners (Texas Parks and Wildlife, 2004), WWDO is an important game bird in the LRGV, which is its historical breeding and nesting habitat (Cottam and Trefethen, 1968).

Since 1920, rural populations of WWDO have suffered a notable reduction. It has been hypothesized that WWDO density in the region has been affected by several factors, such as destruction of natural nesting areas by human development (agriculture, urbanization)(Brown et al., 1977), change in quality of food available (Dolton, 1975), over-hunting and predation (Marsh and Saunders, 1942; Kiel and Harris, 1956), and ingestion of insecticides by drinking contaminated water (Tacha et al., 1994).

WWDO nest in natural mixed woodlands, citrus groves, and trees in urban areas that have dense foliage. WWDO consume primarily grain from agricultural crops, such as sorghum, corn, and domestic sunflower (Dolton, 1975; Schacht et al., 1995). They can feed on seeds on the ground, or feed directly on seed heads elevated above the ground (Schwertner et al., 2003). WWDO normally drink in open areas during short periods of time (seconds to a few minutes) (MacMillen and Trost, 1966). Their mean body mass is approximately 153 g (Zammuto, 1986). Females and males normally take turns incubating the eggs. Males usually stay on the nest from 11:00 to 17:00, whereas females remain on the nest during the rest of the day (Schacht et al., 1995).

3.2. *Irrigated agriculture in the LRGV*

The LRGV is a region of about 11,125 km^2 that extends 160 km upstream from the mouth of the Rio Grande at the Gulf of Mexico in Texas, USA, in Starr, Willacy, Hidalgo, and Cameron counties (Vigness and Odintz, 2004). Agriculture in the LRGV is based on the production of sorghum, vegetables, cotton, sugarcane, citrus, corn and hay-pasture (Chapman et al., 1996); 38% of the region is cropland, of which about 31% is under irrigation. Flooding furrows is the most common irrigation method. About 1307 million cubic meters of water are used annually for irrigation (The Texas Water Development Board, 2004).

3.3. *Methyl parathion and Azinphos methyl*

MP and AM are broad-spectrum agricultural insecticides. They are among the top ten insecticides used in Texas (Texas Center for Policy Studies and Environmental Defense, 2001); MP was the most widely used organophosphate pesticide during the 1980s (Burkepile et al., 2002).

Soils in the LRGV vary from sandy loam to heavy clay, but are predominantly clays. Soil pH ranges between 7.9 and 8.4, and thus are classified as alkaline (Thompson et al., 1972; Williams et al., 1977; Jacobs, 1981; Turner, 1982). For soils with similar characteristics to those of the LRGV, the half-life of MP is equal to 135 h (Sakellarides et al., 2003), whereas the half-life of AM is equal to 770 h (U.S. Environmental Protection Agency, 1998a). The degradation of insecticides in water is influenced by pH (Racke, 1992); half-lives for MP and AM in alkaline water are 600 h and 624 h, respectively (U.S. Environmental Protection Agency, 1998a; U.S. Environmental Protection Agency, 1998b). The degradation rates of pesticides on plant foliage are species-specific. Half-lives of approximately 3.6 h and 10.4 h have been estimated for MP and AM, respectively (U.S. Environmental Protection Agency, 1998a; U.S. Environmental Protection Agency, 1998b).

3.4. *Simulation of the field study*

3.4.1. *Model parameterization*

Custer and Mitchell (1987) measured brain ChE activity in several wildlife species, including WWDO, after the application of various insecticides, including MP and AM, to several crop fields via fixed-wing aircraft. We simulated chemical treatment of a particular cotton field (Santa Maria) in which AM was applied at a rate of 280 g of active ingredient (a.i.) ha^{-1} on May 18, June 4, 9, and 27, and July 1, and MP was applied at a rate of 560 g a.i. ha^{-1} twice on July 10 and twice on July 16. Application drift was set at 8%. Custer and Mitchell did not provide information about the time of day that insecticides were applied, nor about irrigation events. In the LRGV, pesticide applications usually are performed in the morning or evening, when there is less wind and most of the pollinating insects are inactive, thus we simulated AM applications at 8:00 and MP applications at 8:00 and 10:00. An irrigation of 115 mm was simulated 24 h after each pesticide application or after the last application when two applications were performed at the same day. Every time the field was flooded, birds were forced to drink (satisfy completely their daily water requirement) in the cotton field at 9:00. Runoff was set at

Table 1 – Average water intake, percentage of reduction in water intake, brain ChE activity, and percentage of reduction in brain ChE activity of white-winged doves exposed to methyl parathion in drinking water

Methyl parathion concentration (ppm)	Average water intake (ml day^{-1})	Reduction in water intake (%)	Average brain ChE activity (μmol min^{-1} g^{-1})	Reduction in brain ChE activity (%)
0.0	29.6 (7.3)	0.0	21.0 (1.8)	0.00
2.6	20.3 (2.3)	31.4	14.3 (4.5)	31.90
5.2	18.0 (5.4)	39.2	14.2 (7.1)	31.90
7.8	14.7 (3.9)	50.3	7.5 (2.6)	32.38
10.4	10.5 (3.9)	64.5	4.6 (1.7)	64.29

Data from Small et al. (1998). Numbers in brackets are standard deviations.

10%. Because canopy cover changes seasonally, the amount of insecticide that comes from runoff and accumulates on the ground (floodable and non-floodable areas) is a function of plant cover changes. We represented the change in percentage of plant cover (y) over time (x [days]) in the cotton field as: $y = a/(1 + b \times \exp^{(-cx)})$; where $a = 100.18$, $b = 134.86$, and $c = 0.126$ (Norman, 2003; Norman, personal communication).

To parameterize infiltration and evaporation rates, we used data from Fipps (2004) to estimate an infiltration rate of 7.62 mm h^{-1} and an evaporation rate of 1390 mm year^{-1}, which are representative values for LRGV. The evaporation rate was calculated as:

$$\frac{0.8 \times \text{peak Class A pan evaporation} \times \text{floodable area}}{100} \quad (26)$$

The *peak class A pan evaporation* occurs in July and equals 6.35 mm per day (Fipps, 2004). We assumed the *floodable area* represented 60% of the field.

To parameterize the dose–response curve relating the concentration of MP in drinking water to ChE inhibition in WWDO, we drew upon experimental data reported by Small et al. (1998). They exposed captive WWDO to various levels of MP in drinking water to determine the effects of water intake on ChE activity in the brain (Table 1), and also on productivity and reproductive behavior. Based on these data we estimated the relation between MP dose per gram of body weight (BW)(assuming BW = 153 g) and ChE inhibition by linear regression $Y = a + bX$; where Y is the percentage of ChE inhibition and X is the MP dose (μg g BW^{-1}). The resulting equation was:

$$Y = -2.121 + 90.91X (r = 0.918, \quad r^2 = 0.843,$$
$$P = 0.03, \quad SE = 14.03) \quad (27)$$

To our knowledge, there are no data relating ChE inhibition in WWDO to AM concentration in drinking water. Thus we estimated a dose–response curve for AM using experimental data from a study conducted by Thompson et al. (1995), which related the activation of organophosphorus pesticides to oxon metabolites and sensitivity of 'B' sterases to inhibition by these metabolites in the brain of pigeons (*Columba livia*). They found that MP oxon inhibits brain ChE 48.26 times more than AM oxon. Based on the relatively close phylogenetic relationship between WWDO and pigeon, we assumed that they have similar activation and detoxification metabolic pathways to both AM and MP oxon metabolites. Based on this assumption, we corrected the MP dose–response curve (Eq. (26)) to estimate a dose–response curve for AM:

$$Y = \left(\frac{(-2.121 + 90.91X)}{(48.26 \times 1.21)} \right) \quad (28)$$

where Y is the percentage of ChE inhibition and X is the AM dose (μg g BW^{-1}). The value 1.21 corresponds to the AM oxom weight-based equivalent, which results from dividing the molecular weight of MP (263.21 g mol^{-1}) by the molecular weight of AM (317.33 g mol^{-1}). This correction standardizes the effect of molecular weight on application rates based on the g of active ingredient per ha.

3.4.2. *Simulation results*

The highest accumulations of residue in the floodable area were 15.46 g a.i. ha^{-1} for AM and 61.51 g a.i. ha^{-1} for MP (Fig. 4), which resulted in maximum concentrations in drinking water of 0.013 ppm for AM and 0.048 ppm for MP. Maximun levels of ChE inhibition were reached during the last application for both AM and MP, 0.27% on July for AM and 0.65% on July 16 for MP (Fig. 5). These simulated levels of ChE inhibition are well below both the diagnostic level of exposure (20%) and diagnostic level of severe risk (50%), and are consistent with the lack of ChE inhibition reported by Custer and Mitchell (1987).

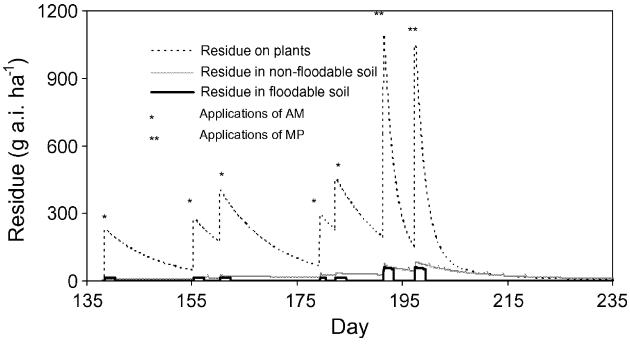

Fig. 4 – Simulated amount of methyl parathion and Azinphos methyl residue accumulated on plants in the non-floodable area and in the floodable area of a cotton field. Irrigations of 115 mm were simulated 24 h after each Azinphos methyl application and 24 h after the first and third applications of methyl parathion.

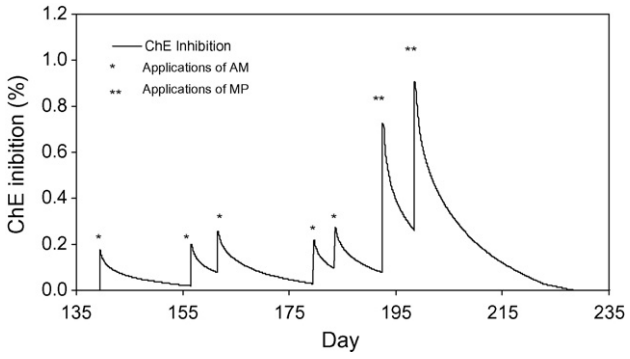

Fig. 5 – Simulated brain ChE inhibition in a white-winged dove that drank water from an irrigated cotton field treated with methyl parathion and Azinphos methyl. Irrigations of 115 mm were simulated 24 h after each Azinphos methyl application and 24 h after the first and third applications of methyl parathion.

3.5. *Simulation of a "worst case" scenario*

We used the model to search for possible "worst case" scenarios that might arise from alternative combinations of irrigation and pesticide application schemes, which were slightly different from those of the simulated field study reported above, but feasible within the context of cotton agriculture in the LRGV. Here, we report the simulation of one particular scheme that resulted in markedly increased levels of ChE inhibition in WWDO.

The "worst case" scenario differed from that of the simulated field study in that we simulated a rainfall event of 15 mm in place of the last 115 mm irrigation. We set the percentage of pesticide washoff at 65 and 90 for AM and MP, respectively (Knisel and Davis, 2000); since there were no rainfall events in the simulated field study, there was no washoff. When the WWDO drank rain water after the rainfall event, it exhibited a level of ChE inhibition (>78%) that greatly exceeded the diagnostic level of risk (50%). This high level of ChE inhibition resulted from the fact that more pesticide was washed off the canopy and the non-floodable soil, and this washoff was dissolved in less water. Concentrations of AM and MP dissolved in water were 5.8 and 10.4 times higher, respectively, than the simulated field study, and concentrations of AM and MP dissolved in water were 7.5 and 92.8 times higher, respectively, than in the simulated field study (Table 2). In fact, during the "worst case" simulation, levels of ChE inhibition were >50% for a total of 1.2 days, and were >20% for 6.5 days. Survival and reproduction of an animal with this level of ChE inhibition would be seriously compromised.

4. Discussion and conclusion

Custer and Mitchell (1987) did not find WWDO with inhibited ChE activity after they were collected from fields that had been sprayed the previous days. Although the likelihood of exposure to AM and MP in the simulated field study might have been higher than in the study of Custer and Mitchell, the simulated WWDO also exhibited unmeasurably low levels of ChE inhibition. However, as shown in the simulation of a "worst case" scenario, there is a risk of dangerously high levels of exposure to insecticides under certain conditions, such as occurrence of a rainfall event just after an insecticide application. The probability of such risk depends not only on the frequency and intensity of irrigation and rainfall events, but also on the availability of non-contaminated sources of drinking water. The probability that WWDO drink in a cotton field depends on the distribution of different crops and, hence, alternative sources of water across the landscape. For instance, the simulated WWDO spends 2% of its time in cotton fields (Schacht et al., 1995). Since the probability of finding water in any simulated ALU after a rainfall event is the same, the probability of drinking in the cotton field is 0.02, which, when multiplied by the probability of a rainfall event occurring soon after an insecticide application, results in an extremely low risk. Furthermore, rain may have two opposite effects on risk of exposure to pesticides of wildlife using agricultural fields for foraging or drinking. Whereas rain may threaten the health of animals that drink in agricultural fields treated with pesticides, rain may favor herbivores because of the washoff of pesticides from the canopy (Wang et al., 2000).

It should be a useful tool to help assess the ecological risk to non-target wildlife of exposure to pesticide-contaminated water in irrigated agricultural fields. Conceptual development (Section 2), parameterization (Section 3.4.1), and application (Section 3.4.2) of the model paralleled the three phases used by the Environmental Protection Agency (EPA) of the United States to conduct an ecological risk assessment: problem formulation, analysis, and risk characterization (EPA—Environmental Protection Agency, 1998). The present

Table 2 – Maximum ChE inhibition in a white-winged dove, and maximum residues of Azinphos methyl (AM) and methyl parathion (MP) in the floodable soil and dissolved in water, occurring during simulations of the field study of Custer and Mitchell (1987) and a "worst case" irrigation/pesticide application scenario

	Pesticide	Field study	"Worst case" scenario
ChE inhibition (%)	AM	0.27	8.09
	PM	0.86	70.58
Residue in floodable soil (g a.i. ha^{-1})	AM	15.46	89.05
	PM	61.51	642.02
Residue dissolved in water (ppm)	AM	0.013	0.618
	PM	0.048	4.454

simulation model could be considered as a submodel of the EPA-Terrestrial Level II Model (TLIIM) that deals with the ingestion of insecticide through drinking water. Also, it fulfills almost all the recommendations that the Scientific Advisory Panel of the Federal Insecticide, Fungicide, and Rodenticide Act, which met in 2001, made on the TLIIM Version 1.0, and then incorporated in the TLIIM Version 2.0 (EPA—Environmental Protection Agency, 2004).

To demonstrate application of the model, we focused on assessing the risk of exposure to ChE-inhibiting pesticides for birds drinking water from agricultural fields under several combinations of environmental conditions and agricultural practices typical of the LRGV of Texas (assessment goal), as indicated by levels of ChE inhibition in individual birds (assessment endpoint). We parameterized the model to simulate the exposure of a WWDO to organophosphorus and carbamate pesticides (exposure to stressors) and the resulting levels of ChE inhibition in the brain (relationship between stressor levels and ecological effects); ChE inhibition can be a direct (lethal dose) or indirect (sub-lethal, behavior-altering dose) cause of death, and can impair reproduction. The present model could be adapted to help assess the ecological risk to a variety of non-target wildlife of exposure to a variety of environmental contaminants. The present submodels generically represent periodicity and magnitude of contaminant arrival in the environment (submodel I), contaminant transport in the environment (submodels II, III, and IV), exposure of non-target wildlife to contaminants (submodel V), and ecological impact of exposure on non-target wildlife (submodel VI). These submodels could be re-formulated, re-parameterized, and/or "turned off" without changing the general structure of the model. Obviously, the amount of actual programming necessary to re-formulate submodels will depend on the particular system of interest. But we suspect many scenarios of interest, for example, assessing the ecological risk to non-target wildlife of exposure to heavy metals in the environment, would require relatively little re-programming.

This model should be particularly useful in identifying specific situations in which the juxtaposition of environmental conditions and management schemes could result in a high risk to non-target wildlife. However, usefulness of this simulation model, like others, could be improved by the inclusion of new data on basic parameters, such as species-specific dose–response curves for pesticide-induced ChE inhibition and half-lives of pesticide residues in plants, water and soil. Environmental agencies use a few species as surrogates for risk assessment of the impact of environmental pollutants; however, species tolerance to the exposure to these substances is variable, even in species that are phylogenetically closely related (Mineau, 1991; Thompson et al., 1995; Blakley and Yole, 2002). Also, the assumptions that there is no toxic action of inert ingredients, adjuvants, and diluents, and that there are additive but no synergistic or suppressive effects of insecticide mixtures, should be reviewed.

Thus, we suggest investing more effort in studying (1) the degradation of insecticide residues in soil and water under different natural conditions, (2) the relationship between the amount of insecticide ingested and the resulting level of brain cholinesterase activity on a species- and age-specific basis not only for the active ingredient but also for diluents and adjuvants if they are toxic, (3) the effects resulting from the interaction of different insecticides, and (4) the relationships among levels of ChE inhibition and survival and reproductive risk.

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