PESTICIDE IMPACT ON NON-TARGET WILDLIFE IN IRRIGATED CROPS: SIMULATED IMPACT OF CHOLINESTERASE-INHIBITING PESTICIDES ON WHITE-WINGED DOVES IN THE LOWER RIO GRANDE VALLEY OF TEXAS

A Dissertation

by

JORGE MARCELO PISANI

Submitted to the Office of Graduate Studies of
Texas A&M University
in partial fulfillment of the requirements for the degree of

DOCTOR OF PHILOSOPHY

May 2006

Major Subject: Wildlife and Fisheries Sciences

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Approved by:

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ABSTRACT

Pesticide Impact on Non-Target Wildlife in Irrigated Crops: Simulated Impact of Cholinesterase-Inhibiting Pesticides on White-Winged Doves in the Lower Rio Grande Valley of Texas. (May 2006)

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Chair of Advisory Committee: Dr.William E. Grant

I present a simulation model that should be a useful tool for risk assessment of the impact of insecticide inhibitors of cholinesterase (ChE) applied in irrigated agricultural fields on non-target wildlife. I developed the model as a compartment model based on difference equations (Δt = 1 hour) and programmed with Stella® VII software. Conceptually the model is compartmentalized into 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, I simulate historical, current, and "worst-case" scenarios, that examined the impact of ChE-inhibiting insecticides on white-winged doves (WWDO - *Zenaida asiatica*) in the Lower Rio Grande Valley of Texas (LRGV), USA. To my knowledge, there are no field data verifying that the cause of ChE deprivation in WWDO is due to the ingestion of ChE-inhibiting insecticide residues

dissolved in drinking water. I parameterized the model to represent a system composed of fields of cotton, sorghum, corn, citrus, and brushland that encompasses the activity range of a WWDO in the LRGV. I simulated situations representing the typical scenario of WWDO using irrigated crop fields in the absence and in the presence of rain. I also simulated "worst case" scenarios in which methyl parathion was applied at high rates and high frequency.

Based on results of the simulations, I conclude that it is unlikely that WWDO are seriously exposed to ChE-inhibiting insecticides by drinking contaminated water. Only in rare cases, for example, when a rain event occurs just after the application of insecticides, are levels of ChE inhibition likely to approach diagnostic levels (20 %).

The present simulation model should be a useful tool to predict the effect of ChE-inhibiting insecticides on the ChE activity of different species that drink contaminated water from irrigated agricultural fields. It 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.

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CHAPTER I

INTRODUCTION

About 6.5 billion human beings are living on the earth, and the earth's population is expected to be 9.1 billion in 2050 (UN Department of Economic and Social Affairs Population Division, 2005). The most basic need for this expanding population is food. All basic products of the food industry come from agricultural systems. The duplication of population density in the last 65 years was supported by the Green revolution, a new tide of agricultural technologies in the 1960s and 1970s, such as use of fertilizer, pesticides, increased irrigation, and improved seeds. At present, although irrigated farmland represents 20 % of total farmland, it produces about 40 % of global foods (FAO, 2003).

However, the maximization of the efficiency of agricultural production has caused collateral undesirable effects. For instance, a massive use of pesticides has resulted in an accumulation of toxic residues in the environment that threatens the health of people, plants, animals, and ecosystems. In 1939, a new chemical with the most powerful properties ever seen was patented, DDT, a general insect killer with long residual effect. But it was not until the late 1950s that DDT became widely used in agriculture. A few naturalists became concerned after the first evidence of animal die-offs related to DDT

This dissertation follows the style of Ecological Modelling.

sprays. One of them was Rachel Carson, who, in 1962, published the book "The Silent Spring", in which she explained the side effects of DDT. Such was the impact of this book on American society that it not only caused DDT to be banned but also provided the basis of a new environmental safety awareness among the public.

After Carlson's book, a new question emerged. What insecticide properties would be more desirable? In the light of this question Organophosphorus (OP) and Carbamate (CA) insecticides became important. Compared with organochlorine insecticides like DDT, OPs and CAs have higher acute toxicity (their effects are produced at lower doses) but shorter degradation times. Because of their relatively fast decay they were assumed to be environmentally safer; they neither accumulate in soils or water for long time periods nor bioaccumulate throughout trophic chains (Pope and Rall, 1995).

With the aim of minimizing the use of pesticides, about 3 decades ago the idea of eradicating pests began to be replaced by the idea of reducing them to levels that do not produce economic damage (Odum and Barret, 2000). This new concept about pest control led President Nixon, in 1972, to formally commit the U.S. government to the development and promotion of Integrated Pest Management (IPM). IPM is a set of agricultural practices, such as crop rotations, biological control, and use of resistant crop varieties for long-term prevention of pest damage. Pesticides are only used if the density of a pest has reached the economic damage threshold. Although IPM is widely used, many pesticides still are being applied. For instance, in 1997, 41,305 Tons of insecticide active ingredients were applied in the Unites States, of which 54 % and 22 % were OPs and CAs, respectively (Gianessi and Silvers, 2000; Cuperus et al., 2005).

Factors such us new crop varieties, pest resistance, pesticide accumulation in the environment, new pesticides on the market, recommended application rates, and evidence of pesticide side effects are constantly changing. Therefore, governmental environmental agencies like the US Environmental Protection Agency (EPA), Canadian Pest Management Regulatory Agency (PMRA), and the Council of the European Community for Pesticide Regulation must continually evaluate the conditions under which pesticides are registered to minimize the harmful consequences of their use. This is a hard task because people in charge of decision making about pesticide registration have to deal with the tradeoff of proximate benefits of using a pesticide (e.g., increase in production, incomes from pesticide production, control of parasites and disease vectors) versus the ultimate consequences (e.g., environmental pollution, and other negative impacts on plants, animals, and human health).

To accomplish this regulatory decision making, EPA follows two processes, risk assessment and risk management. Risk assessment defines the potential probability that the adverse effects of a pesticide occur to individuals or populations, while risk management weighs the appropriate risk reduction alternatives considering risk assessment, social, economic, legal, political, and ecological factors. The basic process in risk assessment is to compare the toxicity information available for a pesticide with the level of exposure to which the organism may be subject (EPA - Office of Pesticide Programs, 2000).

Due to the acetyl cholinesterase (ChE) inhibitory characteristic of OPs and CAs, the principal element considered in ecological risk assessment of the exposure to these

insecticides is the level of ChE activity of animals. Effects of exposure to OPs and CAs can range from no visible sign of intoxication, to alteration of behavior, physiology, and reproduction, to death (Rattner and Fairbrother, 1993; EPA - Office of Pesticide Programs, 2000).

The Office of Pesticide Programs of the EPA is in charge of proposing and reviewing the guidelines for pesticide risk assessment. Different types of data may be used for risk assessment, such as data collected from laboratory studies, field studies, or data produced as output from a simulation model. Laboratory data are more accurate; however, they cannot represent complex natural processes (e.g., animals subjected to potentially lethal doses in laboratory experiments may have higher survival rates than animals subjected to similar doses living in the wild). On the other hand, field data represent real processes, but lack of control variables may make it difficult to identify interactions among variables and, consequently, complicate the recognition of cause-effect relationships (EPA - Environmental Protection Agency, 1998).

Often, simulation models are developed as a means of incorporating data from laboratory and field studies, as well as information that exists in the form of expert opinion, into an integrated tool that can help inform management decisions. The usefulness of model outputs depends on the appropriateness of model structure (appropriateness of the conceptual model) and the reliability of parameter estimates (reliability of information drawn from laboratory and field studies and from expert opinion). Model outputs (simulated data) represent changes in the simulated system, and, by inference, in the real system, over time (Grant et al., 1997). They not only can

simulate systems under circumstances that have been observed but also they can simulate hypothetical situations (e.g., predict the transport of pesticides under different sets of climatic conditions, or predict the impact of a pesticide not already registered). This feature makes simulation models a valuable tool for risk assessment and also a powerful learning and communication tool because they provide an explicit expression of the assumptions and understanding of a system for others to evaluate (EPA - Environmental Protection Agency, 1998).

In this dissertation, I present a simulation model that should be a useful tool for risk assessment of the impact of insecticide inhibitors of ChE applied in irrigated agricultural fields on non-target wildlife. In Chapter II, I describe a simulation model that estimates the level of ChE inhibition of an animal that drinks contaminated water from irrigated agricultural fields treated with ChE-inhibiting pesticides. In Chapter III, I use the model to simulate current agricultural scenarios in the Low Rio Grande Valley of Texas to evaluate the hypothesis that white-winged doves (*Zenaida asiatica*) exposed to OPs and CAs when they drink contaminated water from irrigated agricultural fields exhibit increased levels of ChE inhibition. I present conclusions of the previous chapters and research recommendations in Chapter IV.

CHAPTER II

SIMULATING THE IMPACT OF CHOLINESTERASE-INHIBITING PESTICIDES ON NON-TARGET WILDLIFE IN IRRIGATED CROPS

1. Introduction

Approximately 40% of global food production is supported by irrigated agriculture, which comprises 20% of 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 USA during 1997 (Gianessi and Silvers, 2000) (Table 1). Whereas 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; Flickinger et al., 1991; Grue et al., 1991; Smith et al., 1995; Mineau et al., 1999; Goldstein et al., 1999; Fleischli et al., 2004; Wobeser 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

Table 1.

Amount of active ingredient (ai) in tons and percentage of different types of insecticides applied in USA in 1997. Data from the National Pesticide Use Database (NCFAP, 2003)

Insecticide type	Tons a.i. applied	% by type
Organophosphates	22,245	53.9
Carbamates	9,166	22.2
Chlorinated	6,672	16.2
Sulfites	1,152	2.8
Synthetic pyrethroids	987	2.4
Cyclodienes	726	1.8
Nitroguanidines	123	0.3
Organotins	120	0.3
Antibiotics	60	0.1
Insect growth regulators	54	0.1

degrades the neurotransmissor acetylcholine. This enzyme is responsible for nervous firing within the peripheral and central nervous system. In addition, CAs and OPs bind to others cholinesterases (e.g. butyrylcholinesterase in liver and plasma) and insecticide detoxifying enzymes. Animals with ChE depression show anorexia, lethargy, behavioral and physiological disorders (Grue and Shipley, 1981; Grue et al., 1991; Grue et al., 1997; Bishop et al., 2000a; Bishop et al., 2000b; Bishop et al., 2000c; Solecki et al., 2001a; 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 I present a model that simulates the level of ChE inhibition in animals drinking pesticide-contaminated water from flood-irrigated crop fields. I 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, I 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 water concentration is a function of amount of insecticide residue and 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 metabolization 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 in difference equations (Δt = 1 hour) 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

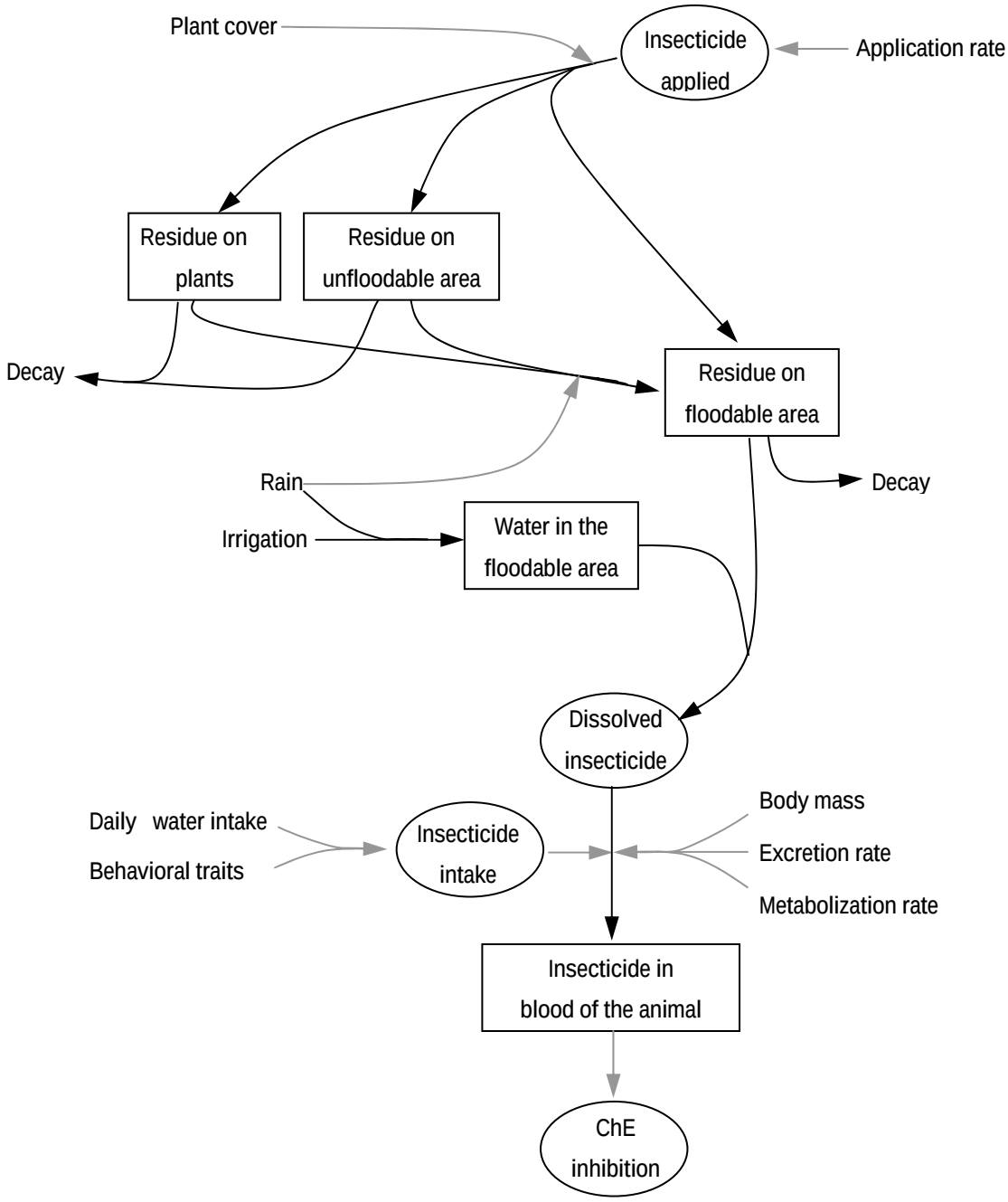

Fig. 1. General conceptual model. Gray and black arrows represent information and material flows, respectively.

replicated under an array of n insecticides x 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); or 2) trees, shrubs or vines (crop type 1 = citrus, apples, pears, plums, peaches, pecans, grapevines, etc.); or 3) range. Under the classification of range 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. 2).

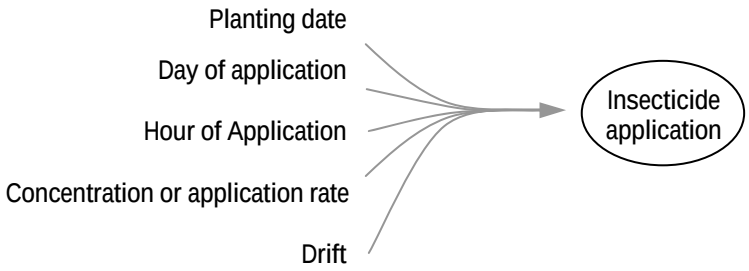

Fig. 2. Conceptual submodel of insecticide application. Arrows represent information flows (Appendix B.1).

2.2.1. Quantitative development

Insecticide applied (*iap* in g of active ingredient ha^{-1} hr^{-1}) can be represented by the equation:

$$iap_t = iar_t \times (1 - d_t / 100) \quad (1)$$

where *iar* represents the pesticide application rate (g of active ingredient ha^{-1} hr^{-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 Julian day 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 hour system. The application rate or concentration is entered as grams of insecticide active ingredient per hectare (g a.i. ha^{-1}) (Fig. 2). Drift is entered as a percentage. No pesticides are applied on range; therefore, it is considered an area free of pesticides.

2.3. Submodel II - Insecticide into floodable soil

2.3.1. Granulated insecticides

The model predictions are based in 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 I 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 ground surface and they could potentially be dissolved in the irrigation water; and also, ingested directly by birds, which might cause 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 and/or is carried out from the crop through drift (Salyani and Cromwel, 1992; Stover et al., 2002; Siebers et al., 2003) (Figs. 3 and 4). Because plant cover increases throughout the growing season, there is a time when plants start to grow above the

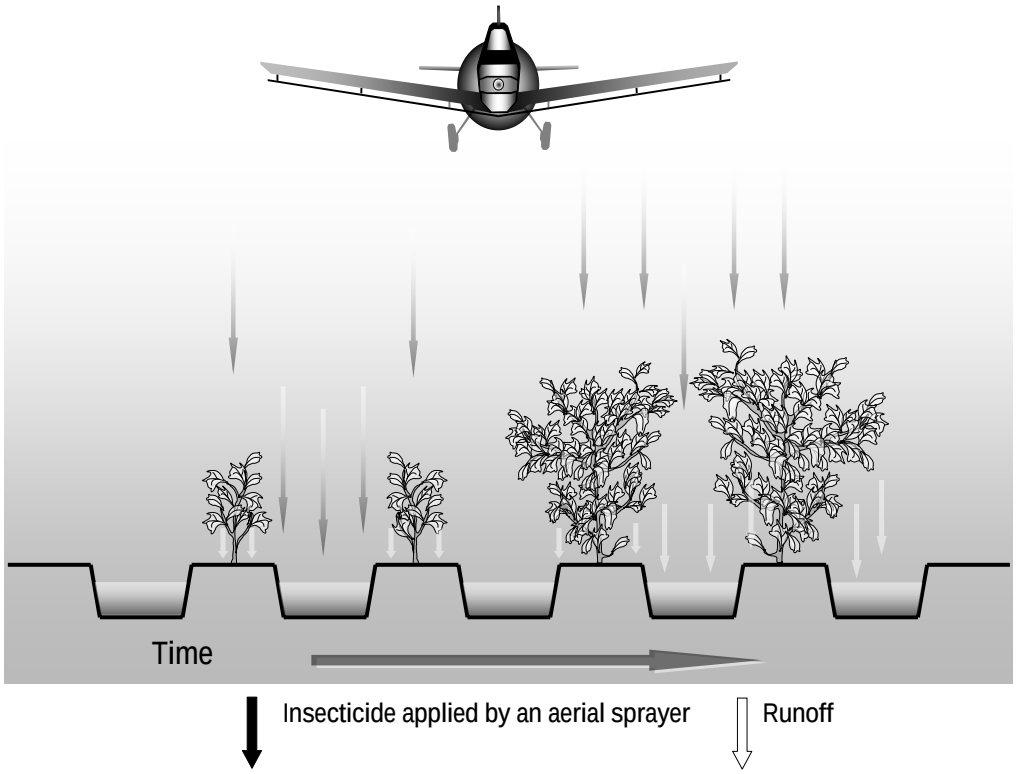

Fig. 3. Pathway followed by insecticides after being released from an aerial or ground sprayer in a type 0 crop such as cotton. The amount of pesticide that drops on the floodable area depends on the application rate and plant cover. The insecticide sprayed may drop directly on the soil and plants; another portion of the insecticide is lost as drift. After the application, a portion of the insecticide that drops on plants reaches the soil as runoff.

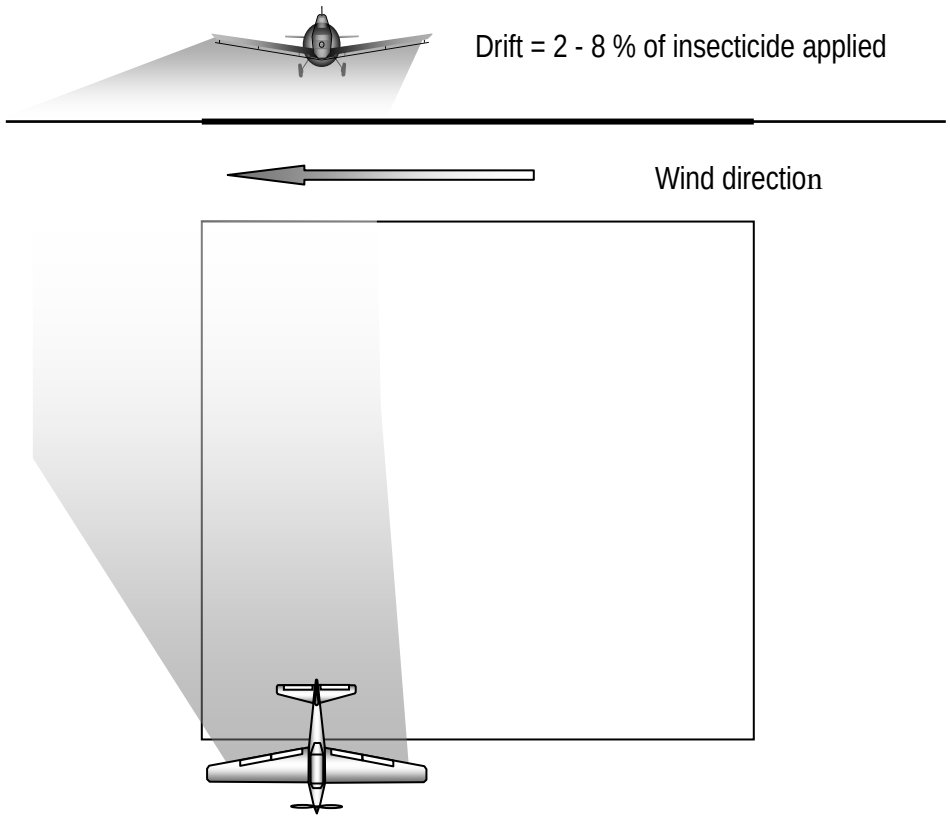

Fig. 4. Drift of a typical aerial application. About 2 - 8 % of insecticide applied, with an average 16 km h^{-1} crosswind, moves out of the target site.

floodable rows. From this point, the amount of insecticide that directly reaches this area decreases over time (Himel et al., 1990) (Fig. 5.); yet, runoff from the canopy above the floodable begins (Fig. 3). Runoff is defined as the insecticide that rolls down from leaves, fruits and branches and falls on the ground; plus, the insecticide that reaches the ground after crossing through the canopy without being intercepted. The runoff from the portion of the canopy above the non floodable area (Fig. 5) is not taken into account in the model. However, if a rain event happens (above 13 mm), the model assumes that insecticide residue accumulated on the non floodable area plus a portion of the residue on plants will be washed-out 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 (Fig. 6). 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 Cromwel, 1992; Cunningham and Harden, 1998; Stover et al., 2002; Salyani, 2004). Small droplets produce better insecticide coverage, but they are prone to be drift or evaporated (Salyani and Cromwel, 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 to coalesce with other droplets and fall 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. It is assumed that there is bare soil under the trees or vines.

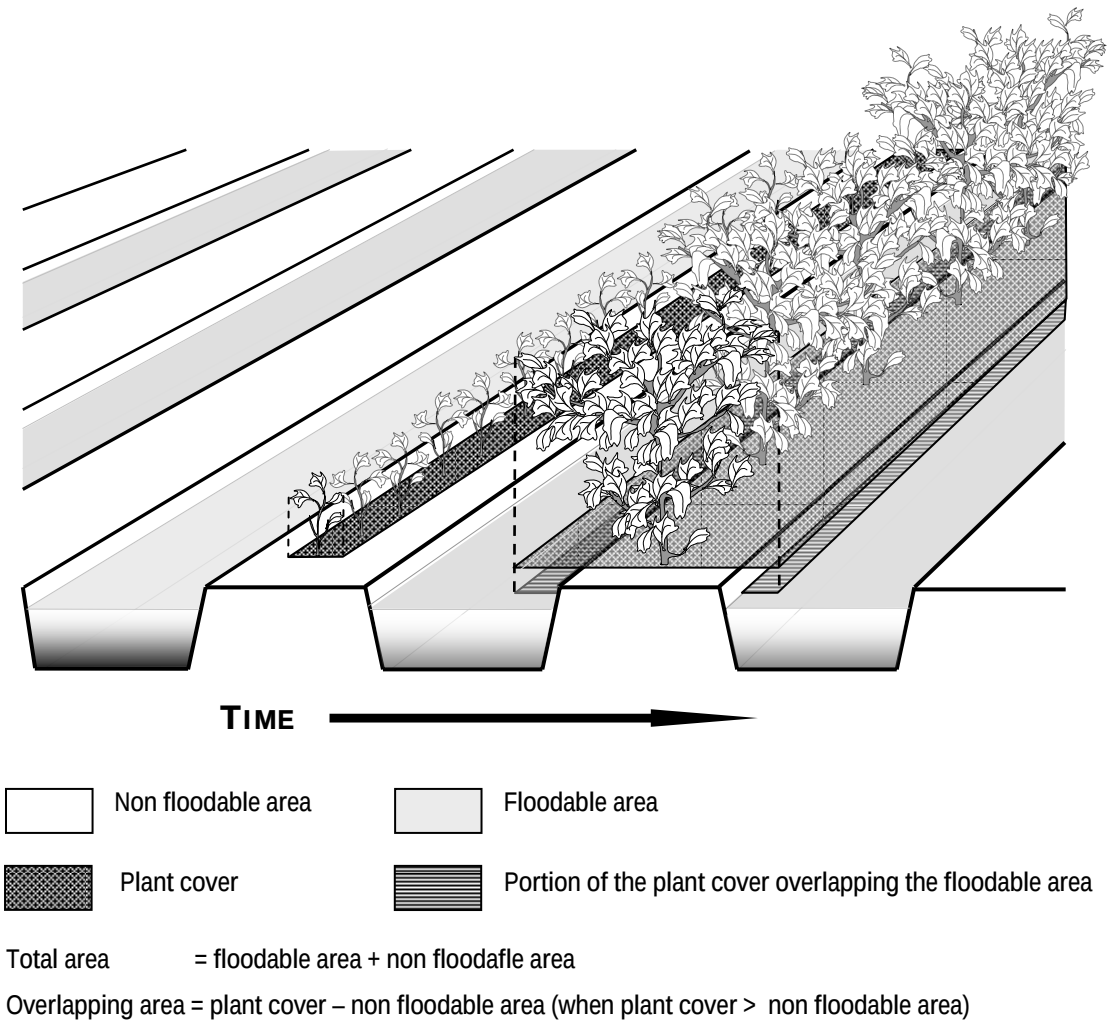

Fig. 5. Seasonal changes in plant cover of a type 0 crop such as cotton. The plant cover increases, covering first the non floodable area and then the floodable area.

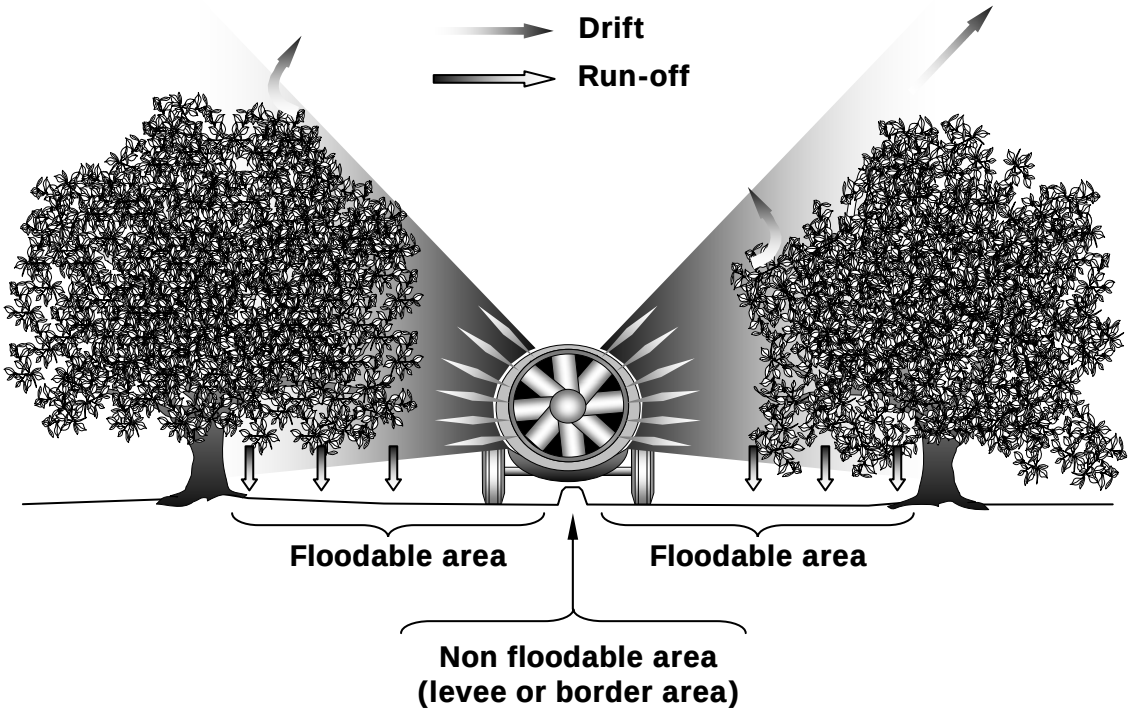

Fig. 6. Pathway followed by insecticides after being released from an air-carrier ground sprayer in a type 1 crop such as citrus. The amount of pesticide that drops on the floodable area comes from runoff. The other portion of the applied insecticide is lost as drift.

2.3.3. Insecticide residue degradation

While the field is not flooded, I 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, $C_t = C_0 \times e^{-k \times t}$ 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:

$$C_{t+1} = C_t + A_t - (C_t \times (1 - e^{-(ln2)/t\,½})) \quad (2)$$

where C_t represent concentration of residue in g/ha 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 sun light, 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; Liu et al., 2000; Karpuozas and Walker, 2000; Rao and Hornsby, 2001; Sanchez-Martin and Sanchez-Camazano, 2003; Sakellarides et al., 2003). The fraction of humic substances within the soil organic matter has strong adsorptive power on organothiophosphate and carbamates 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), I assume that insecticide in this fine layer can be totally dissolved during an irrigation or rain event.

The decay of insecticides starts immediately after the 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, I 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 in 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 it 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 (Table 2).

Table 2.

Water solubility of organophosphate and carbamate pesticides at 20-25 °C. Data from Extoxnet (2005).

Pesticide	Type	Water solubility ($mg\ l^{-1}$)	Pesticide	Type	Water solubility ($mg\ l^{-1}$)
Acephate	OP	790,000.0	Methyl parathion	OP	55.0-60.0
Azinphos methyl	OP	30.0	Phorate	OP	50.0
Chlorpyrifos	OP	2.0	Phosmet	OP	250.0
Diazinon	OP	40.0	Terbufos	OP	5.0
Dicrotophos	OP	miscible			
Dimethoate	OP	25,000.0	Aldicarb	CA	6,000.0
Disulfoton	OP	25.0	Carbaryl	CA	40.0
Ethion	OP	1.1	Carbofuran	CA	320.0
Ethyl parathion	OP	12.4	Methomyl	CA	57,900.0
Malathion	OP	130.0	Oxamyl	CA	280,000.0
Methamidofos	OP	90,000.0	Ziram	CA	65.0
Methidation	OP	240.0			

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. 7):

$$IRP_{t+1} = IRP_t + (ifp - idp) \times \Delta t \quad (3)$$

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

$$= IRF_t + (iff - idw) \times \Delta t \quad \text{if soil is flooded} \quad (4b)$$

$$IRN_{t+1} = IRN_t + (ifn - ids_n) \times \Delta t \quad (5)$$

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} hr^{-1}, and:

$$ifp = iap_t \times pc_t / 100 \times (1 - irp / 100) \text{ (see equation 1 for } iap_t) \quad (6)$$

$$iff = iap_t \times fa / 100 \quad \text{if } pc_t <= nfa \quad (7a)$$

$$= iap_t \times (1 - pc_t / 100) + iap_t \times ((pc_t - nfa) / 100) \times irp / 100 \quad \text{if } pc_t > nfa \quad (7b)$$

$$ifn = iap_t \times ((nfa - pc_t) / 100) + iap_t \times pc_t / 100 \times irp / 100 \quad \text{if } pc_t <= nfa \quad (8a)$$

$$= iap_t \times nfa / 100 \times irp / 100 \quad \text{if } pc_t > nfa \quad (8b)$$

$$nfa = 100 - fa \quad (9)$$

$$pc = f(t) \quad (10)$$

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 run-off. Once plants start to grow above the floodable area, $(1 - pc_t / 100)$ represents the

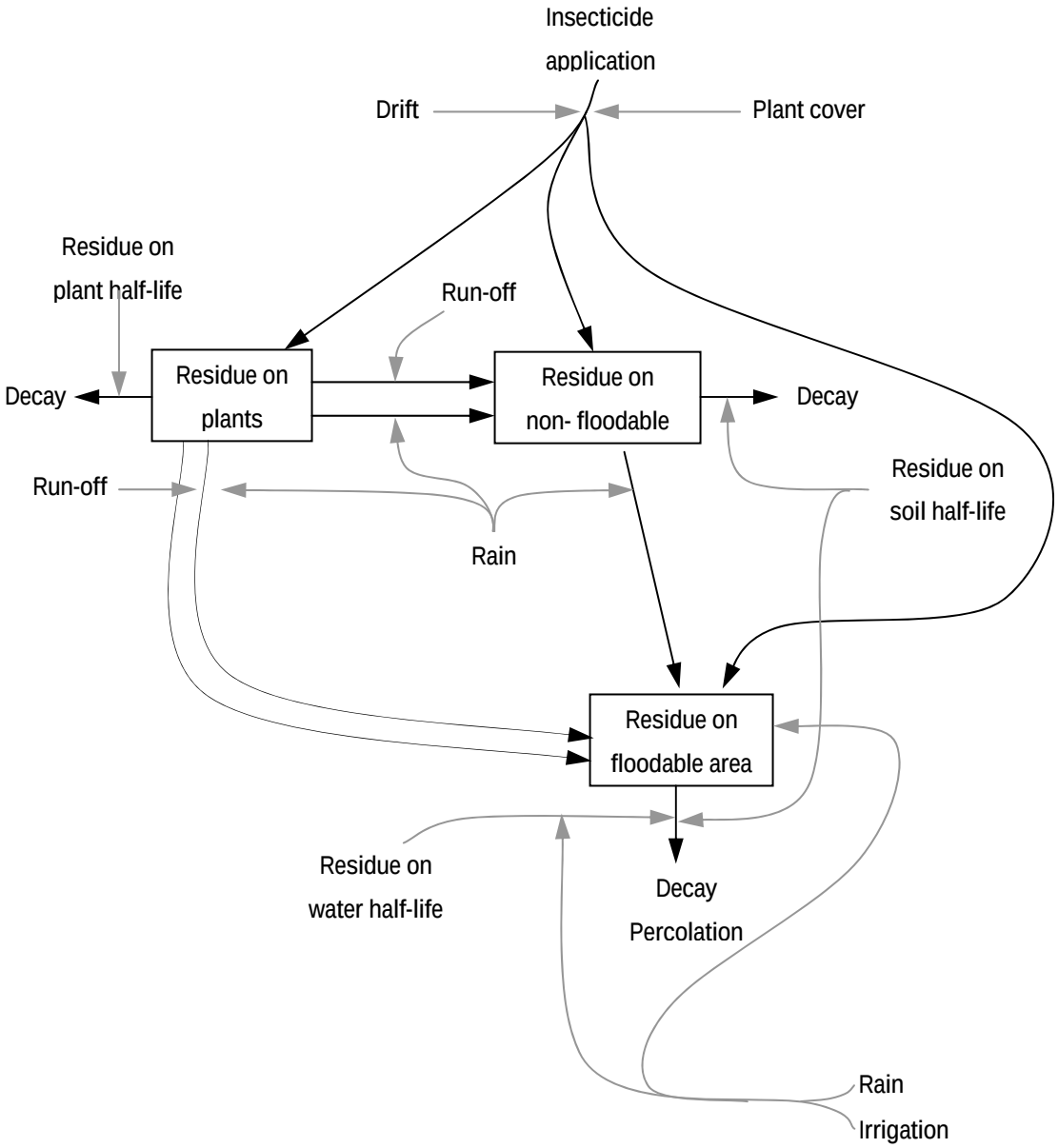

Fig. 7. Conceptual model representing the accumulation of insecticide in the floodable soil. Gray and black arrows represent information and mass flows, respectively (Appendix B.2).

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* are the degradation rates of insecticide on plants, soil and water, respectively. They can be represented as:

$$idp = IRP_t \times (1 - EXP(- (LOGN(2) / T_{1/2\,p})))$$ (11)

$$ids_f = IRF_t \times (1 - EXP(- (LOGN(2) / T_{1/2\,s})))$$ (12)

$$ids_n = IRN_t \times (1 - EXP(- (LOGN(2) / T_{1/2\,s})))$$ (13)

$$idw = IRF_t \times (1 - EXP(- (LOGN(2) / T_{1/2\,w})))$$ (14)

where $T_{1/2\,p}$, $T_{1/2\,s}$ and $T_{1/2\,w}$ 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:

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

where *wff* represent 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.8). Therefore, how fast the water disappears is a function of the amount of water covering the field combined with the evaporation and infiltration rates.

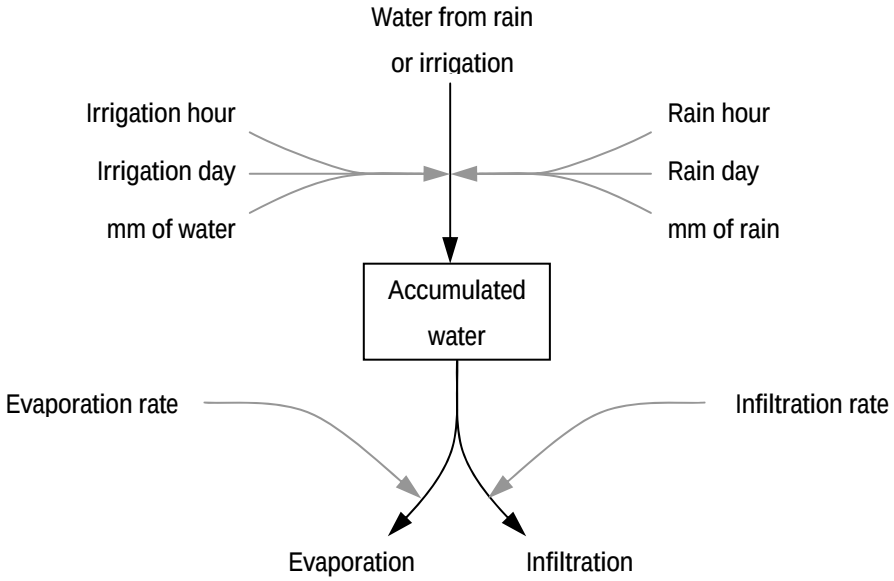

Fig. 8. Conceptual model representing irrigation and rain. Gray and black arrows represent information and mass flows, respectively (Appendix B.3).

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 liters ha^{-1} hr^{-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 Julian day; hour of irrigation and hour of rain are entered based on a 1-24 hour 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 (Table 2). 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. 9).

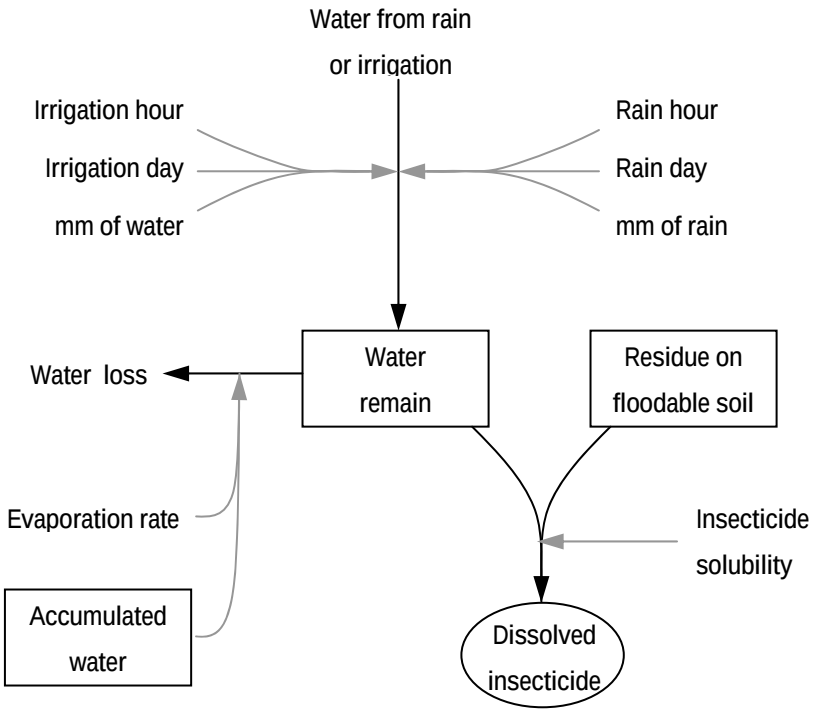

Fig. 9. Conceptual model representing insecticide dissolution in water. Gray and black arrows represent information and mass flows, respectively. The concentration of insecticide residue in water is related to the amount of accumulated water and the residue in the floodable area. Water disappears by evaporation and infiltration. Residue decays in both soil and water, and by leaches into the soil. Water remain represents the initial amount of water accumulated each hour, which subsequently is affected only by evaporation; this keeps the concentration of insecticide dissolved independent of water lost by infiltration (Appendix B.4).

2.5.1. Quantitative development

The concentration of residue in water is represented by *IRW* in ppm or μg g^{-1} or μg ml^{-1}.

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

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

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

$$= 0 \quad \text{if } AW_t(16) = 0 \quad (18b)$$

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

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 in an environment composed by different ALUs, I will focus in a bird species. As an example, the hypothetical bird lives in an environment consisting of four ALUs and range.

The place where the bird forages is important because it determines where the animal drinks. Two modes can be used for simulations: in mode 1, the model user specifies in which ALU the bird forages, whereas in mode 2, the bird forages according to the bird's foraging rules (Fig. 10). 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 an 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 (Fig. 10). It is also assumed that the previous choice does not affect the choice of the next drinking site.

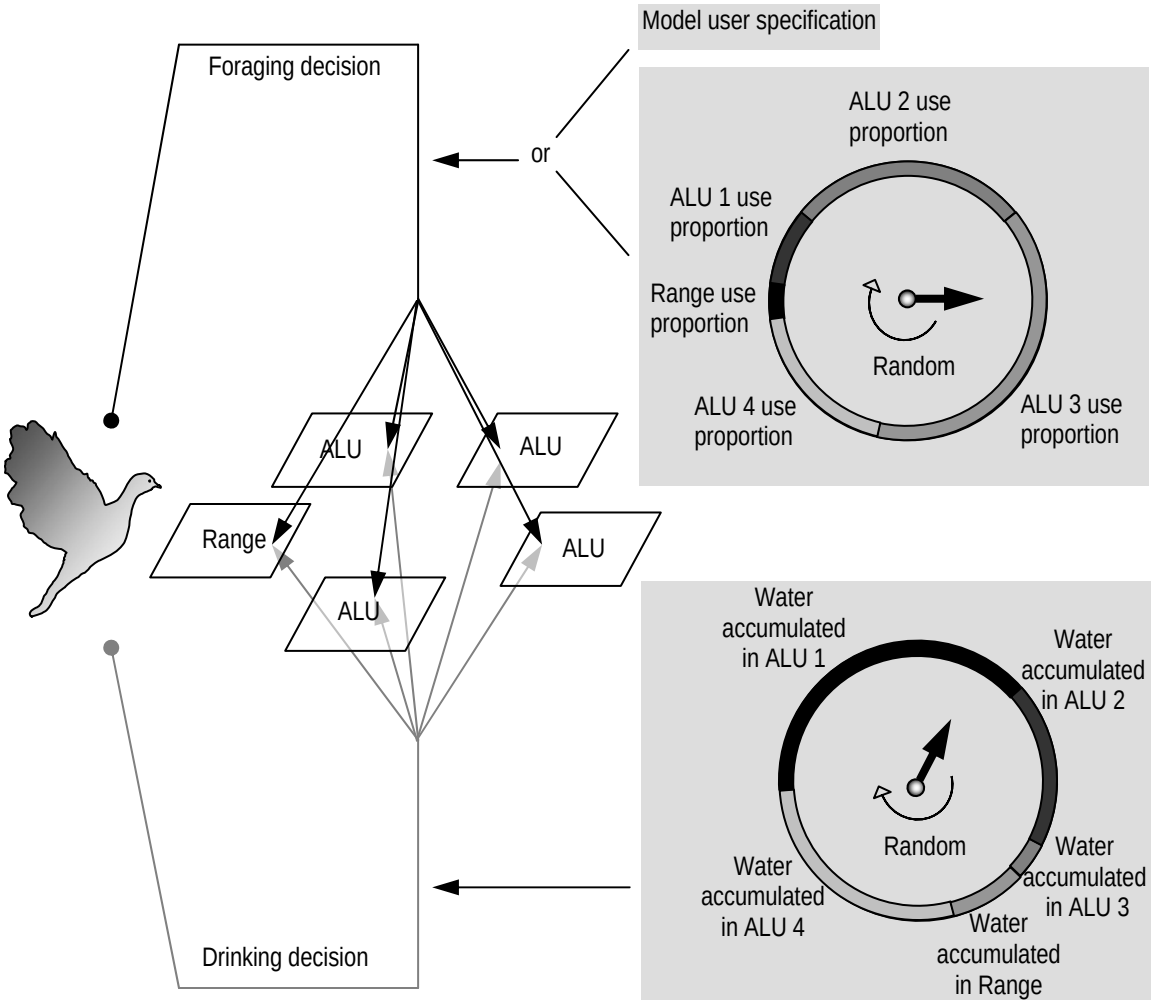

Fig.10. Spatial foraging and drinking rules. The place where a bird drinks depends upon where it is foraging. Where the bird forages can be specified by the model user, or the bird can chose an ALU randomly, conditioned on its ALU use proportions. The bird will drink in the ALU where it is foraging if the ALU is flooded. Alternatively, the bird will choose an ALU randomly, with the probability conditioned on the amount of water accumulated in each ALU. The greater the amount of water accumulated in an ALU, the higher the probability of being chosen.

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., 1998c; Mineau, 2002; Burkepile et al., 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 (Fig. 11). It is assumed that the duration of these bouts is equal to, or shorter than, one hour. 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. 12).

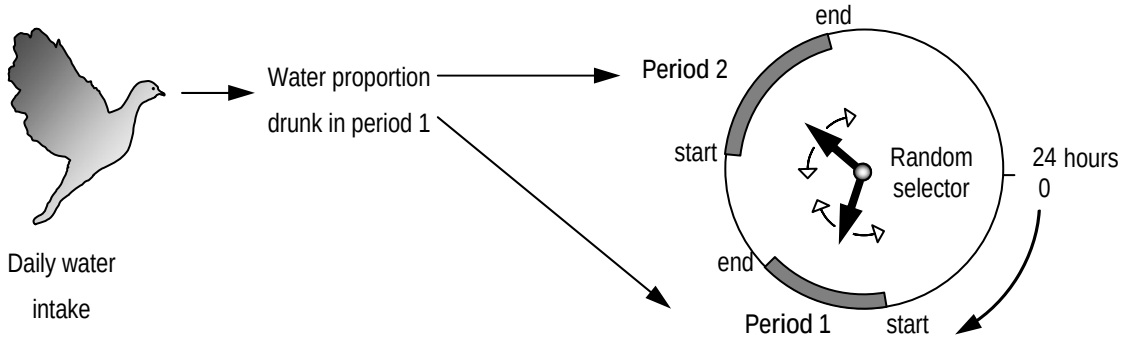

Fig. 11. Time drinking rules. The model allows two periods each day for the bird to forage and drink. It is assumed that one hour is enough for a bird to satisfy its daily water needs. Within each period, the time at which the bird drinks is chosen randomly. The model user has to specify starting and ending times of each period, daily water intake, and the proportion of the daily water intake drunk during the first period.

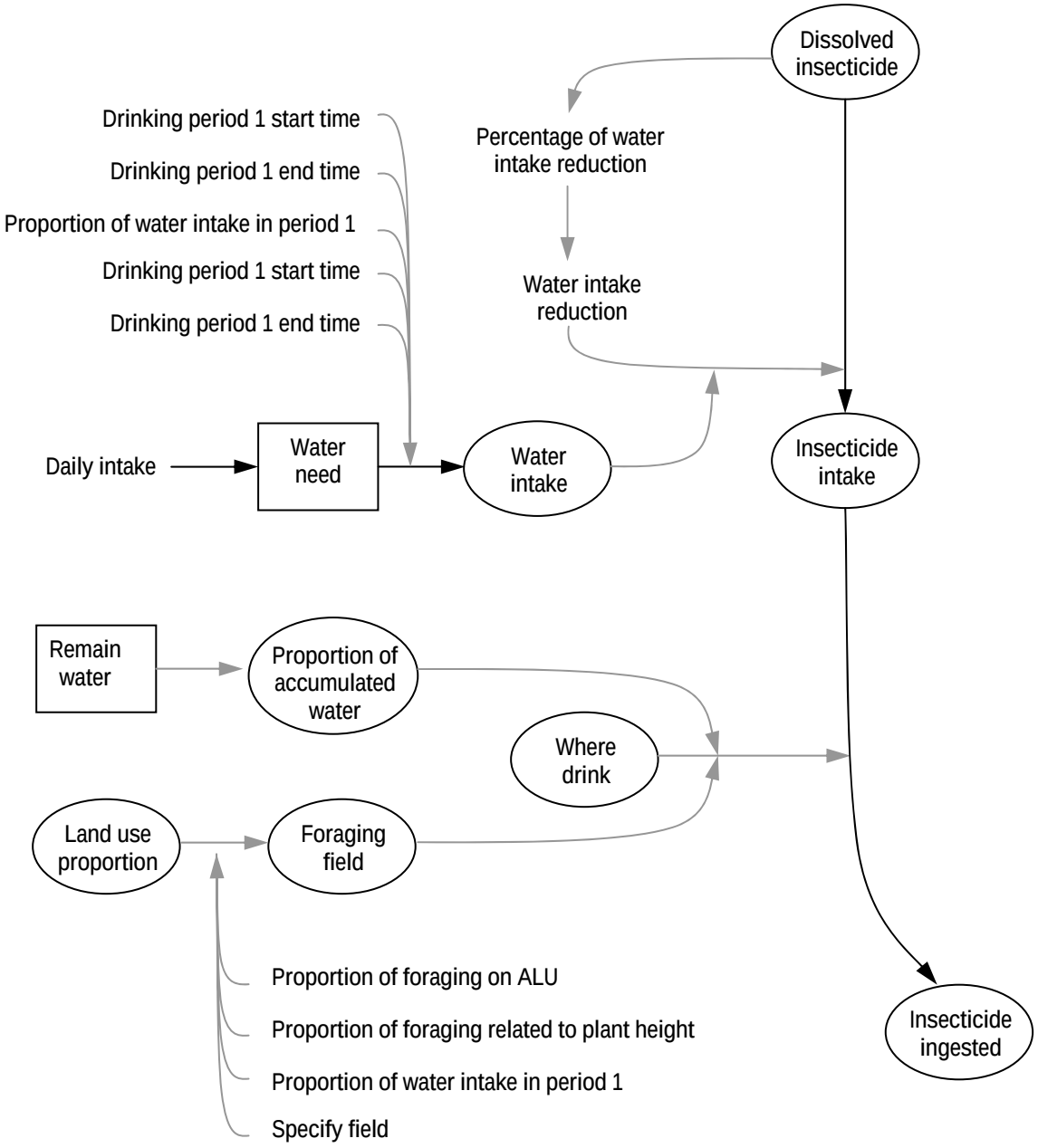

Fig. 12. Conceptual model representing animal contamination with insecticide. Gray and black arrows represent information and mass flows, respectively. Drinking water needs and the amount of insecticide dissolved determine the potential insecticide intake; the actual insecticide ingested depends on where the animal drinks (Appendix B.5).

2.6.1. Quantitative development

Amount of insecticide ingested is represented by *IIN* in μg hr^{-1}.

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

$$WIN_{t+1} = WIN_t + (WIN1 + WIN2 - WIR) \times \Delta t \qquad (21)$$

where *WIN* corresponds to water intake measured in g or ml hr^{-1}. See equation 12 for *IRW*. *WIN1* and *WIN2* in g or ml hr^{-1} symbolize water intake during period 1 and 2 respectively. *WIR*, in g or ml hr^{-1}, represents the reduction of water intake after drink 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 a Julian day. Starting and ending times of drinking periods are entered based on a 1-24 hour system.

2.7. Submodel VI - Cholinesterase inhibition and recovery

The ChE inhibition in a bird is related to load the of 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. 13). A portion of OPs is activated to oxon-form (toxic form of organophosphates) via desulfuration by mono-oxygenases, P450-dependent or flavin-containing. Part of the oxon form is inactivated or degradated by "B" esterases or "A" esterases, mono-oxigenases and glutation tranferases, respectively (Sultatos, 1987; Thompson et al., 1991; Parker and Goldstein, 2000). The remaing 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

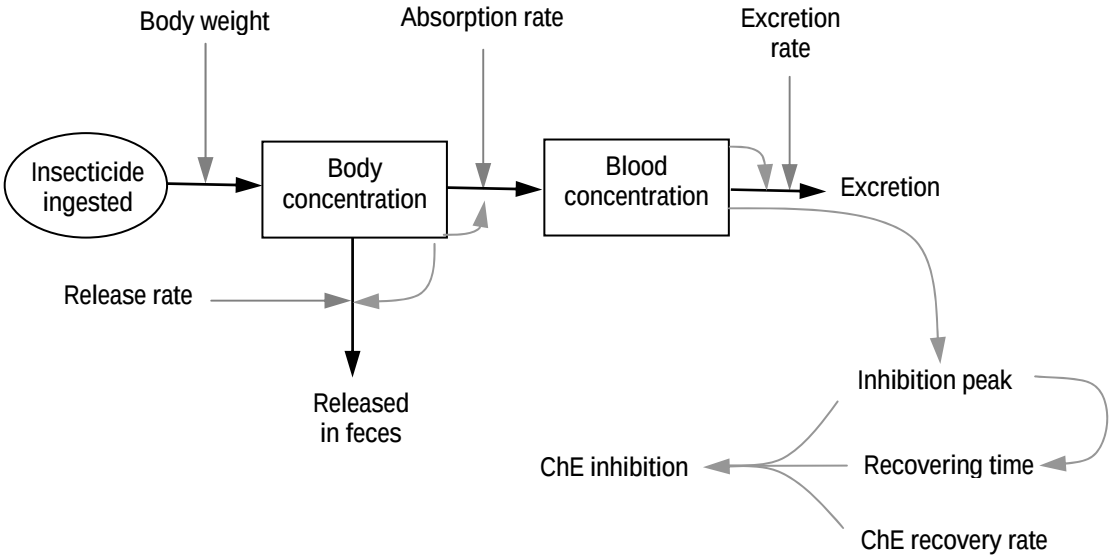

Fig. 13. Conceptual model representing insecticide ingested and ChE inhibition. Black arrows represent the flow of insecticide residue and byproducts of their metabolization. Gray arrows represent information flows. The concentration of insecticide in the body of the animal depends on the insecticide ingested and its body weight. Part of the insecticide ingested is released in feces. The remaining portion is absorbed into the bloodstream. The insecticide concentration in blood is a balance between the absorption and excretion. Metabolic byproducts are excreted mainly in urine. The appearance of insecticide in the blood stream triggers a peak of ChE inhibition, the magnitude of which is related to insecticide blood concentration. Level of ChE inhibition begins to decrease immediately, at the specified recovery rate (Appendix B.6).

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 $Y = a + b \times (logX)$, which is supported by evidence obtained by other authors cited 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 the 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 insecticides 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., 1998b; 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).

I use the equation $Y = a + b \times (logX)$ 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 *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:

$$IDS_{t+1} = IDS_t + ((IIN_t / bw) \times (1 - ife/100)) \times \Delta t \quad (22)$$

$$IBT_{t+1} = IBT_t + (IDS_t - iex_t) \times \Delta t \quad (23)$$

$$Iex_t = IBT_t \times (1- EXP(- (LOGN(2) / T_{1/2a}))) \quad (24)$$

$$ChE = f(IBT_{t+1}) \quad (25)$$

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 equation 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, I 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). I 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 are not ChE-inhibiting pesticides, therefore were not included in the model.

In the following sections, I first provide pertinent background information on WWDO, irrigated agriculture in the LRGV, and characteristics of AM and MP. I then describe parameterization and use of the model to simulate part of the field experiment

of Custer and Mitchell (1987). Finally I 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 Jr. and Harrs, 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 average 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).

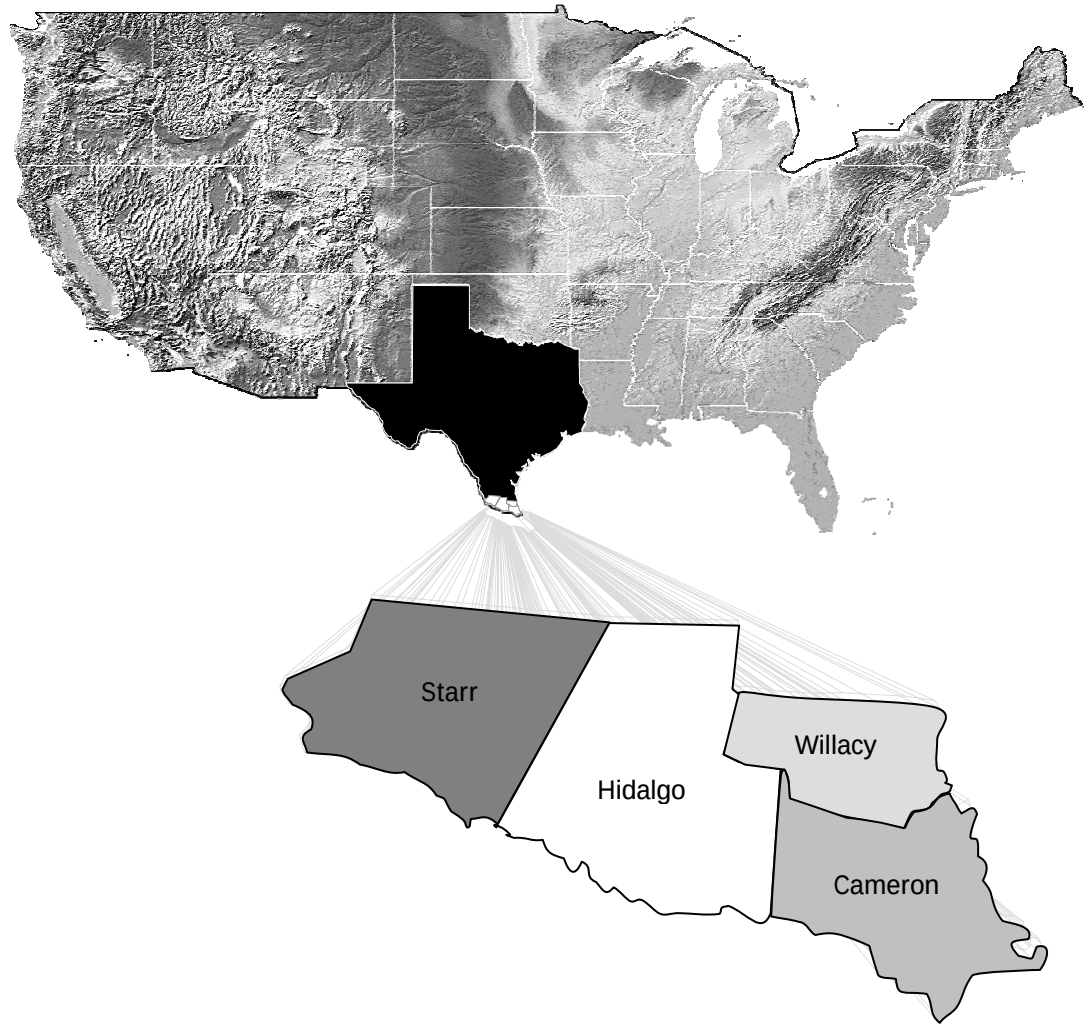

Fig. 14. Location of the Lower Rio Grande Valley. This region comprises the southeastern Texas counties of Starr, Hidalgo, Willacy, and Cameron.

3.2. Irrigated agriculture in the LRGV

The LRGV is a region of about 11,125 square kilometers that extends 100 miles upstream from the mouth of the Rio Grande at the Gulf of Mexico. It comprises the southern Texas counties of Starr, Willacy, Hidalgo and Cameron (Vigness and Odintz, 2004)(Fig. 14). 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 1,307 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 hours (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 crops fields via fixed-wing aircraft. I 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 I simulated AM pesticide applications at 8:00 am and, for MP, again at 10:00 am. Every time the field was flooded, birds were forced to drink (satisfy completely their daily water requirement) in the cotton field at 9:00 am. Runoff was set at 10 %. Because canopy cover changes over time, the amount of insecticide that comes from runoff and accumulates on the ground

(floodable and non floodable area) is a function of plant cover changes. Typical seasonal changes in the canopy cover of cotton in the LRGV are shown in Figure 15.

To parameterize infiltration and evaporation rates, I 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:

0.8 × peak Class A pan evaporation × floodable area / 100 (26)

The *peak class A pan evaporation* occurs in July and equals 6.35 mm per day (Fipps, 2004). I 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, I drew upon experimental data reported by Small et al. (1998)(Appendix A.1). 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 3), and also on productivity and reproductive behavior. Based on

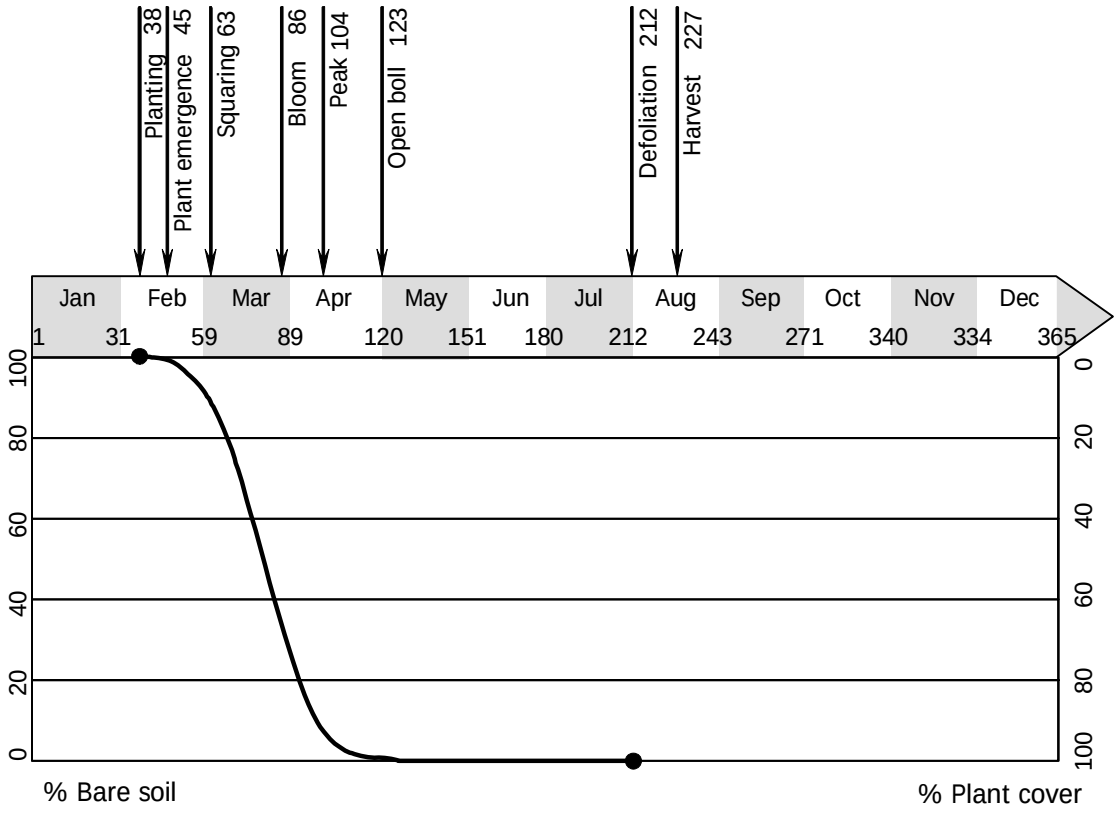

Fig. 15. Phenological stages and change in the percentage of plant cover of a cotton field in the LRGV. Data from Norman (2003) and Norman (personal communication). The percentage of plant cover was estimated by multiplying the average width of plants times 100 meters (length of a row) times 103 rows per hectare (each row is 0.96 m wide). The change in percentage of plant cover (y) over time (x [days]) was represented as: $\boldsymbol{y = a / (1 + b \times exp^{(-c \times x)})}$; where $\boldsymbol{a}$ = 100.18, $\boldsymbol{b}$ = 134.86, and $\boldsymbol{c}$ = 0.126.

Table 3.

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. Data from Small et al. (1998).

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

Numbers in brackets are standard deviations.

these data, I estimated the relation between MP dose per gram of body weight (BW)(assuming BW = 153 g) and ChE inhibition by linear regression $Y = a + b \times X$; where Y is the percentage of ChE inhibition and X is the MP dose (µg gBW^{-1}). The resulting equation was:

$$Y = -2.121 + 90.91X \quad (27)$$

To my knowledge, there are no data relating ChE inhibition in WWDO to AM concentration in drinking water. Thus I 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 libia*). They found that MP oxon is 48.26 times stronger as an inhibitor of brain ChE than AM oxon (Fig. 16). Based on the relatively close phylogenetic relationship between WWDO and pigeon, I assumed that they have similar activation and detoxification metabolic pathways to both AM and MP oxon metabolites. Based on this assumption, I corrected the MP dose-response curve (equation 26) to estimate a dose-response curve for AM:

$$Y = (-2.121 + 90.91 \times X) / (48.26 \times 1.21) \quad (28)$$

where Y is the percentage of ChE inhibition and X is the AM dose (µg gBW^{-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})(Fig. 16). This correction standardizes the effect of molecular weight on application rates based on the g of active ingredient per ha.

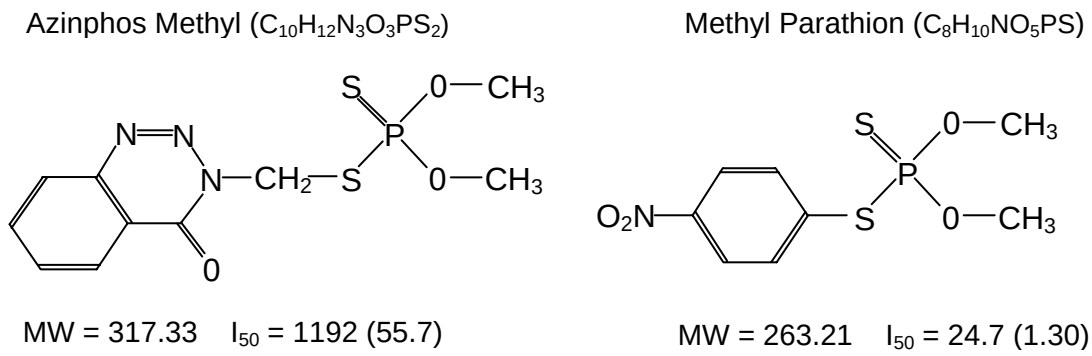

Fig. 16. Molecular formulae, molecular weight, and I_{50} of azinphos methyl and methyl parathion. I_{50} represents the mean (n = 6) concentration of oxon in nmol g^{-1} of wet brain tissue required for inhibiting 50% of ChE activity. Standard errors are shown in parentheses (Thompson et al., 1995).

3.4.2. Simulation results

The highest accumulations of residue in the floodable area were 15.46 g of a.i. ha^{-1} for AM and 61.51 g of a.i. ha^{-1} for MP (Fig. 17), 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. 18). 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).

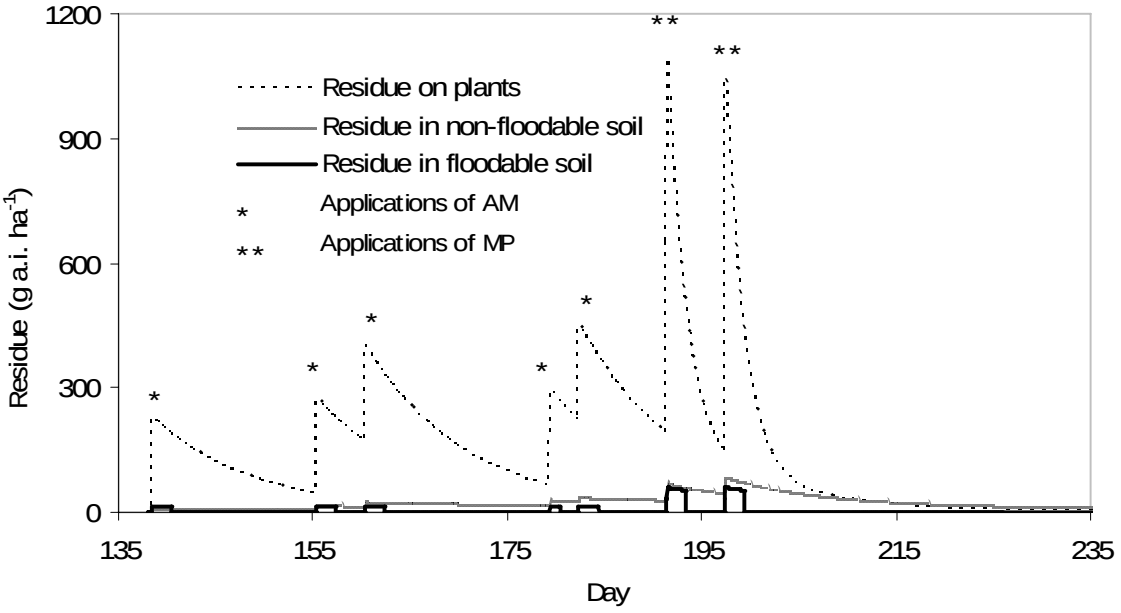

Fig. 17. Simulated amount of methyl parathion and azinphos methyl residue accumulated on plants, in the non-floodable area, and in the floodable area of a cotton field. Irrigations of 115 mm were simulated 24 hours after each azinphos methyl application, and 24 hours after the first and third applications of methyl parathion.

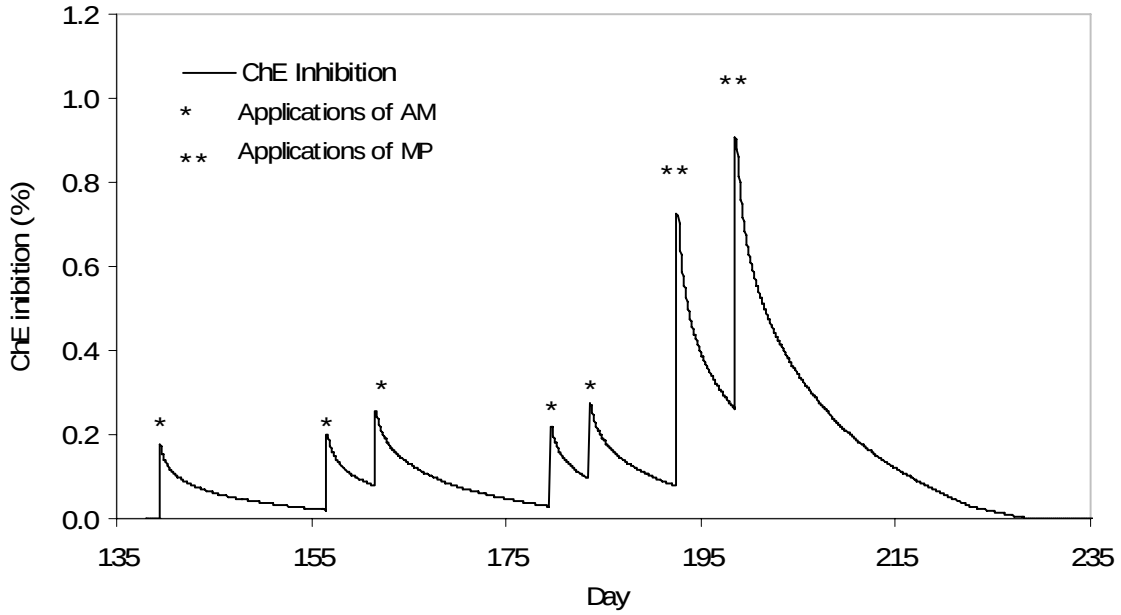

Fig. 18. Simulated brain ChE inhibition in a white-winged dove that drank water from an irrigated cotton field treated with methyl parathion and azinphos methyl. Irrigations of 115 mm were simulated 24 hours after each azinphos methyl application, and 24 hours after the first and third applications of methyl parathion.

3.5. Simulation of a "worst case" scenario

I 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, I 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 I simulated a rainfall event of 15 mm in place of the last 115 mm irrigation. I 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. Levels 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 4). 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.

Table 4.

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

4. Discussion and conclusion

Custer and Mitchell (1987) did not find WWDO with inhibited ChE activity after 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).

The present simulation model should be a useful tool to predict the effect of OPs and CAs on the ChE activity of different species that drink contaminated water from irrigated agricultural fields. It 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., 1995a; 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, I 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.

CHAPTER III

EXPOSURE OF WHITE-WINGED DOVES IN THE LOWER RIO GRANDE VALLEY OF TEXAS TO CHOLINESTERASE-INHIBITING PESTICIDES

1. Introduction

The Lower Rio Grande Valley (LRGV) of Texas has an intensive agricultural activity. Even though an integrated pest management (IPM) program is being achieved in the region (Bohmfalk et al., 1999; Parker et al., 1999; Anciso et al., 2002; Norman, 2003), crops are currently treated with moderate to high rates of insecticides. In 1997, 92.5% of the insecticides applied in Texas belonged to the group of cholinesterase-inhibiting pesticides that comprise Organophosphates (OPs) and Carbamates (CAs)(Gianessi and Silvers, 2000)(Table 5). Approximately 35% of the cropland of the region is under irrigation (The Texas Water Development Board, 2004)(Table 6). Information about how contaminated water from irrigated fields may impact wildlife that drink that water is scarce (Small et al., 1998a). For example, Tacha et al. (1994) found that white-winged dove (WWDO - *Zenaida asiatica*) had been exposed to cholinesterase-inhibiting pesticides and hypothesized that the contamination of irrigation water with pesticides has been one of the causes of decline of rural WWDO populations in the Lower Rio Grande Valley of Texas (LRGV). However, to my knowledge, there are no field data verifying that the cause of cholinesterase (ChE) deprivation in WWDO is the ingestion of OP and/or CA residues dissolved in drinking water.

Table 5.

Amount of active ingredient (a.i.) in tons and percentage of different types of insecticides applied in Texas, USA in 1997 (NCFAP, 2003).

Insecticide type	Tons a.i. applied	% by type	Insecticide type	Tons a.i. applied	% by type
Organophosphates	4619.49	67.1	Synthetic pyrethroids	147.74	2.1
Acephate	117.77		Bifenthrin	24.62	
Azinphos-methyl	152.96		Cyfluthrin	4.19	
Chlorpyrifos	600.9		Cypermethrin	14.52	
Diazinon	47.95		Deltamethrin	7.83	
Dicrotophos	75.19		Esfenvalerate	11.09	
Dimethoate	262.59		Lambdacyhalothrin	13.33	
Disulfoton	53.95		Fenpropathrin	0.05	
Ethion	3.75		Permethrin	66.89	
Ethoprop	31.82		Tralomethrin	5.22	
Ethyl-parathion	95.08		Sulfites	144.18	2.1
Malathion	1649.33		Propargite	144.18	
Methamidophos	6.19		Cyclodienes	141.35	2.1
Methyl-parathion	360.26		Endosulfan	141.35	
Methidathion	4.5		Insect growth regulators	34.38	0.5
Oxidemeton-methyl	0.71		Cyromazine	0.35	
Phorate	159.22		Tebufenozide	34.03	
Phosmet	40.3		Organotins	20.98	0.3
Profenofos	216.17		Fenbutatin oxide	20.98	
Terbufos	740.85		Chlorinateds	20.61	0.3
Carbamates	1747.29	25.4	Chloropicrin	4.91	
Aldicarb	338.13		Dicofol	14.12	
Carbaryl	413.84		Lindane	1.58	
Carbofuran	207.65		Nitroguanidines	8.32	0.1
Methomyl	52.01		Imidacloprid	8.32	
Oxamyl	181.52		Antibiotics	0.17	0.0
Thiobencarb	319.97		Abamectin	0.1	
Thiodicarb	208.99		Spinosad	0.07	
Ziram	25.18				

Table 6.

Area of irrigated crops (ha) in the Lower Rio Grande Valley (LRGV) for the year 2003. (The Texas Water Development Board, 2004).

Crop	County				LRGV
	Cameron	Hidalgo	Starr	Willacy	
Sorghum	21,885	17,803	673	2,463	42,823
Cotton	11,243	15,592	1,058	2,215	30,108
Veg(deep)	507	21,572	834	279	23,193
Veg(sha)	509	20,036	452	113	21,109
Sugarcane	6,409	7,583	0	2,746	16,739
Corn	3,839	4,133	0	321	8,292
Hay-pasture	4,159	2,408	75	866	7,508
Citrus	1,860	1,675	0	142	3,677
Others	5,836	8,585	485	519	15,425
Total by county	56,248	99,386	3,577	9,663	168,874

In this Chapter, I use the simulation model described in Chapter II to estimate the effect of OPs and CAs dissolved in irrigation water on the activity of brain cholinesterase of WWDO in the LRGV. I first present background information on WWDO in the LRGV, ChE-inhibiting insecticides, and agriculture in the LRGV. I then present a brief overview of the simulation model. Finally, I describe parameterization and use of the model to simulate the impact of OPs and CAs on WWDO in the LRGV.

2. Background information

2.1. White-winged doves in the Lower Rio Grande Valley

WWDO is considered one of the most important game birds of the southeastern United States. Roughly $200 million are generated each fall in Texas as revenue of the activity of about 460,000 dove hunters (George, 2004). Currently there are two types of populations of WWDO; rural populations which have been nesting historically in the LRGV, and urban populations which have been spreading northward since the mid 1970's, probably attracted by bird feeders, water, and urban forestation for nesting. In this Chapter, I focus on the rural populations which are more at risk of being affected by agricultural pesticides.

WWDOs arrive in the LRGV from wintering areas in Mexico and Central America. Normally, WWDOs nest in thick native brush and citrus groves. The rural population of WWDO increased abruptly in the early 1900's, reaching a peak of more than 4 million individuals in 1923 (Marsh and Saunders, 1942). This increase was attributed to the introduction of irrigation and grain farming at the beginning of the 20th century (George

et al., 1994). Subsecuently, the number of WWDOs decreased severely until the late 1980's to about 300,000 individuals (George et al., 1994). Seemingly there has been a slow recovery during the last decade (Schwertner et al., 2003)(Fig. 19), but recent estimates of mortality and survival rates of Texas WWDO are not available (Martinez et al., 2003; George, 2004).

Reduction of WWDO density has been attributed to several causes: loss of natural breeding areas, over hunting, egg predation by grackles, low quality of the available food, and effect of pesticides. About 95% of the natural breeding habitat of the species has been lost due to human disturbances, such as agricultural, industrial, and urban development (Marsh and Saunders, 1942; Cottam and Trefethen, 1968; Brown et al., 1977). However, WWDO populations appear to have shifted their nesting areas from the lost brushland to citrus groves. In 1950, almost 80% of WWDOs were nesting in citrus trees. Severe freezes in 1951, 1962, 1983 and 1989 dramatically affected citrus plantations. Thus, it has been hypothesized that the damage to citrus trees has been responsible for the WWDO population decline (Cottam and Trefethen, 1968; Swanson and Rappole, 1993). Under the rationale that, if nesting habitat is limiting population growth, a dense-dependent factor should be involved, Swanson and Rappole (1993) determined the effects of intra-specific competition for nesting territories in breeding populations of WWDO in the LRGV of Texas. They pointed out that native nesting habitat suitable for breeding is being underused, which suggests that processes other than habitat loss might be involved.

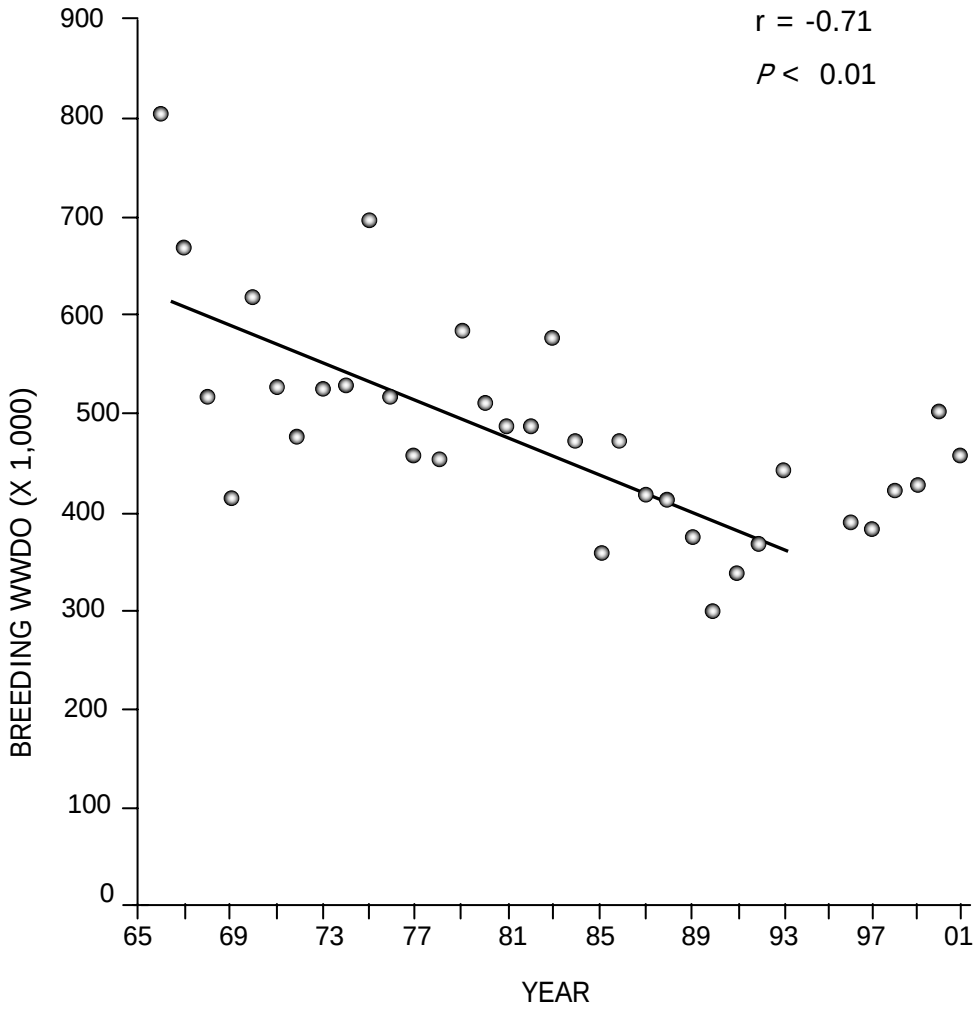

Fig. 19. Temporal changes in the rural breeding populations of white-winged dove (WWDO) in the Lower Rio Grande Valley. Data from George et al. (1994) and Schwertner (2003).

According to Dolton (1975), shortage of preferred high quality natural foods as a consequence of habitat shrinkage could become a limiting factor in WWDO population growth. However, studies carried out by Schacht et al. (1995) showed that nesting populations of WWDO in the LRGV were not limited by the availability of high quality foods. Sorghum and sunflower grains replaced the high quality items of the diet of doves feeding in natural woodlands.

Grackle (*Quiscalus mexicanus*) predation is another factor that may affect WWDO populations (Kiel Jr. and Harrs, 1956; Blankinship, 1996). Blankinship (1996) found that grackle density reduction locally increased WWDO fledging; however the density of grackles in the LRGV did not increase sufficiently during the past century to be considered an important factor in the WWDO decline (Hayslette et al., 1996).

Overhunting has been hypothesized as another factor involved in the WWDO decline (Marsh and Saunders, 1942; Kiel Jr. and Harrs, 1956). According to Brown et al. (1977), harvest during hunting season should not exceeded 25% of the breeding population. During the 1960's, the number of individuals killed exceeded the breeding population (Hayslette et al., 1996). Determination of bag-limits and length of the hunting season each year depends on population size and number of birds harvested. The breeding population is estimated using call-count surveys. This method is based on the premise that each calling male represents a breeding pair (Rappole and Waggerman, 1986). Often the call-count method overestimates the number of breeding birds because of the louder calling of unpaired males, calling females, hearing subjectivity of the people performing the survey, and clumped distribution of nesting areas (West et al., 1998). However, it can

be an appropriate method to estimate nesting pairs in areas with high levels of nests (West et al., 1998). Harvest surveys may underestimate the actual number of birds killed due to unretrieved kills. Reported unretrieved loss of birds has reached values greater than 50% of bagged birds during some years (Kiel Jr. and Harrs, 1956). Martinez et al. (2003) simulated the annual productivity and long-term population trends of WWDO in the Tamaulipan Biotic Province. Their model was parameterized using information synthesized from decades of field data on WWDO. They could not generate a stable long-term population trend with the model parameterized based on suggested sustainable harvest rates and empirically-based estimates of migratory return rates. They suggested that more studies that produce unbiased estimates of nest success and the proportions of migrant adults and juveniles that return annually are necessary. Also they pointed out that new hypotheses regarding factors limiting WWDO density should be considered.

Purdy (1983) hypothesized that pesticide use in LRGV could be one of the factors implicated in the WWDO population decline. There is evidence that WWDO have been exposed to pesticides used in agricultural fields (Tacha et al., 1994), and Tacha et al. (1994) hypothesized that WWDO are exposed to anticholinesterase compounds by ingesting contaminated water from irrigated cotton fields. A prediction derived from the Tacha et al. (1994) hypothesis is that WWDO that drink water in agricultural fields of LRGV are ChE deprived.

2.2. Cholinesterase-inhibiting insecticides

The functioning of several important organs of vertebrates, arthropods and mollusks are controlled by electrical impulses transmitted through nervous fibers. These impulses are stimulated by the neurotranmissor acetylcholine and inhibited by acetylcholinesterase (ChE), an enzyme that breaks down acetylcholine in the synapses between the neurons of nervous fibers. OPs and CAs inhibit the activity of the ChE, producing a continuous firing in the fiber, and, consequently, a non-normal functioning of organs. Animals with sublethal cholinesterase depression show physiological and behavioral disorders that may diminish their ability to survive, reproduce, or adapt to the environment (Grue and Shipley, 1981; Grue et al., 1991; Grue et al., 1997; Bishop et al., 2000a; Bishop et al., 2000d; Solecki et al., 2001b; Burger et al., 2002). A 20% inhibition of brain ChE indicates that an animal has been exposed to ChE inhibiting pesticides, while a 50% inhibition is considered lethal (Ludke et al., 1975; Hill and Fleming, 1982).

Custer and Mitchell (1987) studied the pattern of insecticide use in agricultural fields of LRGV and the level of cholinesterase inhibition in grackles, mourning doves (*Zenaida macroura*), and WWDOs living in brushlands surrounded by those fields. While they found ChE-inhibited grackles and mourning doves, they did not observe ChE-inhibited WWDOs. Tacha et al. (1994) found that during 1991-92 WWDOs from 6 locations in the LRGV had been exposed to anticholinesterase compounds. They hypothesized that OPs ingested with contaminated water from irrigated cotton fields had been responsible for the cholinesterase-depressed birds because: 1) CAs are rarely used in the LRGV, 2) WWDOs spent almost all of their time in brushlands, sunflower,

sorghum, and cotton fields (Schacht et al., 1995), 3) WWDO diets were composed almost exclusively of sorghum and sunflower which are rarely sprayed during the breeding season, 4) WWDOs spent less than 2% of their time in cotton fields, which they only visited for drinking, and 5) cotton fields were regularly sprayed with organophosphate insecticides.

2.3. Agriculture in the Lower Rio Grande Valley

Willacy, Star, Hidalgo, and Cameron counties compose the LRGV of Texas (Vigness and Odintz, 2004)(Fig. 14). Since the building of railroad and irrigation systems at the beginning of the past century, the LRGV has been transformed from a semiarid rangeland to a well developed agricultural region (Chapman et al., 1996). Of the 1,112,659 ha in the LRGV, 38 % are in cropland, and 11% of the cropland is under irrigation. Sorghum, cotton, vegetables, sugarcane, corn, and citrus are the principal crops (Table 2). Ninety percent of the water rights are held by agriculture. The water distribution network comprises about 642 miles of canals, 10 miles of pipelines, and 45 miles of resacas (Fipps and Pope, 1999). Flood irrigation is the most common type of irrigation (Norman, personal communication). Common field size is 13 ha, the predominant furrow length is 366 m, and each irrigation event, or application, is about 115 mm (Falkner and Fipps, 2002).

The climate ranges from subtropical subhumid, characterized by hot summers and dry winters, in the eastern part of the region, to subtropical steppe westward, typified by

semiarid conditions (Larkin and Bomar, 1983). Climate parameters of the region are shown in the climate diagram of the city of McAllen in Figure 20.

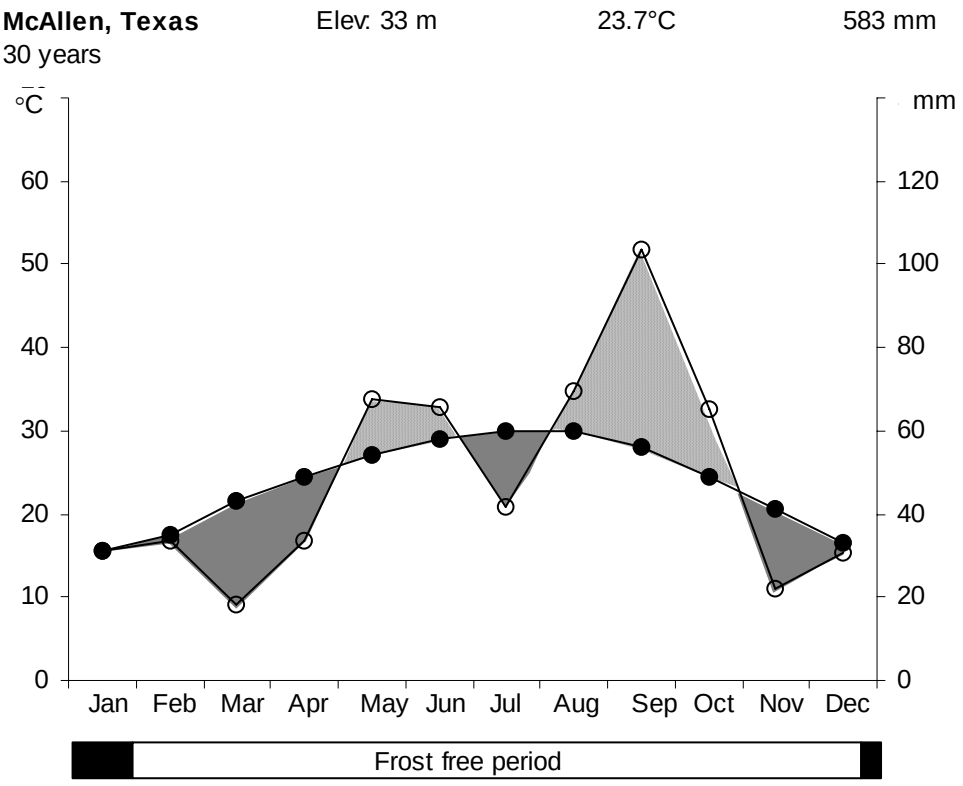

Fig. 20. Climatic diagram of McAllen, Texas. moist and dry periods. Open circle: precipitation, solid circle: temperature. Data from (NOAA - National Weather Service Forecast Office, 2005).

Most of the soils of the LRGV are alfisols of the subgenus ustisols. The pH of these soils ranges between 7.9 and 8.4, and thus they are classified as alkaline. Although their texture varies from sandy loam to heavy clay, these soils are predominantly clays (Thompson et al., 1972; Williams et al., 1977; United States Department of Agriculture Soil Conservation Service, 1981; Jacobs, 1981; Turner, 1982).

2.3.1. Cotton crops

Texas ranks first among U.S. states as a producer of cotton. Three percent of Texas cotton is produced in LRGV, of which 60 % is irrigated. Cotton is the crop that receives the most insecticide applications. The OPs such as methyl parathion, malathion, azinphosmethyl, profenofos, and dicrotophos make up 66% of all insecticides used in Texas cotton, while CAs such as aldicarb, oxamyl, and carbofuran comprise 11 %. On average, there are 1.2 insecticide applications across all planted hectares (Smith and Anisco, 1999). About 37 % of all insecticides applied are used against the boll weevil, of these, azinphos methyl, methyl parathion, and oxamyl are used in 31 %, 22 %, and 11 % of applications, respectively (Table 7). Farmers in some zones are involved with the Boll Weevil (*Anthonomous grandis grandis*) Eradication Program in which several applications at a low application rate of malathion are carried out. Climate and agricultural activities are the principal driving forces that control pest populations. Fleahoppers (*Pseudatomoscelis seriatus*) before bloom, boll weevils during the entire year, and bollworms (*Heliothis zea*) and tobacco budworms (*Heliothis virescens*) after bloom until harvest are the most common pests in cotton fields. There is not a regular

Table 7.

Most common cotton pests, organophosphate (OP) and carbamate (CA) insecticides applied, and application rate in g a.i. ha^{-1} (Norman, 2002).

Insect pest	Insecticide	Insecticide type	Application rate
Fleathooper[1]	Acephate	OP	210.9-280.2
	Chlorpyrifos	OP	213.0-560.4
	Dicrotophos	OP	896.7-1793.2
	Dimethoate	OP	123.3-280.2
	Methyl parathion	OP	112.2
	Oxydemeton-methyl	OP	280.2
	Oxamil	CA	280.2
Boll weevil[2]	Azinphosmethyl	OP	280.2
(overwintered)	Malathion	OP	683.7-1367.5
	Methyl parathion	OP	280.2-560.4
	Oxamil	CA	280.2
Boll weevil (In-	Azinphosmethyl	OP	280.2
season)	Dicrotophos	OP	560.4
	Malathion	OP	1031.2-1367.5
	Methyl parathion	OP	420.3-560.4
	Oxamil	CA	280.2
Cotton aphids[3]	Chlorpyrifos	OP	280.2-1127.5
	Dicrotophos	OP	140.1-280.2
	Dimethoate	OP	140.1-280.2
	Profenofos	OP	560.4
	Methomyl	OP	1127.5
	Methyl parathion	OP	280.2-420.3
	Ethyl parathion	OP	280.2-420.3
Bollworm[4] and	Acephate	OP	1127.5
Tobacco	Methyl parathion	OP	1401.1-2241.7
budworm[5]	Profenofos	OP	560.4
	Thiodicarb	CA	672.6-1008.7

[1] *Pseudatomoscelis seriatus*, [2] *Anthonomous grandis grandis*, [3] *Aphis gossypii*, *Aphis craccivora*, *Myzus persicae*, [4] *Heliothis zea*, [5] *Heliothis virescens.*

application schedule; insecticides are applied if, after a systematic monitoring, it is found that a pest will surpass the economic damage threshold. However, a usual application schedule of a typical year is 1-2 applications to control fleahoopers and overwintered boll weevils, and 1-2 applications after bloom to control boll weevils and bollworms or cutworms. Pre-bloom applications usually are no closer than 10 days before bloom to allow the reestablishment of populations of beneficial insects that will control the outbreak of bollworms and tobacco budworms. These two species are often controlled by natural enemies and weather conditions. The most common pest and pesticides used to control them are shown in Table 7.

Cotton habitually is irrigated 2-3 times during the entire growing season. The maximum water requirement of cotton is from the time the first flowers open (first bloom) until the maximum number of flowers are open (peak bloom). Normally, this coincides with the driest period of the year, therefore, the first and second irrigations usually are applied 3-4 days before and 10 days after first bloom, respectively. The last irrigation is usually applied about 90 days after planting. The amount of water supplied per irrigation is about 100-115 mm (Stichler, personal communication, Norman, personal communication).

2.3.2. Corn crops

Corn earworm (*Heliothis zea*) is the only major pest of corn in the LRGV (Norman, personal communication). Corn earworm moths lay eggs in the recently exposed silks. Because silks of an ear are constantly emerging from the husks during 2-3 days, several applications are needed to treat the unexposed portion of the silks. After hatching, the

larvae move, feeding on silk, towards the apical kernels and begin to feed on them. About 99 % of the corn acreage is not treated because treatments are usually costly and not always effective (Norman, personal communication, Porter et al., 2005). Some suggested insecticides to treat this pest are shown in Table 8.

Depending on soil moisture, corn may be irrigated 1 or 2 days after planting. Then during the growing season corn is usually irrigated 2 times before silking and 2 or 3 times between silking and dent reproductive phenological states. Usually 76 to 100 mm are applied in each irrigation.

Table 8.

Most common corn pests, organophosphate (OP) and carbamate (CA) insecticides applied, and application rate in g a.i. ha^{-1} (Porter et al., 2002).

Insect pest	Insecticide	Insecticide type	Application rate
Earworm[1]	Carbaryl	CA	2241.7-2689.9
	Methomyl	OP	246.6-504.4
	Ethyl parathion	OP	560.4
Cutworm[2]	Chlorpyrifos	OP	1120.8-1681.3
Flee beetles[3]	Carbaryl	CA	1345
	Methyl parathion	OP	134.4

[1] *Heliothis zea*, [2] *Agrotis* and *Euxoa spp.*, [3] *Chaetocnema pulicaria*

2.3.3. Sorghum crops

Greenbugs (*Schizaphis graminum*) and sorghum midgets (*Stenodiplosis sorghicola*) are the most common pests of sorghum. They are present in almost all fields, even in those with different management history. All other pests are occasional (Cronholm et al., 1998b) (Table 9).

Sorghum plants have a fast growth stage between 40 and 65 days after planting. This is the period when plants have the highest water requirements. Water stress during this period seriously affects grain production (Stichler et al., 1997; Rogers and Alam, 1998; Stichler and Fipps, 2003). Although the amount of irrigation water needed depends on the season and the amount of soil water stored in the root zone, about 530 mm ensures a growing season without stress. This amount is normally scheduled as 200, 100, 78, 76, and 76 mm the days 2, 30, 50, 60, and 90 after planting, respectively (Stichler and Fipps, 2003).

2.3.4. Citrus orchard

Almost all Texas citrus is produced in the LRGV. Texas is among the top ten world citrus producers. The production of grapefruit in 1999 was 241,000 Ton (Anciso et al., 2002). The most common insect pests are citrus rust mite (*Phyllocoptruta oleivora*), California red scale (*Aonidiella aurantii*), and Florida red scale (*Chrysomphalus aonidum*). Citrus rust mites and other species of mites damage leaves causing defoliation. Although this damage in vigorous trees is seldom important, they may affect fruit rind, size, and appearance of fruits. Infestation of citrus mites increases after the

Table 9.

Most common sorghum pests, organophosphate (OP) and carbamate (CA) insecticides applied, and application rate in g a.i. ha^{-1} (Cronholm et al., 1998a).

Insect pest	Insecticide	Insecticide type	Application rate
Greenbug[1]	Carbofuran	CA	340.2-453.6
	Chlorpyrifos	OP	113.4-453.6
	Dimethoate	OP	113.4-226.8
	Disulfoton	OP	113.4-226.8
	Malathion	OP	557
	Ethyl parathion	OP	113.4-226.8
Sorghum	Chlorpyrifos	OP	280.2
Midge[2]	Malathion	OP	672.5-1008.8
	Methomyl	OP	246.6-504.4
	Ethyl parathion	OP	560.4
Yellow	Carbofuran	CA	560.4-1120.8
sugar	Dimethoate	OP	560.4-1120.9
cane aphids[3]	Disulfoton	OP	560.4-1120.10
	Ethyl parathion	OP	1120.9

[1] *Schizaphis graminum*, [2] *Stenodiplosis sorghicola*, [3] *Sipha flava.*

heaviest rains (May/June, August/September), reaching thresholds at which chemical treatment must be used. Natural enemies usually cannot control citrus mites under the economic damage threshold. Generally, most of the damage occurs from bloom to November, when winter conditions are no longer favorable for population outbreaks (Anciso et al., 2002). California (*Aonidiella aurantii*), Florida (*Chrysomphalus aonidum*), and other scales normally become dangerous when the populations of their natural enemies are disrupted by chemical treatments on other pests. Oil application is an effective tool to treat heavy scale infestations and also is safe for their natural enemies (Smith et al., 1997; Anciso et al., 2002). Table 10 shows the common OP and CA insecticides used to treat mite infestations.

Citrus trees in LRGV have a minimum requirement of about 1140 to 1270 mm of available soil moisture per year. Because normal rain in the region ranges from 432 to 610 mm, about 635 mm of water has to be supplemented by irrigation. Fruit quality and quantity may be seriously affected if water stress occurs from January to June, thus water usually is applied when soil moisture depletion is about 40-50 %. Normally about 5 irrigations of 127 mm are carried out annually from early February to November (Anciso, personal communication; Sauls, 2002a; Sauls, 2002b).

Table 10.

Most common citrus pests, organophosphate (OP) and carbamate (CA) insecticides applied, and application rate in g a.i. ha^{-1} (Smith et al., 1997).

Insect pest	Insecticide	Insecticide type	Application rate
Citrus rust mite[1]	Formetanate	CA	313.8-627.6
	Ethion	OP	2241.7-3362.6
	Azinphos methyl	OP	1120.8-1681.2
	Chlorpyrifos	OP	1120.8-3923.0
	Oxamyl	CA	175.0-700.5
Texas citrus mite[2]	Azinphos methyl	OP	1120.8-1681.2
Citrus red mite[3]	Ethion	OP	2241.7-3362.6
	Methidathion	OP	700.5-1401.1
	Carbaryl	CA	2241.7-2689.9

[1] *Phyllocoptruta oleivora*, [2] *Eutetranychus banksi*, [3] *Panonychus citri.*

3. Overview of the simulation model

I developed the model as a compartment model, based on difference equations (Δt = 1 hour) and programmed with Stella® VII software (High Performance Systems, Inc., New Hampshire, USA), designed to simulate the level of ChE inhibition in an animal that drinks water in an agricultural system composed of different irrigated crops and rangeland (Fig. 1). Insecticides applied to crop fields accumulate in 3 compartments: plants, floodable areas, and non-floodable areas of the field. Change in plant cover throughout the growing season affects the amount of insecticides that falls directly on

those compartments. The degradation of the insecticides follows a first-order degradation curve which is a function of their half-lives. During irrigation events or rainfall, insecticide residues accumulate in floodable areas (e.g. furrows, and basins) and dissolve in water, which is used by animals for drinking. When a rainfall occurs, remnant insecticides on plants and on non-floodable beds are washed off to floodable areas. ChE inhibition is estimated as a dose-response function of the amount of insecticide load in blood after being ingested with drinking water. Factors and processes such as insecticide application rate, drift, crop types, insecticide degradation rates, seasonal plant cover changes, irrigation and rain water accumulation and disappearance, insecticide dissolution, behavioral foraging and drinking rules, insecticide intake, insecticide excretion and release in feces, the relationship between concentration of insecticide in the blood and ChE inhibition, and ChE activity recovery all are represented in the model.

4. Simulating the impact of OPs and CAs on WWDO in the LRGV

4.1. Model parameterization

I parameterized the model to represent a system composed of fields of cotton, sorghum, corn, citrus, and brushland that encompases the activity range of WWDO in the LRGV, which is an area of the approximately 250 ha. Parameterization of the model required information on application rates, application schedules, and washoff characteristics of the five insecticides used, irrigation rates and irrigation schedules of

the four crops planted, proportion of time WWDO spent foraging in each of these crops, and dose-response curves for insecticide-ChE inhibition.

4.1.1. Insecticide applications

Common insecticide types, application dates, and application rates used in the LRGV were applied as shown in Table 11. Simulated insecticide applications were applied at 8:00 am; insecticides are usually applied in the early morning and in the evening to avoid drift, caused by strong afternoon wind, and because the pollinating and other beneficial insects are mostly inactive during these hours (Norman, personal communication). Insecticide solubility in water, percentage of washoff of insecticide residues on canopy after a rain of 13 mm, and half-lives of insecticide applied in plants, soil, water, and bird body are shown in Table 12. Cotton and sorghum fields typically are sprayed with ground sprayers, while citrus orchards are sprayed with airblast sprayers; consequently, drift was set at 0.5 % for simulated applications to cotton and sorghum fields and 4 % for simulated applications to citrus orchards.

4.1.2. Irrigation schedules

Irrigations were simulated at dates and rates typically used in LRGV. Floodable area for cotton, corn, and sorghum fields was set at 60 %, and for citrus orchards was set at 90 %. Soil infiltration rate was 7.62 mm h^{-1} and evaporation rates were 1585 l h^{-1} ha^{-1} for cotton, corn, and sorghum fields, and 2378 l h^{-1} ha^{-1} for citrus fields.

Table 11.

Dates and rates of insecticide and irrigation applications in cotton, corn, sorghum, and citrus fields used in a simulated agricultural scenario in the Lower Rio Grande Valley.

Insecticide applications			Julian day	Irrigation	
Chemical	g a.i. ha^{-1}	Crop		mm	Crop
			52	203	Sorghum
			56	127	Citrus
Oxamyl	280	Cotton	70		
			74	100	Corn
			74	120	Sorghum
Oxamyl	280	Cotton	75		
Dimethoate	1120	Sorghum	76		
			80	100	Cotton
			92	100	Cotton
			93	76	Sorghum
			104	76	Sorghum
			105	100	Corn
Oxamyl	700	Citrus	106		
			110	127	Citrus
Azinphos methyl	280	Cotton	110		
Methyl parathion	1400	Cotton	115		
			122	100	Corn
			127	100	Cotton
Chlorpyrifos	543	Sorghum	131		
			135	100	Corn
			135	76	Sorghum
			144	127	Citrus
			145	100	Corn
Azinphos methyl	1680	Citrus	177		
			213	127	Citrus
			317	127	Citrus

Table 12.

Insecticide solubility in water, percentage of washoff of insecticide residues on canopy after a rain of 13 mm, and half-lives of insecticide applied in plants, soil, water, and bird body.

Insecticide	Type	Half-lives (h)				Solubility in	Washoff
		Plants	Soil	Water	Birds	water (mg l^{-1})	%
Azinphos methyl	OP	172.8[1]	770.0[1]	624[1]	10.3[5]	30	65
Chlorpyrifos	OP	79.2[2]	720.0[2]	1,728[7]	24.0[3]	2	65
Dimethoate	OP	72.0[2]	118.0[2]	192[3]	12.0[3]	25,000	95
Methyl parathion	OP	31.2[2]	135.0[2]	600[6]	3.6[3]	55	90
Oxamyl	CA	96.0[2]	96.0[2]	48[3]	13.8[4]	280,000	95

[1] (US EPA - Environmental Protection Agency, 1998a), [2] (Knisel and Davis, 2000), [3] (Extoxnet, 2005), [4] (Harvey and Hanh, 1978), [5] (Kidd and James, 1991), [6] (US EPA - Environmental Protection Agency, 1998b), [7] (Racke, 1992)

4.1.3. Crop use by WWDO

Planting days for cotton, corn and sorghum were 40, 20, and 59, respectively. Phenological stages and changes in canopy cover of these crops are shown in Figures 15, 21, and 22. The proportions of time that WWDO spent in cotton, corn, sorghum, citrus, and range, were 0.02, 0.08, 0.13, 0.70, and 0.07, respectively. WWDO usually nest in citrus orchards because there are few remnants of natural vegetation in the LRGV. Simulated males foraged from 9:00 to 11:00 and from 17:00 to 19:00 hours, while simulated females foraged from 12:00 to 16:00 hours. Assuming an average weight of 153 g (Zammuto, 1986), WWDO have a daily water intake requirement of approximately 29.5 ml d^{-1} (Small et al., 1998d). They are capable of drinking and rehydrating in a very short time (seconds-minutes), normally twice a day (MacMillen and Trost, 1966). Both simulated males and females satisfy their daily water requirements in two drinking bouts.

4.1.4. Dose-response curves

To parameterize the dose-response curves relating the concentrations of insecticides in drinking water to ChE inhibition in WWDO, I drew upon experimental data from several sources. Small et al. (1998) 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 3)(Appendix A.1), and also on productivity and reproductive behavior. Based on these data, I estimated the relation between MP dose per gram of BW (assuming BW = 153 g) and ChE inhibition by linear regression $Y = a + b \times X$, where Y is the percentage of ChE inhibition and X is the MP dose (μg gBW^{-1} day^{-1}).

$Y = -2.121 + 90.91\ X \quad (r = 0.91 \quad P = 0.03)$

To my knowledge, there are no data relating ChE inhibition in WWDO to AM, Chlorpyrifos (CH), Dimethoate (DI), and Oxamyl (OX) concentrations in food or drinking water. Thus, I used available data from studies were brain ChE activity of other birds was related to different doses of these insecticides. For example, Thompson et al. (1995) related the activation of organophosphorus pesticides to oxon metabolites and

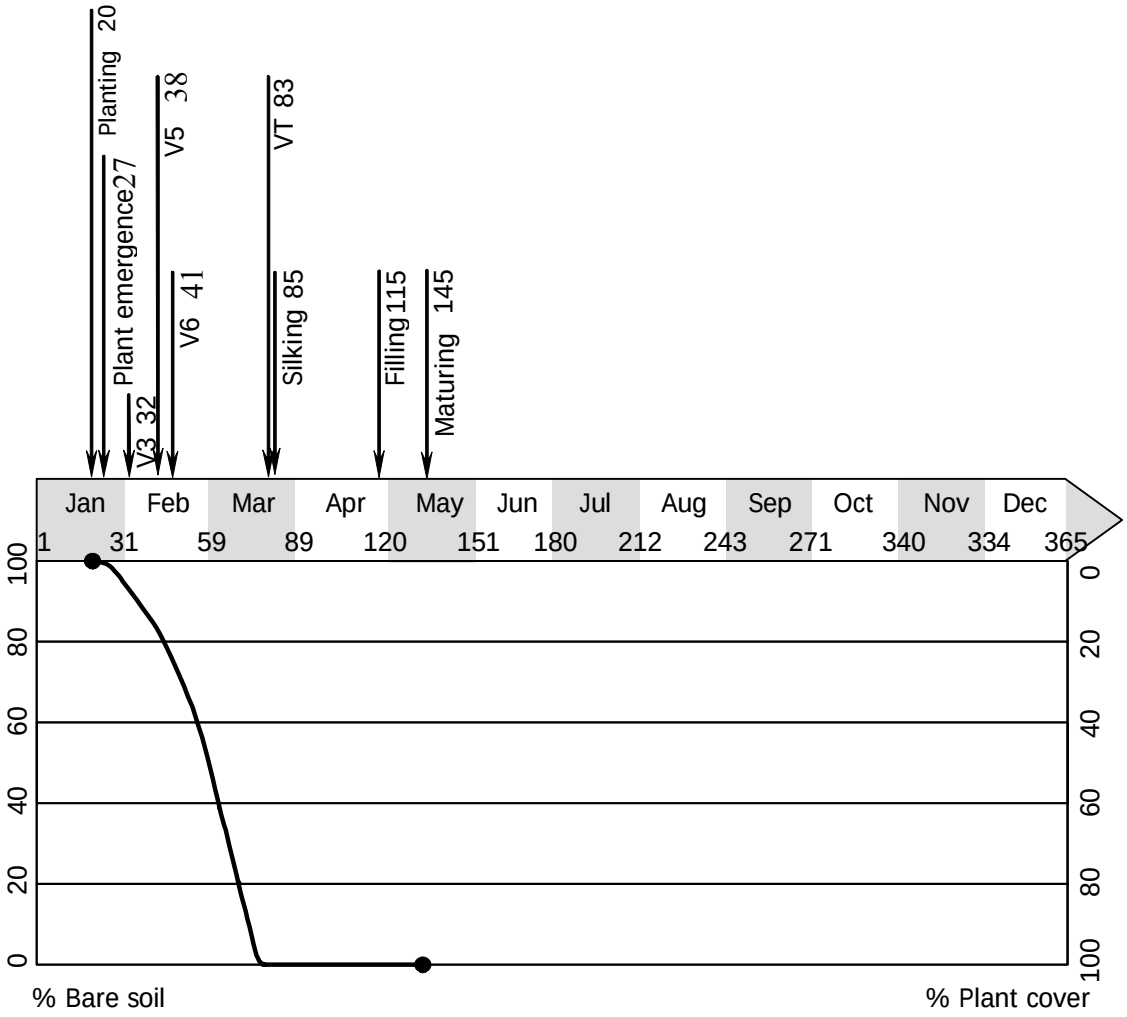

Fig. 21. Phenological stages and change in the percentage of plant cover of a corn field in the LRGV. Data from Rhoads (1986), Carter (1993), Urias-Lopez et al. (2000), Bean and Gerik (2000), and Andreotti et al. (2001). The relationship between height and width of corn plants was estimated from scale-referenced photographs of different growth stages. The percentage of plant cover was estimated multiplying the average width of plants times 100 meters (length of a row) times 130 rows per hectare (each row is 0.76 m wide). The change in percentage of plant cover (y) over time (x [days]) was represented as: $\boldsymbol{y = a / (1 + b \times exp^{(-c \times x)})}$; where $\boldsymbol{a}$ = 141.63, $\boldsymbol{b}$ = 66.99, and $\boldsymbol{c}$ = 0.087.

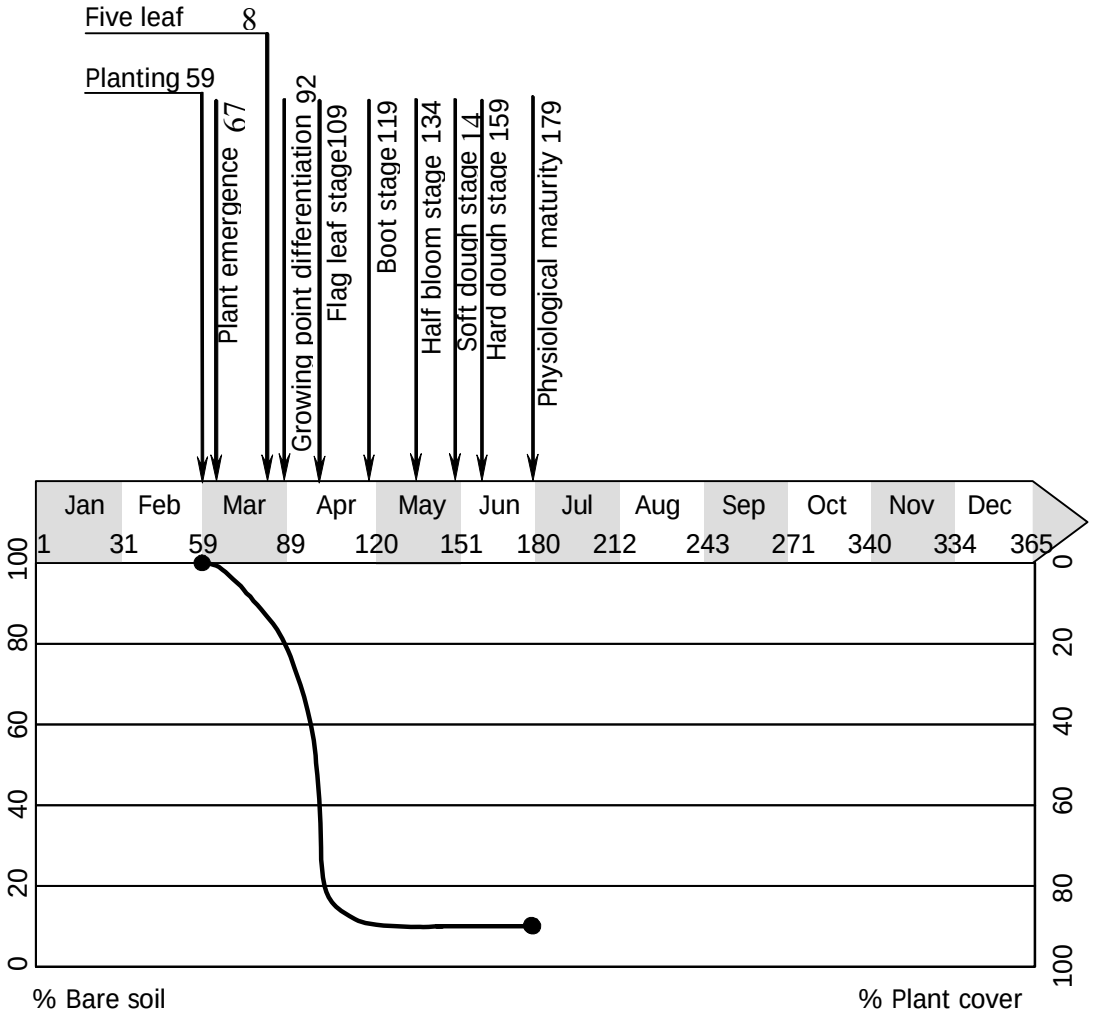

Fig. 22. Phenological stages and change in the percentage of plant cover of a sorghum field in the LRGV. Data from Stichler et al. (1997), Vanderlip (1998), Gerik et al. (2003), Stichler and Fipps (2003), and Warrik (2003). The relationship between height and width of sorghum plants was estimated from scale-referenced photographs of different growth stages. The percentage of plant cover per hectare was estimated by multiplying the average width of plants times 100 meters (length of a row) times 99 rows per hectare (each row is 1 m wide). The change in percentage of plant cover (y) over time (x [days]) was represented as: $\boldsymbol{y = a / (1 + b \times exp^{(-c \times x)})}$; where $\boldsymbol{a}$ = 88.69, $\boldsymbol{b}$ = 253.73, and $\boldsymbol{c}$ = 0.146.

sensitivity of 'B'sterases to inhibition by these metabolites in the brain of pigeons (*Columba libia*). They found that MP oxon is 48.26 times stronger as an inhibitor of brain ChE than AM oxon. Due to the relatively close phylogenetic relationship between WWDO and pigeons, I assumed that they have similar activation and detoxification metabolic pathways for both AM and MP oxon metabolites. Based on this assumption, I corrected the MP dose-response curve (equation 1) to estimate a dose-response curve for AM:

$$Y = (-2.121 + 90.91\,X) / (48.26 \times 1.21)$$

where Y is the percentage of ChE inhibition and X is the AM dose (μg gBW^{-1} day^{-1}). The value 1.21 corresponds to the AM oxon 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.

For CH, I used data from Cairns et al. (1991), who studied the brain ChE activity of Bobwhite quail (*Colinus virginianus*) acutely exposed to this insecticide (Table 13)(Appendix A.2). Based on these data, I estimated the relation between CH dose per gram of BW and ChE inhibition by linear regression:

$$Y = -1.869661 + 0.443918\,X \quad (r = 0.99 \;\; P = 0.0001)$$

where Y is the percentage of ChE inhibition and X is the CH dose (μg gBW^{-1} day^{-1}).

Table 13.

Relationship between doses of chlorpyrifos and ChE inhibition in Bobwhite quail (*Colinus virginianus*). Data from Cairns et al. (1991).

Doses		ChE inhibition
(Mg $bird^{-1}$)	(μg gBW^{-1})	(%)
0.0	0.0	0
0.5	14.9	3
1.0	29.9	10
1.5	44.8	17
2.0	59.7	22
2.5	74.6	34

Table 14.

Relationship among doses of dimethoate, ChE activity, and ChE inhibition in Japanese quail (*Cutornix cutornix japonica*). Data modified from Solecki et al. (2001). Numbers between parentheses are SD.

	Doses			
	0 ppm	10 ppm	35 ppm	70 ppm
Males	6.14 (1.04)	6.18 (0.90)	4.87 (0.70)	3.65 (0.83)
Females	5.90 (0.62)	5.80 (0.71)	4.90 (0.54)	3.46 (0.56)
Average	6.02	5.99	4.89	3.56
ChE inhibtion	0	3.39	21.13	42.58

I estimated the dose-response curve of ChE inhibition for DI from data in Solecki et al. (2001) who studied the effect of intake of several doses of DI on ChE activity, reproduction, and successful hatchability of eggs of Japanese quail (*Coturnix coturnix japonica*)(Table 14)(Appendix A.3). During 6 weeks birds received diets containing either 10, 17, or 70 ppm of DI. Assuming an average body weight of 130 g, I estimated a daily food intake of 15.4 g day^{-1} using Nagy's equation (Nagy, 1987):

$$Daily\ food\ intake = 0.648 \times BW^{0.651}$$

Relating the proportion of DI in the diet offered to birds, daily food intake, and average BW, I estimated the daily intake of DI per g of BW. Thus Japanese quail received 0, 1.19, 4.15, and 8.30 ppm of DI per g of BW per day. Then, I obtained the linear relationship between doses of DI and ChE inhibition:

$$Y = -1.209935 + 5.274175 \times X \qquad (r = 0.98 \quad P = 0.02)$$

where *Y* is the percentage of ChE inhibition and *X* is the DI dose in μg gBW^{-1} day^{-1}.

I estimated the relationship between doses of OX and ChE inhibition from *in vitro* data obtained by Parker and Golstein (2000). They exposed brain ChE of nestling European starlings (*Sturnus vulgaris*) to doses of OX that ranged from $1x10^{-8.5}$ to $1x10^{-2}$ M (molar) (Table 15)(Appendix A.4). The following equation describes the relationship between OX and ChE inhibition:

$$Y = 97.978846 \times (1 - e^{-0.959676 \times X})$$

where *Y* is the percentage of ChE inhibition and *X* is the OX dose μg gBW^{-1} day^{-1}.

Table 15.

Relationship between doses of oxamyl and ChE inhibition in nestling European starlings (*Sturnus vulgaris*). Data modified from Parker and Golstein (2000).

Doses		ChE inhibition
(Log [Mole l^{-1}])	(μg g^{-1})	(%)
-8.5	0.001	2.0
-8.0	0.002	2.5
-7.5	0.007	3.0
-7.0	0.022	3.5
-6.5	0.069	8.0
-6.0	0.219	20.0
-5.5	0.693	48.0
-5.0	2.193	84.0
-4.5	6.935	97.0
-4.0	21.929	100.0

4.2. Experimental design for simulations

I simulated 3 situations representing: 1) the typical current scenario of WWDO using irrigated fields of cotton, corn, sorghum, and citrus, with the model parameterized as described in Section 4.1., in the absence of rain; 2) the same typical scenario, but with a rainfall event of 15 mm at 8:00 am on day-of-the year 117, which corresponds to the beginning of the late April to June rainy season; and 3) an historical scenario typical of the 1980's, when MP frequently was applied at high rates to cotton fields to treat boll weevil outbreaks, with the model parameterized as described in Section 4.1. except that MP was applied to cotton fields every 5 days from day-of-year 70 to day-of-year 110 at a rate of 1400g/ha of a.i., and OX and AM were not applied. I simulated the first 2 scenarios to assess possible impact of the current use of OPs and CAs in the LRGV on WWDO under "normal" and "worst case" environmental situations, respectively. I ran the third simulation to explore the idea that the impact of MP might have contributed to the WWDO population decline during the 1980's (Tacha et al., 1994). For each of the 3 situations, I ran 50 replicate stochastic (Monte Carlo) simulations and monitored the level of ChE inhibition.

4.3. Simulation results

The mean (n=50) maximum level of ChE inhibition for simulations of the typical scenario without rain was 1.33 (SD 0.03) % (Fig. 23 (A)); WWDO arrive in LRGV approximately on day-of-year 80 (late March) and depart day 280 (early October).

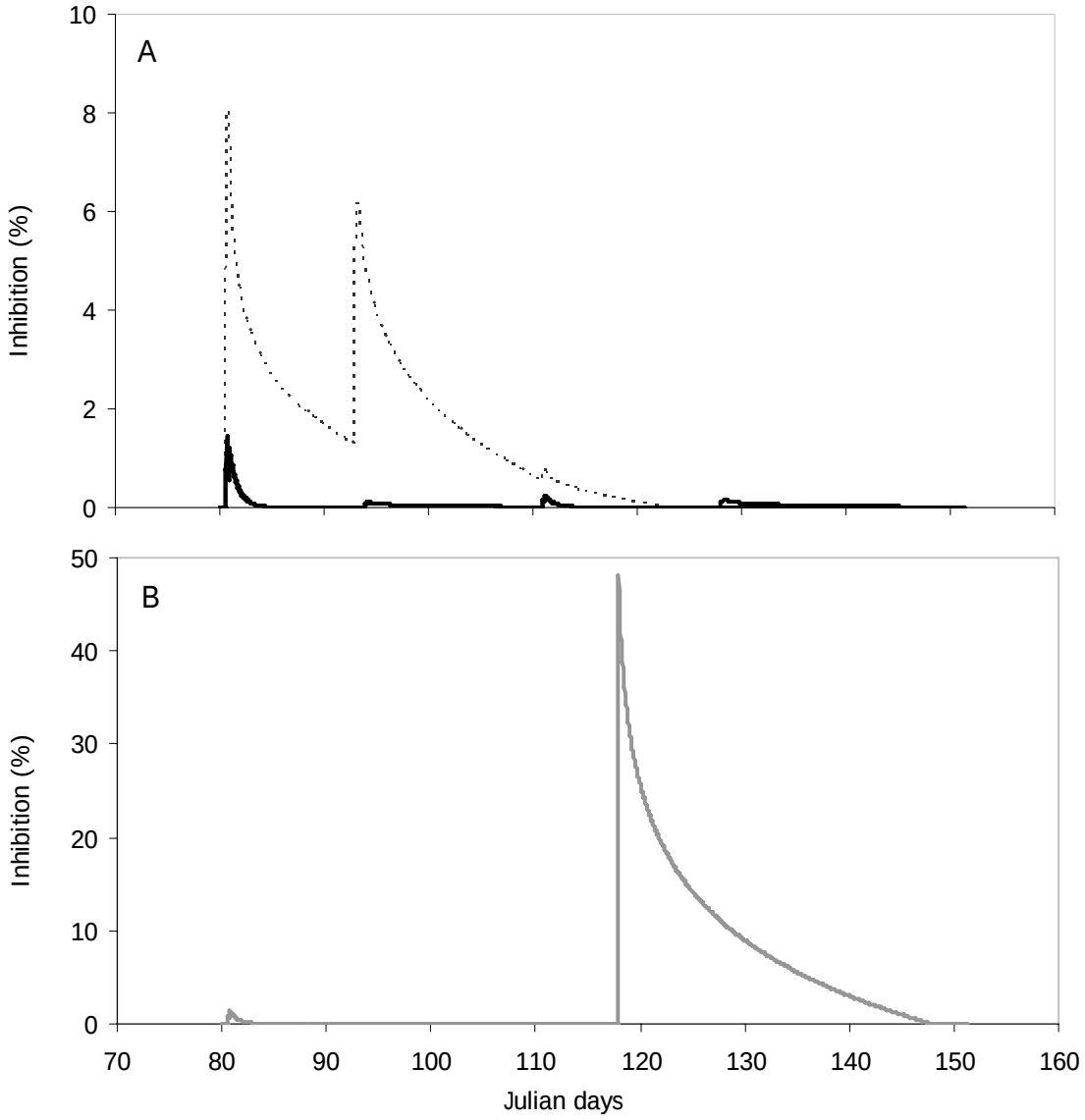

Fig. 23. Mean maximum cholinesterase (ChE) inhibition in the brain of a simulated white-winged dove that drank water from irrigated crop fields in the Lower Rio Grande Valley of Texas, USA. The dotted line in (A) represents situation #3 (historical (1980s) scenario), with high rates of pesticide application, n = 50). The continuous lines in (A) represents both situation # 1 (typical current scenario, without rain, n = 50) and the most common result under situation # 2 (typical current scenario, with a rainfall event on day-of-year 117, n = 49). The line in (B) represents the single replicate (n = 1) under situation #2 in which the simulated individual drank water from a cotton field (rather than exclusively from, sorghum, corn, and citrus fields). See text for details.

In 49 out of 50 repetitions of the typical scenario with rain, the WWDO drank rain water exclusively from, citrus, corn, and sorghum fields, while in only 1 out of 50 repetitions did the WWDO drink water from a cotton field. The WWDO that drank rain water from citrus, corn, and sorghum fields, had a mean maximum level of ChE inhibition equal to 1.43 (SD 0.03) % (Fig. 23 (A)), while the single WWDO that drank rain water from cotton fields had a maximum level of ChE inhibition of 48.07 % (Fig. 23 (B)). When I simulated the historical scenario with high rates of MP applied at high frequency (n=50), the WWDO had a mean maximum level of ChE equal to 8.09 (SD 0.33) % (Fig. 23 (A)). Except for the animal that drank in cotton fields after the rainfall, which suffered levels of ChE inhibition close to the lethal limit (50 %), simulated levels of ChE inhibition were well below the level which is diagnostic for exposure to CAs or OPs (20 %).

Canopy cover of annual crops such as cotton, sorghum, and corn, increases very fast during the growing season. Therefore, over time, less of the applied insecticide drops on the ground and more stays on the plant canopy. Because the simulated rain surpassed the threshold for washoff (13 mm in 1 h), remnant insecticide residues on the canopy and in the soil beneath the canopy were 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). Thus, the concentration of residues in rain water was higher and represented a high-risk situation for animals drinking that water.

5. Discussion and conclusions

In 1987, Custer and Mitchell studied the exposure to insecticides of WWDO in heavily treated cotton and sugarcane fields in the LRGV. Most of the insecticides applied were OPs. They found that grackles and mourning doves had significantly lower ChE activity than non-exposed controls, but they did not find WWDO with deprived ChE activity.

In 1991 and 1992, Tacha et al. (1994) studied the exposure to anticholinesterase insecticides of WWDO captured in 5 locations within remnants of native brushland and 1 citrus orchard. In the first and second years of their study they found 76 % and 39 %, respectively, of the birds captured had levels of ChE inhibition > 16.1 %, which they considered diagnostic for exposure to ChE-inhibiting pesticides. Tacha et al. (1994) hypothesized that WWDO were being exposed to ChE-inhibiting pesticides by drinking contaminated water; WWDO commonly were seen drinking in cotton fields, which usually receive high loads of insecticides.

Based on simulation results, I conclude that is unlikely that WWDO are seriously exposed to ChE-inhibiting pesticides by drinking contaminated water. Only in rare cases, for example, when a rain event occurs just after the application of insecticides, are levels of ChE inhibition likely to approach diagnostic levels (20 %). Other ChE inhibiting substances like heavy metals (Tacha et al., 1994), or other routes of exposure to OPs and CAs, such as inhalation of airborne residues of recently applied insecticides or dermal exposure to insecticides, may be more likely causes of ChE inhibition in WWDO and should be investigated.

Data needed to estimate several parameters used in the model are lacking or inaccurate, such as data on degradation half-lives of insecticides on plants, in water and in soils, and transportation of dissolved insecticide along flooded furrows. Of particular importance is the lack of ChE-inhibition/insecticide dose-response curves for WWDO; the current model draws upon data from other bird species, although it is well known that tolerance to particular insecticides may be very species-specific (Mineau, 1991; Thompson et al., 1995; Blakley and Yole, 2002).

CHAPTER IV

CONCLUSIONS

I developed a simulation model to help assess the ecological risk to non-target wildlife of exposure to pesticide-contaminated water in irrigated agricultural fields. Conceptual development (Chapter II, Section 2), parameterization (Chapter III, Section 4.1), and application (Chapter III, Section 4.2) of the model paralleled the three phases used by the Environmental Protection Agency of the United States to conduct an ecological risk assessment: problem formulation, analysis, and risk characterization (EPA - Environmental Protection Agency, 1998). Problem formulation includes identification of assessment goals and selection of appropriate assessment endpoints, and development of a conceptual model. Analysis includes evaluation of exposure to stressors and the relationship between stressor levels and ecological effects. Risk characterization includes integration of exposure and exposure-response profiles to determine ecological adversity.

To demonstrate application of the model, I 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). I 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. Simulation results indicated that levels of ChE inhibition in WWDO remained far below the diagnostic level for pesticide exposure (20%) under most circumstances (exposure profile). These results do not support the suggestion of Tacha et al. (1994) that drinking water from agricultural fields in the LRGV poses a significant risk of pesticide exposure for WWDO. However, simulation results also indicated that under certain rarely occurring ($P < 0.02$) circumstances, ChE inhibition approached lethal (exposure-response profile) levels (50%); for example, when a rain event occurs within 24 hours of a pesticide application.

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 sub-models generically represent periodicity and magnitude of contaminant arrival in the environment (sub-model 1), contaminant transport in the environment (sub-models 2, 3, and 4), exposure of non-target wildlife to contaminants (sub-model 5), and ecological impact of exposure on non-target wildlife (sub-model 6). These sub-models 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 sub-models will depend on the particular system of interest. But I 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.

The primary factor limiting usefulness of the model is the lack of reliable data. Important data gaps are identified clearly during conceptual model development. For example, except for methyl parathion I could not find data on pesticide-dose/ChE-inhibition curves for WWDO. These curves represent the specific tolerance of species to different pesticides. To parameterize the model I had to draw upon experimental estimates of the effect of insecticides on other species. Also, half-lives of pesticides in soil, water, plants, and the body of animals are unknown or are very variable. The model only takes into account the toxicity of pesticide active ingredients and assumes no interaction among the effects of pesticides on animals. However, although scarce, there are studies that demonstrated that components in pesticide formulations other that active ingredients (adjuvants, diluents), and pesticide interactions (synergistic or suppressive), should be considered into the evaluation of pesticide impact on non-target wildlife.

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APPENDIX A

ESTIMATION OF THE INSECTICIDE-DOSE – CHE INHIBITION CURVES

1. Estimation of the methyl parathion-dose - ChE inhibition curve for white-winged dove (*Zenaida asiatica*). Data modified from Small et al. (1998).

Methyl parathion concentration (ppm)	Methyl parathion dose (μg gBW^{-1} day^{-1})	Average brain ChE activity (μmol min^{-1}g-1)	Brain ChE activity (% of inhibition)
0	0.00	21.0	0.00
2.6	0.35	14.3	31.90
5.2	0.58	14.2	32.38
7.8	0.75	7.5	64.29
10.4	0.71	4.6	78.10

Results of linear regression ($Y = a + b \times X$) methyl parathion dose (μg gBW^{-1} day^{-1}) to brain ChE inhibition (%):

a = -2.121	r = 0.918	SE = 14.03
b = 90.91	r^2 = 0.843	P = 0.03

2. Estimation of the chlorpyrifos-dose - ChE inhibition curve for bobwhite (*Colinus virginianus*). Data from Cairns et al. (1991).

Dose		Brain ChE inhibition
(mg $bird^{-1}$)	(μg gBW^{-1} day^{-1})	(%)
0.0	0	0
0.5	14	3
1.0	28	10
1.5	42	17
2.0	59	22
2.5	76	34

Results of linear regression ($Y = a + b \times X$) relating chlorpyrifos dose (ug gBW^{-1} day^{-1}) to brain ChE inhibition (%):

a = -1.869661	r = 0.99026	SE = 1.97508
b = 0.443918	r^2 = 0.98062	P = 0.0001

3. Estimation of the dimethoate-dose - ChE inhibition curve for Japanese quail (*Coturnix coturnix japonica*). Data from Solecki et al. (2001).

Dimethoate concentration in the diet (ppm = μg g^{-1})	Dimethoate intake per animal (μg day^{-1})	Dimethoate intake per g of animal body weight (μg gBW^{-1} day^{-1})	Brain ChE inhibition (%)
0	0	0	0
10	154	1.19	3.39
35	539	4.15	21.13
70	1079	8.30	42.58

Japanese quail average BW = 130

Daily food intake = $0.648 \times (BW^{0.651})$ (Nagy, 1987) = 15.41 g day^{-1}

Results of linear regression ($Y = a + b \times X$) relating dimethoate dose (ug gBW^{-1} day^{-1}) to brain ChE inhibition (%):

a = -1.209935	r = 0.990854	SE = 1.49644
b = 5.274175	r^2 = 0.99604	P = 0.02

4. Estimation of the oxamyl-dose - ChE inhibition curve for European starlings (*Sturnus vulgaris*). Data from Parker and Golstein (2000).

Doses		Brain ChE inhibition
Log [Mole l^{-1}]	(ug gBW^{-1} day^{-1})	(%)
-8.5	0.001	2.0
-8.0	0.002	2.5
-7.5	0.007	3.0
-7.0	0.022	3.5
-6.5	0.069	8.0
-6.0	0.219	20.0
-5.5	0.693	48.0
-5.0	2.193	84.0
-4.5	6.935	97.0
-4.0	21.929	100.0

Oxamyl MW = 219.29

Fitted equation of the form $Y = a \times (1 - \exp^{(-b \times X)})$(Hyams, 2001) relating oxamyl dose (ug gBW^{-1} day^{-1}) to brain ChE inhibition (%):

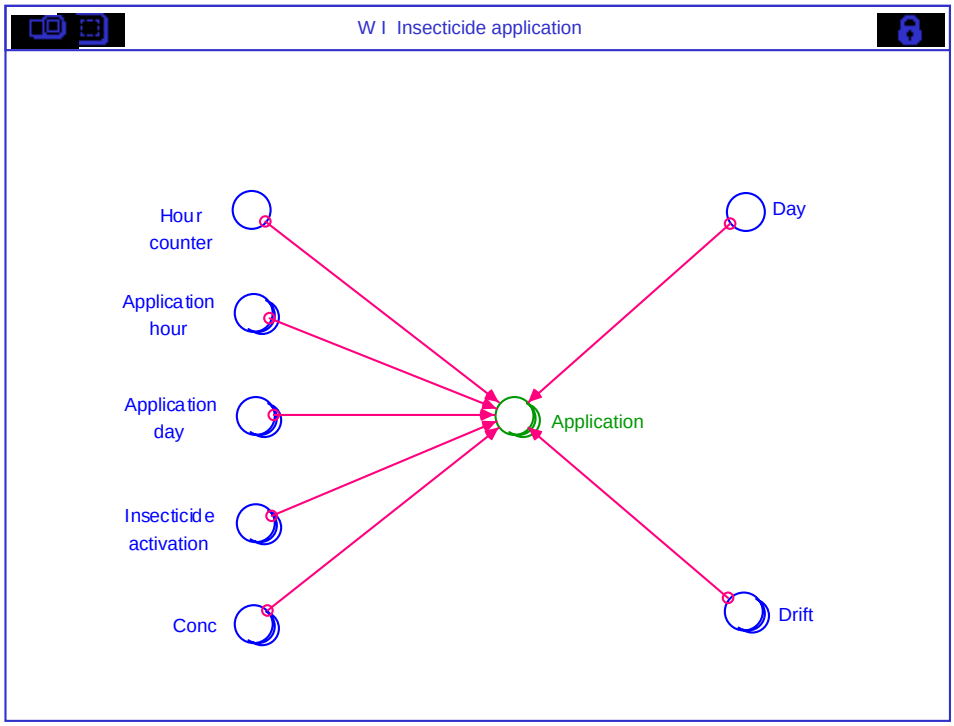
a = 97.973571 r = 0.9990939 SE = 1.8471856

b = 0.960326

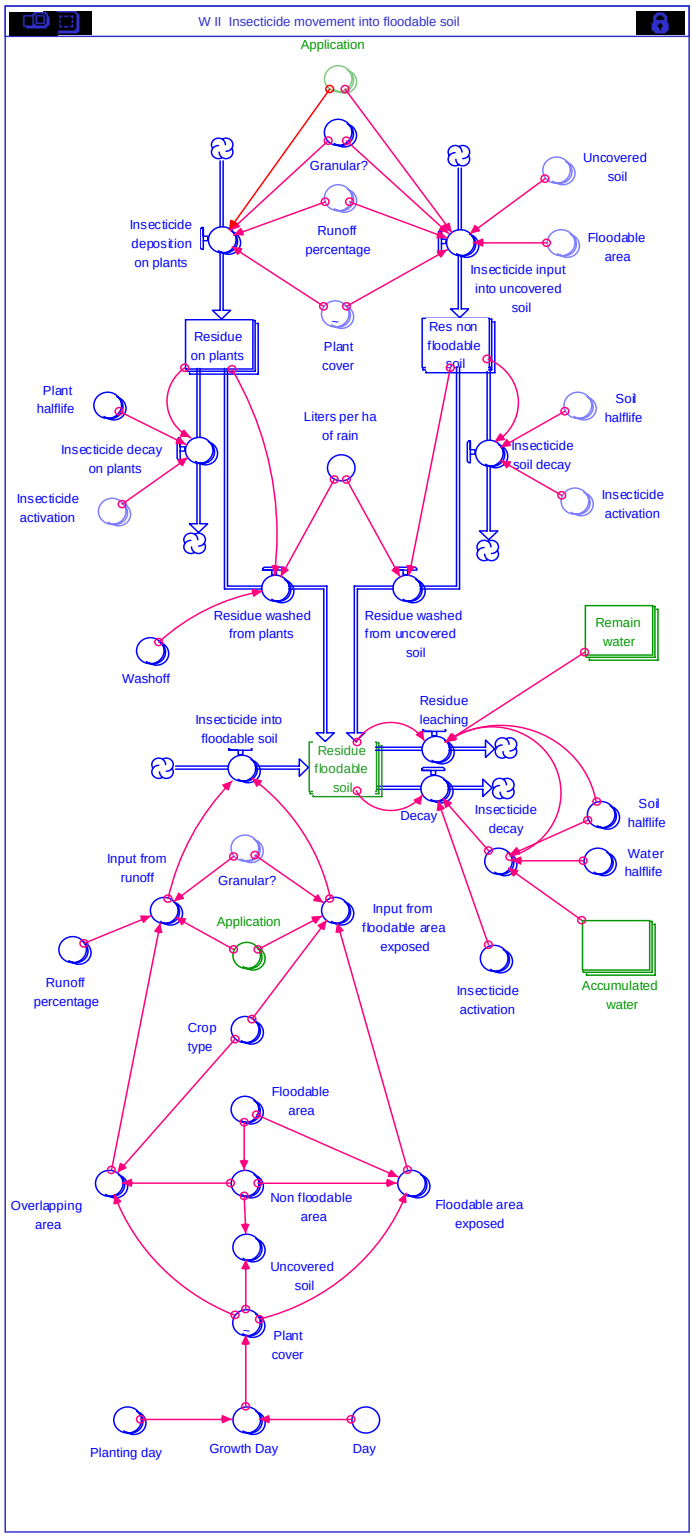
APPENDIX B

CONCEPTUAL SUBMODELS AS REPRESENTED IN STELLA®VII

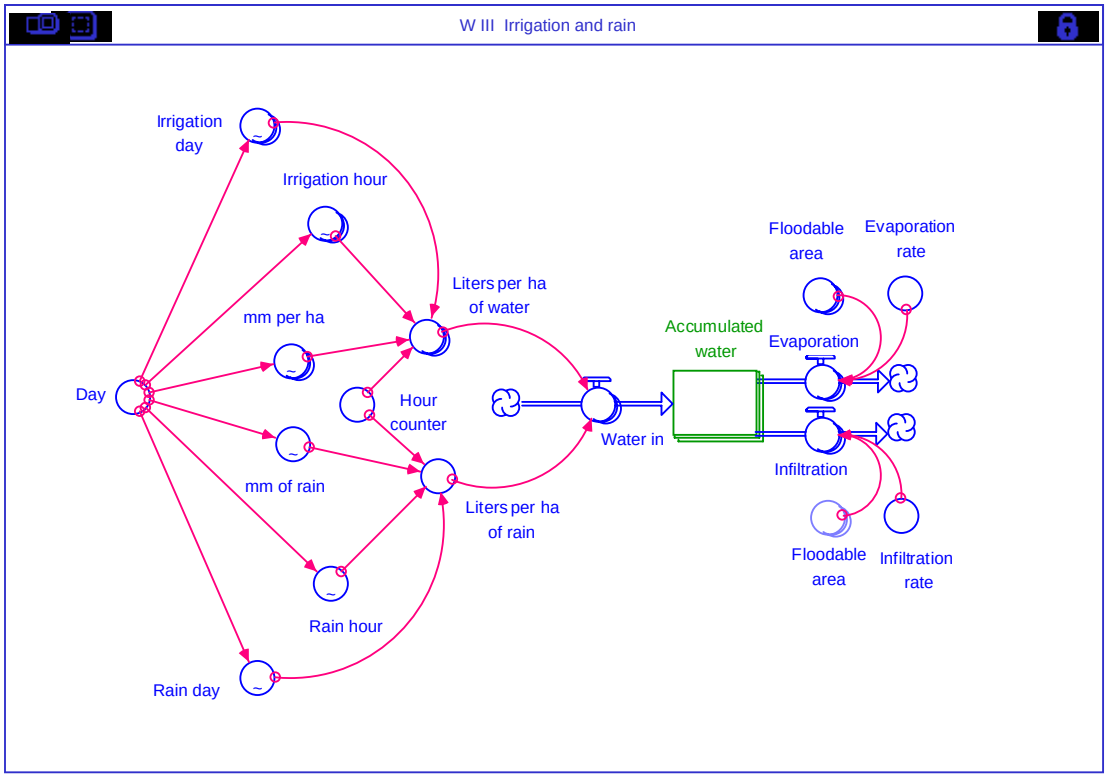
Submodel 1. Insecticide application



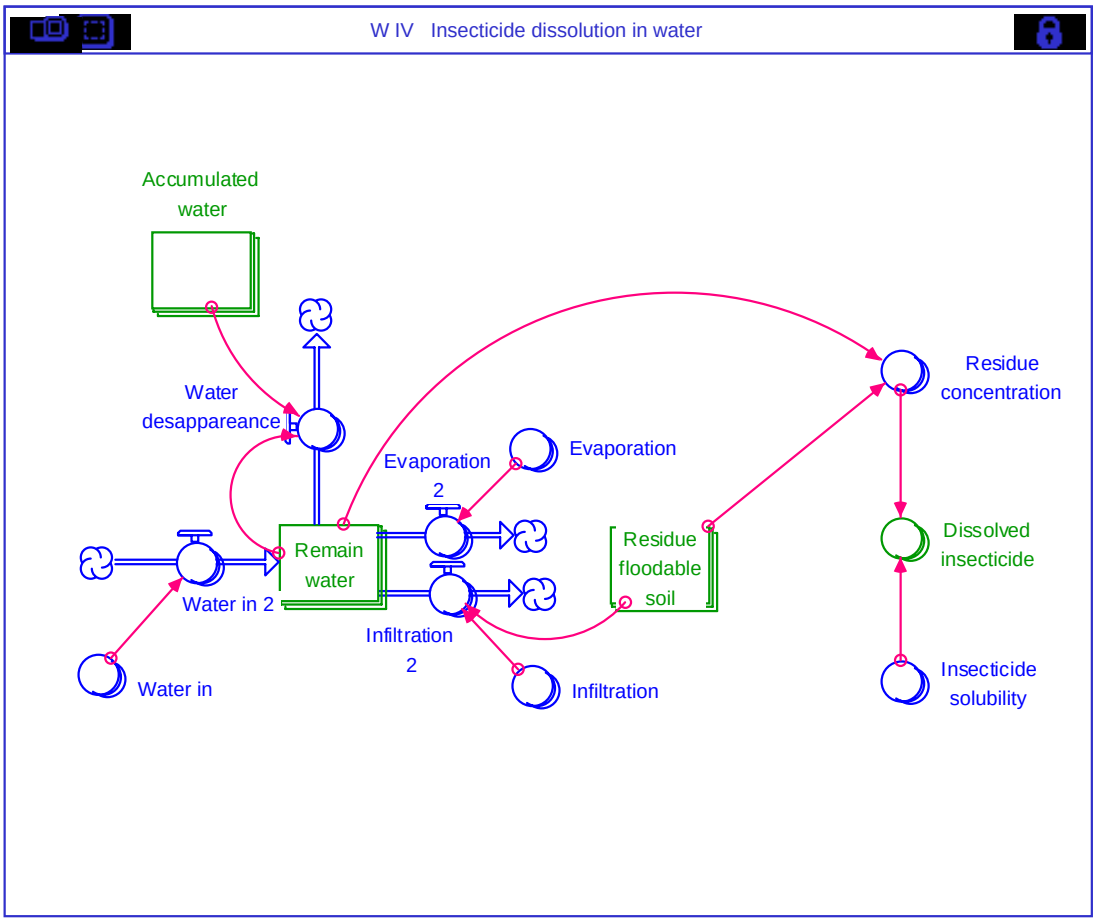
Submodel 2. Insecticide movement into floodable soil



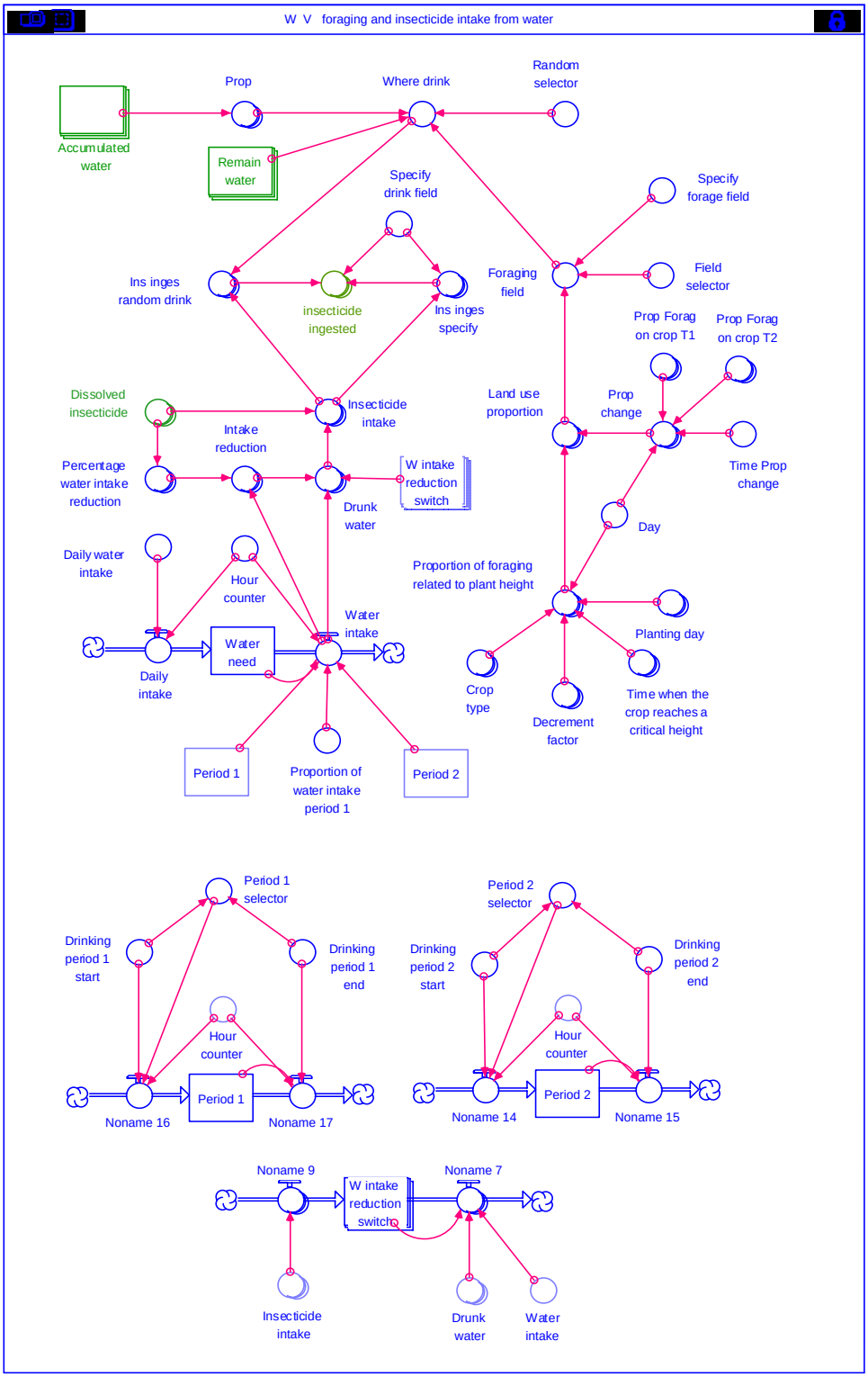
Submodel 3. Irrigation and rain



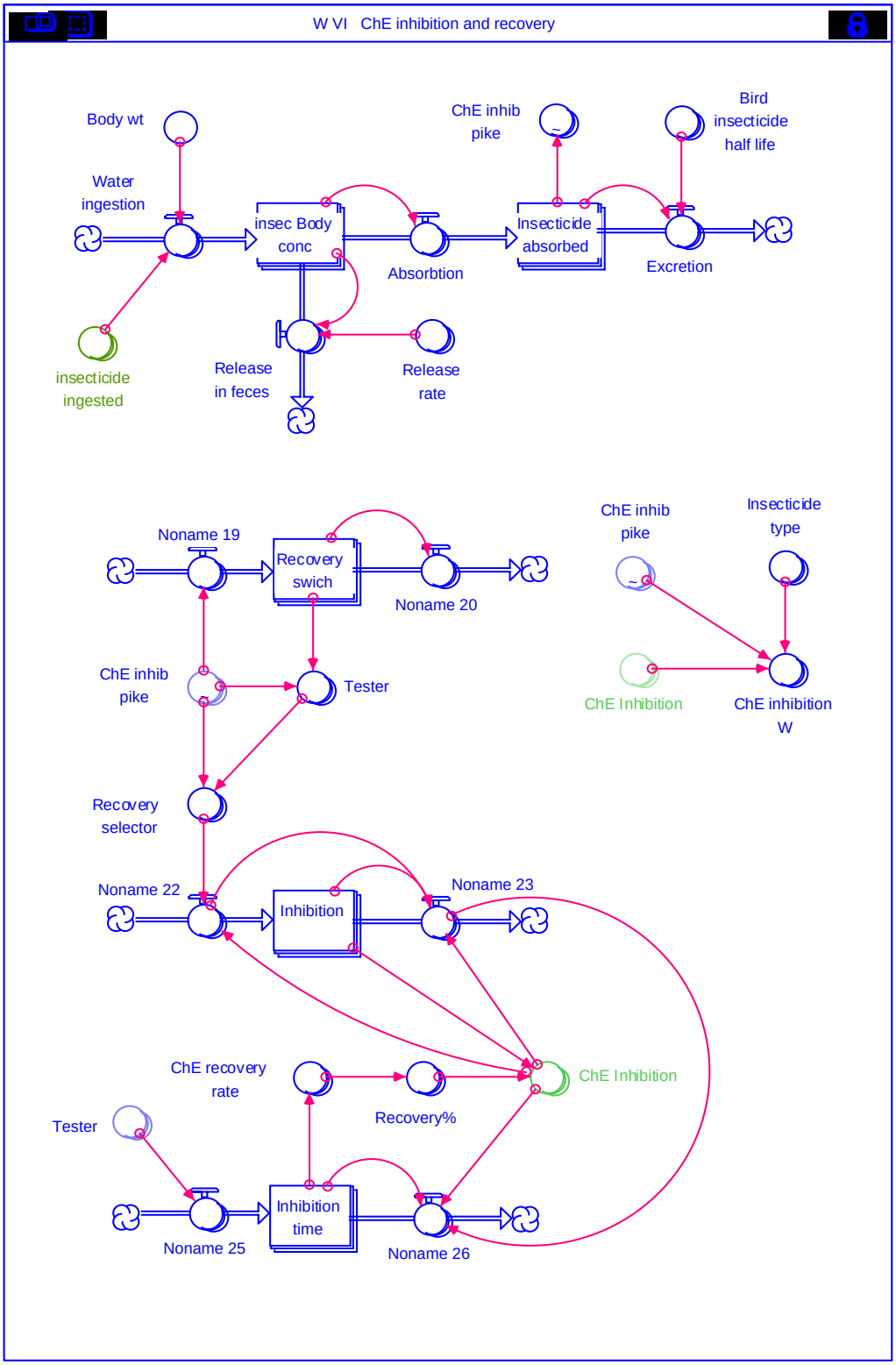
Submodel 4. Insecticide dissolution in water



Submodel 5. Foraging and insecticide intake from water



Submodel 6. ChE inhibition and recovery



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