Pesticide Regulatory Actions and the Development of Pest Resistance:

A Dynamic Bioeconomic Model

by

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(ABSTRACT)

Pest resistance to pesticides can have severe impacts on both commercial agriculture and the environment. But many resistance problems are exacerbated because pest susceptibility is a dynamic, common-property resource subject to inefficient allocation by the market. Theoretically, the impact of resistance can be mitigated through regulatory management of the control technology set. However, the current pesticide regulatory process does not include resistance considerations in its quantitative analyses due to the computational difficulties encountered when trying to optimize complex bioeconomic models. As a result, regulatory efforts may actually promote increased susceptibility depletion and the rapid emergence of resistance. This study overcame these problems by forming a dynamic bioeconomic model that combined: 1) a widely accepted genetic simulator used by entomologists; 2) an aggregate economic surplus model with nationwide regulatory relevance; and 3) an improved simulation optimization algorithm that conserved computational resources. For the purpose of illustration, the bioeconomic model was parameterized to represent the U.S. apple production system.

Information generated through optimization of the dynamic bioeconomic model suggested that resistance becomes quantitatively important when planning horizons exceed 10 years, confirming that the economic performance of the production system becomes severely sub-optimal when susceptibility depletion is not incorporated into decision-making. Furthermore, insecticide withdrawals from an initial control technology set led to large additional losses in economic surplus, although the exact magnitude of these impacts varied depending on the characteristics of the insecticide withdrawn. Substantial withdrawal-induced losses in

economic surplus occurred regardless of the planning horizon, and they were accompanied by temporal shifts in insecticide applications. The need to incorporate a dynamic, bioeconomic simulation analysis in the regulatory process was demonstrated by comparing statically optimal and extant insecticide use recommendations with the dynamically-optimal solutions. Optimal solutions drastically reduced economic surplus losses, although they did lead to increased levels of insecticide use. Ultimately, management of the resistance/regulation nexus requires that both current economic data and the time-dynamics of system biology play a prominent role in the benefits assessment process. This can only be accomplished if an investment is made in the necessary basic research and model development.

Acknowledgements

Although a single name appears under the title, in no way can I presume to take full credit for the research detailed upon the pages of this dissertation. In terms of logistical support, this work owes its existence to the financial resources provided by the United States Environmental Protection Agency and the United States Department of Agriculture, and particularly to the project officers and administrators who promoted the underwriting of what was, at the time, a highly speculative project. It is my hope that any measure of success achieved by this research will help provide the impetus for increased funding of other complex, interdisciplinary topics.

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willingness to share his extensive knowledge and experience concerning the development of resistance in apple pests.

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Coming closer to home, this dissertation could never have been attempted, much less completed, if it were not for the unending Love and support provided since childhood by my parents, Richard and Lorraine Kazmierczak. To them alone I credit my enjoyment of reading, thirst for knowledge, and the intellectual confidence that I could achieve anything with hard work and persistence. In addition, they instilled in me a valuable notion that is lamentably rare in graduate education; that a fulfilling life must be built upon a broad foundation of work and recreational interests. While I am certain that there were times when they felt despair for my future, I hope that the completion of this dissertation, and my acceptance of an academic position, will make them at least fractionally as proud of me as I am of them.

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CHAPTER I: INTRODUCTION

The recent widespread emergence of pest resistance to chemical controls poses fundamental challenges to producers, government regulators, environmentalists, and the agrichemical industry. Resistance threatens the ability of producers to control economically damaging pests, leading to alarm over the prospects for long-run sustainable yield and product quality. Significant production failures, poor quantity and quality of foods and fibers, loss of efficacious pesticides, and additional expenditures on research, development, and registration of replacement pest controls may be the legacy of policies that did not adequately consider the impact of resistance (Dover and Croft). In addition, increasing resistance may imply increasing total pesticide use and subsequent dangers to human and environmental health, especially if regulations on chemical use inadvertently encourage the use of single-chemical pest control programs (Knight and Norton). Taken together, emerging resistance problems and higher residual levels of pesticides in food, water, air, and soil have fueled intense public controversy over the kinds of chemicals used in agriculture, how they are applied, and the ways in which they may promote pest resistance and instability in the U.S. food production system.

It is difficult to overstate the devastating impact pest resistance can have on an agroecosystem. A graphic example of these effects can be found in the recent history of southwestern cotton production. Following years of extensive insecticide use that saw fields

being treated 15 to 20 times per season with myriad combinations of chemicals, major cotton pests developed widespread resistance to controls (Adkisson et al.). Although the U.S. cotton industry was saved from destruction by a carefully timed and coordinated integrated pest management system (Dover and Croft), Mexican cotton production fell from over 370,000 bales in 1960 to 50,000 bales in 1965 (van den Bosch). Today, little cotton is grown in the Mexican regions that once had over 710,000 planted acres (Adkisson et al.).

Resistance Defined

Resistance can be defined as the genetic changes that occur in a pest population in response to the selection pressure generated by repeated control actions (Sawicki). Resistance arises because pesticides kill only those organisms that are genetically susceptible to the particular chemical compound used. Individual organisms that are capable of surviving exposure to a pesticide are often confronted with significantly less competition for food and space, along with a decreased probability of predator-induced mortality. These individuals then have the opportunity to reproduce and establish a new population that is genetically resistant to the chemical. In other words, the resistant pests are more fit than their susceptible conspecifics when the environment includes pesticides. But if use of the pesticide is eliminated, susceptibles will often have superior relative fitness. Combined with the knowledge that a pesticide's initial effectiveness depends on the formulation used, dosages, length of exposure, and various other factors, the relative fitness differences between resistant and susceptible conspecifics suggests that selection for genetic resistance may occur due to the way in which a pesticide was developed and/or how it was employed in a control program. If this is true, then pest susceptibility to chemical control may be viewed as a resource stock subject to manipulation and management (Hueth and Regev).

Problem Statement

Because of the potentially severe impacts of resistance on commercial agriculture and the environment, pesticide resistance and the management of pest susceptibility have become policy issues for many public and social institutions, particularly the U.S. Environmental Protection Agency (EPA). But, while the EPA continues to search for a way to include resistance considerations in the static procedures for analyzing pesticide benefits and costs, pesticide regulatory actions themselves may hasten the onset of pest resistance. Because the EPA's benefit analyses only include qualitative judgements of resistance impacts, current decisions about pesticide use will affect future pest susceptibility (and thus future control options) in ways that are quite unpredictable. A solution to this problem requires that pest susceptibility, pesticide use, and regulatory actions be dynamically modelled. It is the empirical modelling of the dynamic economic impacts associated with pesticide resistance, and how the resulting information might be used to enhance pest management and the EPA's benefit assessments, that are the focus of this study. Arthropod management and insecticide resistance problems in an apple agroecosystem will be used for illustration throughout the study, both due to the significant monetary value of the apple industry and the fact that apple producers often apply large amounts of insecticides annually (Rajotte et al.).

Justification of the Study

The emergence and proliferation of pest control failures points to the need for a nationwide policy addressing the problem of pest resistance. Development and implementation of a unified public policy is especially appropriate given that pest susceptibility is best characterized as a common property resource subject to sub-optimal market allocation. Given the food production, health, and environmental impact of pest resistance, society appears to have

a legitimate claim to maintained pest susceptibility. The EPA already is responsible for assessing the relative risks and benefits of pesticides, and it has the legal power to restrict or ban the use of any agricultural chemical. With this power, and the acknowledgement that resistance might be affected by the regulatory process, the EPA initiated this examination of how pesticide use, pest resistance, and pesticide regulation interact to affect the long-run economic performance of agricultural production systems.

Although the potential problems of pest resistance have been known for some time, the difficulties associated with dimensionality and access to adequate databases have plagued the development of realistic bioeconomic models of resistance. In a 1979 review of pest management optimization models, Varadarajan found that although 32 dynamic studies had been attempted since Watt first suggested the approach for pest management in 1963, only 10 studies dealt with specific pest complexes, and fewer still with pest resistance. However, even these 10 studies tended to incorporate highly simplified representations of the agroecosystems, resulting in little empirical application and almost no impact on the development of actual pest management programs.

Perhaps the best attempts at empirically examining the effects of resistance on the optimal allocation of pesticides was a 1983 study by Regev et al. and a 1984 study by Archibald. Using the alfalfa weevil complex, Regev et al. suggested that the conflicting dynamic roles of pesticides and pest resistance implies that an optimal steady-state solution is not attainable. However, they also indicated that there were problems associated with their study's ecological model and in determining the appropriate way to incorporate the relevant biological characteristics into an economic model. Archibald's study determined the optimal input use given resistance and various regulatory strategies in cotton production. But, by using an aggregated econometric approach, the author avoided the genotypic detail that is necessary for the model to be applied to other systems.

A comprehensive, dynamic approach is needed to address the issues behind the simultaneous management of pests and pest resistance. This study extends previous work by formulating a dynamic bioeconomic model that is not only economically realistic, but that also

incorporates a high degree of biological realism. This approach allows the empirical economic evaluation of alternate policy prescriptions for pesticide regulation and for dealing with the development of resistance.

Study Objectives

The encompassing objective of this study is to develop an empirical procedure capable of incorporating the time-dynamic impacts of pest resistance into the policy-making process that forms federal pesticide regulations. Given the complexities inherent in many agroecosystems, the subtle interactions between regulations and the evolution of resistance, and the need for a high degree of realism, the empirical procedure cannot be based on simple single- or multiple-equation biological models. Instead, sophisticated simulation models of the biological systems are required. Thus, a secondary objective of the study is to demonstrate the feasibility of combining a dynamic biological simulator, an economic market model, and an efficient search optimizer into a combined model capable of yielding realistic estimates of the consequences of various policy actions. In addition, a specific crop and pest system is used for illustration. As a result, this study also strives to provide estimates of the economic effects of insecticide regulatory actions on the mid-Atlantic apple production system. To accomplish all of these goals, the study is divided into a number of specific steps:

1. Develop a economic model of the aggregate apple market that is flexible enough to examine the potential effects of pest control measures on emerging pest resistance to insecticides;

2. Combine the economic model with a biological simulator to form a bioeconomic model capable of realistically representing the development of pest resistance through time;

3. Optimize the bioeconomic model to determine the long-run impacts of resistance on system performance; and

4. Use the bioeconomic model to describe the economic effects of various regulatory actions on the insecticides used in apple production.

The Study Agroecosystem

The tufted apple bud moth (*Platynota idaeusalis* Walker), European red mite (*Panonychus ulmi* Koch), black ladybird beetle (*Stethorus punctum* LeConte) pest, pest-predator complex in apples was chosen as the study system. This complex is prevalent in the Cumberland/Shenandoah production region that covers parts of Pennsylvania, West Virginia, Virginia, Maryland, and New Jersey. The choice of this agroecosystem was prompted by a number of factors. First, apple production in the U.S. is a high-value enterprise dominated by a strict quality grading system composed, in large part, of characteristics related to visual appearance. This leads to numerous preventative insecticide applications per year and, as a result, recurring problems with the development of pest resistance. Secondly, the insecticides used in apple production are frequently the subject of EPA benefit analyses. This regulatory focus on chemicals used in fruit production has arisen because of their tendency to be toxic to humans and other non-target organisms, the possibility of worker exposure due to application procedures, and the potential for residue accumulation on fresh marketed fruit. Given that these concerns could also be expressed with respect to other crops, the use of the apple agroecosystem in this study should provide a good example of how to examine the dynamic impacts of resistance in other systems as well. Thirdly, the investigation had access to 20 years of data from the Biglerville Fruit Research Laboratory (Pennsylvania State University) covering this particular pest/predator complex. Lastly, and perhaps most importantly, the biological interactions in this apple system, both between species and with control chem-

icals, are about as intricate and complex as they can be in an agroecosystem. If this system can be modelled by the procedures detailed in this study, it would suggest that most other agroecosystems and resistance/regulatory problems could also be investigated using a similar approach.

The tufted apple bud moth (TABM) is the major direct pest on apple fruit in the Cumberland/Shenandoah production region (Hull et al. 1983b) and has been successfully controlled in the past using organophosphate insecticides. While using these chemicals against TABM, an apple grower's most effective total pest control strategy has been to apply limited amounts of acaricides to keep European red mite (ERM), an indirect pest feeding on apple tree foliage, below their economic threshold[1] until the black ladybird beetle (BLB) appears in the orchards to control ERM naturally (Hull et al. 1983b). This integrated pest management system is possible because of BLB's tolerance to the organophosphate insecticides used for TABM control. However, with TABM resistance to organophosphates, growers cannot rely as heavily on BLB to control ERM because BLB is generally not as tolerant of the other insecticides that are effective against TABM, such as the carbamates and synthetic pyrethroids. Growers must then resort to increased use of acaricides to control mites. But it is from the extensive use of acaricides that additional problems arise. Most mites have extremely high levels of reproductive capability, with 10 to 20 generations per year not uncommon under favorable environmental conditions. This short generation time, combined with heavy insecticide-induced selection pressure, can produce a rapid emergence and entrenchment of resistance. By itself, this potential for rapid resistance development may not seriously threaten pest control. But combined with the fact that acaricide registrations are dwindling because of demonstrated toxicity to humans and non-target animals, there are potentially catastrophic long-run implications for commercial apple production. As regulatory pressure and TABM/ERM resistance expands, the danger is that the apple pest complex will become uncontrollable and/or that there will be a net increase in the use of insecticides, with

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[1] The pest population level at which a control action must be taken in order to prevent economic damage in the future.

the accompanying environmental and human health risks, over what would have occurred with no regulatory action.

Major Hypotheses

This sub-section describes some of the major scenarios that this study will investigate. But, given the normative confines of the methods employed, these scenarios will not be subject to statistically rigorous attempts at falsification. Thus, they should not be viewed as testable hypotheses. While many assumptions, or maintained hypotheses, underlie the construction of the simulation model and optimization technique used in this analysis, and thus directly contribute to the outcomes generated by the model, the unknown quantitative dynamic interactions of resistance, pesticide use, pesticide regulation, and system economics implies that the scenarios presented cannot, as a practical matter, be *a priori* mapped 1:1 back into the assumptions. In actuality, the assumptions are extant conjectures about long-run behavior that have arisen out of prior knowledge about the short-run biological and economic operation of the agroecosystem. As such, these scenarios are probably best described as working hypotheses for which evidence is sought. The pursuit of evidence for or against these working hypotheses is important because they have begun to enter into the pesticide policy debate as maintained hypotheses and, if erroneous, may have serious implications for the effect of policy tools on the efficiency of agricultural production. Given the tremendous complexity inherent in the long-run pest resistance problem, it is unlikely that positive methods will be capable of fully addressing it in the near future. Therefore, this research will investigate the following working hypotheses:

1. Given an initial static pest control technology set, susceptibility to insecticides is not sustainable when there exists: a) the potential for resistance development; b) a long-run

profit-maximizing objective; and c) a fixed planning horizon and/or present value discounting.

2. Given an initial static pest control technology set, susceptibility to the available chemicals is sustainable if long-run profit maximization is not an objective, even when the potential for resistance exists, there is a fixed planning horizon, and/or there is present value discounting.

3. Given an initial static pest control technology set and the potential for resistance development, any reduction in the number of available insecticides will hasten the development of resistance to the remaining chemical alternatives.

4. Given an initial static pest control technology set, any reduction in the number of available insecticides will decrease the long-run economic surplus obtainable from the apple production system.

5. Given an initial static pest control technology set and the presence of resistance, any reduction in the number of available insecticides will increase long-run total use of insecticides.

6. Given an initial static pest control technology set and the current importance of organophosphate insecticides to apple production, regulatory withdrawal of an organophosphate will result in larger long-run decreases in economic surplus compared to the decreases experienced following the withdrawal of a non-organophosphate control.

7. In terms of resistance development, economic surplus losses, and total use of insecticides, regulatory action on individual insecticides is sub-optimal relative to a simultaneous regulatory evaluation of the entire control technology set.

Major Assumptions

This study will make explicit use of the following assumptions concerning the biological and economic systems being modelled. The details and implications of these assumptions will be discussed in the relevant sections of this dissertation.

Biological

1. The structure of TABM, ERM, and BLB genetic susceptibility to a given insecticide can be modelled as a single gene locus with two alleles.

2. TABM, ERM, and BLB susceptibility to a given insecticide can be a function of their susceptibility to other insecticides, thereby giving rise to the cross-resistance effect.

3. TABM, ERM, and BLB susceptibility to insecticides is determined by multiple factors, including genotype, life-stage, and the presence of refugia.

4. Pest populations can never be permanently eliminated by control actions due to the presence of refugia, pest migration, and/or pest resistance.

5. Pest susceptibility to insecticides is a renewable resource stock in the sense that reversion to pre-resistance levels of susceptibility can potentially occur in the very-long-run if insecticide use is suspended.

Economic

1. Sustainable production, maximized long-run total economic surplus, and reduced long-run insecticide loads on the environment are important considerations in the regulatory decision-making process.

2. When considering resistance, a regulatory region can be defined as a contiguous geographic area in which the crop-pest complex of interest exists.

3. All available production and pesticide technology is given and assumed to be unchanging over the regulatory time horizon, thereby making this a worst-case analysis.

4. The economic damage caused by TABM is restricted to the growing and marketing season in which the infestation occurred.

5. Because of important multi-season effects on production, economic loss due to ERM may not be restricted to the growing and marketing season in which the infestation occurred.

Summary of Procedures

Long-run optimal insecticide use in an agroecosystem with developing pest resistance and potential insecticide regulation depends not only on current insecticide productivity and efficacy, but also on the productivity, quantity, timing, and efficacy of all future applications. Because the time-patterns of insecticide use, genotypic frequency, life-stage distribution, and interactions between predators and pests all affect the development of pest resistance, the problem of controlling pests under resistance/regulation conditions is inherently complex and

dynamic. In an attempt to address these complex dynamics, the modelling proceeds within a combined simulation and control theoretic framework.

The objective of this study's economic model is to maximize, over a given planning horizon, the total economic surplus associated with the U.S. apple market. Gross benefits at any time t are derived from the quantity and quality of expected harvest yield, and were generated with the use of an estimated supply and demand model of the U.S. apple market. Given the pest complex being examined, potential changes in total U.S. production only include that part arising from the pest damage in the mid-Atlantic region. Thus, production was assumed to be at steady-state for the rest of the country and the world. In keeping with the dynamic nature of the problem, maximization of the economic model was subject to the time-varying interactions of pest densities, predator density, pest susceptibility to insecticides, predator susceptibility to insecticides, immigration of susceptible pests and predators, environmental carrying capacities, and crop yield.

The biological characterization of the apple agroecosystem, including the evolution of pest resistance, was simulated using a modified version of the Pascal-coded computer program "Simulating the Evolution of Resistance of Arthropods (SERA)" (Tabashnik and Croft 1982, 1985; as modified by Knight 1990; Kazmierczak 1991). SERA embodies a flexible and biologically realistic framework that integrates user specified life history parameters, population genetic characteristics, migration patterns, and pesticide mortality functions for a pest-predator complex. From this information, SERA generates the time-path of resistance and population levels under user-specified insecticide application regimes. SERA has been shown to accurately reflect patterns of resistance development and will be parameterized for this study using experimental field data and expert opinion.

After parameterization, the economic and biological models were linked to form a comprehensive bioeconomic model. This linkage occurred by having the dynamic relationships embodied in the biological model form the basis for constraints in the economic model. Constraints could also be placed on the range of permitted pest controls, thereby allowing the examination of different regulatory policy options. The qualitative nature of the theoretical

solution to the problem was first explored to gain insight and provide structure for an empirical evaluation. Secondly, an empirical solution to the maximization problem was obtained by optimizing the bioeconomic model, with respect to total economic surplus, using a modified simplex search procedure (Azadivar and Lee). This approach yielded the optimal trajectory of the decision and state variables through time for any initial conditions, even though the model specification was not analytically tractable. In addition, non-optimal, heuristic decision variable trajectories were also used with the simulation model to generate state variable trajectories. These state trajectories were then compared to the optimal solution in order to discern the impact of deviations from optimal conditions.

Overview of the Dissertation

The remainder of this dissertation is organized as follows. Chapter II sets the stage for the problem addressed by examining how arthropod pest management is affected by resistance, including a description of the biological basis for resistance and some of the resistance management tactics that have been developed by entomologists. Chapter III considers the economic and regulatory forces acting upon and reacting to pest resistance. Chapter IV details the specific system used for the current investigation. Included is a brief discussion of potential arthropod management problems in mid-Atlantic apple production. Chapter V describes the theoretical model used to guide the empirical analyses, and explores the qualitative nature of the theoretical solution. Chapter VI begins an examination of how the theoretical model was transformed into an empirical model, focusing on the biological relationships in the model and the specifics of the agroecosystem simulation. Chapter VII presents the economic surplus framework used to guide the optimization analysis. Chapter VIII provides the linkages between the simulation and economic surplus models. In addition, a complete description of the optimization process is included. Chapter IX then presents and discusses the results of the analysis. Lastly, Chapter X offers a summary of the dissertation, conclusions concerning

the regulatory policy issues examined in the study, and provides suggestions for further research.

CHAPTER II: RESISTANCE AND MANAGEMENT

Agriculture has always been vulnerable to pest organisms during crop growth, harvest, and storage. Traditional attempts to prevent widespread pest damage focused on long-term approaches to whole-farm management and resulted in yearly shifts in the crop species grown, cultivation methods, cropping intensity, waste disposal, and soil conservation practices (Francis et al.). Labor intensive and implemented with family survival as the major objective, traditional pest management was effective enough to promote slow, steady growth in food production. But with the post-World War II introduction of synthetic chemical insecticides and other new technologies, agriculture experienced dramatic increases in crop yield and quality. Insecticides reduced the perceived need for diversified production practices as a pest control method and helped lead to farm specialization. This marked the beginning of modern commercial agriculture, with its wide geographic dispersion and large-scale, monocultural cropping patterns.

As might be expected, the initial success of synthetic insecticides led to their widespread adoption, and they were considered by many to be the final solution to agriculture's insect damage problems (Turpin and York; Dover and Croft). But, reliance on intensive monocultural cropping created tremendous opportunities for pest infestations and damage if a control agent suddenly lost its effectiveness. In essence, insecticides helped form a highly productive, but potentially unstable, agroecosystem open to substantial reduction or elimination of

marketable yield by pests. While it was obvious that the potential benefits of pesticides were accompanied by many costs, including undesirable environmental effects, endangered human health, and various long-run pest problems induced by pesticide use, most of these negative effects were not explicitly valued in markets and thus not included in the decision-making process of individual growers. The resulting symptoms of this market failure can be readily seen in the emergence of pest resurgence, non-target effects, and the rapidly expanding difficulties associated with pest resistance to insecticides.

Pest resurgence can occur following the use of any broad-spectrum insecticide. Although the chemical may be quite effective at controlling the target pests, its lack of toxic specificity can lead to the elimination of predator and/or parasite organisms that were exerting some control over the pest populations. Reduction in the magnitude of biological control produces conditions in which, following chemical applications, a pest can rebuild its population to levels higher than those existing prior to treatment. Secondary pests, or those that were previously not economically important, can also become problems as they are released from the competitive pressures that existed between them and the original pest target, predators, or parasites. This situation prompts the need for repeated control actions aimed at the target pest and promotes a positive feedback process that tends to amplify resurgence, non-target, and pest resistance effects (Balk and Koeman).

The non-target effects of insecticides can be broadly split into those related to direct impacts on farm-workers and farm families, and those related to residual impacts on off-farm human health and the environment. Because many insecticide formulations are prone to generating dust and fumes during handling and application, individuals that work with the chemicals on the farm are often subject to acute and chronic poisoning. In addition, inefficient or inappropriate application procedures can directly expose other farm workers, families, and rural residents to insecticides via spray drift or premature entry to the treated areas. Although protective clothing, modern application techniques and formulations, and re-entry regulations were designed to alleviate these secondary health effects, enforcement is often problematic due to the extensive use of pesticides, lack of training on the part of applicators, and

negligence (Environmental Protection Agency). Indirect residual impacts on the environment can also be traced to insecticides, with the most obvious symptoms being the poisoning of non-target birds and animals, contamination of surface water, and long-term seepage into groundwater reserves (Grue et al.; Batie et al.). Most residual impacts on the environment are not traceable to willful pesticide misuse, but instead result from an incomplete understanding of how a given pesticide reacts in the environment. The cases of DDT, aldrin, and dieldrin are examples where the lack of attention to the prospect of bioaccumulation led to long-term environmental contamination (Koeman). Another unintended and generally unforeseen impact of insecticides has been the accelerating emergence of pest resistance.

Pest resistance poses a long-term, dynamic threat to the economic viability of commercial agriculture, primarily because of the multi-seasonal interactions among pesticide use, pest evolution, and crop production practices. As will be discussed later, short-run profit-oriented pest management may accelerate the emergence of resistance and the economic collapse of the agroecosystem. In response, agriculturalists have attempted to develop control strategies that explicitly recognize and attempt to reduce the detrimental effects of pesticide use while protecting society's interest in maintaining high levels of quality production in the future. To help appreciate the difficulties involved in developing such a comprehensive approach to pest management, the remainder of this chapter:

1. Describes the biological basis for the development of arthropod resistance to insecticides, both in terms of the genetic mechanisms involved and the various selection pressures that propel resistance through identifiable stages; and

2. Examines some of the potential tactics and strategies that can be used to manage resistance in arthropod populations.

Pest Resistance

Resistance to control emerges in arthropod populations because insecticides often promote the change through an organism's physiological reaction to their presence and/or through the genetic changes arising from evolutionary selection pressures placed on the pest population. But resistance is not synonymous with control failure, for there can be a change in the response of a segment of the pest population to a chemical without immediately losing the ability to suppress the total pest population below economically damaging levels (Sawicki). In addition, the ability to develop resistance to an insecticide, the final level of resistance, and the speed of susceptibility depletion are not constant across all arthropod species. For example, despite the intensive use of DDT in U.S. corn production, the corn borer never showed evidence of emerging resistance. The common house fly, however, developed resistance to DDT in many areas of the country after only three years of exposure (Georghiou and Taylor). Even within a species, resistance can develop more rapidly in one specific population, as has occurred with the Colorado potato beetle in Long Island agroecosystems (Forgash). Of the many factors that can potentially affect the development of resistance, the most important appear to be the genetic, population, and management characteristics associated with a particular pest population (Table 1). While most of these characteristics are discussed in relation to the theoretical and empirical model in later chapters, the following two sub-sections give a concise explanation of how they can act to promote resistance.

Basic Genetics of Resistance

Standard neo-Darwinian theory suggests that evolutionary change results from the accumulation of many separate, random mutations. Each mutation is either maintained or eliminated from the genome by environmental selection pressures, with each individual mutation contributing infinitesimally to the total change seen in any population (Moore). Ac-

Table 1. Characteristics Influencing the Selection of Resistance to Insecticides.

GENETIC

- Frequency of resistance alleles in the population
- Number of resistance alleles in the population
- Dominance of resistance alleles
- Penetrance, expressivity, and interactions of resistance alleles
- Past selection pressure from other chemicals
- Extent of integration of resistant genome with other fitness factors

POPULATION

- Number of generations per production season
- Number of offspring per generation
- Mode of reproduction (monogamy/polygamy, parthenogenesis)
- Mobility (immigration, emmigration)
- Food source specificity (monophagy/polyphagy)
- Extent of fortuitous survival and refugia

MANAGEMENT

- Chemical nature of the insecticide
- Relationship to earlier-used insecticides
- Persistence of insecticide residues
- Specific formulation of the insecticide
- Application threshold for the population
- Selection threshold for the population
- Life stage selected via use of the insecticide
- Mode of insecticide application
- Extent of space-limited selection
- Extent of alternating selection

Note: Adapted from Georghiou and Taylor (1986), p. 158.

ceptance of this model would suggest that all arthropod species have the same potential to develop resistance to any insecticide, and that this adaptation should lead to a polygenic form of insecticide resistance.[2] But this "adaptationist" approach to general evolution has been strongly criticized (for example, see Lewontin and Gould; Gould, S.J.) and, even if valid for some cases, may not be an appropriate insecticide resistance model because most documented cases of resistance in field populations suggest the involvement of a single major gene (Whitten and McKenzie). In addition, some arthropods have demonstrated little or no ability to develop resistance to certain insecticides (as was apparent for the corn borer and DDT), although the Darwinian model would suggest that herbivorous arthropods should be preadapted for resistance development to pesticides due to their constant contact with toxic plant enzyme systems (Croft and Brown). Even those arthropods that do develop resistance tend to do so at a rate far faster than that predicted by the adaptationist model.

Instead of using an adaptationist model, recent studies of resistance development have focused on the regulatory gene hypothesis (Plapp 1986).[3] Insecticides operate by interfering with an organism's internal energy production, cuticle deposition, hormone action, and/or nervous system function. Thus, the emergence of resistance implies biological changes that reduce the penetration of the chemical into the organism, reduce the chemical's reactive availability, increase the organism's detoxification ability, or change the action site in the organism so that the chemical no longer binds in sufficient quantities to be lethal (Baillie). These biological changes can also interact to produce rapid resistance development or a high degree of resistance, as when reduced penetration promotes greater opportunity for detoxification. But the timing and nature of these changes, which reflect the underlying genetic structure of the population, appear to change too quickly and in a too consistent pattern to be proximately caused by the random mutation of Darwinian evolution. By asserting a role for regulatory genes, a model can be devised that provides a genetic basis for the emergence

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[2] Polygenic resistance is defined as resistance conferred by the expression of many individual structural genes, each providing some characteristic that enables the pest to resist control.

[3] Regulatory genes are most easily thought of as master genes that determine the final effect of other genes. Thus, regulatory genes act to control the expression of a population's genetic structure.

of resistance while recognizing the contradictions between observed resistance and the adaptationist model (Levin).

Regulatory genes appear to become involved in resistance development in one of two ways (Plapp 1986). First, a regulatory gene can lead to quantitative resistance by increasing an organism's bio-synthesis of compounds that promote some mechanism of resistance. This type of gene tends to exhibit all-or-nothing, fully dominant or recessive, inheritance. A second type of regulatory gene controls the specific nature of the compounds synthesized by an organism, thereby conferring qualitative resistance by increasing the resistance producing qualities per unit of synthesized compound. This type of gene usually exhibits intermediate, or codominant, inheritance. While the exact process by which these genes biochemically interact to produce metabolic and non-metabolic resistance is complex and outside the bounds of this study (for examples, see Oppenoorth; Lund and Narahashi; Kadous et al.), some important resulting characteristics, and their practical implications, are summarized below:

1. Although the precise biochemical mechanisms for resistance to insecticides are not yet known with certainty, most evidence suggests that a single gene locus is probably involved in a majority of resistance cases (Plapp 1986). If this is true, then population models of the inheritance and expression of resistance can be relatively simple and based on straightforward application of known genetic theory (May and Dobson).

2. Because the genes that lead to the detoxification of specific insecticides are probably not all located at the same site on a chromosome, observed cases of multiple and cross-resistance suggest that a common resistance controlling mechanism may be operating (Plapp 1986).[4] This allows models to be constructed on the basis of single regulatory

[4] If the genes leading to individual insecticide resistance are not close together on a chromosome, then the meotic process of gamete production, with its splitting and recombination of chromosomes and chromosome segments, would lead to the separation of resistance genes and a random distribution among individual organisms. But the presence of multiple and cross-resistance suggests either that this separation does not take place and that the genes really are close together, or that there is a single gene controlling the expression of different types of resistance.

genes, rather than on the basis of multiple regulatory or structural genes, each with its own insecticide specific action.

3. Alleles[5] that confer resistance are thought to be mildly deleterious to individual organisms prior to the use of insecticides, leading to low population frequencies for resistance alleles (10^{-2} to 10^{-4}) (Georghiou and Taylor). This deleterious effect of resistance when insecticide pressure is not present allows the possibility of reversion, or the return of susceptibility to populations after discontinuing insecticide use. Thus, the concept of fitness, or the differential survival and reproductive ability of organisms given specific environmental stresses, may be important when discussing the development and potential control of resistance.

4. Although resistant populations tend to exhibit lower fitness levels than susceptible populations when insecticides are not present in the environment, the resistant genome may coadapt[6] over time and lead to a stable, non-revertible resistance (Georghiou and Taylor). The tendency for this to occur, and the relatively low fitness disadvantages associated with most resistance, results in a reversion process that is usually many times slower than the original build-up of resistance.

5. Much resistance, and in particular metabolic resistance, appears to be due to the process of induction. In this case, the production of detoxifying agents is coordinated so that exposure to a chemical that gives rise to a specific detoxifier also induces the production of several other detoxifiers (Plapp 1984). This mechanism can lead to the development

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[5] As a simple definition, each gene can be thought of as composed of 2 parts, or alleles. With respect to resistance, each allele in a gene carries the genetic code for resistance (R) or susceptibility (S). Thus, for a quantitatively resistant regulatory gene where resistance is fully recessive, an allele sequence of SS or SR would indicate homozygotic (a gene composed of 2 identical alleles) or heterozygotic (a gene composed of 2 different alleles) susceptibility (respectively), while an allele sequence of RR would indicate homozygotic resistance.

[6] Coadaptation involves the inheritable association of resistant genes with other genes that confer survivability in general circumstances.

of cross-resistance, where resistance to a single chemical implies some level of resistance to other chemicals as well.

6. Because of the possibility of cross-resistance (regardless of its mechanism), past selection pressure from insecticides may lead to a faster development rate of resistance to new insecticides. In fact, certain mechanisms of resistance have been found to confer resistance to new insecticides that are not even chemically related to the original insecticide (Georghiou and Taylor).

This overview of the genetic basis for resistance gives some indication of the intricate biological mechanisms that allow pest organisms to change in response to attempted controls. These genetic changes occur in pest populations in response to the selection pressures generated by insecticides. But experience has shown that these selection pressures and the development of resistance are not haphazardly expressed. In fact, resistance tends to progress through relatively distinct stages.

The Stages of Resistance

When a synthetic insecticide is first introduced into an agricultural production system, control of target arthropods is often universal and dramatic. But control does not imply that all pest organisms are eliminated. Incomplete insecticide coverage, natural refugia, or tolerant life-stages may result in survival for a significant fraction of the pest population. In addition, there may be a number of individual organisms that, although assumed to be in a susceptible life-stage and exposed to the insecticide, possess a greater degree of fitness that allows them to survive (Gould, F.; Roush and McKenzie). It is from these few individuals that genetically resistant populations develop.

The temporal emergence of resistance can be divided into four more or less distinct stages (Conway and Comins):

1. An initial resistant and susceptible gene frequency equilibrium supported by mutation;

2. Rapid selection for resistance in an apparently susceptible population;

3. A mounting pest control problem where increasing insecticide dosages are needed to enact diminishing control; and

4. Abandonment of the insecticide as a control agent, perhaps followed by reversion of the pest population to susceptibility.

Each of these stages is characterized by a particular state of the pest population genome that is expressed in aggregate population behavior and physiology. Examining the stages in detail will help provide an understanding of how an initially effective pest control can end up promoting resistance.

In the first stage of resistance development, pest populations are highly susceptible to specific insecticide controls, often with low dosages and infrequent applications. The use of the insecticides may prove so effective at maintaining pest populations below economically damaging levels that growers come to regard it as a permanent cure for the particular pest problem being targeted. This was essentially the reaction to all synthetic pesticides in the two decades following their introduction. But as use of an insecticide continues, regulatory genes that might confer resistance to the specific insecticide become segregated in the pest population. In addition, the regulatory genes may become associated with genes for other characteristics that help the organism survive in the presence of the control chemical, thereby increasing fitness. Initially, this process is either unobserved in the field or hinted at by slowly falling control efficacy. As a result, it presents only minor problems, and growers compensate by marginally increasing dosages or shifting application schedules to exploit more susceptible life-stages. Historically, growers have also used insecticides prophylactically during this period on the assumption that the chemicals provide a form of insurance against pest damage (Carlson). As insecticide use intensifies, genetic segregation continues and a readily ob-

served sub-population of the pest emerges that can only be controlled through progressively higher dosages or more frequent applications (Balk and Koeman). This emergence of a resistant sub-population, and the move towards dramatically increased insecticide use, marks the initiation of the second stage of resistance development.

The second stage of resistance development is primarily characterized by an increasing level of selection pressure against those pest individuals that are less fit for an environment with insecticides. During this time, resistant individuals may experience reduced competition for food and habitat as the majority of the susceptible pest population is eliminated. The broad-spectrum action of most insecticides may also help reduce pressure on the emerging resistant sub-population by eliminating the natural enemies of the pest. Survival of the resistant sub-population and its ability to escape control depends, to a large extent, on the degree of its reproductive isolation and success. If the sub-population is large enough to prevent constraints on the location of mates, and if resistance does not also imply drastic reductions in the efficiency of reproductive physiology, then the resistant sub-population will rapidly expand even as compensatory insecticide use increases. This marks the beginning of the third stage of resistance development.

In the third stage of resistance, extensive insecticide use and increasing control failures predominate. Although resistance in the majority of the pest population accounts for this, questions have been raised as to why natural enemies of the pest do not also develop resistance and provide some control. In fact, some natural enemies do become resistant or were originally tolerant to the insecticide. These species are often employed, along with chemicals, in integrated pest management systems (Croft and Bode). But most natural enemies do not develop resistance as rapidly as pest arthropods. Two main theories exist concerning this puzzling observation (Croft and Morse):

1. As suggested earlier, herbivorous arthropods may be preadapted to detoxifying insecticides because of their constant exposure to toxic plant chemicals. This mechanism could be the result of a common regulatory gene being used to promote the synthesis of

more than one type of detoxifying enzyme. Under this scenario, natural enemies do not have the same kind of rapid-response regulatory genes because they do not feed on plants directly, but on the pests that have already detoxified the harmful plant chemicals.

2. During the initial stages of insecticide use, natural enemies that avoid contact with or are tolerant of the chemical may suddenly be faced with a drastically reduced food supply. Under intense competition, entire populations of natural enemies may be eliminated through starvation, emigration to more suitable habitat, or reproductive failure due to low energy intake or the inability to find mates as total numbers decrease. In addition, natural enemies tend to have lower intrinsic rates of reproduction than the pests that serve as their food source. Thus, once removed, natural enemy populations will reappear only to the extent that immigration or artificial release programs are large enough to provide a viable base population that can respond to the gradual increases in the resistant pest sub-population. Unfortunately, natural enemies are rarely efficient enough to provide complete control of pests in monocultural systems, and developing pest resistance often leads to the use of alternate insecticides that the natural enemy cannot avoid or to which it may not be tolerant. This can effectively prevent the use of natural enemies and eliminate the viability of an integrated pest management program.

Regardless of the cause for maintained susceptibility in natural enemies, their inability to control pest populations in which resistance is developing, along with increased insecticide use and high selection pressure for resistant pests, leads to a crisis where the insecticide can no longer provide reliable control and prevent economic damage. At this point, the fourth stage of resistance is reached, entailing the abandonment of the insecticide. Once the pest is no longer subject to the selection pressure provided by the insecticide, it is possible for susceptibility to gradually reappear. However, this is likely to occur only if the fitness of the resistant pest is lower than that of susceptible individuals that might immigrate into the previously treated area.

Given that resistance appears to be intimately linked to shifts in the genetic composition of the pest population, there are a number of biological factors that may be important to addressing the resistance problem. These factors include the frequency of the gene conferring resistance, the dominance of resistant genes, the fitness of resistant individuals, the generation time of the pest, the number of offspring per generation, the mode of reproduction, the extent of pest mobility, the specificity of any natural enemies, and the availability of refugia within the treatment area. However, none of these characteristics are easily manipulated to forestall or eliminate resistance development. But there are also a number of other management factors that can directly affect the speed and magnitude of resistance development, such as insecticide persistence, dosage, targeted life-stage, treatment threshold, use of biological controls, and the use of multiple, mixed, or alternate applications.

Tactics For Managing Resistance

The potentially serious consequences of wide-spread agricultural pest resistance has promoted considerable effort into the development of resistance management strategies (for example, see Georghiou and Taylor 1977a, 1977b; Comins 1977, 1979, 1986; Croft and Morse). In fact, it was the stimulus of resistance that provided much of the support for the development of early integrated pest control programs (Croft; Hoyt). Most recent investigations have concentrated on prospects for decreasing the rate and level of resistance development in pests, although promoters of integrated pest management systems have also sought ways to increase resistance in natural enemies. Because resistance development can be conceptualized as a sequence of stages, management strategies have generally tried to target specific stages where resistant sub-populations are still small and perhaps vulnerable (Keiding; Brown).

Theoretical models and laboratory experiments have demonstrated that the rate of resistance can be directly linked to the degree of resistant gene dominance, the level of resist-

ant gene frequency, and the comparative fitness advantage of resistant genotypes (Futuyma; Georghiou and Taylor 1977a). This suggests that resistance might be controlled or delayed by an intervention that prevents the emergence of these resistance-linked characteristics. But until recently, the selection of management strategies and tactics has depended on past field experience, with success seemingly based on intuition and luck (Leeper et al.). Recent advances, however, indicate that resistance management might be obtainable through planned manipulation (Georghiou and Taylor 1977b; Comins 1977, 1979). This process could result in pest populations that might initially show increases in resistance to an insecticide, but that would then revert back rapidly or frequently to susceptibility so that resistance never entered the third, or crisis, stage (Croft and Bode). The factors that might be manipulated include the application and selection of treatment thresholds, the mode of insecticide application, the spatial patterns of selection that result from the insecticide applications, and the particular pattern of insecticide use (Georghiou and Taylor 1986).

The process of resistance management can be divided into one of two general categories; 1) attempts to indirectly manipulate the fitness of resistance in pest populations by altering natural population ecology, or 2) attempts to directly influence the development of resistance through the pattern and timing of control actions. Indirect approaches include many "soft" biotechnological methods, such as the use of sterile pest releases, mass releases of susceptible pests, mass releases of resistant natural enemies, *in situ* alteration of pest fecundity, hormonal attraction of migratory susceptible pests, and/or the cultivation of crops genetically engineered to resist damage by pest organisms (Proshold et al.; Karban and Carey; Leeper et al.; Curtis et al.). Also included in this group would be the application of chemicals that stimulate the plant's natural defenses and the quarantining of resistant populations in order to prevent their spread (Leeper et al.; Sutherst and Comins). These methods focus on treatment thresholds, either by trying to slow the increase of pest populations and total insecticide use, or by attempting to keep the frequency of resistant genotypes below levels that require alternative controls. Although these indirect techniques have much intuitive appeal, particularly at a time when the use of synthetic chemical controls is undergoing

intense public scrutiny, their acceptance has been limited because many have yet to be proven to work effectively in commercial production systems. In fact, most attempts to indirectly manipulate the biological characteristics of pest populations have been operationally disappointing, with future success dependent on advances in low-cost resistance detection, pest population monitoring, and mass arthropod breeding techniques (Leeper et al.).

With the commercial application of indirect resistance management still to be perfected, perhaps the most promising approach involves the direct manipulation of resistance through sophisticated pesticide application programs. From the standpoint of implementation, this approach to resistance is likely to gain the greatest acceptance in agriculture because it utilizes familiar insecticide controls and can potentially be effective without causing any significant decreases in crop yield or quality. The primary rationale behind these methods is the observation that the reproductive spread of resistant alleles can only be reduced by: 1) preserving the sub-population of pests that are susceptible homozygotes, thereby providing a breeding pool of controllable pests that will act as a dilution factor to prevent or delay the emergence of resistance as the major genotype in the overall pest population, or 2) eliminating, to the fullest extent possible, heterozygotes and resistant homozygotes, thereby preventing the resistant genotype from establishing a reproductively viable sub-population. In essence, management effort is centered on changing the effective dominance of resistance (Curtis et al.). This manipulation acts to either support a susceptible sub-population, or to create an environment where normally resistant organisms are less fit for survival. This approach to resistance management generally uses insecticides in sequential, mixed, rotational, mosaic, low-rate, and/or high-rate application schedules.

Sequential insecticide application involves the progressive use of chemicals, from those conferring the lowest levels of simple resistance with limited cross-resistance potential, to those conferring high levels of complex resistance and cross-resistance. Studies suggest that this approach tends to minimize the selective advantages of resistant genotypes (Devonshire and Moorse) and may provide the maximum possible delay in the development of uncontrollable pest populations (Keiding). If the number of generations or length of time between the

use of the initial insecticide and complete control failure is long enough to allow resistance to decline below a critical frequency, then the sequential use of the chemicals can begin again and, in principle, forestall complete control failure indefinitely (Georghiou; Georghiou et al.). The problem with this approach, however, is that its applicability is generally recognizable only in hindsight because complete information on the nature of resistance to all chemicals is rarely known before their wide-spread use (Sawicki).

Insecticide mixtures, or the simultaneous application of more than one insecticide, are another potential way of managing resistance. In this approach, each chemical used in the combination has a different mode of action, and they are all applied at levels high enough to individually lead to effective control. The main assumption behind this method is that most pests will not have the ability to develop resistance to more than one form of control at a time. Thus, individuals surviving exposure to one insecticide are likely to be killed by another in the mixture (Georghiou). In addition, insecticide combinations may act synergistically, rather than merely additively, to delay resistance even longer than expected (National Research Council 1986). But, in general, this approach will only be biologically successful if there is substantial immigration of susceptible pests into the agroecosystem or if mass releases of susceptibles occurs after control treatments. If not, then the small number of surviving individual pest organisms can potentially develop into multiple-resistant populations, a situation that can also occur if reduced dosages of each insecticide are used in the mixtures (Sutherst and Comins). These problems, along with the obviously increased use and cost of chemicals, makes this mixture approach to resistance management particularly risky.

In much the same way as mixtures, insecticide rotations can be used to reduce resistant allele frequencies in a pest population. In this approach, the same chemical control is never applied twice in a row, but instead is alternated with other effective chemical controls. By doing so, pressures are, in theory, constantly shifted for selection of different genotypes. Of course, this assumes that the likelihood of a single genotype conferring resistance to more than one or two chemicals is small. Like mixtures, this process will only work if the controls have sufficiently different modes of action and if susceptible immigration occurs following

treatment. In addition, it assumes that resistant genotypes have substantially lower fitness, and thus a rapid reversion potential, compared to susceptibles (Georghiou). Although resistance to an insecticide can decline during the chemical's absence, the rate of decline may be too slow for any practical benefit unless negative cross-resistance effects are also present (Georghiou et al.; Roush and Plapp).[7] However, negative cross-resistant effects have yet to be demonstrated in non-experimental, field conditions (Sawicki).

Perhaps the most controversial of the potential direct resistance management tactics are those of mosaic, low-rate, and high-rate pesticide applications. Mosaic pesticide applications involve deliberately not treating a certain proportion of the crop or field with the intent of creating refugia for susceptible homozygote pests. This method attempts to provide a geographic reservoir of susceptibility from which emerging resistance in the treated population can be diluted. Untreated areas can also serve as refugia for the pest's natural enemies, and may reduce the need for some future applications (Tabashnik and Croft 1982). In addition to mosaic applications, both geographic and temporal refugia can be created through the use of higher action thresholds, limited-residual chemicals, highly pest selective compounds, and/or non-chemical pest controls (Georghiou and Taylor 1986; Denholm et al.).

In contrast to attempts at providing refugia for susceptibility, the tactic of high-rate pesticide use attempts to eliminate all potential refugia while at the same time applying sufficient levels of the control to eliminate both heterozygotes and resistant homozygotes (Taylor and Georghiou). Problems arise, however, when incomplete information on the nature of resistance and its frequency in the pest population prevent elimination of the pest from the treatment area. This increases the likelihood that high-dose approaches will only increase the selection pressure for resistant genotypes, thereby leading to the more rapid development of uncontrollable pest populations. In addition, the intrinsic uneven distribution of insecticide application leads to low dosage areas even in high-dose applications. Because of this uncertainty, high-dose approaches need to be coupled with mass releases of susceptible pests

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[7] Negative cross-resistance occurs when the use of a specific insecticide reduces the potential for the development of resistance to some other insecticide even while it may be promoting resistance to itself.

in order to dilute potential resistance, a tactic that is not only expensive but also counter-intuitive given the severity of the high-dose controls.

As is obvious from the above discussion, most resistance management tactics have some flaw that renders them a potential mechanism by which resistance could increase. Most of these difficulties are related to the fact that the timely and accurate detection of resistance is currently problematic, making the choice of a resistance management strategy somewhat ad hoc. But equally important, and often overlooked, is the fact that the success of a resistance management tactic is not only dependent on its technical efficacy, but also on how it effects the economic decision-making process of the grower and the long-term operation of the agroecosystem. Commercial implementation of any specific management program critically depends on its economic viability and on the nature of pest susceptibility as a common-property resource. Given that common-property resources are subject to sub-optimal market allocation, it is important that the economic implications of any resistance management strategy be explicitly considered. Therefore, following a brief summary, the next chapter will concentrate on the economics of resistance management.

Summary

In discussing the biological basis and management tactics for arthropod pest resistance, a number of important concepts were introduced. These ideas, listed below, will be utilized in the discussion of the economics of resistance and in the chapters devoted to the development of theoretical and empirical models.

- Arthropod resistance to insecticides is genetically based and capable of being represented by a single gene locus, two allele model.

- Resistance develops at different rates in different species, and is linked to a number of biological and operational factors (Table 1).

- Although possibly a renewable resource, the replenishment of a stock of pest susceptibility to a given insecticide usually occurs at an extremely slow rate.

- Given the limited number of chemicals from which insecticides are formed, cross-resistance and multiple-resistance are critical factors to consider in a resistance management program.

- Effective resistance management may require action before resistance clearly manifests itself in the form of control failures.

- Although indirect means of managing resistance through the alteration of population ecology may prove cost-effective in the future, current technology appears to favor direct management through manipulation of insecticide application regimes.

- Even with direct resistance management, certain behavioral and ecological characteristics of a population, such as the rate of migration for both pests and natural enemies, can critically affect the success of a management program.

CHAPTER III: ECONOMIC AND REGULATORY DIMENSIONS

Pesticides have become increasingly important in U.S. agriculture. Total use of pesticides in the U.S. has risen from 540 million pounds of active ingredients in 1964 to over 1 billion pounds in 1985. Agriculture's intense use of this input is reflected in the fact that U.S. growers annually apply over 75 percent, or 770 million pounds, of the active ingredients (Environmental Protection Agency). Pesticide expenditures account for 2 to 23 percent of total variable costs of production, depending on the crop, geographic location, and the severity of pest problems in any particular year, with U.S. producers having spent over $4.6 billion on pesticides in 1985 alone (Stemeroff and Culver; Environmental Protection Agency). But the economic importance of pesticides is not restricted to growers. Thirty major companies produce most of the basic active ingredients used in the U.S., with over 3,000 separate companies generating formulations and 29,000 companies operating as pesticide distributors. More than 11,000 people are employed in the physical production of pesticide chemicals, a figure that does not include research and development personnel or the tens of thousands employed as formulators and distributors. In addition to the normal economic activity generated by such a large industry, pesticide producers spend over $400 million annually on research and development, of which $100 million is directed towards generating the data required by the EPA for registration (Environmental Protection Agency). Taken together, these statistics suggest that pesticides, and their use, are an important component of agricultural

and rural economies. Thus, regulation and/or restriction of pesticide use needs to be carefully considered, as the economic impacts of these decisions may be extensive.

Similar to other agricultural inputs, pesticides are used to enhance crop productivity. In the case of insecticides, this is generally accomplished not by directly stimulating crop growth, but by protecting the crop from pest damage and thus helping to provide optimal growing conditions. The productivity gains attributable to pesticide use are commonly thought to be above the average gains for other agricultural inputs, especially in large commercial operations. Some studies have suggested that private economic returns to pesticides average as much as 300 to 400 percent, with specific high-value crops such as apples showing pesticide rates of return perhaps as high as 1,300 percent (Pimentel et al.; Stemeroff and Culver). Aggregate monetary gains from pesticide use may also be substantial and in the hundreds of million dollars for any single pesticide/crop combination (Krystynak; Dunnett). Of course, the total public plus private rates of return from pesticide abatement programs such as integrated pest management may also range between 300 to 500 percent for some crop systems (Napit et al.). But from a psychological viewpoint, growers may tend to use pesticides as a form of crop insurance, or actually be compelled to use them by the lending institutions that finance their operations (Carlson; MacIntyre). In addition, the influence of the agrichemical industry's sales promotion and advertising campaigns can make the idea of not using pesticides seem ridiculous (Stemeroff and Culver; Jordan).

Although the arguments for and against continued pesticide use are numerous, with each having persuasive elements, decisions concerning the intensity of use often appear to be based on custom or emotion and without adequately considering the long-term, dynamic impacts of the chemicals. In particular, the use of static economic analysis may be invalid when the agroecosystem is subject to dynamic biological fluctuations such as emerging resistance. In an attempt to address part of these deficiencies, this chapter will

1. Examine why traditional static economic analysis is insufficient when dealing with problems concerning pesticide use;

2. Detail the economic implications of resistance in an agricultural production system, both as it pertains to individual insect species and with respect to the presence of multiple-species complexes;

3. Present some possible linkages among resistance biology, the economics of resistance, and attempts to regulate the use of insecticides; and

4. Set the stage for an examination of a specific pest management problem where resistance and pesticide regulations play an important role in the dynamic evolution of the agroecosystem.

Pesticides in the Static Neoclassical Model

The introduction and adoption of synthetic chemical pesticides was widely expected to help eliminate pest damage in commercial agriculture. In order to accomplish this goal, decisions needed to be made with respect to type, timing, and dosages of the pesticides used. Producers incurred real expenditures when using pesticides, and thus they needed to know how and when to apply the chemicals most effectively and at the least cost. In essence, there was a need to determine the relationship between pest levels and economic damage, as well as a way of relating current pest population levels with the need for control actions (Tillman and Baum). Not surprisingly, these needs were initially addressed by the entomologists and plant pathologists responsible for developing pest management programs.

One of the first attempts to relate pest population levels with pest damage and the need for control action came packaged in the concept of "economic thresholds" (Stern et al.). These authors defined economic thresholds as the density of a pest population that would justify control action, with justification subsequently interpreted as the population level where the benefit of treatment just exceeded the cost of control action (Bierne). Economic thresholds

as break-even points, or the levels at which it was desirable to initiate control actions, became widely used by biologists as the basis for decision-making rules (National Academy of Sciences). The major reason for their initial success was the relatively small base of information needed concerning the simple agroecosystems to which they were applied. But it rapidly became apparent that this approach to pest control suffered when the agroecosystem was complex, when control effectiveness was uncertain, or when multiple control actions were needed over the course of a growing season (Mumford and Norton). In fact, it was the seemingly *ad hoc* methods of determining dosage and timing under this original concept of economic thresholds, and concern about how this might affect the economic efficiency of pesticide use, that prompted agricultural economists to introduce marginality concepts into the development of pest management programs.

A critical examination of pest management from a marginalist framework began in the late 1950s. Analyzed for their impacts on the production system, pesticides were treated as normal technological innovations. Economists used production theory concepts to find optimum pesticide dosages, or those where the ratio of pesticide cost to product price equaled the incremental gain in output due to pesticide use (Hillebrandt). Using this approach to the pest management problem, it rapidly became apparent that there were two important pest population levels that needed to be identified: the economic-injury level and the economic threshold. The economic-injury level was defined as the lowest pest population level that would cause economic damage, while the economic threshold was redefined as the pest population level at which a control action must be taken in order to prevent the pest population from reaching and exceeding the economic-injury level (Tillman and Baum). Within this framework, the economic threshold clearly became an action or decision point that would always be less than or equal to the economic-injury level. Used in this way, it also served to formalize the principle that pests can and should be tolerated as long as the expected losses were less than the additional costs of controlling the pest (Fohner et al.). Thus, the economic-injury level, and its corresponding economic threshold, are not only dependent on the biological status of the agroecosystem, but also on the prevailing economic conditions

surrounding crop production and marketing. The economic threshold became responsive to and determined by product and input prices, and it was possible that it would be at levels higher than those at which yield damage first occurred (Headley). In addition, the economic threshold could change not only through a season, but also between seasons, as the biological and economic state of the agroecosystem varied.

Needless to say, the economic definitions of pest injury-levels and thresholds led to an identification process requiring a knowledge of pest biology, ecology, damage relationships, and economics. Although more complicated, this definition was fundamental to the development of integrated pest management programs and has been successfully used in practice to determine whether pest densities warrant control actions (e.g., Apple et al.; Huffaker). This approach, because it is rooted in economic theory, has provided a flexible framework to investigate pest management and pesticide regulatory problems that were beyond the reach of the earlier models. The sophistication of these studies has expanded over the years to include investigations of risk-efficient thresholds for pest control decisions (Moffitt), the role of information in pest control (Stefanou et al.; Szmedra et al.), and the effects of input externalities on optimum pest control decisions (Harper and Zilberman). In all of these studies, a common assumption has been that pest control decisions were either individually or seasonally separable in time, thus making the problem approachable using static production theory.

Traditional static production theory assumes that a producer's short-run objective is profit maximization, with the set of obtainable profit levels being constrained by the technology employed and the levels of factor inputs that are assumed fixed for the production period. If a single-output production function (q) is defined as representing the maximum set of outputs given full use of the available technology (Ω) and the fixed inputs (Z), then the grower's decision process can be portrayed as a search to find the specific set of variable inputs (X) that will maximize enterprise profit (π). By defining profit as gross product revenues (pq) minus total variable costs (WX), or return to the fixed factors of production, then the grower's decision process can be expressed as

$$\underset{X}{\text{MAX}} \quad \pi(X, Z, \theta) \ ,$$

$$\text{subject to} \quad \pi(X, Z, \theta) = pq - WX \ ,$$

$$q = f(X, Z, \Omega) \ ,$$

$$\text{where} \quad \theta = h(p, W, \Omega) \ .$$

The solution to this simple optimization problem can be found mathematically using ordinary calculus. If $f(X, Z, \Omega)$ is monotonic, quasi-concave, and continuous in X, indicating that an interior solution exists, then the optimum level of variable input use (X^*) that produces maximum profit (π^*) is given by the set of first-order conditions

$$p \frac{\partial f}{\partial x_i} = w_i \quad \forall \ i$$

or, after solving for x_i^*,

$$x_i^* = k(p, w_i, Z, \Omega) \quad \forall \ i \ .$$

When firms use the input quantities defined by X^*, they are allocatively efficient. If the firm is able to reach the production frontier, or the maximum amount of output given the available technology, by using any level of X, then it is defined as being technically efficient. A firm that reaches the production frontier by using the variable input level X^* is said to be economically efficient. Although this model of grower decision-making is simplistic, it can be used to demonstrate the traditional economic rationale behind pesticide use.

Consider an agricultural producer in the 1940's. Having adopted a full range of technological innovations before the second world war, this producer could have been operating in an economically efficient manner, producing and earning profits consistent with π^*. But with the war ending, a new product, synthetic pesticides, appeared on the market. While the concept of chemical pest control was not new, pesticides had never been completely reliable, broad-spectrum, or particularly safe. This new technology, however, promised greatly im-

proved pest control. How might this change have affected the producer's decision-making process?

There are two extremes within the range of options available to the producer; 1) adopt no new pest control methods, or 2) completely and immediately adopt the new pest control methods. If the producer chose the first course of action, then in the short-run his production function, maximization problem, and profit outlook would remain essentially unchanged. However, he would no longer be operating in a technically or economically efficient manner because the change in pesticide technology had pushed out the production frontier. It is conceivable that the producer would still be allocatively efficient, but he would be foregoing potentially available profits. In the long-run, other producers that did adopt the new technology would increase aggregate production, thereby leading to a decline in output prices, ceteris paribus, and a decline in the profits obtainable by the non-adopter.

The second course of action available to the producer was to be one of the first individuals to adopt the new pest control technology. Doing so would allow his operation to remain economically efficient, and would yield higher short-run profits until aggregate production increased enough to force down price. To see how this would come about, consider the following example.

Assume that the pesticides used in the pest management process were unaffected by inputs to production used in processes other than pest management. Within the neoclassical framework, this implies a separable production technology[8] where the marginal rate of technical substitution between non-pest management inputs are invariant to changes in inputs used in pest management processes:

$$\frac{\partial}{\partial x_p} \quad \frac{\partial f / \partial x_i}{\partial f / \partial x_j} \quad = \quad 0$$

where

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[8] Strictly defined, a separability based on partition of the input vector X into sub-vectors associated with distinct processes, at least one of which contains more than a single element, is termed weak separability.

x_p = level of pest management inputs, and

x_i, x_j = level of any non-pest management inputs.

Thus, separability implies that the production process can be broken down into stages, so that at each stage some inputs are used to make intermediate inputs.[9] When analyzing pest management activities, economists have made use of this theoretical construction by considering pest management a production sub-process from which service flows are generated to the overall production process (Antle). From this viewpoint, pest management inputs are used in the production of the reduced-pest environment required for the crop production process, and the modelling and measurement of pest management can be separated from concerns with other activities.

Given the separable pest management production technology and a positive marginal product for pest management inputs ($\frac{\partial f}{\partial x_p} > 0$), an increase in the technical efficiency of the pest management process and/or the introduction of improved pest management technology means that more service flows are generated from a given input set. This higher technical efficiency implies increased output and profits for any set level of non-pest management inputs in the overall production process. For example, if m^o denotes the old-technology pest management production function and m^n the new-technology function, such that

$$f[m^n(x_p), \ldots] > f[m^o(x_p), \ldots] ,$$

then for prices taken in a competitive market,

$$p\frac{\partial f[m^n, \ldots]}{\partial x_p} > p\frac{\partial f[m^o, \ldots]}{\partial x_p} ,$$

or the value marginal product (VMP) of pest management inputs to overall production is shifted to the right by the new pest control technology. Because the area under the VMP curve

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[9] In fact, it also implies that in the first stage all "micro-production" functions are independent (Chambers). Although it may be difficult to identify a real agricultural system where this would occur, separability as a simplifying construction has proven to be analytically useful.

represents the total revenue that an input generates, the producer adopting the new technology would increase returns to fixed factors whether he continues to use the input at the old level or intensifies input use (Figure 1). But, in fact, it is the increase in the marginal productivity of the new technology's inputs relative to old technology and non-pest management inputs that encourages their use.

Given the new technology's potential effects on short-run profits, producers would appear to have had an economic incentive to use pesticides. But most producers did not immediately adopt new control technologies. Although the reasons behind this are complex and include various aspects of uncertainty, non-uniform information flow, and grower psychology (Miller; Rogers), it can generally be said that most producers adopted in response to the observed output increases accruing to earlier adopters. As this process continued, the decline in output price caused by the increases in aggregate production tended to reinforce the pressure on non-adopting producers to use the new pest control technology to boost production, even though much of the excess economic benefit for doing so had already been captured by the early adopters.

The above economic argument for pesticide use was instrumental in the development of pest management programs in the U.S. Pesticides were seen as yield protecting inputs, with the only problem being the determination of appropriate applications. However, hidden by these simple analyses was the assumption that pesticides had no external effects not explicitly valued by the market. In particular, the static production theory approach assumed that: 1) the decisions made in each producer's enterprise were completely separable from those made in all other enterprises; and 2) that the impacts of input use did not include fundamental changes in the state or operation of the agroecosystem. But, as the emergence of pest resistance to insecticides has shown, both assumptions were invalid.

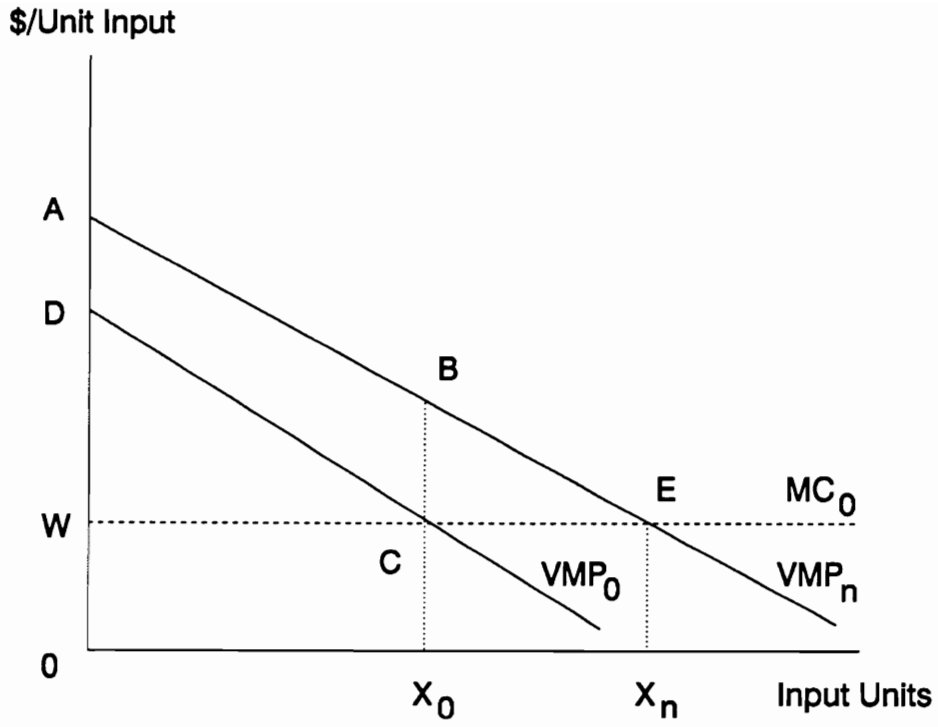

Figure 1. Effect of New Technology on Value Marginal Product and Producer Surplus: Adapted from Antle, p. 21.

Common Property and Dynamic Aspects of Resistance

At the core of difficulties associated with the economically efficient management of agricultural pests is the fact that most pest species do not remain on a single producer's land. As a result, no single producer can claim and enforce ownership of a well-defined set of pests. This is especially relevant for arthropods, whose passive and active migration leads to a situation where a single pest population[10] can cover a large geographic area. While it is true that few producers desire to have property rights to pest populations, it would be in their interests to have rights to a pest population's stock of susceptibility. Without rights to susceptibility, or cooperation on the part of other producers, pest mobility implies that actions not directly controlled by an individual producer can affect the outcomes that a producer attains by the actions he does control (Dasgupta). Thus, control decisions cannot be efficiently made without considering the control decisions of other producers. But in practice, the magnitude of transaction costs makes it nearly impossible for a single producer to integrate, unaided, the decision-making of all other producers who use a pest population's stock of susceptibility. Pest susceptibility, as a common property resource, becomes nonexclusive in the sense that no producer can be prevented from using it. If the resource is degradable, as susceptibility usually is, then it can be demonstrated that common property characteristics will likely lead to its over-utilization and inefficient allocation by the market (Starrett). In short, the externality effects of pesticides are not valued in the market, not accounted for in producer decision-making, and not efficiently utilized. This leads to a divergence between the single producer's optimal pest control policy and the optimal policy for society as a whole.

Although the link between the emergence of resistance and the common property characteristics of pest susceptibility have been recognized for some time, only recently have researchers attempted to address the economic efficiency issues surrounding the phenomenon.

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[10] It may be useful to remember that a single species population can be technically defined as composed of those individual organisms that have the potential to mate and produce viable offspring. Thus, populations may be separated from each other due to geographic or time barriers.

From a theoretical perspective, the problem is well understood (Dasgupta). When the genetic changes that a pest population undergoes in response to pesticide use are hidden from direct view, and when it takes a number of years for clearly observable patterns of resistance to manifest themselves, then individual producers may have little or no information about the specific externality effects of pesticides to include in their decision-making. Moreover, even if the information was available, individual producers would not have an economic incentive to acquire that information or use it in their decision-making, for if they did so alone, they would end up foregoing potential profits without having any guarantee of reducing the aggregate negative impacts of resistance. But by ignoring the affects of pesticides on the common property of susceptibility, an intertemporal externality is imposed in the form of increasing resistance and the future control difficulties it implies.

Because of the common property characteristics of pest susceptibility, and the resulting potential for resistance to develop, the search for a socially optimal use of insecticides depends on a comprehensive knowledge of their productivity over time. Optimal decision-making must include consideration of both application dosage and timing within and across seasons (Knight and Norton). In general, this calculation will not occur in a free-market situation because the intertemporal resistance externalities are, by definition, not explicitly valued. In terms of dosage, private decision-making will result in insecticides being applied until marginal benefit is equated to apparent marginal cost, as was true in the static production case discussed earlier. But when the development of resistance is biologically possible and not included in marginal cost, then insecticides are over-utilized and their productivity, as measured by a response curve, continually shifts down (Figure 2). This leads to a situation where the profit-maximizing producer uses increasing amounts of the insecticide to produce a decreasing amount of output. If the producer attempts to maintain output at pre-resistance levels, then pesticide use can be expected to increase even more drastically, depending on the magnitude of productivity changes, and hasten the onset of control failure.[11]

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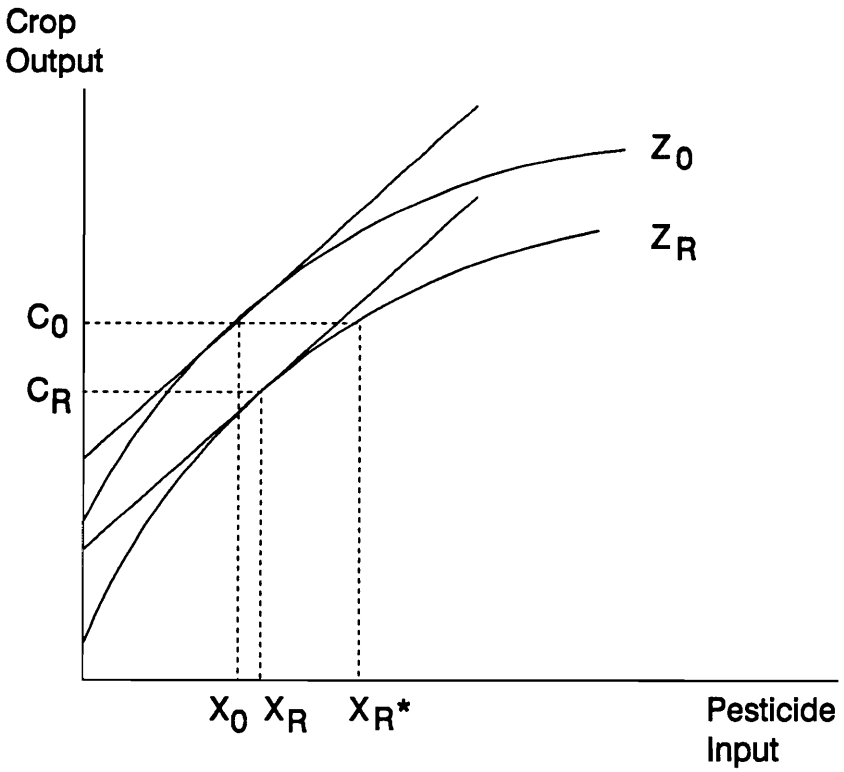
[11] It is theoretically possible for insecticide use to increase and not lead to control failure if a successful high-dose strategy is being employed.



Pesticide productivity, as measured by the response curve, before (Z_0) and after (Z_R) the development of resistance. Given constant relative factor prices, the onset of resistance will cause the profit-maximizing producer to increase pesticide use ($X_0 \rightarrow X_R$) even as crop output falls ($C_0 \rightarrow C_R$). If the producer attempts to maintain old levels of output with resistance, pesticide use will increase greatly ($X_0 \rightarrow X_{R^*}$).

Figure 2. Changes in Pesticide Use and Productivity When Resistance is Present: Adapted from Knight and Norton, p. 301.

Although the theoretical effects of resistance on the long-run dosage use of an insecticide can be examined from a static framework, a more sophisticated model is needed to examine how a producer might change pest control behavior if forced to consider the effects of a control action on the stock of susceptibility. In general, attempts to advance beyond static models have focused on the optimal timing of insecticide applications. This dynamic aspect of pest management with resistance is important because insecticide productivity at any given time is dependent not only on how previous intra- and inter-seasonal insecticide use has affected pest population levels and crop conditions, but also on how the history of chemical use has degraded the stock of pest susceptibility to the control. In addition, when a dynamic economic model is employed to examine resistance, explicit recognition of the time value of money needs to be made. The complexity of these two issues has proven to be challenging even for theoretical examination of dynamic pest management without resistance. Thus, in order to detail the important characteristics of the problem, the following discussion will first concentrate on the general dynamic studies that abstracted from common property issues before reviewing the research that has explicitly considered resistance.

Early studies examining the dynamics of pest management usually focused on how pesticide applications might be timed to control obviously non-static pest populations. These investigations were mostly theoretical in nature, both due to the complexity of the issues involved and the lack of data from which to build an empirical model. For example, Chatterjee developed a dynamic model that was used to find the optimum frequency, intensity, and timing of control actions. In keeping with the author's desire to include as much realism in the model as possible, control actions were allowed to vary between time periods and explicit recognition was made of the tendency for pesticides to have residual, multiperiod effects. In addition, Chatterjee acknowledged the societal interests implicit in pest management systems by minimizing all costs associated with a control program, including the losses attributable to crop damage, program administration, and the impact of the control agent on the environment. As might be expected given the wide range of factors included in the analysis, the author's representation of the agroecosystem and its responses to control actions was highly stylized

and could not be related to any specific system. Nonetheless, the study was important for demonstrating that the complexities of pest management could be examined within the framework of a dynamic model, and it pointed out the important economic and biological characteristics that needed to be considered.

Concurrent with the Chatterjee study, Hall and Norgaard published a theoretical paper that confirmed the need to define thresholds on the basis of pest population levels at control initiation, not on the levels to which pests were to be reduced. This need was related to the observation that agroecosystem complexity and dynamics led to biological response lags that prevented the instantaneous control of pest populations. The authors also demonstrated that, for modelling purposes, a pesticide's effectiveness needed to be directly related to pest population levels, as pesticides tend to become progressively more effective, on a percentage kill basis, as pest populations increase. This observation implied an inherent trade-off between the cost of control actions and the willingness to tolerate some pest damage. Furthermore, their study showed that when the quantity of pesticides to be applied per control action was fixed, the decisions concerning application timing would be independent of prices. Borosh and Talpaz, in commenting on the Hall and Norgaard study, noted that the conditions for a solution to the theoretical model[12] were only necessary, not sufficient, to achieve an optimum. In particular, Borosh and Talpaz indicated that the pest kill function could not be linearly related to pest population levels, thereby introducing further complexity by suggesting that dynamic pest management systems needed to be examined with non-linear models.

The early dynamic studies of pest management provided a theoretical basis and sparked an interest in continued research. Subsequent investigations also attempted to expand into empirical modelling, including the use of experimentally defined population dynamics and pesticide kill functions. For example, Talpaz and Borosh used a simplified cotton agroecosystem model and numerical optimization to demonstrate that models needed to link

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[12] These necessary conditions included that: 1) the number of pests killed increased as the pesticide quantity increased, 2) the number of pests killed increased with pesticide use at a decreasing rate (declining marginal productivity), and 3) that a given quantity of pesticide would kill more individuals as the pest population increased.

multiple treatments per crop season with the pesticide dosages, control costs, and other market prices if an accurate optimal control decision was to result. Although more complex and flexible than most previous models, by their own admission the authors' study suffered from a lack of realism that "would probably prohibit accurate analysis for some time to come" (Talpaz and Borosh). Talpaz et al., followed by Reichelderfer and Bender, attempted to circumvent the inability to realistically specify an analytic model[13] by using complex computer simulations to portray the dynamic operation of an agroecosystem. However, both simulation models were incomplete in that they focused primarily on the interactions of a single pest with the crop of interest, and did not consider the effects of control actions on other potential pests and beneficials in the system. Even so, these two studies were sophisticated attempts to eliminate the need for analytically specifying the system to be modelled. In fact, Talpaz et al., by using an iterative non-linear optimization algorithm, were actually able to determine the optimal policy for pesticide application under deterministic conditions. In contrast, Reichelderfer and Bender used their simulation model to compare predetermined alternative policies without investigating the relationship of the stated policies to the potential system optimums.

In addition to the simulation approaches, researchers have attempted to examine the empirical characteristics of pest management using dynamic programming methods. For example, Shoemaker (1979) examined the optimal timing of multiple pesticide applications when residual pesticide activity is known to occur. This study involved far more complexity than previous analytic and dynamic programming models, including the use of 11 pest age classes with their differential responses to pesticides, and 28 decision periods. The algorithm presented by Shoemaker to solve the dynamic programming problem was unusually efficient at utilizing computer resources, but it still required the analytic specification of some important agroecosystem relationships. Shoemaker (1982) continued to extend the range and complexity of these dynamic programming applications, and provided a clear and concise review

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[13] The term "analytic model" refers to single or multi-equation models that can be solved exactly using standard analytic solution procedures.

of the technique's benefits in a 1981 review. But, in the final analysis, it would appear that studies incorporating this empirical technique will continue to be hindered by the need to analytically specify major agroecosystem and economic relationships, as well as be constrained in the dimensions of the systems that can be examined by the present level of computing technology.

As might be expected, investigations solely into the dynamic aspects of pest management were paralleled by research efforts that attempted to include the potential effects of pest susceptibility as a common property resource. Perhaps the first to incorporate the possible effects of resistance on long-term pest management was Shoemaker (1973a, 1973b, 1973c). Besides demonstrating how the resistance problem and pest management in general could be understood within a general control theory framework, and illustrating the empirical applications possible with dynamic programming, her study provided ample indication of the extreme difficulties that arise when attempts are made to empirically apply dynamic theoretical models. This is especially true when considering the effects of pest resistance, where complex within-season and multiple-season effects are not easily simplified for computation with commonly available algorithms. Shortly after Shoemaker's groundbreaking work, Hueth and Regev investigated the potential effect of pest resistance on optimal pest control choices by constructing, within a discrete-time optimal control framework, a single-pest, single-crop theoretical model of an isolated agroecosystem with no migration of pests. No attempts were made to empirically apply the model, and its formulation was relatively simple. The authors employed the model to: 1) demonstrate the general use of control theory for such a problem, 2) give an example of how costate variables can be interpreted within a pest management context as the current value of foregone future gains from current control actions, or user costs, and 3) examine the general implications of dynamic optimization in the presence of pest resistance. The theoretical solution to their model suggested that the optimal level of pesticide use at any time would be that which equated marginal profits, excluding pest susceptibility, to the user cost associated with depleting a pest's stock of susceptibility. Thus, when the interdependence between producer choices and time periods were considered in devel-

oping pest management strategies, the use of static profit-maximization to measure the benefits and costs of pest control was not an adequate decision criterion (Klonsky). This implied that solutions to dynamic models of pest management would almost certainly require the formulation of variable economic thresholds over the course of a growing season, with the possibility that, under certain conditions, the threshold would even increase as harvest time approached. Interestingly, their analysis also showed that only under the most restrictive conditions would the neglect of resistance result in the overuse of chemicals.

Various subsequent studies have formulated more complex and realistic models of pest management with resistance. Feder and Regev developed a theoretical model of pesticide use and its externalities where the consequences of individual decision-making on pesticide use, pest-predator equilibria, and accumulated pesticide residues could be examined. Their results indicated that, given the common property nature of pest populations, optimal pesticide use could only be attained for society as a whole if centralized control was exercised over individual producer decisions on how, when, and where pesticides would be used. Regev et al. presented a thorough description and model of the optimization problem faced by producers attempting to control the alfalfa weevil. This study made use of empirically based relationships for pest population growth and the damage that pests engendered, and in doing so showed how complex a control model can get. Being unable to solve the problem analytically, the authors made use of a number of simplifying assumptions in order to reduce the problem to one that was approachable with non-linear programming algorithms.[14] The author's concluded that, given the existence of alternative technology, centralized decision making on pesticide use would lead to lower rates of resistance development than private decision making. However, they also demonstrated that a possible alternative was to use Pigouvian taxes on pesticide use, an approach which could theoretically produce a socially optimum solution identical to that attained under centralized control. Similarly, Gutierrez et al. investigated the same agroecosystem, but made explicit use of a biological simulation

[14] To date, the most popular methods of solving complex analytically specified control theory models have been through the use of two-point boundary algorithms, dynamic programming, or the reformulation of the problem into a non-linear programming format.

model. However, instead of taking full advantage of the complexity inherent in the simulation, they concluded that the number of simulation runs required to find an optimal solution was too great, and thus elected to examine only two possible pest management policies that the producer might follow: 1) where information on resistance was available to producers; and 2) where information was not available. The authors found that the mere availability of resistance information itself did not lead to better resistance management, but it did result in lower total pesticide use. Of course, their decision to avoid explicit optimization was based on the idea that, in order to optimize, they would have to completely enumerate all possible states of the system over their single season planning horizon. But, as will be discussed in detail later, various search procedures provide much more efficient ways of approaching this problem.

Other investigations into the effects of resistance on long-term pest management potential have been recently published. For example, Moffitt and Farnesworth specified a mathematical model to help econometrically estimate the effects of resistance development on pesticide demand. Aggregate U.S. pesticide data were then used to estimate pesticide demand elasticities. In addition, Mangel and Plant provided a theoretical framework for analyzing the trade-offs between economic yield from a crop and the development of resistance using a complex intra- and inter-seasonal genetic model of pest dynamics. Their main conclusion was that the timing of pesticide applications could be used to control resistance development, while the dosage per application could control the eventual crop yield.

Of particular interest are three studies that attempted to estimate and use full empirical models in a quest to optimize the management of pest resistance. Lazarus and Dixon utilized a nonlinear programming model to assess potential gains from internalizing resistance through regional coordination. Their model of an Illinois cash grain farm was used to estimate the gains from corn rootworm control, including in the calculations the effects of taxes, subsidies, and environmental costs that would affect the regional optimum by enabling producers to maximize individual profits. Harper used a dynamic simulation model to explore the potential effects of a build-up of resistance to chlordimeform by the pink bollworm on cotton, but,

like Gutierrez et al., ended up not pursuing any explicit optimization of the management procedures. Perhaps the most ambitious empirical attempt to date has been that of Archibald, where a dynamic programming model was used to determine optimal inputs given the presence of resistance under alternate pesticide regulations and pest management strategies. She accomplished this by enumerating a set of states for the system with the use of econometrically derived relationships, thus simplifying the system in order to make it amendable to analysis. However, the advances made by all three of these investigations indicated that a complete empirical analysis of resistance impacts, in all their complexity, might be possible given adequate computing resources and/or the development of improved solution algorithms. To give the reader a better feel for the theoretical intricacies involved in a dynamic pest management model with resistance, the following sub-section details a general optimal control formulation of the problem.

A General Dynamic Model

Consider a continuous-time mathematical model of a general agroecosystem characterized by the following features:

1. There are a number of major marketing channels for the crop. Whether or not a crop can actually be marketed through any given channel may depend on the particular variety of the crop grown, the level of inputs used in production, incidence of pest damage, and/or exogenous factors, such as year to year weather fluctuations. Denote these different marketing channels by the letter h, where $h = 1...\varepsilon$.
2. For each marketing channel there may be a number of different crop grades by which product is priced. The distinction between grades may be based on quality factors that are directly or indirectly affected by crop variety, level of production inputs, incidence of

pest damage, and/or exogenous factors (such as weather). Let these grades be indicated by the letter g, where $g = 1...\tau$.

3. There is a defined pest-predator complex associated with the production of the crop. For generality, each member of this complex may interact in some way with all other members (in addition to interacting with the crop itself). Denote these pest-predator organisms by the letter i, where $i = 1...\gamma$.
4. There are a number of producer-influenced ways in which pests might be controlled, all of which may have lethal or sub-lethal effects on one or more pest-predator organisms. Denote these pest controls by the letter k, where $k = 1...\psi$.
5. It is assumed that pest and non-pest management processes are independent at any stage of production. Thus, the common argument for employing separability conditions in the analysis will be maintained.

Given this agroecosystem and the desire to find the optimal time-path of pest control inputs so as to maximize total economic surplus over time, a dynamic interpretive framework is needed. Optimal control theory, being formally identical to economic capital theory, provides the ideal analytic tool for this interpretation (Dorfman). For example, if we assume that demand may shift exogenously due to non-specific time trends, and that supply shifts both exogenously and endogenously due to changes in input technology sets or total input costs, then this optimization problem can be portrayed as follows:

$$\underset{U}{\text{MAX}} \quad \text{TES} = \int_0^T \left\{ \sum_{h=1}^{\varepsilon} \sum_{g=1}^{\tau} \int_0^{(a_{h,g}\, y_{h,g}(t))} [D_{h,g}(\eta, t) - SY_{h,g}(\eta, U, t)] d\eta \right\} e^{-rt} dt \ ,$$

subject to the equations of motion:

$$\dot{y}_{h,g}(t) = \Omega_{h,g}[y_{h,g}(t), b_i(t), w(t), t] \quad \forall \ h, g, i \ ;$$

$$\dot{b}_i(t) = \Gamma_i[b_j(t), m_j(t), c_j(t), f_j(t), u_k(t), s_{j,k}(t), w(t), t] \quad \forall \ i, j, k, \ \text{where } j = 1...\gamma \ ;$$

$$\dot{s}_{i,k}(t) = \Theta_{i,k}[s_{i,z}(t), u_z(t), t] \quad \forall\ i, j, k, z, \quad where\ z = 1...\psi\ ;$$

$$\dot{f}_i(t) = \Upsilon_i[s_{i,k}(t), t] \quad \forall\ i, k\ ;$$

$$\dot{c}_i(t) = \Psi_i[y_{h,l}(t), t] \quad \forall\ i, h, l\ ;$$

$$\dot{m}_i(t) = \Xi_i[t] \quad \forall\ i\ ;$$

where we define:

U is a fixed set of pest controls in R^{ψ};
$u(t) \in U$;
TES is the total economic surplus over all time periods;
T is the terminal time of the planning horizon;
$D_{h,g}(\eta, t)$ is the demand function for a given crop and grade at time t;
$SY_{h,g}(\eta, U, t)$ is the supply function for a given crop and grade at time t;
$y_{h,g}(t)$ is the yield per acre of grade (g) for crop (h) at time t;
$a_{h,g}$ is the total number of acres producing crop h of grade g;
$b_i(t)$ is the density of pest/predator (i) at time t;
$s_{i,k}(t)$ is the susceptibility of pest/predator (i) to control (k) at time t;
$f_i(t)$ is the fecundity of pest/predator (i) at time t;
$c_i(t)$ is the environmental carrying capacity for pest/predator (i) at time t;
$m_i(t)$ is the immigration of susceptible pest/predator (i) at time t;
$w(t)$ is the deterministic influence of weather in time t;

and the state variables are constrained as

$$y_{h,g}(t) \geq 0\ ; \quad b_i(t) \geq 0\ ; \quad s_{i,k}(t) \geq 0\ ; \quad f_i(t) \geq 0\ ; \quad c_i(t) \geq 0\ ; \quad m_i(t) \geq 0\ ;$$

with $D_{h,g}$, $SY_{h,g}$, $a_{h,g}$, $y_{h,g}(0)$, $b_i(0)$, $s_{i,k}(0)$, $f_i(0)$, $c_i(0)$, and $m_i(0)$ all predefined. It is further assumed that the objective function and the equations of motion are all twice differentiable with respect to each state variable, differentiable with respect to the control set {U}, and continuous with respect to time.

Given a general problem with "pure" state constraints, finding a maximum involves forming a Lagrangian function composed of the unconstrained Hamiltonian and the state variable constraints. With reference to the agroecosystem, the appropriate current-valued Hamiltonian function can be specified as

$$\mathcal{H} = \sum_{h=1}^{\varepsilon} \sum_{g=1}^{\tau} \int_0^{(a_{h,g}\, y_{h,g}(t))} (D_{h,g} - SY_{h,g}) d\eta + \sum_{h=1}^{\varepsilon} \sum_{g=1}^{\tau} \lambda_{h,g}^{y}(t)\ \Omega_{h,g}(t) + \sum_{i=1}^{\gamma} \lambda_i^{b}(t)\ \Gamma_i(t)$$

$$+ \sum_{i=1}^{\gamma} \sum_{k=1}^{\psi} \lambda_{i,k}^{s}(t)\ \Theta_{i,k}(t) + \sum_{i=1}^{\gamma} \lambda_i^{f}(t)\ \Upsilon_i(t) + \sum_{i=1}^{\gamma} \lambda_i^{c}(t)\ \Psi_i(t) + \sum_{i=1}^{\gamma} \lambda_i^{m}(t)\ \Xi_i(t)\ ,$$

where the corresponding Lagrangian function takes the form

$$L = \mathcal{H} + \sum_{h=1}^{\varepsilon} \sum_{g=1}^{\tau} \mu_{h,g}^{y}(t)\ y_{h,g}(t) + \sum_{i=1}^{\gamma} \mu_i^{b}(t)\ b_i(t) + \sum_{i=1}^{\gamma} \sum_{k=1}^{\psi} \mu_{i,k}^{s}(t)\ s_{i,k}(t)$$

$$+ \sum_{i=1}^{\gamma} \mu_i^{f}(t)\ f_i(t) + \sum_{i=1}^{\gamma} \mu_i^{c}(t)\ c_i(t) + \sum_{i=1}^{\gamma} \mu_i^{m}(t)\ m_i(t)$$

and it is understood that the λs and μs denote the current-valued adjoint and state constraint multipliers, respectively. Because the λs can be interpreted as the dynamic, current-value shadow prices, they are equivalent to the marginal value of the state variables at any time (t) (Dorfman; Conrad and Clark). This allows the Hamiltonian to be interpreted as the total rate of increase in the net value of assets derived from the agroecosystem, where:

$$\sum_{h=1}^{\varepsilon} \sum_{g=1}^{\tau} \int_0^{(a_{h,g}\, y_{h,g}(t))} (D_{h,g} - SY_{h,g}) d\eta$$

represents the flow of direct economic surplus from the agroecosystem at time t;

$$\sum_{h=1}^{\varepsilon}\sum_{g=1}^{\tau}\lambda_{h,g}^{y}(t)\ \Omega_{h,g}(t)$$

represents the increase in value of crop yield at time t;

$$\sum_{i=1}^{\gamma}\lambda_{i}^{b}(t)\ \Gamma_{i}(t)$$

represents the net change in the value of pest, pest/predator population levels at time t;

$$\sum_{i=1}^{\gamma}\sum_{k=1}^{\psi}\lambda_{i,k}^{s}(t)\ \Theta_{i,k}(t)$$

represents the net change in the value of susceptibility in the pest, pest/predator complex at time t;

$$\sum_{i=1}^{\gamma}\lambda_{i}^{f}(t)\ \Upsilon_{i}(t)$$

represents the net change in the value of fecundity levels in the pest, pest/predator complex at time t;

$$\sum_{i=1}^{\gamma}\lambda_{i}^{c}(t)\ \Psi_{i}(t)$$

represents the net change in the value of environmental carrying capacity at time t; and

$$\sum_{i=1}^{\gamma}\lambda_{i}^{m}(t)\ \Xi_{i}(t)$$

represents the net change in the value of immigration of susceptible pests and predators at time t. The μs of the final Lagrangian function are, of course, the dynamic shadow prices, or marginal current-values, of the state variables at the constraint bounds. Given these two functions, optimization can be assured by adherence to the sufficient conditions suggested by a modified maximum principle (Seierstad and Sydsaeter).

The exact solution for this general model with state constraints is complex and perhaps impossible to derive. In fact, for any realistically specified functions, this approach usually leads to an intractable analytic problem that can only be approximately solved quantitatively. Nonetheless, qualitative aspects of the potential solution can be explored by assuming that the

solution lies interior to the constraints, implying $\mu(t) = 0$ for each state constraint, and that the terminal time $T = \infty$. Further assuming that sufficiency conditions for a local maximum are met, then the first-order conditions for the current-valued formulation collapse to finding the admissible couplet $(x^*(t), u^*(t))$ that satisfies:

$$\text{control optimality conditions} \quad \frac{\partial \mathcal{H}}{\partial u_k(t)} = 0 \quad \forall \; k \; ;$$

$$\text{adjoint variable conditions} \quad \frac{\partial \mathcal{H}}{\partial x_i(t)} = r\,\lambda_i(t) - \dot{\lambda}_i(t) \quad \forall \; i \; ; \text{and}$$

$$\text{transversality conditions} \quad \frac{\partial \mathcal{H}}{\partial \lambda_i(t)} = \dot{x}_i(t) \quad \forall \; i \; .$$

Of particular interest in the qualitative analysis of this problem are the control optimality and adjoint variable conditions. The control optimality conditions characterize the optimal production rate that maximizes total economic surplus, thereby indicating the optimal use of the pest control inputs. Meanwhile, the adjoint conditions essentially state that the socially required rate of return on the value from the use of a state variable x ($r\lambda(t)$) must equal the state variable's potential change in value ($\dot{\lambda}(t)$) plus the net benefits associated with the state variable's use ($\frac{\partial \mathcal{H}}{\partial x(t)}$).

Assuming the optimal admissible couplet exists, the solution to the general problem takes the form:

$$\frac{\partial \mathcal{H}}{\partial u_k(t)} = \frac{\partial \left[\sum_{h=1}^{\varepsilon} \sum_{g=1}^{\tau} \int_0^{(a_{h,g}\, y_{h,g}(t))} (D_{h,g} - SY_{h,g}) d\eta \right]}{\partial u_k(t)} + \sum_{i=1}^{\gamma} \lambda_i^b(t) \frac{\partial \Gamma_i(t)}{\partial u_k(t)} +$$

$$\sum_{i=1}^{\gamma} \lambda_{i,k}^{s}(t) \frac{\partial \Theta_{i,k}(t)}{\partial u_k(t)} = 0 \quad \forall\ k\ ; \qquad [1]$$

$$\frac{\partial \mathcal{H}}{\partial y_{h,g}(t)} = D_{h,g} - SY_{h,g} + \lambda_{h,g}^{y}(t) \frac{\partial \Omega_{h,g}(t)}{\partial y_{h,g}(t)} + \sum_{i=1}^{\gamma} \lambda_i^{c}(t) \frac{\partial \Psi_i(t)}{\partial y_{h,g}(t)}$$

$$= r\, \lambda_{h,g}^{y}(t) - \dot{\lambda}_{h,g}^{y}(t) \quad \forall\ h, g\ ; \qquad [2]$$

$$\frac{\partial \mathcal{H}}{\partial b_i(t)} = \sum_{h=1}^{\varepsilon} \sum_{g=1}^{\tau} \lambda_{h,g}^{y}(t) \frac{\partial \Omega_{h,g}(t)}{\partial b_i(t)} + \sum_{j=1}^{\gamma} \lambda_j^{b}(t) \frac{\partial \Gamma_j(t)}{\partial b_i(t)}$$

$$= r\, \lambda_i^{b}(t) - \dot{\lambda}_i^{b}(t) \quad \forall\ i\ ; \qquad [3]$$

$$\frac{\partial \mathcal{H}}{\partial s_{i,k}(t)} = \sum_{j=1}^{\gamma} \lambda_j^{b}(t) \frac{\partial \Gamma_j(t)}{\partial s_{i,k}(t)} + \sum_{z=1}^{\psi} \lambda_{i,z}^{s}(t) \frac{\partial \Theta_{i,z}(t)}{\partial s_{i,k}(t)} + \lambda_i^{f}(t) \frac{\partial \Upsilon_i(t)}{\partial s_{i,k}(t)}$$

$$= r\, \lambda_{i,k}^{s}(t) - \dot{\lambda}_{i,k}^{s}(t) \quad \forall\ i,\ k\ ; \qquad [4]$$

$$\frac{\partial \mathcal{H}}{\partial f_i(t)} = \sum_{j=1}^{\gamma} \lambda_j^{b}(t) \frac{\partial \Gamma_j(t)}{\partial f_i(t)} = r\, \lambda_i^{f}(t) - \dot{\lambda}_i^{f}(t) \quad \forall\ i\ ; \qquad [5]$$

$$\frac{\partial \mathcal{H}}{\partial c_i(t)} = \lambda_i^{b}(t) \frac{\partial \Gamma_i(t)}{\partial c_i(t)} = r\, \lambda_i^{c}(t) - \dot{\lambda}_i^{c}(t) \quad \forall\ i\ ; \qquad [6]$$

$$\frac{\partial \mathcal{H}}{\partial m_i(t)} = \sum_{j=1}^{\gamma} \lambda_j^{b}(t) \frac{\partial \Gamma_j(t)}{\partial m_i(t)} = r\, \lambda_i^{m}(t) - \dot{\lambda}_i^{m}(t) \quad \forall\ i\ ; \qquad [7]$$

$$\frac{\partial \mathcal{H}}{\partial \lambda^{y}_{h,g}(t)} = \Omega_{h,g}(t) = \dot{y}_{h,g}(t) \ , \qquad \frac{\partial \mathcal{H}}{\partial \lambda^{b}_{i}(t)} = \Gamma_{i}(t) = \dot{b}_{i}(t) \ , \qquad [8]$$

$$\frac{\partial \mathcal{H}}{\partial \lambda^{s}_{i,k}(t)} = \Theta_{i,k}(t) = \dot{s}_{i,k}(t) \ , \qquad \frac{\partial \mathcal{H}}{\partial \lambda^{f}_{i}(t)} = \Upsilon_{i}(t) = \dot{f}_{i}(t) \ , \qquad [9]$$

$$\frac{\partial \mathcal{H}}{\partial \lambda^{c}_{i}(t)} = \Psi_{i}(t) = \dot{c}_{i}(t) \ , \text{ and} \qquad \frac{\partial \mathcal{H}}{\partial \lambda^{m}_{i}(t)} = \Xi_{i}(t) = \dot{m}_{i}(t) \qquad [10]$$

$$\forall \quad h, \ i, \ k, \ g \ ;$$

where equation [1] is the control optimality condition, equations [2] through [7] are the adjoint variable conditions, and the equation group [8] through [10] are the transversality conditions. Each of these conditions can be given an economic interpretation relative to a specific agroecosystem, a task that shall be undertaken in Chapter V. For now, it is sufficient to realize that any realistic system representation will yield a large set of conditions that must be simultaneously satisfied.

As should be obvious from the discussion in this chapter, investigations into the dynamic impacts of pest management, and resistance management in particular, are capable of being developed theoretically. However, empirical investigations have been complicated by the extreme complexity of most agroecosystems. This complexity makes it impossible to formulate fully specified analytic models that are simple enough to be exactly solved while being detailed enough to have practical application in the development of resistance management schemes. The extent of the complexity problem is further compounded when policy decisions need to be made concerning pesticide regulations.

Regulation and Resistance

The dynamic qualities of public regulations are often ignored when developing and implementing pest management programs. However, they can be extremely important to the

long-term viability of a control scheme. By restricting, encouraging, and/or requiring the use of certain methods and materials, regulations constrain and may eliminate parts of the input set used for pest control (Klonsky). While they take many forms with respect to agriculture, the regulations that most directly impact pest management and resistance are those targeting pesticide use.

Pesticide regulation in the U.S. has evolved from the narrow goal of protecting pesticide users from product fraud to a comprehensive set of objectives that are concerned with the short- and long-run effects of pesticides on the agroecosystem, human health, and the general environment (National Research Council, 1978). The EPA's decisions on pesticide regulations are made within a complex legal and political environment, with the scope of EPA's authority reflecting political compromises between the various constituents of both the legislative and executive branches of the federal government. The decision-making framework was constructed primarily by Congressional statutes, with the EPA being given wide authority to interpret the statutes and develop principles and procedures for their implementation. This section will briefly describe the legislative framework within which the EPA operates, followed by an examination of how it functions with respect to pesticides and a discussion on how the regulation process may interact with the agroecosystem to promote the development of resistance.

Background and Scope of Pesticide Regulation

The first major piece of federal legislation dealing specifically with pesticide use was the Federal Insecticide, Fungicide, and Rodenticide Act of 1947 (FIFRA). FIFRA required that all pesticides be registered with the USDA before sale, with the registration requiring that the pesticide label list ingredients, appropriate uses, and instructions for application. In practice, however, the USDA was unable to strictly enforce regulations on pesticide use because the law allowed unregistered pesticides to be sold under protest by the manufacturer until the

USDA could prove that the pesticide was harmful. This burden of proof overwhelmed the resources available at the USDA. In an attempt to correct this, Congress shifted the burden of proof to manufacturers with the Pesticide Chemical Amendment of 1954.

Although various amendments to FIFRA and other laws regarding pesticide and chemical use on food were passed during the late 1950's and 1960's, the USDA's Pesticide Regulation Division came under increasing congressional criticism. In addition, pesticide research and tolerance setting programs had become inefficiently scattered among the Departments of the Interior and Health, Education, and Welfare. By 1972, public and Congressional pressure for pesticide regulatory reform had reached the point where responsibility for all aspects of pesticide regulation and research were legislated away from the cabinet level departments to the EPA (Committee on Environmental Decision Making).

With the decision to place responsibility for pesticide regulation with the EPA, attention was focused on the development of mechanisms to correct the perceived problems with previous legislation. From a regulator's point of view, the principle weakness of FIFRA and its amendments was that they did not provide any clear way of actually regulating pesticide use once a chemical was registered. Attempting to correct this flaw, the Federal Environmental Pesticide Control Act of 1972 (FEPCA) replaced FIFRA by amendment and went far beyond any previous legislation. It prohibited the sale or use of a pesticide not registered with the EPA, and allowed the EPA to impose restrictions on pesticide use and establish labeling requirements. This amendment established the "no unreasonable adverse effects" standard, the risk/benefit approach to pesticide evaluation, and the task of re-evaluating all pesticides previously registered with the USDA (EPA). In addition, the EPA was granted control over pesticide end uses as well as the movement of pesticides in interstate commerce (Committee on Environmental Decision Making). Specifically, FEPCA charged the administrator of the EPA with (Antle):

1. **Environmental Protection.** The act required that pesticides be regulated to avoid "unacceptable adverse effects on the environment," defined as "any unreasonable risk to man

or the environment, taking into account the economic, social, and environmental costs and benefits of any pesticide."

2. **Registration.** Two classes of registration were created: general, for pesticides which required only labeling; and restricted, for pesticides posing a health threat to applicators or others, and which could only be used by certified applicators. These restricted pesticides could be further regulated by EPA action. In addition, registration rules applied to materials manufactured or traded both intra- and interstate.

3. **Cancellation and Suspension of Registration.** Registration was routinely cancelled after five years, with continued use allowed during the re-registration process. Under suspension, the EPA administrator would immediately stop all sale and use of a pesticide.

Although FEPCA was originally seen as an improvement over FIFRA, the complexity of EPA's mandate, the large number of active pesticide ingredients to be evaluated, and technical difficulties inherent in the determination of benefits, costs, and risk made the system unworkable. Among other complaints was the claim that negative regulatory impacts on agriculture were being ignored (Antle). This problem was addressed by legislative changes in the 1975 extension of FIFRA, which required the EPA to assess the effects of proposed regulations on the agricultural economy. Further modification of FIFRA by the 1978 Federal Pesticide Act attempted to improve the implementation of regulations by creating the Rebuttable Presumption Against Registration (RPAR) process. This amendment provided for a period of public review during which applicants would have an opportunity to contest EPA's finding against a registration.

Even with the additional legislation of the late 1970's, the vast number of pesticides on the market and the rapid introduction of new chemicals and formulations led to administrative gridlock and a breakdown in the registration process. Increasing public and Congressional frustration with the pace of registration led to a 1988 Congressional order that the EPA promptly re-examine all 40,000 pesticide registrations (Devault). To provide the EPA with the

resources to accomplish this, the legislation required, for the first time, that manufacturers seeking a registration pay a fee. This fee, and the costly data required to support registration, was high enough to remove as many as 26,000 pesticides from the registration process. In fact, the House Agricultural Committee estimated that the number of pesticides available for agricultural use would fall at least 30 percent due solely to the legislative mandates (Devault). Many of these voluntarily withdrawn pesticides had completed their economic life-cycle or were produced in limited quantities for specialty markets.

At present, pesticide registration decisions at the EPA are based on test data provided by applicants. Pesticide manufacturers are required to conduct studies covering potential adverse effects to humans, fish, wildlife, and endangered species. Potential human risks include acute toxic poisoning, skin and eye irritation, as well as chronic effects such as cancer, birth defects, and reproductive disorders. The environmental impacts of a pesticide must also be considered so as to determine if the chemical is a potential threat to surface and/or groundwater. Once all the information is received, the EPA must take into account economic, social, and environmental costs and benefits in its decision-making process (EPA). But the economic impact of many of these detrimental effects, or negative externalities, arising from pesticide use are operationally difficult to quantify. In fact, much of the controversy over the regulation of pesticides results from the difficulties in finding acceptable methods for quantifying adverse effects, even though eco-toxicological and human health risk data are submitted to the EPA as part of the registration procedure. An additional complicating factor is that, in order to avoid issues related to uncertainty, the EPA's benefits assessments often adopt a short-run perspective of 3 to 5 years, while many of the negative externalities, such as the development of resistance, tend to appear only in the long-run. As a result, unless a clear and immediate adverse effect of pesticide use can be demonstrated, the benefits assessment procedure may only include consumer and non-user impacts qualitatively, leaving the economic quantitative/qualitative trade-off appraisal to the judgement of the final policy-maker (National Research Council 1978). It is one of the goals of this study to provide a method by which the long-run adverse effects of pest resistance can be quantitatively evaluated and in-

corporated into the EPA's benefits analysis. Before examining the specifics of the interrelationship between regulation and resistance, the following will detail the basic process behind the EPA's current benefits analyses.

The Process of Pesticide Regulation

Because the costs, benefits, and risks of a pesticide are often difficult to quantify, the decision to permit continued use not only involves technical examination of the chemical and its impacts, but also includes policy and value judgements. To be valid, however, these judgements must arise out of a carefully laid scientific foundation (National Research Council, 1978). It is clear from the EPA's legislative mandate that regulators are to account for all the benefits and costs of a pesticide to society before designing policies that would restrict or prohibit use. But current federal law does not require the use of a formal benefit-cost analysis procedure. Instead, the EPA has had the freedom to adapt the analysis process to specific regulatory problems. Nonetheless, even with the changing nature of the process between different analyses, there are still some common approaches and elements used in regulatory decision-making.

Within the framework of the EPA's assessment process, benefits are defined as those desirable effects that result from the continued use of a pesticide. The benefits of a particular pesticide can include (Council for Agricultural Science and Technology):

1. Reductions in crop yield and quality losses on the farm;

2. With lower on-farm losses, increased consumer benefits derived from more product marketed with fewer blemishes at lower prices;

3. Employment and increased economic activity in the pesticide production and marketing sector;

4. Reduced losses from spoilage and trim waste in the food processing sector; and/or

5. Higher productivity in the agricultural sector and a reduced need for food imports.

Examined overall, this description of pesticide benefits can be summarized as (1) the value of productive resources saved due to pesticide use and (2) the value of any additional output, including non-commodity output, arising from the manufacture and use of the pesticide. Thus, the first step in a pesticide assessment usually involves analyzing a pesticide's productivity (National Research Council, 1978).

Pesticide productivity refers to the chemical's effectiveness at providing improved output, public health, or living conditions. This can be contrasted with efficacy, which is the effectiveness of a pesticide in eliminating its target pest. An efficacious pesticide may be relatively unproductive if its target organism does not significantly impact output, health, or the environment. This distinction is important because it implies that pesticide use cannot be justified solely on its ability to control pests, but instead is dependent on its ability to increase output or improve health conditions. Although pesticides targeted for crop pest control can have positive impacts on human health, the EPA usually concentrates on evaluating the specific agricultural productivity of pesticides in terms of their effects on yield and quality. Post-harvest losses due to pre-harvest pest damage are often ignored. Estimating productivity in these terms typically requires knowledge about the substitutability of alternate controls for the pesticide under analysis, as well as an estimate of any output or quality differences expected between use of the pesticide under analysis and its potential alternatives.

Even though it is often the most important positive impact of pesticide use, increased yield or quality are only part of the benefits that accrue to the use of chemical controls. Foregone costs are also important, and within the assessment framework they are defined as the detrimental impacts that would occur if the pesticide were not available for use. Typically, these costs are limited to the increase in variable chemical costs that would occur if the pesticide were withdrawn. In general, this analysis of benefits from continued registration is conducted with partial equilibrium, or partial budgeting, assumptions, often maintaining that

the output prices in the markets being examined are unaffected by any potential changes in output quantity, and that there are no inter-market effects.

Of course, the EPA's assessment procedure also includes consideration of the detrimental effects, or negative externalities, associated with the continued use of the pesticide being analyzed. These costs typically include (Council for Agricultural Science and Technology):

1. Environmental and health degradation due to off-site movement of pesticides during or after application;

2. Health hazards to labor in both pesticide manufacturing facilities and in agriculture;

3. Contamination of food products;

4. Unintended secondary action of pesticides against beneficial organisms, potentially causing an outbreak of previously unimportant pests; and/or

5. Excessive selection pressure against the target pest, leading to the emergence of pest resistance to pesticides.

As suggested from the list above, many of the costs associated with pesticide use are indirect, slow in emerging, hard to define, and very difficult and expensive to quantify. Along with foregone costs, these indirect costs provide much of the ambiguity in analyzing proposed pesticide regulations. Although this study is primarily concerned with the quantitative incorporation of resistance as a foregone cost, in principle the procedure used can be applied to the analysis of many other dynamic indirect costs as well.

The Regulation/Resistance Nexus

While it is true that agricultural chemicals have gradually come under increasing regulatory scrutiny, the relatively new intensity of this situation has confused the development of modern pest management programs and threatened the long-term viability of current strategies. This confusion arises, at least in part, out of the ambiguity associated with the following questions:

1. How will the current regulatory environment affect the development of pest resistance to insecticides?

2. Is there a way to incorporate long-term biological and economic impacts into the regulatory assessment process?

3. Can the potential negative impacts of regulatory action on the development of resistance be mitigated by changing pest management procedures and/or the structure of the regulatory process?

Unfortunately, answers to these questions are either non-existent or based on speculation. Very little empirical work has examined the dynamic interactions between the pesticide regulations and resistance development. However, theoretical investigations focusing on the effects of sudden pesticide withdrawals have given some cause for concern, particularly with respect to their impact on current integrated pest management systems. The pest complexes targeted by IPM control systems often consist of multiple pest and beneficial species, each of which may react differently to any particular management tactic. This interdependence of pests, beneficials, and control methods can result in a management system that is unstable in the sense of being dependent on a very specific set of biological and/or chemical controls. As previously indicated, many integrated systems rely on biological control agents to reduce the need for synthetic chemical controls. In doing so, they tend to eliminate

much of the selection pressure for the development of pest resistance. But problems can develop with these integrated systems because most biological controls have a narrow range of tolerance to insecticides and a limited ability to develop resistance. This characteristic of biological controls requires that integrated systems be carefully formulated to include only those chemicals that do not eliminate the effectiveness of the natural enemies. As a result, integrated systems are subject to breakdown when the critical insecticides can no longer be used and alternate chemicals, to which the natural enemies have no tolerance, are employed. This chemical loss might occur due to outright regulatory withdrawal, which in a worst-case scenario would lead to the complete collapse of the integrated management system and the need for the use of alternative, more toxic controls. However, the likelihood of this occurring is greatly reduced by the fact that these adverse impacts can be anticipated and would have to be included in the economic analysis of a proposed regulation. Only in the case where there were economically viable alternatives to the current chemical and/or system of control would the regulation be instituted. Of more serious concern, primarily due to its insidious nature, is the possibility that regulatory withdrawal of an insecticide will lead to a hastening of resistance development to the remaining chemical controls, a situation that can be anticipated but is not quantitatively considered in regulatory assessments at this point in time.

Consider an integrated pest management system that is vitally dependent on combined biological and chemical controls. Given that the possibility of resistance development to the chemical controls was identified as a threat, the integrated system was developed in a way that permitted the use of a number of specific insecticides from an array of potential controls. The identified insecticides could be tolerated by the biological control, and because there was more than one potential chemical control, problems with pest resistance could be reduced by alternating the use of chemicals, greater reliance on the biological control, or any of the other methods discussed in Chapter II. But while the short-run success of this system is dependent on maintaining pest susceptibility to the available chemicals, stepped-up regulatory pressure has the potential to disrupt the long-term management scheme. As the number of usable insecticides decreases through regulatory withdrawals, the likelihood of resistance develop-

ment to the remaining chemicals increases because, *ceteris paribus*, each remaining chemical may need to be used more often or more intensely in a given time period. This situation raises the possibility that long-run use of the remaining chemical controls might increase because of the biological system's response to the regulation-reduced control input set. Thus, resistance development may imply accelerated insecticide use and the potential for increased residual contamination of the on- and off-farm environment, the very scenario regulations are intended to prevent. Regulation-induced resistance could be especially damaging if cross- or multiple-resistance led to the development of resistance to an entire array of potential controls. Additional complications also arise when the pest complex contains multiple organisms, as will be discussed in the next chapter.

With the recent recognition that regulatory action may promote resistance development, focus has shifted to the regulatory assessment process and potential ways to reduce the impacts on pest management systems. First, the assessment process needs to account for long-term dynamic changes that may result from an insecticide withdrawal. Although this need has long been recognized and addressed qualitatively, it has not been possible to quantitatively evaluate and incorporate these regulation effects into the policy process. This study provides a mechanism to accomplish this empirical objective. Second, and just as important, the rationale behind chemical-by-chemical assessment needs to be critically evaluated. As it now stands, regulatory authorities assess the relative benefits of individual insecticides without quantitatively including the size or future viability of the remaining array of potential chemical controls. With the reality of cross-resistance and the fragility of many integrated control systems, an empirical analysis of system wide regulatory assessments would seem to be warranted. This study also examines this issue and its likely consequences for long-term insecticide use and resistance development.

Summary

Public debate concerning pesticides and the rationales for their use tend to involve at least as much emotion as fact and carefully considered research. This chapter was designed to help avoid including unsubstantiated concepts into the current study by concentrating on the well-documented economic history of pesticides, their role in agriculture, and the potential implications of pesticide regulatory actions. Thus, this chapter provides much of the informational base and motivation for subsequent chapters. In particular, the following points will be important to the continuing development of this current study:

- Although the economic definition of pest thresholds, and their empirical identification, requires complex knowledge about pest biology, ecology, damage relationships, and system economics, thresholds serve to clearly demonstrate that control actions should only be employed when potential injury levels will result in expected crop losses that exceed the additional costs incurred by implementing controls;

- Because the externality effects of pesticides, of which depleting pest susceptibility stocks may be one, are not valued in the market, there will be a divergence between an individual producer's optimal pest control policy and the optimal policy for society as a whole;

- The technical effects of developing pest resistance on production that are not valued in the market tend to promote economic incentives for increased pesticide use, thereby hastening the onset of complete resistance;

- The conflict between the EPA's short-run regulatory perspective and the long-run nature of pest resistance leads to a partial benefits assessment procedure that has, until now, only incorporated resistance on a qualitative level; and

- Pesticides and their widespread use may be associated with substantial private and social short- and long-run costs. However, the benefits associated with pesticides, including the value of productive resources saved and the value of additional commodity and non-commodity output, also tend to be quite high. As a result, pesticides cannot be uniformly characterized as expendable. Instead, careful assessments must be made using a dynamic, agroecosystem approach that can capture the relevant short- and long-run use implications.

CHAPTER IV: THE STUDY AGROECOSYSTEM

Apple production began in the U.S. when European settlers brought the fruit to the new world in the early 1600s (Bultitude). Initially used in subsistence production, the growing of apples became a commercial enterprise as the colonies developed a diversified economy with urbanized centers of population. Commercial production continued to expand throughout the 19th century, eventually becoming widely distributed in order to supply rapidly multiplying local markets. However, by the middle of the 20th century the advent of storage technology, reliable transportation, and advanced agro-management led to a spatial concentration of production into regions endowed with comparative soil, water, and climatic advantages. These technical advances, and the economies resulting from large-scale orchard operations, resulted in dramatic production and distribution increases that marked the beginning of the modern apple industry (Bultitude). Recent production levels have exceeded 8 billion pounds annually (Figure 3), with consistent crops of over 9 billion pounds predicted by the late 1990's. The nominal retail value of the U.S. apple crop has also increased, while the real retail value has held relatively steady since 1980 at $1.0 to 1.3 billion 1967 dollars (Crop Reporting Board, Non-citrus Fruits and Nuts).

As apple production expanded and intensified, orchard management became progressively more complicated and dependent on advanced technology. This was especially true for orchard pest management, where outbreaks of new and sometimes exotic arthropod pests

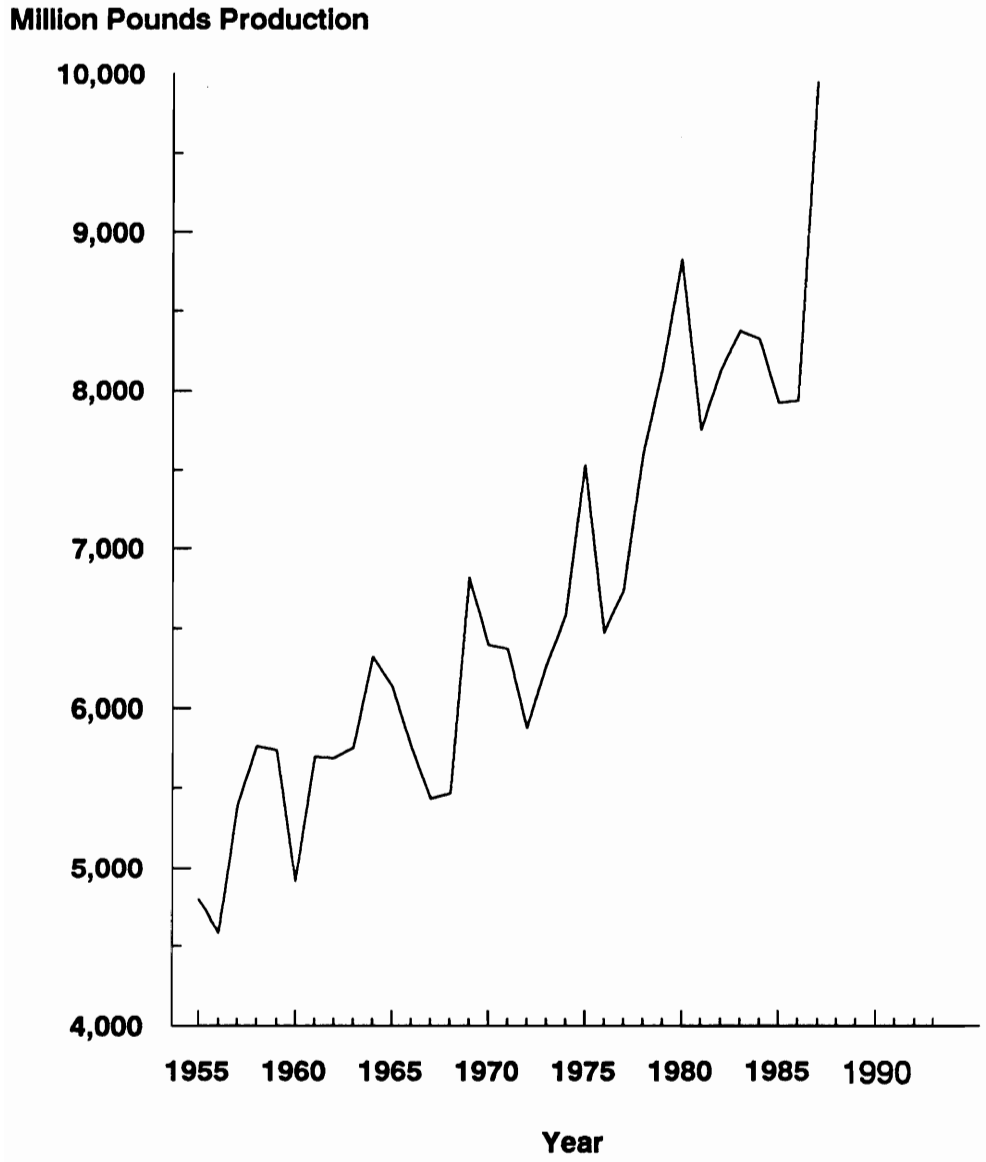

Figure 3. Aggregate Production Trends in the U.S. Apple Market.

would periodically cause significant economic damage that could only be abated by chemical controls. But, these needed controls also seemed to lead to increased levels of pest resistance. In an effort to describe the theoretical relationship among orchard pest management, pesticide regulations, and the development of insecticide resistance in arthropods, this chapter will focus on:

1. a brief introduction to general orchard management procedure and the role of pest controls; and

2. the specific mid-Atlantic apple agroecosystem that was the object of this study.[15]

Orchard and Arthropod Pest Management

Commercial apple orchards in the U.S. generally consist of scion cultivars developed by horticulturalists for specific production regions and/or marketing objectives. Some important characteristics that have been considered during breeding programs include tree growth habit, tolerance to soil and climatic conditions, precocity, anchorage, yield efficiency, and fruit quality in terms of juiciness, flavor, color, firmness, and storability (Hull et al. 1983a). Any particular combination of characteristics will affect the ability to manage the orchard and produce economically viable quantities, with the preference for a given cultivar being jointly determined by producers, processors, and consumer acceptance. Given that many of the forces acting on cultivar choice tend to be uniform throughout a production region, it is not surprising that only a few apple cultivars dominate the mid-Atlantic market, with Red Delicious, Golden Delicious, Rome Beauty, Stayman, and York Imperial having been prominent in

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[15] For the purposes of this study, the mid-Atlantic U.S. is defined as the apple producing areas of Pennsylvania, Virginia, eastern West Virginia, Maryland, Delaware, and western New Jersey. These geographic areas contain the particular pest complex used for illustration in this study, and the area roughly corresponds to the EPA's region III. This area is called the Cumberland-Shenandoah region.

the markets for many years (Crop Reporting Board, Commercial Apples by Variety). Red and Golden Delicious compose over 50 percent of total U.S. production and are the primary fresh market cultivars produced in the region. The main processing cultivar in the area is York Imperial, with Rome Beauty and Stayman also seeing considerable production. Other cultivars, such as Johnathan and MacIntosh, are prevalent in some Pennsylvania localities but mostly absent elsewhere (Figure 4).

Mature orchard manipulation can generally be divided into two categories; non-pest management and pest management. Non-pest management consists of pruning/training and growth regulation, ground cover management, fertilization, and possibly irrigation. Because of the range of climatic conditions in the U.S., it is not surprising that the relative importance of orchard management components vary between production regions (Figure 5). Only ground cover management, fertilization, and "other management" practices, such as financial record keeping, are relatively constant in cost shares across regions. However, not all regional differences can be ascribed to climatic variations, as management philosophy and targeted end-use can affect which management components a grower emphasizes. For example, the thinning and growth regulation of harvestable trees appears to be more important in the Northwest production region than in the midwest, perhaps due to greater reliance on mechanized harvesting and/or product distribution through fresh market channels. On the other hand, pest controls are used more intensively in the midwest, accounting for over 40 percent of the annual production costs.

Although specific apple production practices vary from preharvest to harvestable orchards and from region to region, the typical goal of production management is to produce the maximum net returns that are possible from the enterprise. One of the largest obstacles to achieving this goal, both in the short- and long-run, are the impacts of arthropod pest infestations. Apple trees and fruit are susceptible to a wide variety of pest organisms, with studies indicating that as many as 100 different insect species alone may be feeding in apple orchards in any given year (Oatman et al.). Of course, only a fraction of these arthropod pests reach densities that are high enough to inflict economic damage to the crop. But if left un-

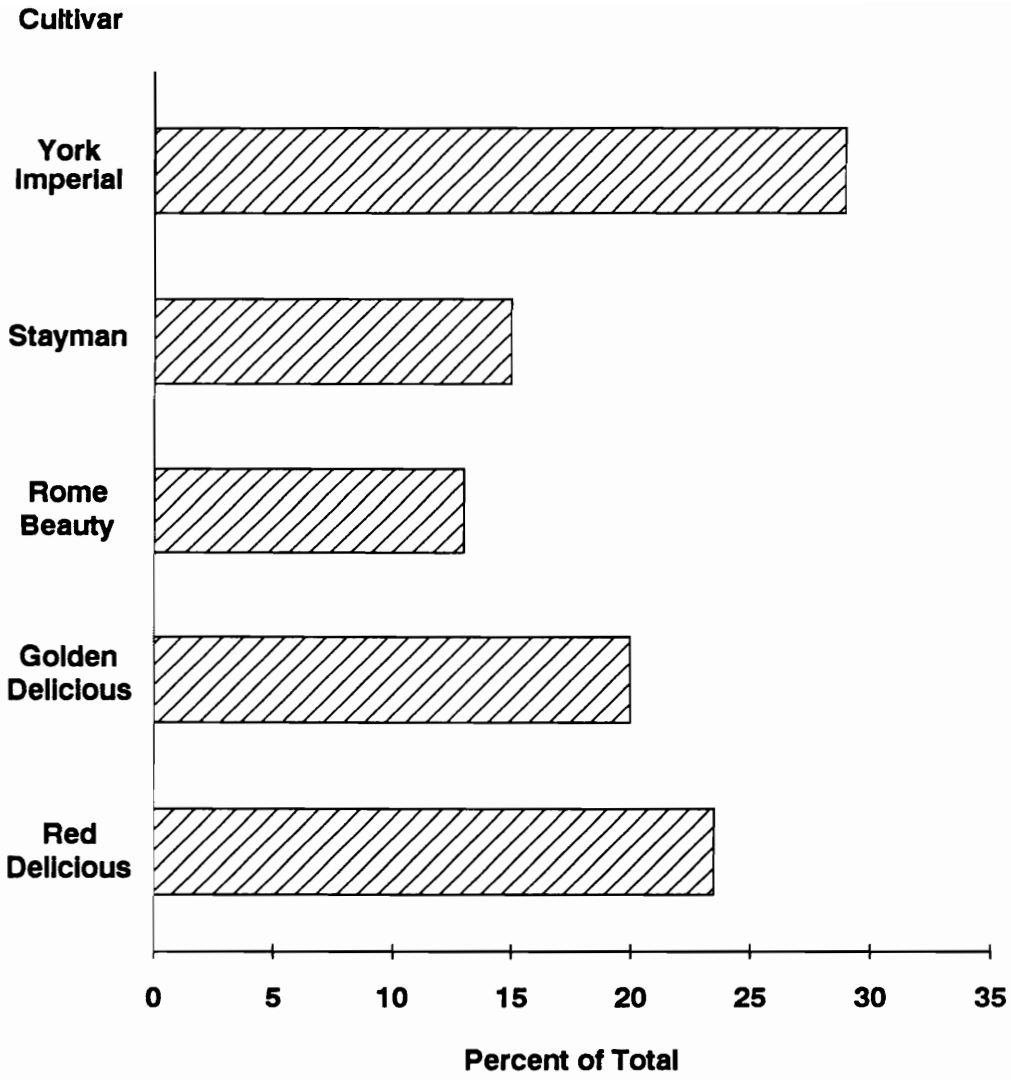

Figure 4. Relative Importance of Apple Cultivars in the Mid-Atlantic Region.

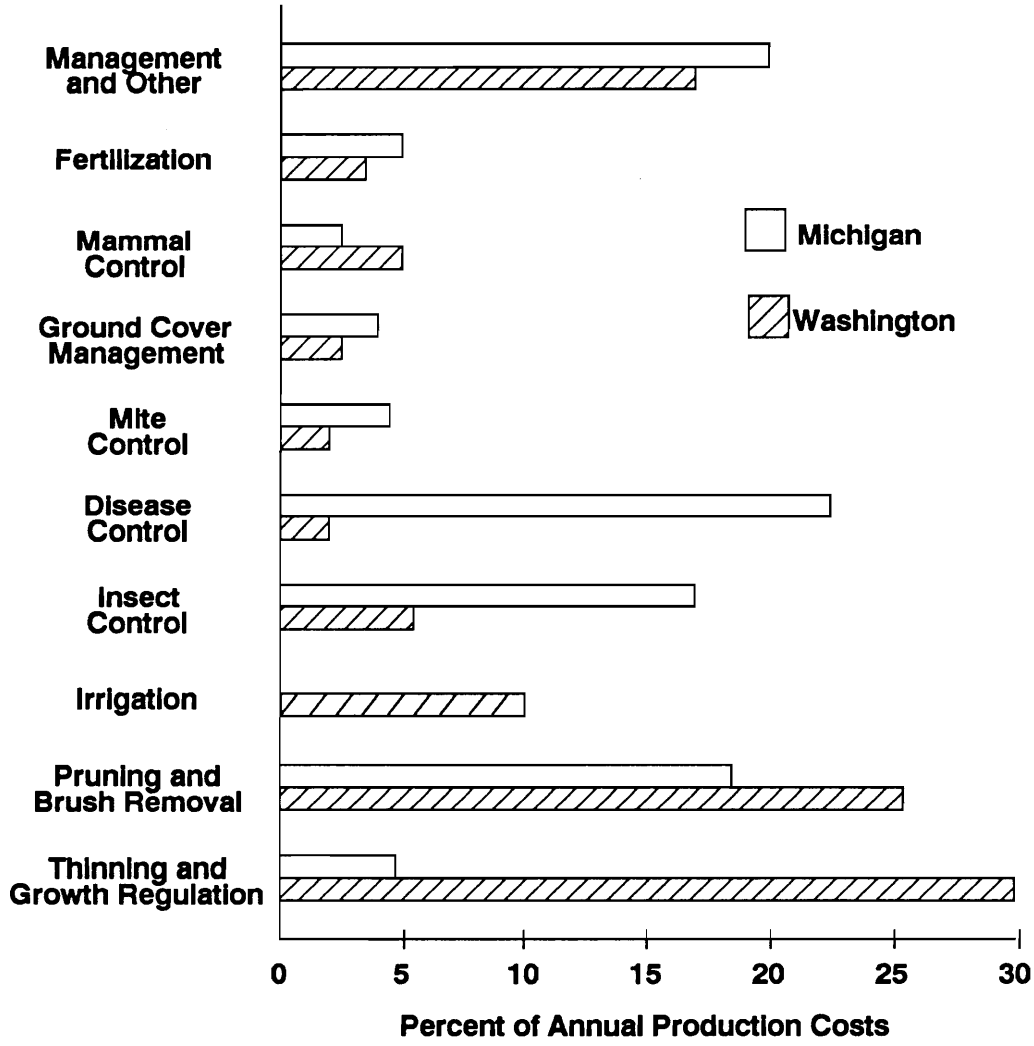

Figure 5. Relative Importance of Orchard Management Components by Region: Adapted from Hull et al. (1983a), p. 52.

checked, the economically important pests can cause dramatic damage, even to the point of eliminating marketable yields (Glass and Lienk). While pest control can be quite simple, management problems are often compounded by the fact that many of the most damaging economic pests have no natural biological controls, have biological controls that are not tolerant of pesticides, and/or have developed resistance to chemical controls. Both the lack of natural enemies and developing resistance have resulted in a production system where total pest management can require as many as 20 to 30 different pesticides used in 10 to 15 annual applications. This per acre rate of pesticide use is one of the highest for any major U.S. crop (Pimentel et al.).

Pest management in apple production has historically been very poor and subject to varying levels of success. For example, pest damage at harvest during the period 1900-1930 was estimated to range between 10 to 40 percent (Croft). At least some of these early management difficulties can be traced to inadequate control technology. In response, pesticide use (initially heavy metals such as lead arsenate) began to accelerate in the early 1920s (Figure 6). Once their effectiveness against arthropod populations were thoroughly documented, the use of synthetic insecticides became widespread, complete with high dosage rates and numerous applications. Apparently as a result of this extensive use, insecticide control failures began to occur. But in fact, control failures were not unique to the post-WWII generation of pesticides. For example, pest resistance to control by lead arsenate compounds was reported in apple production systems as early as the 1930's (Morgan and Madsen). In fact, patterns of slow initial acceptance, rapid expansion of use, and the eventual development of control failure have occurred in many pesticide successors to lead arsenate, including DDT, the organophosphates, and various miticides (Croft).

The repeated emergence and re-emergence of pest control problems in apple production has led to an industry-wide movement towards fine-tuning pest management techniques. With this movement came the realization that there was a real need for more information, understanding, and consideration of the apple ecosystem as a whole if pest management technology was to improve (Croft and Hull). Pest management based exclu-

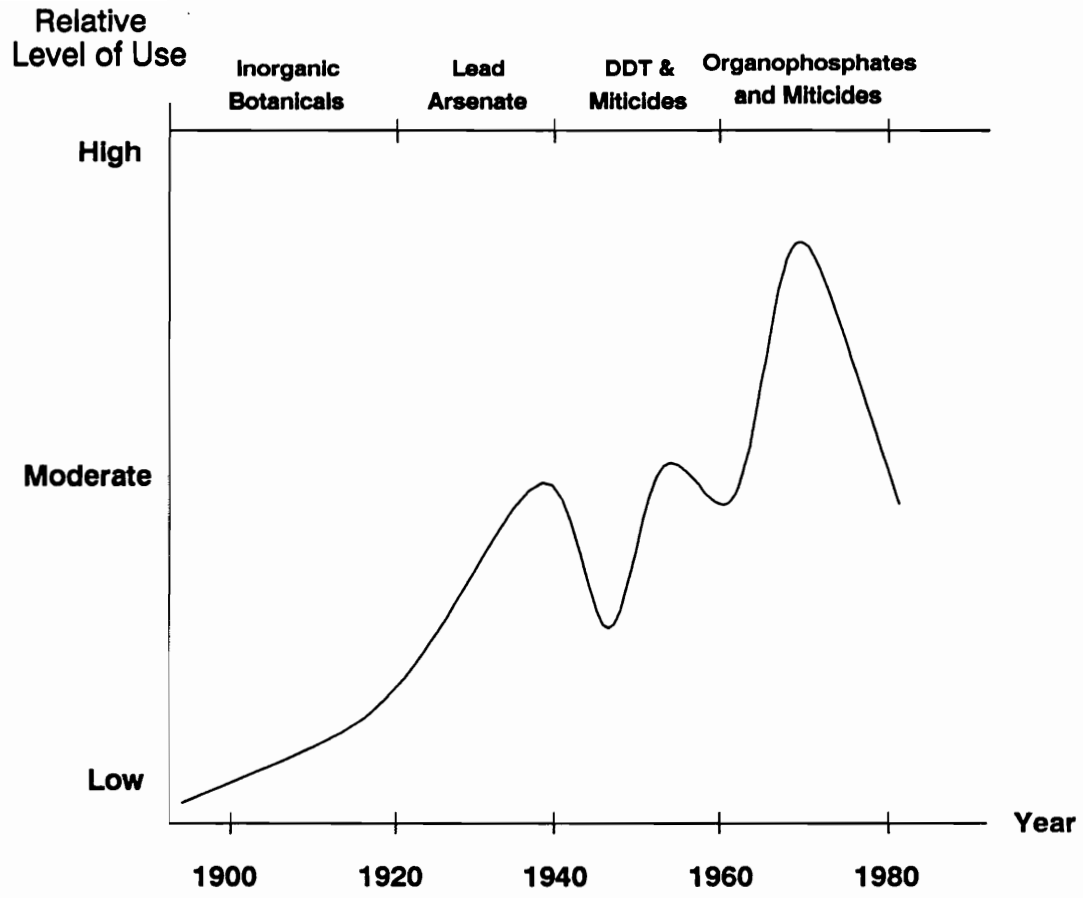

Figure 6. Stylistic Representation of the Historical Trends in U.S. Pesticide Use: Adapted from Croft (1983), p. 6.

sively on synthetic chemicals did provide growers with effective control on a scale not previously enjoyed (Madsen and Morgan), but the deleterious side-effects led to public and regulatory pressure for more selective compounds, better formulations, and more efficient application techniques (Croft). However, accomplishing this within an ecosystem framework proved exceedingly difficult for the complex apple agroecosystem. Because apples are a perennial crop, all production decisions are dynamic in nature, including decisions concerning pest management. This dynamic decision-making arises from the fact that a growers pest management choices not only affect the current year's harvest, but can potentially influence future harvests through the impacts current pest levels and pesticide use have on the future state of apple tree and pest physiology. The existence and implications of this dynamism will become evident through a description of the specific pest complex examined in this study.

A Mid-Atlantic Pest Complex

Empirical economic investigations require that the relevant unit of analysis be carefully defined, thereby identifying the intended scope of implications that may be drawn from the study. In the case of pest resistance, valid economic research could target farm-level, regional, national, or world-wide effects, with the specific focus determined primarily by: 1) the type of questions the study needs to answer; 2) the type of data available for model development and/or estimation; and 3) the structure of the system being examined. But this approach to identifying the unit of analysis generates some apparent conflicts for this study. Because pesticide regulatory agencies are usually elements of federal government, a national model would seem to be the most appropriate, especially if changes in aggregate welfare are of interest. In addition, the marketing of apples occurs on a national scale, with the U.S. market generally assumed to be open to world markets as well. However, agroecosystems that include specific pest complexes are primarily a regional phenomenon, and at their lowest level the impacts of pesticide regulatory actions are generated in specific crop-pest systems. This

apparent need for a regional model is supported by the common-property characteristics of pest resistance, which would place a lower limit on the relevant unit of analysis. While this conflict of problem scope initially appears troublesome, it can be accommodated by the methods used in this study.

Because the apple-pest-predator complex of interest is confined to the mid-Atlantic region, only that region's agroecosystem needs to be biologically modelled. The economic model, on the other hand, needs to recognize the interrelationships between the regional, U.S., and world apple markets. This can be accomplished by assuming that, while the pesticide regulatory actions only directly impact the mid-Atlantic production region,[16] the supply effects of these impacts will be carried through to the national and world markets. In this way, the unit of analysis can, in some sense, be simultaneously regional and larger, with regional impacts relating to pesticide use and resistance development and national impacts relating to changes in aggregate welfare. How this structure is incorporated into the empirical model will be discussed in Chapters VI, VII, and VIII. For now, however, the discussion will concentrate on the mid-Atlantic pest complex.

Pest Categories

Insect pests can be categorized by the type of damage they inflict on the commodity being produced. Direct pests are those that injure the fruit or fruit buds, thereby causing reduced fruit yield and quality in the year of infestation. In general, however, direct pests do not affect tree physiology, and the impacts of direct pests are limited to within year influence on harvest, storability, and marketing. Indirect pests feed on plant fluids, leaves, roots, and/or other plant structures that are not the actual commodity being produced. In doing so, they

[16] The assumption that pesticide regulatory action only affects a given regional crop-pest complex will generally only be valid relative to a specific set of pesticides. If the pesticides being examined have multiple uses across crops and/or pest complexes, then a study such as this one would have to be considered a partial-dynamic analysis, with the partial referring to the fact that other dynamic effects are also likely to result from regulatory action.

only rarely reach sufficient densities to acutely damage the tree or fruit. Instead, indirect pests may cause chronic, multi-year injury to trees. The possible disruption of tree physiology and retarded tree growth can lead to decreased fruit setting, reduced return bloom, and small, poor quality fruit for many years beyond the one in which infestation occurred (Beers and Hull). Thus, pest control decisions involving indirect pests can have long-run, dynamic influences on apple production.

There are a number of factors that determine whether a particular pest complex[17] will become established and economically important in an orchard, with the most obvious being the geographic location and prevailing climate of the region. These factors are important for two reasons. First, the physiological requirements of the complex's organisms must be met, including temperature range, water availability, length of growing season, and food availability. Secondly, the climate and geographic location will determine the type of vegetation existing outside the orchard. If the vegetation is similar to that in the orchard, then the likelihood increases that there will be a natural reservoir to maintain the pest complex regardless of control actions taken in the orchard. This can be especially important in integrated pest management systems, where the reservoir provides a source of natural enemies to respond to pest population increases, thereby reducing the probability that a pest will become economically damaging and uncontrollable. However, when the orchard is managed with the use of broad-spectrum pesticides, any pest is capable of becoming economically important as the natural predator-prey and/or competitive relationships are disturbed. These difficulties, and more, are present in the mid-Atlantic apple pest complex. But before describing the complex and the interrelationships involved, an introduction and biological description of the individual members of the complex is presented below.

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[17] A pest complex can be defined as the combination of direct pests, indirect pests, and natural enemies of the pests that are all interrelated due to ecological or economic processes active in the production system.

Tufted Apple Bud Moth

The Tufted Apple Bud Moth (TABM), *Platynota idaeusalis* Walker, is a major direct apple pest in the Cumberland-Shenandoah region of the mid-Atlantic U.S. (Hull et al. 1988b). Although discovered in Cumberland-Shenandoah orchards in 1923 (Frost), it was not until 1969 that TABM was reported as a serious economic pest. TABM damage is characterized by puncture and feeding marks along the surface of the fruit (Bode et al.; Hull and Rajotte). Because visual fruit appearance is very important in the fresh market grading system, TABM injury can result in a reduction of grade and reduced market price to the grower, although some studies suggest that over 80 percent of TABM damaged apples will still be graded as U.S. #1 (Hull and Rajotte). TABM injury can also lead to significant weight, firmness, and decay loss in apples held in long-term storage (Hull et al. 1988b). Although this type of injury may not always affect grower returns, it does suggest that the marketers of fresh apples and apple processors face potential economic losses due to TABM damage. In fact, the accelerated maturation and decay rate found in TABM injured apples held in storage, despite pre-storage treatment for diseases, is a major concern of the processing industry (Hull and Rajotte). Given that TABM problems in the mid-Atlantic region account for 3 to 6 percent of the annual crop damage, TABM control is an important aspect of orchard management (Hull et al. 1983b; Meagher and Hull; Knight and Hull).

Understanding and evaluating the economic impact of TABM requires some knowledge of its life history and the strategies available for its control. TABM populations overwinter as larvae in the litter and ground cover beneath fruit trees, emerging in the spring to complete development on herbaceous plants and apple root suckers (Frost; Bode et al.). Pupating from late April to early May, the larvae emerge shortly thereafter as adults. This first-brood adult population usually peaks in early June, with subsequent mating and egg laying producing larvae that are active in July and early August (Hull and Rajotte). During this period, larvae will feed on apple leaves and undeveloped fruit, as well as on ground cover (Bode). After pupating in August, second-brood TABM adults emerge and produce the larvae that will

overwinter. It is the second-brood larva that cause the most serious problems because they feed on the fruit just before and during harvest (Hull and Rajotte). Although all apple cultivars are susceptible to this pest, fruit injury levels are highest on the late-season cultivars York Imperial and Rome Beauty, amounting to 4.6 percent and 3.4 percent loss of the total fruit numbers, respectively. Stayman and Red Delicious cultivars experience somewhat lower damage levels, amounting to 1.8 percent and 1.7 percent loss of the total fruit numbers, respectively (Knight and Hull).

Apple producers have recently tried to control TABM by applying organophosphate insecticides three to five times per brood in an alternate-row middle spray pattern (Anonymous).[18] However, control of damaging late-season TABM infestation has proven difficult because pesticide coverage, and thus effectiveness, decreases as the apples mature into a heavy fruit load and as growers meet day-to-harvest restrictions (Hull and Rajotte). The recent emergence of organophosphate resistance in TABM also complicates the pest's management, as producers attempt to find new insecticides or other control procedures. One suggestion for managing resistant TABM in regions of heavily mixed fruit production includes area-wide strategies that utilize susceptible sub-populations of TABM from early maturing fruit that do not receive second-brood sprays as reservoirs from which immigration and dilution of resistant populations can occur (Knight and Hull). In practice, however, producers have begun to switch to alternate insecticide controls such as carbamates and synthetic pyrethroids. Although these are capable of adequately controlling TABM, they can potentially lead to decreased yields because of their unintended effects on the pest complex as a whole. The intricacies of these effects will be detailed later.

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[18] Alternate-row middle spraying is a pesticide application method belonging to the group of mosaic procedures discussed in Chapter II.

European Red Mite

The European Red Mite (ERM), *Panonychus ulmi* Koch, is an economically important and troublesome indirect pest of apples. First reported on the west coast in 1911 (Ewing), this mite moved rapidly throughout the U.S. and was being described as a pest in the Northeast by 1915 (Newcomer and Yothers). ERM inflicts damage on apple trees by feeding on the cell contents of leaves. If heavy enough, this feeding may cause decreased net photosynthesis, reduced transpiration, reduced tree growth, smaller fruit, and decreased return bloom and yield in subsequent years (Avery and Briggs; Ferree and Hall; Asquith; Baker; Beers et al. 1984; Lienk; Rabbinge). Damage can occur to both bearing and non-bearing trees, with the greatest economic impact occurring through long-term reductions in fruit production and quality. Although Beers and Hull suggest that lasting damage may vary widely by cultivar, and that the extent of damage may be dependent on the leaf to fruit ratio present on individual trees (Beers et al. 1987), ERM is considered a general pest of all apple cultivars. Economic losses caused by severe infestations of ERM have been estimated to be as high as 30 percent (Chapman et al.; van de Vries).

As is true for most mite species, ERM has an unusual and problem-causing life history. Overwintered as eggs, ERM hatches at or just before bloom in the spring, depending on the cumulative temperature. In Virginia, eggs usually hatch the last week of April (Cagle). After suffering a winter mortality as high as 90 percent (Lienk and Chapman), the first generation requires approximately three weeks to mature, with subsequent generations maturing in 10 to 14 days (Rabbinge). There are generally four to nine generations of ERM each year, but this is highly dependent on the locality and length of the growing season (Metcalf et al.). In addition, ERM can reproduce parthenogenically; eggs from unmated females develop into males, eggs from fertilized females develop into a mixture of about 63 percent females and 37 percent males. Taken together, the biological characteristics of ERM make for a pest that can rapidly develop into a severe problem in any given orchard.

Although ERM can often be controlled with a single petroleum oil spray directed against overwintering eggs, or with the application of an acaricide targeted at newly hatched mites, established summer populations have proven more difficult to control and may require two or three acaricide treatments spaced at two week intervals (Lienk et al.). However, because ERM has demonstrated the ability to rapidly develop resistance to most acaricides, a biological strategy for control was implemented in Pennsylvania in 1969 (Hull). This biocontrol involved use of the predatory Black Ladybird Beetle (BLB -- *Stethorus punctum* LeConte) in conjunction with low doses of miticides applied as needed using the alternate-row middle system of spraying (Asquith and Hull). This integrated approach turned out to be quite successful, only to be threatened by the recent development of organophosphate resistance in TABM. But before examining that issue, it will be useful to consider the biology of BLB.

Black Ladybird Beetle

The BLB belongs to a family of predaceous beetles and occurs locally as an important natural enemy of mites. Adults normally hibernate from early October until spring in wooded areas adjacent to orchards and on the orchard floor. Eggs are deposited on the undersides of leaves, with the larvae hatching out in about a week. After a short 2 to 3 week development period, the larvae pupate and emerge as adults (Alford). Similar to TABM, BLB is at least double-brooded, with the adults surviving for many weeks and being the critical predatory life-stage as far as ERM control is concerned. Although all life stages may be found from May until early August, BLB tends to move into apple orchards from adjacent wooded areas only when ERM population densities are high enough to support their voracious feeding. ERM populations of 2 to 3 mites per leaf have been shown sufficient for this purpose, with BLB actively seeking out areas of the tree containing the highest densities of ERM (Hull et al. 1983a). BLB has been used to effectively maintain ERM populations at acceptable levels with the aid of one-half to two annual acaricide applications aimed at adjusting predator/prey ratios (Hull

et al. 1983b). Since being incorporated into Pennsylvania's integrated apple pest management program, the use of BLB has reduced grower acaricide applications by nearly 80 percent, with growers now spending only 5 to 10 percent of their pesticide bill for mite control (Asquith and Hull). It has also been estimated that, over the life of the program, acaricide use will decrease by more than 1.5 million pounds for a direct cost savings to growers of over $15 million (Rajotte, personal communication).

The Functional Pest Complex

Throughout the middle decades of this century, chemical controls were the primary method of preventing widescale damage in apple production. Broad spectrum insecticides, especially the organophosphates, were extremely effective against many important economic pests, and ERM infestations were controlled with repeated acaricide application. But by the late 1960's, the situation had changed and growers began to search for control alternatives.

There were two factors which led apple producers to adopt a mixed chemical, biological control program. Acaricides had been highly effective and inexpensive, a situation that led to their indiscriminate overuse. Throughout the 1960's, successful chemical control of ERM had gradually diminished. Because of ERM's ability to rapidly develop resistance, newly introduced acaricides were effective for only 2 to 5 years (Hull). Thus, the relative expense involved in multiple applications of a pesticide with diminishing control ability led the industry to seek alternative pest control measures. At the same time, integrated pest management research, development, and implementation programs were beginning to receive government support. The combined grower search for a way to break the continuous cycle of resistance development in ERM and the institutional support for alternatives to strict chemical control led to the introduction of a new strategy for controlling ERM.

The new strategy for controlling ERM involved the use of the natural mite predator BLB. Researchers discovered that this natural enemy of ERM was capable, in conjunction with low

doses of miticides, of preventing ERM populations from reaching levels that would be economically damaging. Introduced in 1969, this system also enabled growers to significantly reduce their pesticide expenditures. In addition, because ERM populations were exposed to greatly reduced cumulative doses of chemicals and thus did not rapidly develop resistance to the compounds, the IPM system provided a mechanism by which the effective life of available acaricides could be lengthened.

As it turned out, 1969 was also an important year in the mid-Atlantic region because the first significant damage by TABM was reported (Bode et al.). Once recognized as an important pest, TABM was targeted for control using familiar organophosphate insecticides. In addition to being relatively inexpensive, some of these chemical controls did not seriously affect BLB because the predator had become tolerant. So even though TABM and ERM often occurred simultaneously in an orchard, a mixed chemical/biological control strategy could be utilized.

Although this integrated control system worked to control TABM and ERM for a number of years, the strategy became threatened in the early 1980's. TABM populations began to exhibit signs of developing resistance to organophosphate insecticides, and scattered control failures developed. Facing potential economic damage, some growers switched to using mixtures of organophosphates and carbamates or pyrethroid insecticides for TABM control, with both being at least somewhat effective in suppressing TABM populations. Unfortunately, they were equally effective at suppressing BLB populations, which did not exhibit the same high degree of tolerance to carbamate and pyrethroids as they had to the organophosphates. This released ERM from predatory pressure and led to scattered outbreaks of economically damaging mite populations. As a result, some growers again resorted to increased use of acaricides to control ERM, and the likelihood of resistance development resurfaced. Thus, if TABM organophosphate and ERM acaricide resistance expands, the danger is that the integrated control system formed around BLB will cease to be effective. Lacking an effective biological control for ERM, producers will be faced with the same situation as in the 1960s, with ever increasing acaricide use and the potential loss of effective chemicals. In the long-run,

this may lead excessive pesticide contamination of the environment and an apple pest complex that becomes uncontrollable.

Summary

Central to the current study are the dynamic biological interactions that occur in the mid-Atlantic apple agroecosystem. As pests and predators affect one another, and in turn are affected by human management, the system evolves in ways that impact its future operation. Some important characteristics of the mid-Atlantic system that will be incorporated into the modelling effort of subsequent chapters include:

- While the majority of apples produced in the mid-Atlantic region are destined for processed marketing channels, significant fresh market production also takes place;

- Because apples are a perennial crop, production management decisions can potentially influence not only the current year's harvest, but also impact future harvests through the cumulative effects of pest levels and pesticide use on apple tree and pest physiology;

- The Tufted Apple Bud Moth (TABM) is a serious pest because its puncture feeding marks on the fruit surface can result in a reduction of marketing grade and reduced market price received by the grower. This is especially critical for fresh market producers, where TABM can damage fruit to the extent that it is only suitable for relatively low-revenue processed marketing;

- The most damaging brood of TABM reaches maturity late in the season when control is difficult due to heavy fruit loads and day-to-harvest pesticide use restrictions;

- The European Red Mite (ERM) can cause long-term damage to apple trees by feeding on leaves, thereby reducing tree growth, apple size, and return bloom in subsequent years. In fact, losses due to ERM have been estimated to be as high as 30 percent;

- Because ERM is capable of reproducing parthenogenically and may have 4-10 generations per year, it has demonstrated the ability to rapidly evolve genetic resistance to most chemicals. Thus, the use of natural predators for integrated control of ERM has been the focus of management effort in the mid-Atlantic for the last 20 years; and

- The Black Ladybird Beetle (BLB), a predator of ERM, can be extremely effective in an integrated pest management system. However, BLB is susceptible to the non-organophosphate chemicals that may be used if TABM continues to develop resistance to organophosphates. This raises the specter of being unable to control ERM in the intermediate- to long-run.

CHAPTER V: A THEORETICAL MODEL

Previous chapters have discussed the time-dynamic nature of: 1) the apple production system; 2) the interaction of pesticides and pest resistance; and 3) the potential impacts of pesticide regulation on long-term pesticide use and the development of pest resistance. Given the temporal complexity of the resistance problem, a static economic model is inappropriate because it will most likely lead to an incorrect estimate of the relative costs and benefits associated with an insecticide's continued registration. In addition, a static framework is not sufficiently flexible to allow an analysis of the relative merits of insecticide assessments based on entire control sets of pesticides.

In order to provide a mechanism to examine some of these inherently time-dynamic problems, this chapter builds on the general theoretical bioeconomic model presented in Chapter III by detailing a control theory model of the mid-Atlantic apple agroecosystem that includes the TABM-ERM-BLB pest complex. Interpretations of the qualitative aspects of the model's general solution are also presented, followed by a discussion of the theoretical implications of the model for pest resistance and pesticide regulation.

The Model and Its Interpretation

Using the general dynamic model outlined in chapter 3, consider the following characterization of the mid-Atlantic apple agroecosystem containing the TABM, ERM, BLB pest pest-predator complex:

1. Apples are produced with the intent of marketing through either fresh or processed channels, each of which has its own product standards. Thus, assume that there are two commodity types ($h = 1,2$), each having three grade classes ($g = 1,2,3$).

2. Because of the differing physiological demands placed on a tree by specific crop loads, changes in crop growth rates may be negatively related to changes in current yields for all commodity classes ($\frac{\partial \Omega_{h,g}(t)}{\partial y_{h,g}(t)} \leq 0 \quad \forall \; h,g$).

3. Two apple pest species (TABM, $i = 1$; ERM, $i = 2$) and one predator species (BLB, $i = 3$) exist in the typical orchard. In this system, the predator preys on pest 2, but pest 1 does not interact in any meaningful way with either pest 2 or the predator ($\frac{\partial \Gamma_2(t)}{\partial b_3(t)} \leq 0$, $\frac{\partial \Gamma_3(t)}{\partial b_2(t)} \geq 0$, $\frac{\partial \Gamma_1(t)}{\partial b_2(t)} = 0$, $\frac{\partial \Gamma_1(t)}{\partial b_3(t)} = 0$).

4. Changes in growth rates for pest and predator populations usually operate in a positive feedback process with respect to current population levels, fecundity, and immigration, as long as the ecological carrying capacity is not reached ($\frac{\partial \Gamma_i(t)}{\partial b_i(t)} \geq 0$, $\frac{\partial \Gamma_i(t)}{\partial f_i(t)} \geq 0$, $\frac{\partial \Gamma_i(t)}{\partial m_i(t)} \geq 0 \quad \forall \; i$).

5. Pest control inputs $k = 1,2,3$ are targeted solely at pest 1, but may have some negative secondary effects on the predator population ($\frac{\partial \Gamma_1(t)}{\partial u_k(t)} \leq 0$, $\frac{\partial \Gamma_3(t)}{\partial u_k(t)} \leq 0$, $\frac{\partial \Gamma_2(t)}{\partial u_k(t)} = 0 \quad \forall \; k = 1,2,3$).

6. Pest control inputs k=4,5,6 are targeted at pest 2, with no secondary effects on the other pest or the predator ($\frac{\partial \Gamma_2(t)}{\partial u_k(t)} \leq 0$, $\frac{\partial \Gamma_1(t)}{\partial u_k(t)} = 0$, $\frac{\partial \Gamma_3(t)}{\partial u_k(t)} = 0 \quad \forall \; k = 4,5,6$).

7. Rate changes in susceptibility to a given pesticide may be negatively or positively related to the use of that pesticide, depending on the specific intensity and mode of application ($\frac{\partial \Theta_{i,k}(t)}{\partial u_k(t)} \gtrless 0 \quad \forall \; i,k$).

8. Rate changes in susceptibility to a given pesticide may result in similar directional changes in susceptibility to other pesticides (the cross resistance effect, where $\frac{\partial \Theta_{i,z}(t)}{\partial s_{i,k}(t)} \geq 0 \quad \forall \; i = 1,3 \;\; z,k = 1,2,3, \quad \frac{\partial \Theta_{2,z}(t)}{\partial s_{2,k}(t)} \geq 0 \quad \forall \; z,k = 4,5,6$).

9. Because there is often a physiological trade-off between resistance and reproductive ability, changes in pest and predator fecundity rates may be positively related to the changes in susceptibility ($\frac{\partial \Upsilon_i(t)}{\partial s_{i,k}(t)} \geq 0 \quad \forall \; i,k$).

10. Rate changes in crop growth may be negatively related to changes in pest populations but not directly related to changes in predator populations ($\frac{\partial \Omega_{h,g}(t)}{\partial b_i(t)} \leq 0 \quad \forall \; h,g,i = 1,2, \quad \frac{\partial \Omega_{h,g}(t)}{\partial b_3(t)} = 0 \quad \forall \; h,g$).

Optimizing the amount of economic surplus obtainable from this system can be accomplished by solving the following dynamic problem:

$$\underset{U}{\text{MAX}} \quad \text{TES} = \int_0^T \sum_{h=1}^{2} \sum_{g=1}^{3} \left\{ \int_0^{(a_{h,g}\, y_{h,g}(t))} [D_{h,g}(\eta, t) - SY_{h,g}(\eta, U, t)] d\eta \right\} e^{-rt} dt \;\; ,$$

subject to the equations of motion:

$$\dot{y}_{h,g}(t) = \Omega_{h,g}[y_{h,g}(t), b_i(t), w(t), t] \quad \forall \; h, g, i \;\; ;$$

$$\dot{b}_i(t) = \Gamma_i[b_j(t), m_j(t), c_j(t), f_j(t), u_k(t), s_{j,k}(t), w(t), t] \quad \forall\ i, j, k, \text{ where } j = 1,2,3\ ;$$

$$\dot{s}_{i,k}(t) = \Theta_{i,k}[s_{i,z}(t), u_z(t), t] \quad \forall\ i, j, k, z, \text{ where } z = 1..6\ ;$$

$$\dot{f}_i(t) = \Upsilon_i[s_{i,k}(t), t] \quad \forall\ i, k\ ;$$

$$\dot{c}_i(t) = \Psi_i[y_{h,g}(t), t] \quad \forall\ i, h, g\ ; \text{ and}$$

$$\dot{m}_i(t) = \Xi_i[t] \quad \forall\ i\ ;$$

where we define:

U is a fixed set of pest controls in R^6;

$u(t) \in U$;

TES is the total economic surplus over all time periods;

T is the terminal time of the planning horizon;

$D_{h,g}(\eta, t)$ is the demand function for a given crop and grade at time t;

$SY_{h,g}(\eta, U, t)$ is the supply function for a given crop and grade at time t;

$y_{h,g}(t)$ is the yield per acre of grade (g) for crop (h) at time t;

$\Omega_{h,g}$ is the instantaneous change in yield per acre;

$a_{h,g}$ is the total number of acres producing crop h of grade g;

$b_i(t)$ is the density of pest/predator (i) at time t;

Γ_i is the instantaneous change in density of pest/predator (i);

$s_{i,k}(t)$ is the susceptibility of pest/predator (i) to control (k) at time t;

$\Theta_{i,k}$ is the instantaneous change in susceptibility of pest/predator (i) to control (k);

$f_i(t)$ is the fecundity of pest/predator (i) at time t;

Υ_i is the instantaneous change in fecundity of pest/predator (i);

$c_i(t)$ is the environmental carrying capacity for pest/predator (i) at time t;

Ψ_i is the instantaneous change in environmental carrying capacity for pest/predator (i);

$m_i(t)$ is the immigration of susceptible pest/predator (i) at time t;

Ξ_i is the instantaneous change in immigration of susceptible pest/predator (i); and

$w(t)$ is the deterministic influence of weather in time t.

The state and control variables are constrained to be non-negative:

$$y_{h,g}(t) \geq 0\,; \quad b_i(t) \geq 0\,; \quad s_{i,k}(t) \geq 0\,; \quad f_i(t) \geq 0\,; \quad c_i(t) \geq 0\,; \quad m_i(t) \geq 0\,; \quad u_k(t) \geq 0\,;$$

while the upper bounds on the use of the control variables are $u_k(t) \leq \beta_k$, and $D_{h,g}$, $SY_{h,g}$, $a_{h,g}$, $y_{h,g}(0)$, $b_i(0)$, $s_{i,k}(0)$, $f_i(0)$, $c_i(0)$, and $m_i(0)$ all predefined. It is further assumed that the objective functional and the equations of motion are all twice differentiable with respect to each state variable, differentiable with respect to the control set $\{U\}$, and continuous with respect to time.

An interior solution to this particular problem, if it exists, can be obtained by comparisons with the general solution of Chapter III. The resulting marginality conditions can then be examined by rearranging the solution into marginal benefits and costs. Beginning with the control optimality conditions, and taking into account the biological interactions outlined above, it can be seen from equation [1] of the general solution that

$$\lambda_1^b(t)\frac{\partial \Gamma_1(t)}{\partial u_k(t)} + \lambda_{3,k}^s(t)\frac{\partial \Theta_{3,k}(t)}{\partial u_k(t)} =$$

$$-\frac{\partial\left[\sum_{h=1}^{2}\sum_{g=1}^{3}\int_0^{(a_{h,g}\,y_{h,g}(t))}(D_{h,g} - SY_{h,g})d\eta\right]}{\partial u_k(t)} - \lambda_3^b(t)\frac{\partial \Gamma_3(t)}{\partial u_k(t)} - \lambda_{1,k}^s(t)\frac{\partial \Theta_{1,k}(t)}{\partial u_k(t)} \qquad [11]$$

$$\forall\ k = 1,2,3\ .$$

Assuming that the marginal value of TABM population levels ($\lambda_1^b(t)$) and BLB susceptibility ($\lambda_{3,k}^s(t)$) are negative, while the marginal value of TABM susceptibility ($\lambda_{1,k}^s(t)$) and BLB population levels ($\lambda_3^b(t)$) are positive, then the first term on the left-hand side of equation [11] represents the marginal benefit derived from the decreased population growth rate of TABM due to the use of control (k), and the second term is the marginal benefit of the increased resistance buildup in the predator to control (k) that arises due to its use. On the right-hand side of equation [11] are the marginal costs associated with the use of control (k), including the

marginal loss of total economic surplus due to the supply shifting effects of increased pest-control input costs;[19] the marginal cost associated with the decreasing rate of predator population growth due to control (k); and the marginal cost of the increasing rate of resistance buildup in TABM to control (k).

Similarly, from equation [1] the following arises:

$$\lambda_2^b(t)\frac{\partial \Gamma_2(t)}{\partial u_k(t)} = -\frac{\partial\left[\sum_{h=1}^{2}\sum_{g=1}^{3}\int_0^{(a_{h,g}\,y_{h,g}(t))}(D_{h,g}-SY_{h,g})\right]}{\partial u_k(t)} - \lambda_{2,k}^s(t)\frac{\partial \Theta_{2,k}(t)}{\partial u_k(t)} \qquad [12]$$

$$\forall\ k = 4,5,6$$

where, if the marginal value of ERM populations ($\lambda_2^b(t)$) is assumed to be negative and the marginal value of ERM susceptibility ($\lambda_{2,k}^s(t)$) is positive, the left-hand side of equation [12] is marginal benefit of the reduced ERM population growth rate engendered by control (k). On the right-hand side, the costs include the marginal loss in total economic surplus resulting from the supply shifting effects of increased pest control inputs[19] and the marginal costs resulting from the increasing rate of resistance buildup in ERM to control (k). Because the predator is not affected by the controls targeted at ERM, this second set of optimality conditions are not as complex as the first, but both [11] and [12] illustrate that the effects of pesticide use are neither confined to the pest populations nor important only in the short-run. In fact, it is entirely possible for the long-run effects of susceptibility to be of greater magnitude than all the other terms combined. This provides further justification for an empirical study that determines the partition between the long- and short-run effects of pesticide use.

The next set of marginal conditions involves the adjoint variables and provides information on the relationship of the marginal product of the state variables to the marginal

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[19] Note, however, that the marginal costs ascribed to changes in economic surplus might actually be marginal benefits for a given system if the pest population levels in $(t-1)$ are high. This is primarily due to the negative effect of chronic pest population levels on output supply ($SY_{h,g}$).

intertemporal opportunity cost associated with the current use of the state variable. Developed from equation [2]:

$$r\lambda^{y}_{h,g}(t) = D_{h,g}(t) - SY_{h,g}(t) + \lambda^{y}_{h,g}(t)\frac{\partial \Omega_{h,g}(t)}{\partial y_{h,g}(t)}$$

$$+ \sum_{i=1}^{3} \lambda^{c}_{i}(t)\frac{\partial \Psi_{i}(t)}{\partial y_{h,g}(t)} + \dot{\lambda}^{y}_{h,g}(t) \qquad \forall\ h = 1,2,\ g = 1,2,3\ ,$$

which indicates that the socially required rate of return on the value of an additional unit of crop yield in a particular sub-market and grade ($r\lambda^{y}_{h,g}$) is equal to the sum of the demand/supply price gap, the value of additional crop growth, the value of the increased environmental carrying capacity associated with the additional unit of crop yield, and the potential change in value of the yield if it is not currently harvested. Note that, in cases of high pest populations at the carrying capacity constraint boundary, the carrying capacity term will be negative because increased carrying capacity implies higher pest fecundity and increased population growth rates. However, realistic pest management schemes will usually keep pest populations well below the carrying capacity of an orchard, thereby effectively making $\lambda^{c}_{i}(t)$ equal to zero.

The next three relationships can be obtained from equation [3]:

$$r\lambda^{b}_{1}(t) = \sum_{h=1}^{2}\sum_{g=1}^{3} \lambda^{y}_{h,g}(t)\frac{\partial \Omega_{h,g}(t)}{\partial b_{1}(t)} + \lambda^{b}_{1}(t)\frac{\partial \Gamma_{1}(t)}{\partial b_{1}(t)} + \dot{\lambda}^{b}_{1}(t) \qquad [13]$$

$$r\lambda^{b}_{2}(t) = \sum_{h=1}^{2}\sum_{g=1}^{3} \lambda^{y}_{h,g}(t)\frac{\partial \Omega_{h,g}(t)}{\partial b_{2}(t)} + \lambda^{b}_{3}(t)\frac{\partial \Gamma_{3}(t)}{\partial b_{2}(t)} + \lambda^{b}_{2}(t)\frac{\partial \Gamma_{2}(t)}{\partial b_{2}(t)} + \dot{\lambda}^{b}_{2}(t) \qquad [14]$$

$$r\lambda_3^b(t) = \lambda_2^b(t)\frac{\partial\Gamma_2(t)}{\partial b_3(t)} + \lambda_3^b(t)\frac{\partial\Gamma_3(t)}{\partial b_3(t)} + \dot{\lambda}_3^b(t) \qquad [15]$$

Each of the relationships above describes the implications of changes in the population level of a predator or pest. Given that only pest 1 and 2 deleteriously affect crop yield, then equations [13] and [14] represent the socially acceptable costs that would be incurred due to a marginal unit increase in pest populations. Equation [15] represents the marginal social benefits obtained from a unit increase in the predator population, and thus in its ability to control pest 2. Specifically, the marginal social costs associated with TABM ($r\lambda_1^b(t)$) is dependent on the value of the yield losses incurred from a population increase in TABM, the costs associated with the increase in the population growth rate of TABM, and the potential increases in the costs associated with TABM if it is not controlled at current population levels. Assuming that $\frac{\partial\Gamma_1(t)}{\partial b_1(t)} > 0$, or no carrying capacity constraints on the organism, then equation [13] suggests that the marginal social costs associated with additional levels of TABM are ever increasing. A similar interpretation holds for equation [14], although the social costs associated with increasing ERM population levels are offset to some degree by the benefits associated with the increasing population growth rate of the predator BLB ($\lambda_3^b(t)\frac{\partial\Gamma_3(t)}{\partial b_2(t)}$). Lastly, equation [15] suggests that the socially required benefits of an additional unit of predators is dependent both on the value of the predator in controlling the growth rate of ERM populations ($\lambda_3^b\frac{\partial\Gamma_2(t)}{\partial b_3(t)}$) and on the value associated with increasing predator populations, along with the potential change in the value of the predators if they are not destroyed.

The next two relationships, describing the implications of changes in pesticide susceptibility, can be obtained by manipulating equation [4]:

$$r\lambda_{i,k}^s(t) = \lambda_i^b(t)\frac{\partial\Gamma_i(t)}{\partial s_{i,k}(t)} + \sum_{z=1}^{k-1}\lambda_{i,z}^s(t)\frac{\partial\Theta_{i,z}(t)}{\partial s_{i,k}(t)} + \sum_{z=k+1}^{3}\lambda_{i,z}^s(t)\frac{\partial\Theta_{i,z}(t)}{\partial s_{i,k}(t)} +$$

$$\lambda^s_{i,k}(t)\frac{\partial\Theta_{i,k}(t)}{\partial s_{i,k}(t)}+\lambda^f_i(t)\frac{\partial\Upsilon_i(t)}{\partial s_{i,k}(t)}+\dot{\lambda}^s_{i,k}(t)\quad\forall\ i=1,3\ \ k=1,2,3\ ; \tag{16}$$

$$r\lambda^s_{2,k}(t)=\lambda^b_2(t)\frac{\partial\Gamma_2(t)}{\partial s_{2,k}(t)}+\sum_{z=4}^{k-1}\lambda^s_{2,z}(t)\frac{\partial\Theta_{2,z}(t)}{\partial s_{2,k}(t)}+\sum_{z=k+1}^{6}\lambda^s_{2,z}(t)\frac{\partial\Theta_{2,z}(t)}{\partial s_{2,k}(t)}+$$

$$\lambda^s_{2,k}(t)\frac{\partial\Theta_{2,k}(t)}{\partial s_{2,k}(t)}+\lambda^f_2(t)\frac{\partial\Upsilon_2(t)}{\partial s_{2,k}(t)}+\dot{\lambda}^s_{2,k}(t)\quad\forall\ k=4,5,6\ . \tag{17}$$

Equation [16] describes the impacts of changes in pesticide susceptibility in both TABM and the predator BLB, both of which are generally affected by the first three pesticides. Specifically, the socially required rate of return on the value of preserving an additional unit of TABM susceptibility to pesticide k is dependent on the sum of the net values associated with: 1) the potential for decreased growth rates through increased pesticide efficacy; 2) the increased susceptibility to other pesticides caused by an increase in susceptibility to pesticide k, or the opposite of the cross-resistance effect; 3) the effect of the current susceptibility on the rate of change in susceptibility; 4) the increases in fecundity caused by an increase in susceptibility; and 5) the potential change in susceptibility if it is not currently depleted through pesticide use. A similar interpretation of equation [16] for BLB and equation [17] for ERM can be used, although in the case of BLB the expression depicts the socially acceptable costs that are incurred due to preserving an additional unit of predator susceptibility. Preserving BLB susceptibility can be interpreted to imply potential costs because a predator susceptible to chemical controls targeted at TABM is less reliable as a non-chemical control against ERM if pesticides are used against TABM.

If we assume that carrying capacity is not constraining in this problem, equation [6] drops out and equations [5] and [7] can be rearranged to yield:

$$r\lambda^f_1(t)=\lambda^b_1(t)\frac{\partial\Gamma_1(t)}{\partial f_1(t)}+\dot{\lambda}^f_1(t)\ ; \tag{18}$$

$$r\lambda_i^f(t) = \sum_{j=2}^{3} \lambda_j^b(t) \frac{\partial \Gamma_j(t)}{\partial f_i(t)} + \dot{\lambda}_i^f(t) \quad \forall\ i = 2,3\ ; \qquad [19]$$

$$r\lambda_1^m(t) = \lambda_1^b(t) \frac{\partial \Gamma_1(t)}{\partial m_1(t)} + \dot{\lambda}_1^m(t)\ ;\ \text{and} \qquad [20]$$

$$r\lambda_i^m(t) = \sum_{j=2}^{3} \lambda_j^b(t) \frac{\partial \Gamma_j(t)}{\partial m_i(t)} + \dot{\lambda}_i^m(t) \quad \forall\ i = 2,3\ . \qquad [21]$$

Equations [18] and [20] characterize the affect of changes in TABM fecundity and migration, respectively. But, because the number and genetic susceptibility of the immigrating organisms will tend to oppose each other in determining their impact on the agroecosystem, these conditions are difficult to interpret and are dependent on second-order effects. In general, however, the socially acceptable cost incurred due to increases in TABM fecundity are dependent on the change in value resulting from changing TABM population growth rates tempered by the potential change in the value of fecundity if it is not allowed to vary. Likewise, the socially acceptable benefit of immigration is dependent on the change in value associated with increased TABM population growth rates, offset to some degree by the potential change in value of immigration if it is not allowed to occur. Equations [19] and [21] describe similar fecundity and migration relationships for ERM and BLB, with the exception of an additional term related to the predator-prey interactions and their affects on population growth rates.

Theoretical Implications of the Model

The control optimality, adjoint variable, and transversality conditions are all important in characterizing the nature of any complete solution to a dynamic, control theory optimization problem. However, within a qualitative analysis of pest resistance, pesticide regulation, and its impacts on pesticide use, equations [11], [12], [16], and [17] provide most of the critical information. These equations describe the optimal use of pest control inputs and the optimal time-path of susceptibility which arises from such use, and thus the optimal time path of resistance development.

Equation [11] describes the optimal use time-path for pesticides targeted at the TABM. Because the mid-Atlantic apple production system relies on an integrated pest management program in which the ERM is primarily controlled by the BLB predator, decisions concerning chemical control of TABM cannot be made without considering the secondary impacts on other organisms in the system. In particular, TABM targeted pesticide use will tend to be limited not only by the potential development of resistance in TABM, but also by any lethal effects of TABM controls on BLB. While there may be a legitimate philosophical reason for preserving a non-pest species like BLB, the motivating force in this model arises from concern over the ability to control ERM. If BLB were a relatively poor control of ERM, then its marginal value at most relative population levels would be small compared to the marginal cost associated with not controlling TABM. This scenario would tend to lower the economic threshold for TABM and lead to an increase in TABM targeted control actions. If, however, BLB was a relatively effective predator of ERM, then it would have a larger marginal value at most population levels and would tend to raise the economic threshold for TABM, resulting in reduced TABM targeted control actions. Of course, the extent of the economic restrictions limiting the chemical control also depends on a number of other factors, including the relative toxicity of the pesticides to TABM and BLB, the monetary cost of the pesticides, and BLB's ability to

develop resistance.[20] Because BLB's ability to develop resistance is small compared to that of TABM, a particular insecticide can be optimally used only if the value of its efficacy toward TABM is higher than the costs of its toxicity towards BLB. This condition occurs because of the need to account for the indirect losses associated with both the pesticide's monetary cost and the differential in resistance development.

Equation [12], which describes the optimal use time-path for pesticides targeted at the ERM, can be interpreted in much the same way as equation [11]. By definition, however, insecticides targeted at ERM are not constrained by non-target effects on BLB. This leads to the condition where the marginal value of the pesticide's efficacy towards ERM only needs to be large enough to compensate for the economic surplus and ERM resistance related value losses it engenders. But, if the short-run evidence from the real mid-Atlantic production system is valid, then the speed at which ERM can develop resistance and ERM's potential multi-year impact on orchard productivity will both result in a high marginal value for continued ERM susceptibility. Considering only equation [12], this implies a high economic threshold for ERM pest populations. If equation [11] and [12] are simultaneously considered with equation [17], then it becomes apparent that there will be a complex trade-off between controlling TABM and preserving BLB populations so that ERM can be controlled with minimal use of chemicals.[21] Thus, the high potential for resistance development in ERM does not necessarily require high economic thresholds and large ERM populations, with the potential for low thresholds dependent, at least in part, on a successful integrated control program.

Given the optimal use of pest control inputs as described in equations [11] and [12], the development of pest resistance (or loss of susceptibility) is detailed by equations [16] and [17]. Of particular interest are the potential effects of cross-resistance. The development of "own-

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[20] Recall that the ability of a natural predator to lose susceptibility is a desirable trait in an agroecosystem managed using IPM techniques. This desirability is reflected in equation [11] by the term

$$\lambda^{s}_{3,k}(t)\ \frac{\partial \Theta_{3,k}(t)}{\partial u_k(t)}\ .$$

[21] It should be mentioned that field experience indicates BLB may not provide complete control of ERM under all conditions. Thus, some chemical use may be inevitable.

chemical" resistance in both TABM and ERM will, as might be expected, increase the social value of preserving the next unit of susceptibility. At the same time, however, the potential for developing cross-resistance over a wide range of chemicals would imply that the value of preserving susceptibility becomes quite large for the pests, while the costs associated with not reducing predator susceptibility also become large. In addition, the importance of resistance in any given time period will be highly dependent on the interest rate, or time preference, used to discount future flows of surplus from the agroecosystem. As the discount rate increases, resistance, being a long-run phenomenon, becomes less important as preferences for short-run surplus are expressed. If, however, preferences are for the extended operation of the mid-Atlantic production system as it is currently managed, then the lower discount rate implied by this objective would result in an increased influence of susceptibility on the optimal time-paths of pesticide use.

As may be obvious from the mathematical and verbal exposition in this chapter, consideration of time-dynamic optimization problems in agriculture can quickly lead to a complex set of conditions that are difficult to analyze individually, let alone as a set. While there may be a number of other theoretical implications that could be drawn from the model above, they mostly relate to biological characteristics that are perhaps better described in the context of an empirical model. Thus, following a brief summary, the next three chapters will detail the development and implementation of an empirical counterpart to the theoretical model.

Summary

This chapter presented a theoretical control model of the mid-Atlantic apple agroecosystem that highlighted the important long-run economic and biological relationships determining the optimal use of pesticides. Consideration of these relationships, and how they change under varying regulatory scenarios, will be the next focus of this dissertation. Therefore, the following list reiterates some of the key results of the above analysis:

- Because the mid-Atlantic agroecosystem consists of interacting, genetically dynamic pests and predators, the effects of pesticide use are neither confined to the targeted pest population nor important only in the short-run;

- The desirability of maintaining viable BLB predator populations, and therefore an active integrated pest management system, depends not only on how effective BLB is in controlling ERM, but also on how costly it will be if TABM populations increase as a result of managing the BLB/ERM subsystem;

- The presence of cross-resistance potential in pest populations greatly complicates the ability to control intricate pest complexes, but it also leads to potential options for the beneficial development of resistance in predators;

- Long-run changes in the genome of a pest, and the way in which this change effects the ability to control pest population levels, may result in a situation where it is economically important to preferentially manage long-run resistance over short-run population levels; and

- The relative trade-off between management of pest population levels and pest resistance will depend on society's preferences for short- versus long-run gain.

CHAPTER VI: EMPIRICAL MODEL I -- SIMULATION

The theoretical model described in chapter 5 presents some imposing problems for empirical research. System complexity, both in the biological and economic processes, all but assures that the computational procedures typically used in pesticide resistance studies will be unwieldy and ineffective. Analytic representation of the objective function and system constraints, while technically possible, leads to such a large set of simultaneous equations that standard solution methods are not practical. Faced with these difficulties, researchers usually simplify the system model. Unfortunately, this simplification often results in a model that is a poor representation of reality and its solution, although theoretically interesting, is of little policy-making value. Because this study is concerned with the development of a relevant policy-making tool, simplification for the purpose of tractability was avoided as much as possible. Given the complexity of the apple agroecosystem and the modelling preferences of entomologists cooperating in the study, simulation was chosen as the best practical method to model the biological system and the evolution of pest resistance. Therefore, this chapter discusses the concept of simulation in research; describes how biological and economic simulation have been previously employed in agricultural economic investigations; and details the specific biological simulation model used in this study.

Using Simulation in Research

Simulation modelling can be defined as the use of a computer to numerically evaluate a system model, with the resulting output taken as an estimate of the real system's true characteristics (Law and Kelton). Simulation models are often characterized based on their treatment of time and uncertainty; they are either static or dynamic, discrete or continuous, and deterministic or stochastic. A static model describes a system at a particular point in time, while a dynamic simulation model attempts to represent the system as it evolves over a period of time. Discrete and continuous simulation models are differentiated based on their concept of how time relates to the state of the system being modelled. If the state variables change only at a finite number of points in time, then the model is discrete; if the state variables change continuously with respect to time, the model is continuous. Deterministic simulations contain no random variables, and therefore generate a unique set of output data for a given set of inputs. On the other hand, stochastic simulation models contain one or more random variables, and thus have output data that are themselves random. As an example of these differing characteristics, a simulation model of a bank would normally treat the inter-arrival and service times of customers as discrete random variables, each with their own probability distributions. Such a model would be termed a discrete-dynamic-stochastic simulation. Alternatively, a typical Monte Carlo simulation, which uses random variables for solving problems where the passage of time plays no role, would be a static simulation model that is either stochastic or deterministic, depending on the problem formulation (Law and Kelton).

Although the approximating nature of simulation modelling may make it imperfect relative to explicit mathematical analysis, it has demonstrated value as a practical substitute for more expensive physical, biological, and social experiments (Anderson; Johnson and Rausser; Miranowski). But simulation modelling does not eliminate the need for careful use of quantitative methods, for its implementation tends to raise theoretical and statistical

questions. In particular, sufficient data to completely parameterize a realistic simulation is rarely available, and the only alternative may be to supply the missing information from prior knowledge about the type of system being studied. This non-statistical parameterization raises issues concerning the integrity of simulation modelling as an experimental device. How these issues are resolved is intimately linked to the way in which simulation models are developed and implemented.

Developing Simulation Models

The process of constructing a simulation model plays a critical role in determining whether or not it will be an appropriate research tool. Five important simulation development steps have been identified (Law and Kelton; Johnson and Rausser):

1. **Specification** concerns the operational structure that must be given to a model once it has been logically conceived and designed. This step requires that behavioral relationships be identified in a way that avoids embedding conflicting hypotheses in the model. The easiest way to accomplish this is to specify the model in a very basic form, usually consisting of a well-defined and inter-related system of equations. However, many large-system models tend to be specified *ad hoc*, primarily because of gaps in the theoretical understanding of how the system operates or the inability to gather information on actual system relationships. Shortcomings in the specification step often make it difficult to objectively evaluate the information a simulation model generates, but these difficulties may be overcome to some degree by carefully identifying the degree of *ad hocness* in each system relationship so that the model can be independently evaluated by individuals other than those that constructed it.

2. **Parameter estimation** for specified system relationships can occur by methods that range from standard econometrics to subjective expert opinion, depending on the availability

of data, the likelihood of structural change, and a modeller's own simulation philosophy. Although many researchers would insist that the only valid parameter estimates are those that are based in statistics, it is important to note that simulation modelling was originally conceived and is uniquely designed to accommodate a wide range of information types (Law and Kelton).

3. **Verification**[22] is the process by which a simulation model's performance is evaluated relative to its intended purpose. In large, dynamic simulations, verification includes tests for syntax, specification, and programming errors. The operation of the computer code must be in accordance with the original logic and design of the systems model, and output needs to be checked in order to insure that the program can reproduce the data upon which it is based. In short, "...review of a model to determine if its behavior is as anticipated during construction can be regarded simply as an essay in applied common sense" (Anderson).

4. **Validation** is concerned with how well the simulation model corresponds to the real system. All models of complex systems are partially reduced forms because they represent incomplete specifications of the real system being examined. Thus, it is always possible to imagine a more fundamental description of the system (Johnson and Rausser). This suggests that a model cannot be solely judged by the resemblance between its specification and the real system. The ability to generate accurate predictions can, at least in part, be a measure of model validity (Anderson). Another standard is the correspondence of the simulation model's parameter values with the theoretical model's restrictions. While this entire validation process is clearly subjective and presents a number of interesting problems (Naylor and Finger), it should be noted that validation of simulation and analytic models is not compatible or comparable. The philosophical framework upon

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[22] Although verification and validation were once thought to constitute a major roadblock to the scientific use of simulation, research over the last decade has provided methods for assuring not only that the simulation is operating as intended, but also that it corresponds to the operation of the real system (Law and Kelton).

which most large-scale simulation modelling is based assures that simulation and analytics become competing alternatives in the modelling process.

5. **Revision**, which continually occurs as simulation models are designed and constructed, focuses on altering the model to make it more suitable for the intended objectives. The type of revisions and their impact are usually based on: 1) comparisons with the real system; 2) the implications of model output; 3) refinements of modelling objectives; and/or 4) improvement in the operation of the simulation model itself. This process is as much a part of the prior information used to construct the model as the more frequently discussed theoretical justification and data sources. Thus, revisions need to be systematically detailed if the model is to be amenable to evaluation for other research efforts (Johnson and Rausser).

When simulation studies are criticized, it is usually for not explicitly describing the process by which the above steps are implemented. This kind of omission usually arises because the interdisciplinary nature of simulation research makes it difficult to justify development and evaluation based on the norms of any single discipline. With this potential problem in mind, the simulation model used in this study will be detailed as completely as possible (primarily in Appendices A through E). But before giving an overview of this process, a summary of the advantages/disadvantages of simulation modelling and a discussion of its previous use in agricultural economics is presented.

Advantages and Disadvantages of Simulation

The advantages of simulation for empirically analyzing complex problems tend to revolve around the simplicity of implementation relative to applied analytic techniques (Smith 1973a). Even though advanced mathematical skills are usually not required, highly refined and intricate interactions among system components can be modelled within a simulation frame-

work. Simulation may take the form of an analytic, multi-equation model, but it can also be based on relationships that employ stochastic and fuzzy logic in their representations of the system and the details of related human decision-making. This approach to modelling has proved useful in analyzing complex problems, and provides a process-oriented structure through which various disciplines can interact (Trapp). An additional advantage of simulation is that many policy-makers,

> ... being operationally oriented, tend to put more faith in the results of a simulation than in the results of mathematical abstractions because the former frequently appear to be directly simulating operational procedures (Koopman and Berger).

This ability to generate and maintain credibility with policy-makers can be an important consideration for an analyst interested in seeing his recommendations considered and implemented (Majone).

Although simulation has many advantages as an investigative technique, it does have one major disadvantage, especially for agricultural economists. Once a verified and validated simulation has been developed, optimization of the system should entail parameter sweeps, a process where one or more model parameters are systematically and exhaustively varied and their effects on simulation responses noted (Minkoff). But the extent of this process usually promotes the less ambitious task of developing limited comparisons of potential policy options, with little or no information generated regarding the relationship of these policy options to the potential optimums for the system. The *ad hoc* nature of these investigations can lead to reduced credibility for simulation studies (Boggess; Musser and Tew). This, in turn, results in a general unwillingness on the part of many economists to engage in simulation studies. Combined with an economist's tendencies toward deterministic optimization and mathematical rigor, these disadvantages have contributed to some of the uneasiness generated when working on interdisciplinary problems with other professionals. The situation is unfortunate, for the principles of rigorous decision-making and the behavioral insights that economists can bring to interdisciplinary simulation studies are often sorely lacking. In fact, some agricultural economists have actually been attempting to optimize simulation models for some time, but without clearly defining the nature and importance of their investigations

(Trapp). These and other uses of simulation within agricultural economics are discussed in the next section.

Simulation in Agricultural Economics

Simulation was introduced as a potential research tool in agricultural economics at about the same time that the definition of an agricultural problem was being expanded to include issues surrounding natural resources, economic development, and national economic policy (Johnson and Rausser). Although the early realization that simulation did not necessarily imply optimization and closed-form mathematical solutions resulted in some frustration and concern that simulation models could, like statistics, "prove anything" (Dillon), a number of agricultural economists began to employ the method in non-optimizing studies of agricultural policy-making. As early as 1974, review papers were published concerning the application of simulation in agricultural economics (Anderson). Over time, simulation proved useful for realistic modelling of agricultural problems that incorporated multi-disciplinary economic, biological, sociological, and physical information (Trapp).

Simulation models in agricultural economics have been developed primarily for analyses of farm- and region-specific problems, although a number of large-scale macroeconomic simulations have also been attempted. Investigations covering the timing of product sales (Eidman et al.), feed management rules (Dent), the economics of soil conservation (Dumsday), and general farm planning in stochastic environments (Halter and Dean; Hinman and Hutton) comprised some of the earliest work. These studies tended to be simple relative to current applications, with simulation being used to tentatively address inherent dynamic and stochastic aspects of the problems posed (Anderson). But with time came increasing sophistication. For example, Boehlje and Griffin studied the effect of alternative price-support programs on income and land values for different size farms. Holland and Young analyzed the impacts of crop acreage limitations on varying farm sizes. Agricultural simulation studies

even branched out to address potential policy options concerning pest management and the regulation of pesticides (Reichelderfer and Bender).

While simulation originally held promise as a method by which economic experiments could be performed without having to manipulate real social systems, none of the studies mentioned above had the capability to determine a local or global optimal strategy. Instead, the authors usually investigated some small set of potential options. But because neoclassical economics is grounded in optimization, researchers eventually began to link simulation models with various optimization methods. Many of these studies combined simulation with established economic optimization approaches like linear, non-linear, or recursive programming (Chien and Bradford; Kingma; Richardson and Condra; Dudley and Burt). Some studies also applied advanced, general purpose optimization algorithms. For example, Zusman and Amiad examined a simulated firm model response surface using steepest ascent pattern searches. This method is a type of numerical gradient optimization that allowed the authors to direct their sequences of policy simulations away from inferior regions of solution space. Schechter modelled both macro and micro components of a farm management model to develop a response surface generator, after which a random search for the optimal policy was conducted. More recently, Talpaz et al. used a boll weevil-cotton simulator to provide the system dynamics in a model that employed the Fletcher-Powell-Davidson method for non-linear optimization, while Guimaraes and Kingsman utilized a grain terminal simulator and Powell's DSC method to investigate delivery/inventory problems.

Various studies have demonstrated that simulation optimization can be used to investigate problems with a specific structure or with limited dimensions. But the apparent inability to successfully link a general purpose simulation optimization process with complex models has contributed to the underutilization of simulation as a modelling tool in economics, particularly when compared to its widespread adoption in other disciplines. While correcting this discrepancy is dependent, for most economists, on overcoming the non-optimizing nature of many simulation efforts, simulation optimization procedures have not been enthusiastically accepted because they frequently limit the state-size of research models. However, general

methods for simulation optimization that do not inhibit system characterization or analysis have been reported in the operations research literature, and they would appear to provide much of the mathematical and optimizing rigor demanded by economists. The specific approach used in this study, the Modified Complex Method, will be detailed in chapter 8. Given that a general simulation optimization method is available, the remainder of this chapter will concentrate on a description of the biological simulation model that forms the backbone of the current investigation.

Simulating the Mid-Atlantic Pest Complex

An empirical analysis of the interactions between pest resistance and pesticide regulations is dependent on the biological processes that occur in specific agroecosystems. Critical links exist among a pest organism's genetic constitution, the ability to control the organism's population levels, and the economic output obtainable from crop production. Given the intricacies of the mid-Atlantic pest complex, its operation was modelled using an advanced computerized simulation called SERA (Simulating the Evolution of Resistance in Arthropods). SERA was originally designed to allow dynamic investigations into the evolution of a single organism's genome and its reaction to the use of a single insecticide (Tabashnik and Croft 1982). It was subsequently validated and used in other entomological research (Tabashnik and Croft 1985; Tabashnik). As modified for this study, SERA incorporates the relevant pest/predator population dynamics and the use of multiple pesticides (Knight; Kazmierczak). Before describing the specific operation of the SERA model, a conceptual explanation of the genetics underlying the analysis is in order.

Basic Genetics of SERA

The main purpose behind modelling the evolutionary genetics of a system is to predict the rate change in the frequencies of genes present in the modelled population. Accomplishing this within the framework of an economic analysis, however, presents some interesting difficulties. In particular, agroecosystems that display pest resistance have a mixture of renewable and non-renewable resource characteristics. Perennial crops, such as apples, can generally be considered a renewable resource in the intermediate- and long-run. But, pest susceptibility is fundamentally a short-run exhaustible resource, perhaps becoming renewable only in the very-long-run (Mangel). Modelling this exhaustibility requires a truly dynamic[23] simulator that can generate sequential genome shifts. The simplest approach to these calculations, and the method utilized in the SERA model, is to assume that resistance can be described as a single-locus, two-allele semi-dominant inheritance mechanism. Given this assumption, the evolution of resistance to insecticides can be modelled with the well-known Hardy-Weinberg formula.

In a biological population that is well-mixed, at equilibrium, and possessing of the following characteristics (Nisbet et al.):

1. The inheritance mechanism consists of one locus, two alleles;

2. There are no spontaneous or induced mutations;

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[23] Truly dynamic economic models are those that explicitly recognize the time-dependent characteristics of the system being modelled and the need to optimize intertemporally across the entire planning horizon. These dynamic models can be contrasted with pseudo-dynamic models, which approach the time-dependent analysis of a system by breaking the planning horizon into multiple periods. Each period is then modelled using some form of static simulator, with the state of the system projected over the planning horizon by optimization in period t, followed by updating the state of the system before optimization in period $t + 1$ (McDill). Typically, the discrete time-periods chosen for pseudo-dynamic models are determined by data availability or convenience. If, however, the time periods are chosen based on knowledge of system function, and they reflect periods during which little relevant change in system state can be expected, then pseudo-dynamic models will closely approximate a truly dynamic model.

3. Mating is random;

4. Generations are non-overlapping in their mating patterns;

5. Differential survivability prior to reproduction (viability) is the only selective force; and

6. There is constant (*inter alia* density independent) relative individual fitness;

the frequencies of zygotic genotypes can be calculated using the Hardy-Weinberg equilibrium relationship

$$P = p^2, \quad Q = 2pq, \quad R = q^2$$

where p represents the zygotic frequency of the susceptibility allele S, q represents the zygotic frequency of the resistance allele R, P represents the homozygotic susceptibility genotype SS, Q represent the heterozygotic genotype RS, and R represents the homozygotic resistant genotype RR.[24] If $q(n)$ is defined as the frequency of the R allele in generation n, then a dynamic Hardy-Weinberg relationship can be formulated according to the following difference equation (Comins):

$$q(n+1) = \frac{V_{RR}\, q(n)^2 + V_{RS}\, q(n)\,[1 - q(n)]}{V_{RR}\, q(n)^2 + 2V_{RS}\, q(n)\,[1 - q(n)] + V_{SS}[1 - q(n)]^2} \qquad [22]$$

where V_{ij} is the survivorship probability of an individual of genotype ij in generation n. Perhaps the most important aspect of equation [22] is that, as $q(n)$ becomes very small,

$$q(n+1) \sim \left(\frac{V_{RS}}{V_{SS}}\right) q(n) + f[q(n)]$$

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[24] Refer back to Chapter 2, footnote 5, for definitions of the genetic notation used in this section.

so that there is exponential growth or decay of resistance for small levels of resistance, and the rate of growth or decay depends primarily on the relative survivorship levels of homozygous and heterozygous susceptible genotypes (Mangel). In the absence of cross-resistance, this relationship may help to explain why studies repeatedly find a relative constancy in the number of generations it takes for resistance to develop (May and Dobson).

Based on the genetic inheritance model outlined above, the SERA model tracks the dynamic fluctuations in an organism's susceptibility to pesticides. By recognizing that potential differences in survivability are dependent not only on pesticide susceptibility but also on the life-stage of the organism, fertility, environmental carrying capacity, and pest/predator interactions, SERA is capable of modelling an agroecosystem with extremely complex pest and predator population ecologies.

Structure of the SERA Model

SERA can best be described as a discrete-dynamic simulation model with the capability of being either deterministic or stochastic, depending on how weather data, and thus the persistence of pesticide residue, is generated. The simulation is parameterized based on user inputs that 1) define the life-history of the species being simulated, and 2) detail the technical characteristics of all potential pest controls. Required life-history information includes initial growth rates; daily and winter natural mortality; fecundity; population age structure; immigration rates from a variety of sources; densities; environmental carrying capacities; potential interactions with prey or predators; initial zygotic frequencies for resistance and susceptibility; potential cross-resistant genetic linkages; and the fitness effects of the resistance alleles. The data is either specified for each relevant developmental stage of each species or it is subsequently apportioned by developmental stage. As the level of detail suggests, SERA actually tracks the emergence of resistance in each developmental stage of a species and not just for the population as a whole. This complexity is necessary given that

natural and control-induced mortality rates can vary widely depending on the age of an organism. Required pest control information includes the timing and dosages of control; the relative toxicity of each control by species developmental stage; the extent of potential refugia from controls; and chemical half-lives in the environment. All totalled, this information allows the SERA model to generate the discrete time-paths, under any control regime, of: 1) species population densities by life-stage; 2) species resistance levels to each control by life-stage; and 3) the cumulative environmental chemical loadings. A summary description of the SERA model is given in Figure 7.

The application of the SERA model to the resistance/regulation problem required that two versions of SERA be parameterized and run sequentially. Separate parameterizations allowed TABM and ERM/BLB populations to be modelled distinct from each other, an approach suggested by the apparent biological isolation observed between TABM and ERM/BLB in the real system. The sequential execution of the simulations was determined by computer hardware constraints -- parallel execution would be preferred on the basis of speed. But regardless of how the simulation was conducted, the SERA model executed in a similar fashion for any parameterization.

The SERA model begins a simulation by reading the relevant life-history and control parameters from a data file (Figure 8). Initial population densities and resistant alleles are then distributed among life-stages so as to approximate observed field relationships. Following this setup, the simulation enters the main calculation phase. First, a determination is made as to whether a control action is scheduled for the currently simulated period.[25] If a pest-control action is scheduled, then the current environmental load of each control is updated to include both the current period's action and any residual controls that might be present from action in previous periods. If this environmental dose is positive, then control-induced mortality is calculated for each life-stage, taking into account the distribution of genotypes within each life-stage and the possibility of escape from control via refugia. Sec-

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[25] For the purposes of this study, specific chemical and dosage application schedules were determined external to the SERA model, for all but the initial time period, via the optimization algorithm discussed in Chapter 8.

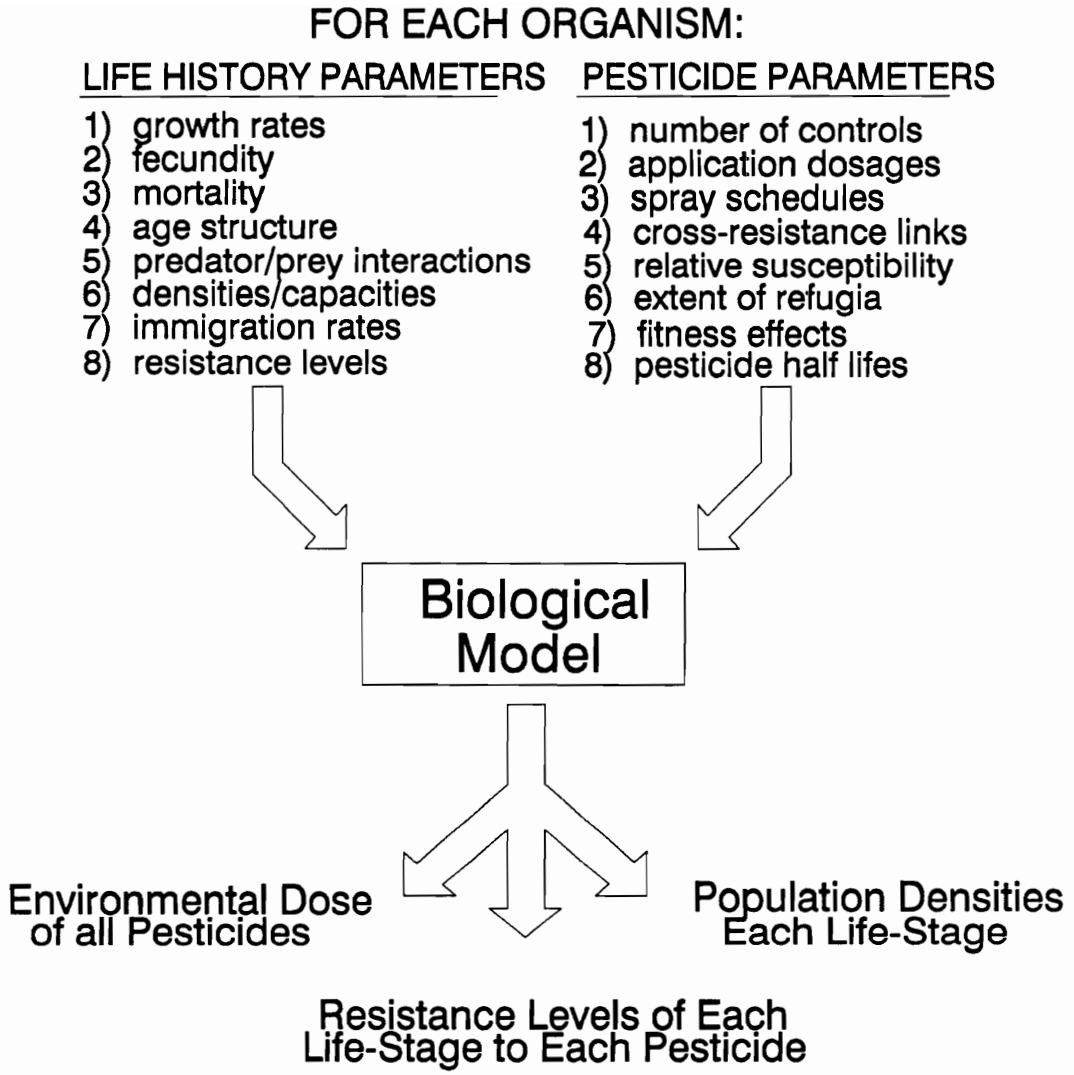

Figure 7. Summary Description of the SERA Model and its Operation.

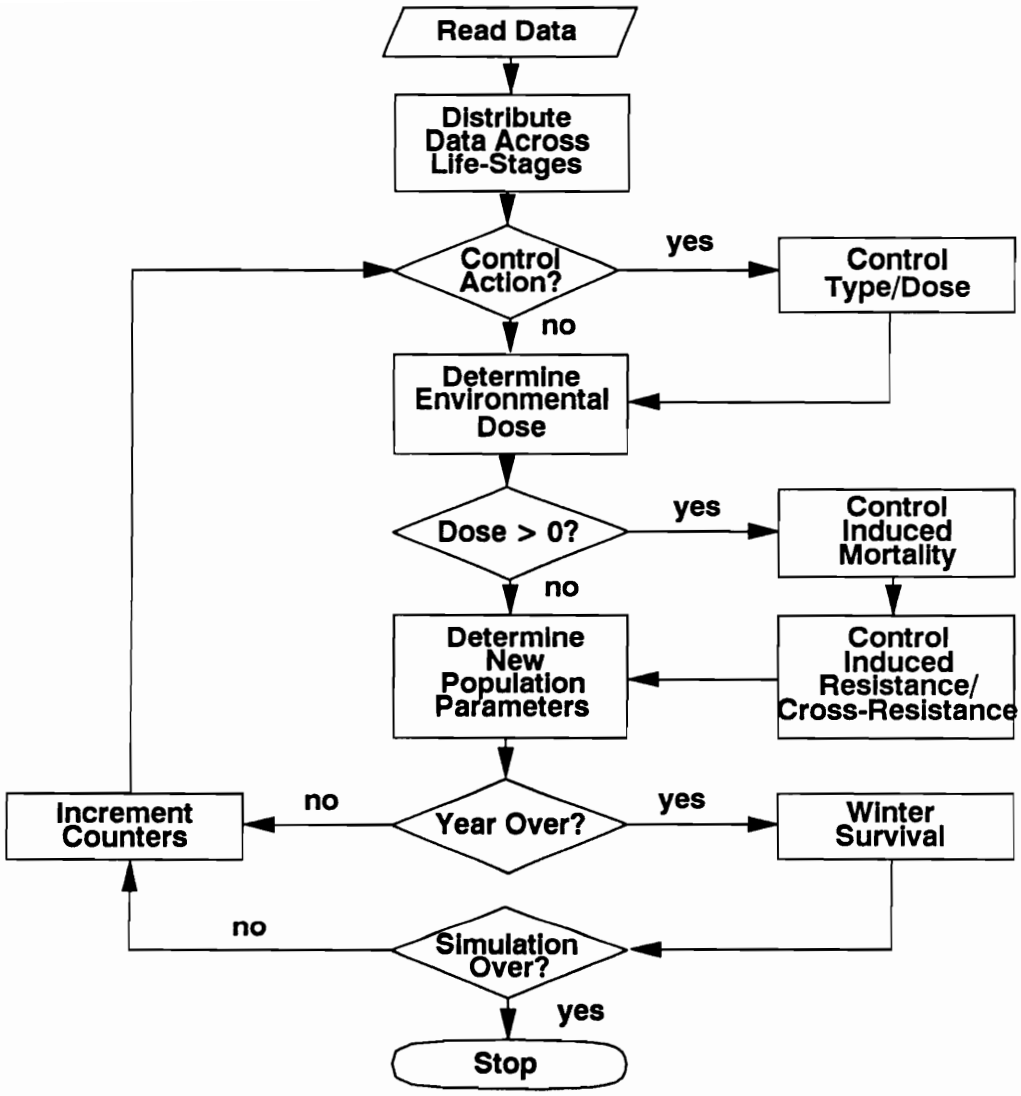

Figure 8. Summary Flowchart of the SERA Model and its Execution.

ondly, the resulting own-chemical and potential cross-resistance effects of the control actions on genotypic frequencies are calculated, as are the impacts of the distributional shifts in genotypes on a species fecundity, overall mortality (including any prey or predator effects), and population growth for the next period.

Once the primary impacts of current and previous control use are determined, the SERA model combines them with information on immigration and movement into refugia in order to calculate each life-stage's active population levels and genotype frequencies for the next period. If the yearly allotment of periods has already been simulated, then pre-defined winter mortality rates are used to initialize the population levels and genotypic frequencies for the next simulation year. If all the allotted years have been simulated, then execution stops. More detailed information on how SERA operates can be obtained from the annotated program codes in Appendices A and C.

Like many ecological simulations, SERA can be considered a member of the "advanced-bookkeeping" class of discrete models. This designation serves to highlight the fact that the simulation is not necessarily mathematically complex in any of its components. Instead, complexity arises from the time-dynamic characteristics of each component, the varying levels of interaction between components, and the number of components. But, even with this complexity, SERA is only as realistic as the information used to parameterize it. While the information requirements needed to characterize the mid-Atlantic pest complex are substantial, they were available from various field observations, laboratory experiments, and expert opinions. The origin and specifics of the data used to parameterize SERA for TABM, ERM, and BLB simulation are discussed in the next 3 sections.

TABM Population Ecology in SERA

The TABM version[26] of the SERA model was designed to simulate genetic and population dynamics for a typical 160 day growing season that lasts from 23 April to 30 September. Each growing season consisted of 2 complete generations, with each generation composed of 20 developmental life-stages. Transition probabilities between these life-stages were determined by natural and pest-control induced mortalities. Although kept distinct for calculation purposes in the simulation model, these life-stages were aggregated for output statistics in the following way: egg (stage 1 to 3); small larvae (stage 4 to 6); medium larvae (stage 7 to 9); large larvae (stage 10 to 12); pupa (stage 13 to 16); and adult (stage 17 to 20). This particular organization, its relationship to TABM development, and, by consequence, the implied 4-day time-step of simulation, was chosen so as to accurately depict the stage-structured population dynamics that are observed in the field. Insecticide induced mortality also tends to be similar for similar stages of insect development, thereby making the stage organization convenient for the purposes of some aggregate calculations.

Initial TABM population density for the first day/first year of simulation was set at 1250 individuals. This parameter value was based on experimental data indicating that an average orchard with typical levels of fruit damage has overwintering populations of 12.5 individuals beneath each tree in a 1 acre, 100 tree plot. Initial densities in subsequently simulated years were determined by a winter mortality rate that was adjusted to yield approximately 20 percent survival. Within season natural mortality rates were set at a level that generated a 4-fold population increase if no control measures were used.[27] Specifically, survival rates consisted of the following: 73 percent of all eggs hatch; 12 percent of all eggs reach the small larvae

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[26] Some additional information on the TABM's life-history parameter values is provided in Appendix B.

[27] In the absence of immigration, these levels of natural mortality would actually lead the simulated TABM populations to decrease about 10 percent between years. This construct was included to approximate the importance of immigration from surrounding orchard and woodlot acres to long-term TABM population dynamics.

stage; 8 percent of all eggs reach the medium larvae stage; 7 percent of all eggs reach the the large larvae stage; and 6 percent of all eggs reach adulthood. These levels of natural mortality allowed the simulation to closely conform to observed age profiles for initial adult emergence, peak adult emergence, and egg mass deposition in actual orchards (Figure 9).

Fecundity for homozygotic susceptible TABM individuals, and thus the 100 percent fecundity rate, was initially set at 1.5 egg masses every 4-day simulation time-step. Reproductive disadvantages associated with resistance alleles were set at the conservatively low levels of 5 percent for heterozygotes and 10 percent for homozygotes. Conservatively low initial frequencies of the resistant allele (10^{-6}) were also used. Previous studies have utilized frequencies that ranged from 10^{-3} to 10^{-6}, thereby making the estimates of time-to-resistance generated in this study on the upper end of the potential range. Based on field observations that indicate (Knight, personal communication): 1) a maximum of 100 egg masses per tree for a 100 tree per acre plot; 2) approximately 100 eggs per mass; and 3) an 8 percent survivability to the medium larvae stage; the maximum environmental carrying capacity was set at 80,000 individuals. However, density-dependent mortality began at 10 percent of the carrying capacity, or 80 larvae per tree. In the absence of pest control measures, this parameterization will result in approximately 10 percent fruit injury at the initial density dependent mortality level, a value typically seen in experimental observations of chemical-free orchards. Additional details and the annotated program code for the TABM version of the SERA model can be found in Appendices A and B.

ERM/BLB Population Ecology in SERA

The ERM/BLB version[28] of the SERA model was designed to simulate the genetic and population characteristics for the predator-prey sub-system of the mid-Atlantic pest complex. As in the TABM version, each growing season consisted of 160 days (23 April to 30 September)

[28] Some addition information on ERM's life-history parameter values is provided in Appendix D.

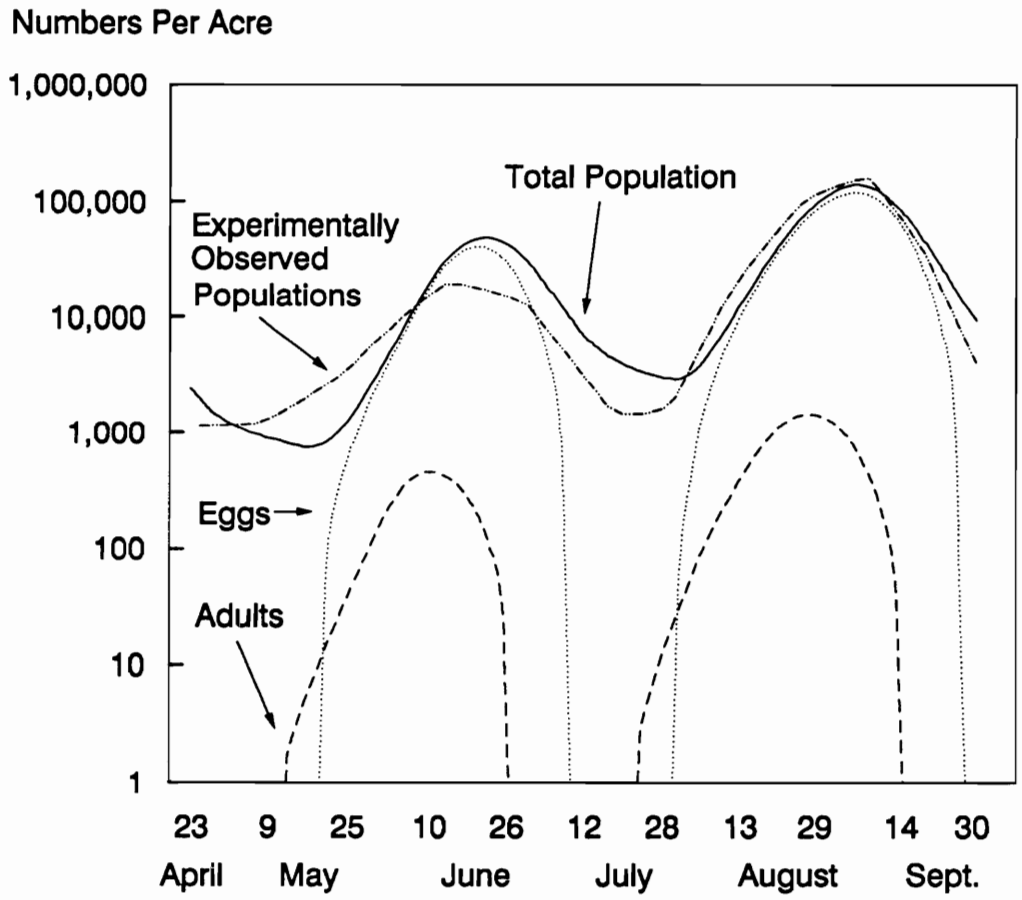

Figure 9. Comparison of Experimentally Observed and Generated TABM Age Profiles: Experimental Observations Adapted from Published and Unpublished Data.

simulated in 4 day increments. In terms of generations, this growing season length consisted of 4 to 5 complete generations for the BLB and 7 to 8 generations for ERM, with the exact number dependent on the development of a particular season's population age structure. Also, as in the TABM version, the ERM/BLB version of SERA aggregated 20 developmental life-stages into six general groups. For ERM, these groups were termed egg, larvae, protonymph, deutonymph, and two adult stages, with reproductive capability beginning in developmental substage 6. The double-length adult group explicitly recognizes the long adult lifespan relative to the passage through the other groups. For BLB, the six life-stages consisted of egg, small larva, medium larva, large larva, pupa, and adult, with reproduction beginning in substage 8.

Initial population densities for the first day/year of simulation were set at 1.0 individual per leaf for ERM and 0.01 individual per leaf for BLB. As with TABM, these initial densities are typical of those found in commercial orchards. Age profiles of the populations were distributed among the 20 developmental stages so as to conform to observed field data. In particular, ERM is known to overwinter entirely as eggs, whereas BLBs overwinter as adults. Time step survival rates were initially set at approximately 90 percent for all stages, with winter survival adjusted to produce approximately 50 percent survival with the same relative population age structure each year for both species. The dynamics of these modelled populations were further initialized to create, in the absence of pesticide pressure, peaks in ERM density of between 10 to 20 individuals per leaf occurring at simulation steps 29 to 33, and BLB peaks of 1.2 to 1.7 individuals per leaf occurring at simulation steps 33 to 40. Without the influence of the predator BLB, these initial conditions would lead to ERM populations of up to 45 individuals per leaf by simulation step 40. These levels of natural mortality allowed the simulation to closely conform to observed age profiles for initial adult emergence, peak adult emergence, and egg depositions in actual orchards (Figure 10).

Fecundity for homozygotic susceptible individuals, and thus the 100 percent fecundity rate, was initially set at 12 per simulation time-step for ERM and 20 per time-step for BLB. Reproductive disadvantages associated with resistance alleles were identical to those used

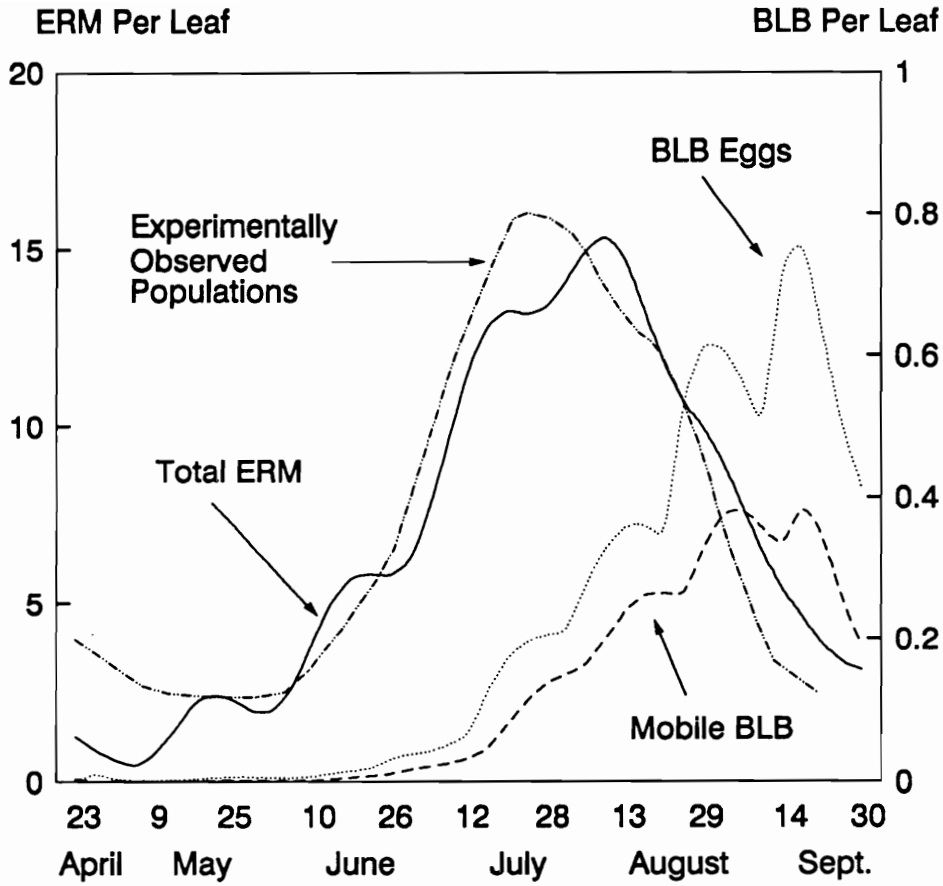

Figure 10. Comparison of Experimentally Observed and Generated ERM/BLB Age Profiles: Experimental Observations Adapted From Published and Unpublished Data.

for TABM; 5 percent for heterozygotes and 10 percent for homozygotes. Conservatively low initial frequencies of the resistant allele (10^{-6}) were also used. Environmental carrying capacity was initialized at 400 per leaf for ERM, which results in density-dependent mortality that begins at 40 individuals per leaf. For BLB, carrying capacity will be more than a function of ERM supply, as large numbers of BLB's can cause feeding interference. Thus, BLB carrying capacity was set at 100, which leads to the initiation of density dependent mortality at 10 individuals per leaf.

Immigration rates are another factor that can be important in determining the combined ERM/BLB population and genetic dynamics in any given orchard. Individuals migrating from other fruit producing acreage into the simulated orchard would be expected to have some level of resistance already developed, whereas individuals migrating from untreated areas should have very little resistance to the chemical controls used in the orchards. Although ERM generally does not immigrate into orchards in large numbers, BLB adults are very mobile and capable of extensive immigration. Thus, immigration rates for ERM were set at 0.43 percent of the total reproductive females per simulation time-step, while immigration for BLB was set at 5.0 percent of the total population per time-step, with the numbers assumed to originate equally from other apple orchards, other non-apple fruit orchards, and non-treated areas, such as woodlots. Immigrants from other apple orchards were assumed to have the same genetic characteristics as the modelled orchard. Immigrants from non-apple orchards were assumed to somewhat less resistance to the chemicals used in apple production. Immigrants from untreated areas were assumed to have the initial frequency of resistant alleles.

The predator-prey interactions between BLB and ERM were based on information found in the entomological literature and on expert opinion (Dover et al.; Knight, personal communication; Hull, personal communication), with all active predator stages consuming prey and all active prey stages subject to predation. Feeding rates were affected by prey density, interference among predators, and predator spatial distribution. The interaction model assumed uniform prey distribution and a predator searching efficiency of 0.2 for BLB. Predator functional response was described by the Royama equation

$$Z = \frac{\ln\left(e^{-YBP} - e^{-BX-YBP} + e^{-BX}\right)}{B}$$

where Z is the number of prey eaten per leaf per day; P is the predator density per leaf; Y is the maximum feeding rate per predator, set at 2.5 prey per day; X is the prey density per leaf before consumption; and B is predator searching efficiency. Predator distribution among leaves was described by a negative binomial equation (Bliss and Fisher) with a clumping parameter and truncations at the 95th percentile (Dover et al.). Total prey consumption was determined by: 1) calculating the proportion of leaves having each nonzero predator density; 2) applying Royama's equation for each predator density; 3) weighting the Z value for each predator density by the appropriate proportion; and 4) summing the weighted X values. Because prey were the only food source for predators, predator fecundity was a function of prey consumed. Predator starvation and emigration out of the orchard due to lack of prey were not distinguished as each effectively removed predators from the system being modelled. Minimum necessary consumption rate of ERM by BLB was set at 4.0 individuals per time-step, a level at which predator survival rate in the orchard was 50 percent. Below this critical consumption rate, survival declined linearly to 0 percent at 0 prey consumed. From a consumption rate of 4 to 40 per time-step, survival rate increased linearly to 100 percent, where it remained for any additional increases in consumption. Fecundity rates for BLB were also linked to consumption, and determined as the same percentage of maximum as the survival rates at different consumption rates. Further specifics on the ERM/BLB version of the SERA model and its parameterization can be found in Appendices C and D.

The Role of Chemical Controls in SERA

Critical to the operation of the SERA simulation model are the parameters related to

pest-control measures.[29] This information determines, to a large extent, how fast resistance will develop. For this study, six insecticides were included in the control technology set: azinphosmethyl, chlorpyrifos, phosmet, microencapsulated methyl parathion, methomyl, and esfenvalerate. Maximum chemical application rates were set 25 percent above those currently recommended by labelling in order to provide for the possibility that the labelled rates were too low to be optimal. However, actual applications were not required to be as large as either labelled or maximum rates. In addition, actual exposure rates were not simply a function of the current period's application. Instead, total exposure was calculated as the sum of current period applications and the residue left from previous applications. This level of environmental insecticide loading was determined from experimentally estimated and published chemical half-lives.

The effective toxicities for exposure to each chemical control in each time-step, expressed as LC50's and LC90's,[30] are based on experimental residue assays and expert opinion. Toxicity across life-stages and species were calculated by using the approximate relative ratio of toxicity observed for TABM exposed to the control chemical Guthion. Thus, it was assumed that the distribution of life-stage mortality affects for all control chemicals were identical to that observed for Guthion. Toxicity affects on resistant individuals were based on a study by Travis, where homozygous resistance was related to a 30-fold increase in LC50's and a 10-fold increase in LC90's.

Cross-resistance was another potentially important factor in determining the rate of resistance development. Research has suggested that cross-resistance may occur among organophosphate controls, but little evidence exists for its occurrence between organophosphate and carbamates/pyrethroids, or between carbamates and pyrethroids. Thus, simulations were initially conducted with no cross resistance among the

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[29] A brief technical characterization of all chemical controls used in the simulations can be found in Appendix E.

[30] LC50 defines the lethal concentration of a control needed to kill 50 percent of the exposed organisms in a timed bioassay. Similarly, LC90 defines the lethal concentration needed to kill 90 percent of the exposed organisms in a timed bioassay. This information is used to form probit mortality curves, from which mortality rates for any environmental concentration of the control can be interpolated.

organophosphates. However, given the potential implications of this factor in the emergence of resistance, sensitivity analysis was performed with the cross-resistance parameters. Additional detail on the procedures used to include chemical control in the simulations can be found in Appendix E.

Summary

This chapter presented an overview of the construction and parameterization of an agroecosystem simulation model used to estimate long-run dynamics for population density and genetic frequencies in the mid-Atlantic apple pest complex. The information derived from this simulation is used to determine the economic impacts of any given pest-control scenario, and therefore represents a fundamental database of this study. Because of its importance, a number of key simulation characteristics are summarized below:

- The SERA versions implemented in this study can best be described as discrete-dynamic-deterministic simulation models.

- Genome shifts are calculated from the Hardy-Weinberg formula with the assumption that resistance can be described as a single-locus, two-allele semi-dominant inheritance mechanism.

- Two versions of the SERA model are parameterized for this study -- one for TABM and the other for the ERM/BLB pest-predator system. These versions can be calculated separately because there are no apparent direct linkages between the dynamics of TABM populations and the pest-predator interactions of ERM/BLB.

- While the information requirements needed to characterize the mid-Atlantic pest complex and chemical controls are substantial, they help provide for a realistic simulation and

were available from various field observations, laboratory experiments, and expert opinions. The origin of some key parameters are discussed in this chapter, with the remainder being detailed in the relevant appendices.

CHAPTER VII: EMPIRICAL MODEL II -- ECONOMIC SURPLUS

A quantitative evaluation of long-run insecticide use, and the effects of regulatory actions, requires identification and estimation of the physical system being studied. In addition, society's interest in

> ... securing that level of pesticide usage, given the technology at any point in time, that provides the maximum positive benefits over and above the negative benefits or costs .... (Headley and Lewis, p. 24)

suggests a need for an economic assessment of insecticide use and regulatory impacts (CAST). Chapter VI focused on the development of a simulation model through which the dynamic influences of insecticide use on the biological structure of an agroecosystem could be empirically discerned. In this chapter, attention will be directed toward the economic manifestations of these agroecosystem impacts and the methods used for determining their value. First, an expected economic surplus framework is introduced, with the discussion concentrating on what is being measured, how it is measured, the appropriateness of the measure, and previous uses of the surplus framework in agricultural economics. Second, the general empirical economic model used in this study is presented, along with the specific parameterization relevant to the mid-Atlantic apple agroecosystem. Lastly, the data and estimation procedures that support the parameterization are described.

Economic Surplus Analysis

Any economic study must begin by identifying the relevant group to which costs and benefits accrue. Given the impact insecticides have on final crop production, any reduction in the control technology set will most immediately affect individual producers. And, in fact, most economic studies of insecticide use and resistance have been conducted at the farm level. But from a regulator's point of view, producers are not the only group whose economic well-being is influenced by insecticide use. Consumers, the ultimate users of food and fiber end-products, also will be affected by technology-induced changes in product supply. In addition, the common property nature of pest susceptibility, and the negative insecticide externalities it implies, ensures that individual producer actions will affect not only themselves, but also other producers and consumers. As a result, a complete economic evaluation of the interactions among insecticide use, resistance, and regulation requires that producer and consumer impacts be jointly analyzed. Because producers and consumers combined can be considered to constitute society as a whole, it is at the societal level that this economic analysis is conducted. One common framework employed for such an analysis is the partial equilibrium expected-surplus approach.

General Framework

The expected-surplus approach to measuring economic impacts is based on the concept of putting a money value on any potential net changes in utility or welfare. Money units may be the best empirical measure of society's preferences for certain outcomes because members of society constantly express their preferences when they exchange money for goods.[31]

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[31] Because money valuation of utility is intimately linked to the idea that individual preferences are the only relevant factor in analyzing social impacts, rejection of preferences as a basis for social decision-making requires rejection of the use of money values as a measure. But while some social groups are explicitly antagonistic to the supremacy of individual preferences, the dominant societal

Aggregating individual actions of consumers can provide an approximate identification of the market demand curve for the product being studied.[32] Similarly, aggregation of feasible marginal cost curves for individual producers leads to market supply schedules. Static equilibrium in the market can then be identified as that price where the quantity supplied by producers is exactly equal to the quantity demanded by consumers. Within this framework, consumer surplus is defined as the excess total money-valued utility of a product, realized by consumers as a group, over and above the market value of the total quantity of the good consumed. This is often depicted graphically as the area under the ordinary market demand curve and above the price line. Producer surplus is the difference between the gross income received by the sale of the total product quantity and the total variable costs of production. Graphically, this is the area above the market supply curve and below the price line.[33] Taken together, consumer plus producer surplus is termed total economic surplus. Thus, the impact of exogenous factors on market supply and demand schedules can be measured as the resulting changes in consumer, producer, and total economic surplus (Mishan).

Criticisms and Support

Any implementation of applied welfare analysis must consider a number of important, and at times conflicting, questions (McKenzie and Pearce):

1. According to economic theory, what societal attributes correspond to utility or welfare?

viewpoint, as imbedded in law and custom, is that individual preferences are the base upon which society should be organized (Pearce and Turner).

[32] Of course, this aggregation, and the methods used to accomplish it, can be quite controversial. Given that virtually all reasonable assumptions about consumer behavior quickly lead to problems with impossibility theorems (Sen), the approach used in this analysis can only be theoretically justified if restrictions are imposed on the types of individual preferences allowable as inputs to the social choice process (Starrett). Namely, analysis is confined to social decisions made on the basis of the Pareto principle.

[33] Producer's surplus, as defined here, does not necessarily accrue to the actual growers of the product, but rather to the owners of the fixed factors of production and those supplying intermediate inputs to the grower.

2. In principle, what indicators can be used to measure these social attributes?

3. How can these indicators be mathematically expressed so as to exactly replicate, at least in principle, the possible states of social welfare?

4. Given the limitations inherent in any empirical study, how can the theoretically ideal representation of welfare be best approximated?

Although a superficially simple concept, economists have had some difficulty in devising exact empirical approaches for welfare analysis. Procedures employing economic surplus as a welfare measure, although widely used, have been criticized because of their 1) normative nature; 2) general exclusion of transaction costs and the effects of incomplete risk markets; 3) often perceived lack of policy relevance; 4) lack of relationship with underlying utility; 5) inability to draw exact general equilibrium inferences; and 6) various empirical problems related to the choice of supply and demand functional forms (McKenzie and Pearce; Richter; Harberger; Burns). The first three objections, while often valid within a theoretical context, can be overcome for empirical work if the orientation, assumptions, and deficiencies of the analysis are made explicit (Norton et al.). In addition, it should be clearly understood that surplus measures can serve as little more than a first-order approximation to any welfare change.[34] For example, an important assumption explicit in surplus analysis is the ability of the analyst to identify the relative importance each individual's preferences should have in societal decision-making. Welfare gainers also are assumed to compensate losers to the extent that any change induced by exogenous factors is at least Pareto neutral. In reality, however, compensation is rarely made, and expected-surplus will only measure true welfare change if the preference weights are all assumed equal (Beiri, de Janvry, and Schmitz). If equal weights are a gross distortion, as would likely be the case for investigations of localized environmental contamination, then some attempt to derive weights and a transfer scheme

[34] This point is more rigorously demonstrated in the next subsection.

might be required. The importance of incomplete risk markets and policy relevance can also be reduced by refining the surplus calculations and presenting the results of the analysis in widely understandable price and quantity terms (Norton et al.).

Perhaps the most serious objections to the use of expected-surplus analysis, and the ones needing the closest consideration, are those that question the relationship of surplus to utility, both statically and in a general equilibrium context. If those issues can be satisfactorily addressed, then the use of surplus measures for obtaining estimates of welfare change hinges on finding adequate solutions to the functional form problems that face all empirical studies. So, before discussing the specifics of how the expected-surplus model was implemented in this study, it may be useful to briefly examine some of the philosophical, theoretical, and practical debates that have surrounded this topic.

The Relationship of Surplus to Welfare

Studies that utilize economic surplus to approximate changes in total social welfare generally do so to compare the relative costs and benefits of potential policy actions. However, the use of expected-surplus implies that it measures the utility changes of shifts in social welfare in a way that: 1) accounts for all the social attributes that define utility relative to the policy actions, and 2) allows the determination of preferences for different policy actions based on the value of the economic surplus measure. Theoretically, the first assumption will only hold in the special case where all the effects of the policy-action are monetized, and specifically where no income effects arise from shifts in individual and/or market supply and demand schedules (McKenzie and Pearce). Income effects, from the view of willingness-to-pay for a given product, are those surpluses that are gained or lost because of changes in real income. For example, the withdrawal of an insecticide from a static pest-control technology set may lead to a backward shift in the market product supply curve, implying less equilibrium output quantity at a higher equilibrium price. A given consumer can still purchase the same quantity of output as before the withdrawal, but to do so would cost more and reduce the

residual income available for the purchase of other goods. This scenario implies that surplus will decrease by more than the extra amount that must be paid for the product on which the insecticide was used. In addition, the implied change in real income may also affect the position of the individual or market demand curve itself, for any specific curve is based on the *ceteris paribus* condition of constant real income and prices for all other goods. Thus, income effects may be important precisely because, in a partial equilibrium framework, they are assumed not to exist, thereby making the measurement issue and the general equilibrium issue closely related.

In order to understand the theoretical basis for the income-effect criticism, consider how the algebraic representations of surplus changes are derived.[35] Defining ΔW as an exact expression for the willingness-to-pay for a welfare change from state 0 to state 1,

$$\Delta W = W(Q^1\,;\,P^0) - W(Q^0\,;\,P^0) \quad , \qquad [23]$$

a Taylor series expansion of equation [23] yields

$$\Delta W = \sum \frac{\partial W}{\partial Q_i}\Delta Q_i + \frac{1}{2}\sum\sum \frac{\partial^2 W}{\partial Q_i \partial Q_j}\Delta Q_i \Delta Q_j + f(\Delta^3) \qquad [24]$$

where $f(\Delta^3)$ represents all polynomial terms of third order or higher. Assuming smoothly differentiable functions and the relationship

$$\frac{\partial W}{\partial Q_i} = \lambda P_i \quad ,$$

where λ is the marginal utility of income at price P_i, manipulation of equation [24] results in

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[35] The following explanation is based on the arguments presented by Weitzman for surplus as an exact approximation.

$$\Delta W = P^0 \Delta Q + \frac{1}{2} \Delta P \Delta Q + \frac{1}{2} \Delta \lambda P^0 \Delta Q + f(\Delta^3) \quad . \qquad [25]$$

The first two terms on the right-hand-side of equation [25] correspond to the change in total surplus under the demand curve, including that portion accruing to the owners of variable inputs to the production process if supply is not perfectly price inelastic. But if equation [25] is derived from the exact change in welfare defined in equation [23], then the change in surplus is only a partial second-order approximation to the actual underlying change in utility. The magnitude of the error introduced by ignoring the third and fourth terms of equation [25] are the source of the measurement debate (McKenzie and Pearce).

If the use of partial-equilibrium surplus measures do indeed result in clearly identifiable errors, what alternative approaches might be employed? Depending on whether producers or consumers hold the property rights to the product, the theoretically correct measures would either be compensating or equivalent variation (Pearce and Turner). Based on the Hicksian demand curve, these measures provide theoretically sound indications of willingness-to-pay and willingness-to-accept compensation for any potential welfare change, and thus give an exact money-metric of utility change. However, Hicksian demand curves cannot be easily observed as manifestations of market processes because they are based on constant utility across their entire length, or constant real income even as prices change. While complete information could potentially be gathered to estimate the higher-order elasticities necessary to fully evaluate equation [25], the effort may be excessive relative to the improvements in accuracy of the welfare measure. In fact, it has been demonstrated that the errors introduced by using partial equilibrium surplus as a welfare measure are likely to be very small if: 1) the income elasticity of demand is small (inelastic), and/or 2) the budget share of the product is small (Willig). Both of these conditions are met by the econometrically estimated U.S. apple market, with the specific details given in the latter part of this chapter. In addition, for relatively small changes about the original price and quantity, the first order effects in equation [25] will always dominate the second and higher order effects.

Returning to the second implied assumption in the use of surplus as a utility measure, it must be determined whether, and in what way, surplus can be used to compare potential policy options. An analyst's ability to meaningfully measure differences among policy options is partly dependent on whether it is assumed that those affected have ordinal, weak cardinal, or strongly cardinal preference orderings (Morey). Care must be taken not to ascribe more information to the surplus measure than is allowed by the assumed preference structure. If ordinal preferences are assumed, as is usually the case, then surplus analysis would yield information on the magnitudes of each policy-option outcome, with these outcomes conforming to a complete, reflexive, and transitive ordering. But because only the ordering property of numbers can be utilized under an ordinal preference structure, a numerical measurement of economic surplus would be unique only up to a monotonic transformation, even though all numerical scales have more extensive properties. Thus, potential policy-options could be ordered as to their preference, but the differences between policy outcomes would not be meaningful.

The problem with applying knowledge of preference structure effects is that only monotonic transformations of utility have been developed to measure welfare, and, in general, no affine transformations can ever be obtained (Morey). For example, equivalent variation will unambiguously rank consumption bundles in the same order that a consumer would. This is why McKenzie and Pearce so strongly advocate its use as an index of utility. In addition, because economic surplus is derived, with a few simplifying assumptions, from the same expression generating equivalent variation (equation [23]), Willig advocates economic surplus as a useful approximation to equivalent variation. But both of these applications must be cognizant of the ordinal preference structure underlying the derivations:

> ... the cardinal properties of an individual's cardinal preference ordering cannot be inferred from the cardinal properties of a money measure of that ordering .... One can never determine the cardinal properties of the individual's cardinal preference ordering ... solely on the basis of the individual's behavior in the market. It cannot be done because any monotonic transformation of the true cardinal utility function will explain behavior just as well as the true function (Morey, p.169).

The use of any common welfare measure can only be objectively used to rank the alternatives, even though the use of a numerical scale appears to confer cardinality on the measure. Thus,

extreme care must be taken when presenting the results of *any* welfare analysis to decision-makers, whether or not it includes economic surplus measures.

Given the difficulties and caveats associated with even the simplest welfare analysis, it is not surprising to find a high level of debate in the literature concerning the appropriateness of various techniques. In the end, however, it must be recognized that obtaining an exact welfare measure may be empirically impractical. Under such conditions, and with the need to provide analysis for critical problems, the applied economist must utilize the most appropriate tools available (Mishan). Considering the state-of-the-art in welfare analysis, economic surplus would appear to be the best empirical approach to determining the potential consumer and producer impacts arising from insecticide use, its regulation, and long-run resistance. It remains, however, to discuss an important implementation hurdle of this empirical procedure.

The Functional Form Debate

Another long-running debate among researchers involved in empirical welfare analysis concerns the choice of functional forms for estimating market schedules. Whether in the context of a production, supply, or demand study, economic theory provides precious little guidance on appropriate functional forms (Fuss, McFadden, and Mundlak; Zarembka). This is especially true for market-level studies, where even theoretical requirements on the number of parameters tends not to preclude any appropriately modified functional form (Zeimer, Musser, and Hill). As a result, functional forms are usually chosen on an *ad hoc* basis, even though it has been demonstrated that functional form can significantly affect the statistical properties of a surplus welfare measure (Adamowicz, Fletcher, and Graham-Tomasi). Often, choices are made on some goodness-of-fit criterion or to simplify an extended analysis that might be based on the estimated schedules. With this type of criterion, it is not surprising to discover that most expected-surplus studies have employed linear functional forms to esti-

mate market supply and demand. However, the advisability of choosing linear forms must rest on the trade-off between easy analytic manipulations and accurate system modelling.

The linear functional form has a number of attractive features. Foremost among these is its high degree of parsimony. Not only does this characteristic facilitate data collection and statistical estimation, but it also allows partial-equilibrium consumer and producer surplus changes to be interpreted and calculated in terms of regular Euclidean rectangles and triangles. This is usually the cited reason for the linear form's use in welfare analysis (Norton et al.), and it has been widely applied to a variety of problems. In addition, the linear form is perhaps the best developed from a theoretical econometric viewpoint, and most common estimators can be easily applied without concern about the functional form's effect on the properties of the error term. However, many observed supply relationships do not exhibit linear behavior over their entire ranges, and demand relationships are theoretically prevented from being globally linear. This tends to lead to estimated curves that poorly fit the observed market data, thereby failing one of the important *ad hoc* criteria. And even if the estimated linear curves do have a relatively close fit to the observed data, their function form can generate gross over-estimations of surplus changes.

Attempts to avoid the problems associated with linear functional specifications have led to proposals for application of alternate functional forms, including the use of non-linear quadratic, double-log, semi-log, and other power transformations. While these alternate forms may provide a closer correspondence to the observed data, their use in welfare studies has been limited by extensive data requirements and the complications they inject into the calculation of relevant surplus changes. Given the nature of this study, the desire to compare the *relative* performance of various policy scenarios, the quantity and quality of available data, and the previously discussed limitations on the use of surplus for comparing policy actions, it was decided that the accuracy gained from estimating non-linear supply and demand schedules would be small relative to the data and computational difficulties this choice would introduce. Thus, linear functional forms were used as an approximation to the true functional forms for both supply and demand in the retail apple market.

Economic Surplus in Agricultural Economics

Expected economic surplus as a marginal measure of utility has found extensive use in economics, particularly with respect to project evaluation, governmental planning, and regulation problems (Currie, Murphy, and Schmitz; Starrett; Dasgupta and Heal). These studies have been approached in two distinctly different ways (Dasgupta; Schuh and Tollini). In the first, termed *ex post* analysis, economic surplus is taken to represent the aggregation of revealed individual preferences over allocations at each state of nature that has already occurred. The second, termed *ex ante* analysis, extends this approach by incorporating some form of aggregate beliefs about future outcomes. Thus, as the names suggest, *ex post* is primarily used to evaluate outcomes that have already occurred, while *ex ante* analysis is employed to choose between actions that will affect future welfare levels. Given the planning nature of this study, the following discussion will concentrate on some examples of *ex ante* surplus measures in agricultural economics.

One of the most frequent uses of *ex ante* expected economic surplus analysis has been in the evaluation of potential returns to various investments in agricultural research.[36] Fishel described a computerized model developed for collecting and processing data related to research activities, thereby providing a framework through which an efficient allocation of resources could be determined. The study included the calculation of potential benefit-cost ratios, benefits minus costs, and internal rates of return from subjective information obtained by surveying scientists for estimates of average annual expenditures, time requirements, and technical feasibility of various research activities. Similarly, studies have demonstrated that historical data need not be available to conduct an *ex ante* analysis if one can obtain subjective estimates of potential yield and/or cost effects from scientists (Easter and Norton; Araji, Sim, and Gardner). An important aspect of the Easter and Norton study was the demonstration that benefit-cost ratios were sensitive to variations in expected yield increases, and

[36] The review of this part of the literature is taken, in part, from Norton and Davis.

thus should be carefully analyzed using sensitivity analysis. Pinstrup-Anderson, Ruiz de Londono, and Hoover developed a procedure to estimate the nutritional implication of alternative commodity priorities in agricultural research and policy in Colombia. This study provided the background used to support the derivation of surplus change formulas presented later, and was followed by a more extensive empirical application that included the effects of demand shifts and government pricing policy changes over time on research and extension benefits in Peru (Norton, Ganoza, and Pomareda). Using a somewhat different approach, Ramalho de Castro and Schuh developed a model that focused on growth and distributional effects of technical change along with direct and indirect effects of research. Their calculations, based on secondary yield data and an assumption that technological change caused shifts in the supply curves for various crops, compared the resulting distributional effects on consumers and producers.

In addition to general investigations of research and extension programs, other published studies have used the expected economic surplus approach to measure the potential welfare changes associated with policy-actions aimed at specific commodities. Kaiser, Streeter, and Liu examined the distributional impacts of incorporating mandatory supply controls into the dairy policy mix. The authors used a dynamic econometric model to simulate changes in equilibrium prices and quantities, from which producer and consumer welfare changes were determined. Gallagher used the theoretical framework of expected-surplus to study the European Community's grain sector and the trade implications of various policy and price formation mechanisms, while Miller, Rosenblatt, and Hushak examined the impact of different assumptions concerning the technology-driven shifts in market supply curves. An evaluation of agricultural biotechnology and its economic impact on the pork industry, conducted by Lemeiux and Wohlgenant, also used econometric simulation to generate potential yield changes. From these changes, the authors calculated the potential impacts on producer, consumer, and total economic surplus. In addition, Lemeiux and Wohlgenant demonstrated a way in which extreme-bound estimates of important parameters can be included to check the sensitivity of simulation results. Investigations into the effects of pesticide regulations

have also been conducted using the general economic surplus framework (Lichtenberg, Parker, and Zilberman). In these cases, a marginal analysis of environmental policy based on expected economic surplus can be a viable alternative to econometric models, especially when the available econometric techniques are inapplicable or impractical.

Market Models for Mid-Atlantic Apples

Given the decision to accept expected economic surplus, based on linear market models, as an appropriate empirical measure of welfare changes in this study, its application remains to be demonstrated. But before doing so, a brief review of the apple market's structure will provide a useful context for the modelling effort that follows.

General Market Structure

The U.S. apple production and distribution system has changed dramatically over the last few decades. In the early 1950's, as much as 70 percent of the crop was utilized in the fresh market. Current fresh utilization, however, has fallen to approximately 55 percent of total production as processed products have grown in importance. Although processed products have definite marketing advantages over fresh apples in terms of storage and convenience (Pasour), it appears that the shift towards processed utilization has been primarily fueled by the increasing popularity of juice-based beverages in the U.S. The fact that apple juice can also be used as a volume expander for many types of juice cocktail products has reinforced the trend towards processing. The importance of juice products has also been mirrored in the recent increase of apple products imported by the U.S. (Table 2), of which the majority arrives in the form of juice concentrate.

Table 2. U.S. Imports, Exports, Production, and World-Wide Production of Apple Products.

Year	Fresh		Processed		U.S. Production	World-wide Production
	Imports	Exports	Imports	Exports		
	million pounds					
1955	54.65	94.46	10.37	2.38	4,803.09	18,399.32
1956	78.55	102.11	6.52	4.89	4,579.44	17,793.03
1957	44.35	84.46	7.36	2.10	5,390.46	20,213.53
1958	48.70	248.73	3.87	17.31	5,762.32	22,486.92
1959	56.55	138.21	8.30	7.35	5,738.20	47,178.44
1960	42.20	167.57	7.31	3.81	4,917.50	31,305.32
1961	44.30	181.43	14.35	5.11	5,696.30	46,957.98
1962	60.00	176.64	7.78	5.63	5,689.10	33,509.92
1963	83.15	146.64	40.82	3.18	5,752.60	43,651.08
1964	57.95	215.59	50.49	4.44	6,319.40	40,564.64
1965	34.35	252.17	62.28	3.33	6,140.20	42,769.24
1966	31.54	226.72	88.83	2.65	5,761.40	45,467.72
1967	92.48	177.54	106.14	2.07	5,436.00	47,881.71
1968	106.88	109.52	137.18	1.31	5,469.00	44,107.43
1969	93.60	89.57	176.58	0.83	6,819.60	49,993.71
1970	75.74	108.91	207.60	1.07	6,397.70	46,305.42
1971	95.84	111.20	414.05	0.96	6,373.20	46,682.41
1972	99.61	127.62	311.31	1.02	5,878.80	43,137.41
1973	109.48	168.55	274.44	1.21	6,265.00	49,737.98
1974	79.59	188.27	263.32	1.26	6,579.70	47,301.90
1975	85.96	242.12	259.44	1.09	7,530.00	52,711.99
1976	116.20	255.15	420.45	1.09	6,472.20	49,078.81
1977	108.99	316.16	396.00	1.81	6,739.60	67,399.03
1978	132.29	317.77	541.64	1.79	7,596.90	71,492.97
1979	164.87	462.69	802.95	2.08	8,143.10	80,584.74
1980	156.87	553.11	525.82	1.26	8,828.40	75,174.66
1981	169.08	666.29	984.58	2.28	7,753.60	72,233.72
1982	158.44	566.67	1,249.87	4.32	8,122.00	91,358.62
1983	216.49	565.84	1,798.20	3.03	8,378.50	86,748.81
1984	228.47	455.12	2,026.30	3.10	8,331.00	89,017.34
1985	273.60	389.92	2,584.34	1.90	7,923.50	74,165.91
1986	290.45	398.97	2,773.16	2.55	7,933.00	76,425.46
1987	294.14	461.45	2,726.49	2.07	9,944.40	85,678.33

Note: Non-citrus Fruits and Nuts, various years; FAO Production Yearbook, various years; FAO Trade Yearbook, various years.

Given the extent of changes in the utilization of apple products, it is not surprising to find that the demands of marketing have significantly affected on-farm production practices. Perhaps the most obvious result is that apple growers no longer have the continuous flexibility they once did to determine whether their products will end up in fresh or processed market channels. In fact, growers generally must target their output specifically for either the fresh market or processed market before the growing season, and often even before planting an orchard. This fixity in marketing decisions has arisen because the processed and fresh markets have each tended to develop their own specific set of varietal, quality, and size standards as their operations have become more technologically advanced. In turn, these standards not only constrain a producer's choice of cultivars, but they also influence the management practices employed, especially in terms of pest control. These standards tend to predetermine the market destination of a grower's product before the season begins.

Although the advent of separate market standards for fresh and processed apples has severely constrained marketing options, it has not yet led to complete rigidity. Apples originally targeted for the fresh market but rejected because they do not meet quality standards can often be redirected to some types of processed market uses. However, given the significant price premiums paid for quality fresh apples, this outcome is definitely not preferred by fresh apple producers and usually produces a negative net return on their crop. On the other hand, apples targeted for processed markets tend to have size and appearance characteristics that make them unsuitable for fresh marketing except in years of extreme shortage. Thus, the overall apple market is actually composed of two more-or-less distinct sub-markets, with the primary connection between them being the flow of fresh targeted apples to the processed market. Although some evidence suggests that decreasing fresh relative to processed marketing margins will also divert fresh apples into processing channels (Pasour), a static price relationship between the markets is typically the rule.

In terms of pricing, the domestic U.S. market for apple products is relatively free from the direct distortions often seen in markets for other agricultural products. Major commodity programs do not exist for apples, and governmental intervention is generally restricted to the

maintenance of quality grading standards, with some market-order activity occurring in the fresh market. Thus, the intervention-free assumptions of the closed-economy example are generally applicable. However, given the magnitude of net processed apple imports into the U.S. (Table 2), producers clearly do not operate within a closed market and the domestic equilibrium pricing of the closed-economy example cannot be assumed. This fact requires that the market be modelled to include trade-induced pricing characteristics. Given the historical record of the U.S. as a net exporter of fresh apple products and a net importer of processed apple products, and the knowledge of substantial barriers between the fresh and processed markets, separate fresh and processed market surplus relationships also appear to be required. But before detailing these relationships, a brief discussion about the direction of potential supply-schedule shifts is needed.

When dealing with potential reductions in production technology-sets, economic theory suggests that market level supply schedules should shift to the left or backwards, resulting in less quantity and higher price for any given demand schedule. This was the general scenario outlined in previous chapters when describing the potential effects of regulations and resistance. However, when the market is sub-divided on the basis of quality, then supply does not necessarily shift back in both sub-markets because there is the possibility of quantity transfers between markets. For example, if pest damage in fresh apples increases at all levels of production due to the withdrawal of a particular insecticide, then the fresh market supply curve would be expected to shift backwards. But, the resulting quantity losses in the fresh submarket may not be entirely realized as losses to the overall apple market. At least some of the inferior fresh fruit has the potential to find uses in the processed market. This being the case, it is possible for a backward shift in the fresh market supply curve to lead to an outward shift in the processed market supply curve as more producers target their product to the processed market because of their inability to control fresh apple damage.

Of course, there are limits to the kind of scenario outlined above. Severe pest damage may prevent the utilization of fresh targeted apples in the processed markets, or pest damage may affect both fresh and process targeted apples, causing a backward supply shift in both

markets. This latter scenario may be especially true in the long-run for pests, such as ERM, whose damage reduces the absolute quantity of fruit produced by trees. In general, however, indeterminacy in the direction of shift effects requires that the model be developed to handle both backward and forward shifts in each sub-market.

Open-Economy Export Relationships

The export model used to represent the fresh apple market in the U.S. is graphically described in Figure 11 and Figure 12. For outward supply shifts (Figure 11), the change in consumer surplus is geometrically defined as the area

$$\Delta CS = P_0 cdP_1 = P_0 ceP_1 + ced$$

and can be calculated[37] from

$$\Delta CS = P_0 Q_D^0 g(1 + \frac{1}{2} g|\eta|) \quad ,$$

where:

$$g = \frac{L\rho}{(\rho + |\gamma|)} \quad ;$$

$$L = \frac{Q_S^0 K\varepsilon}{\rho(Q_S^0 - Q_D^0)} \quad ;$$

$$K = \frac{J}{\varepsilon} \quad ;$$

ε = supply price elasticity in the exporting country;

[37] The specific derivation of formulas for the export and import models are presented in Appendix F.

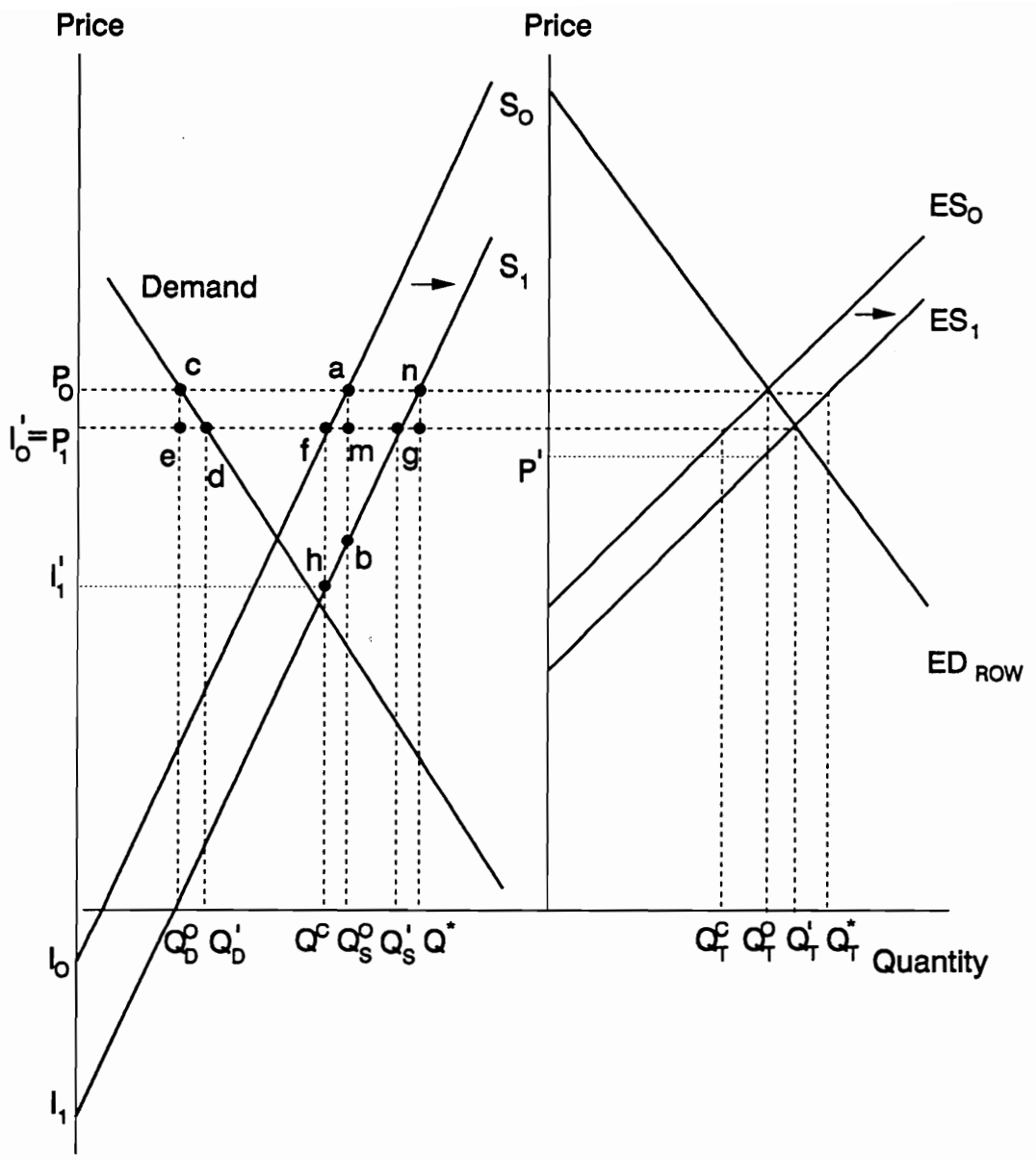

Figure 11. Surplus Changes in a Large-Country Exporter Model with Outward Supply Shifts.

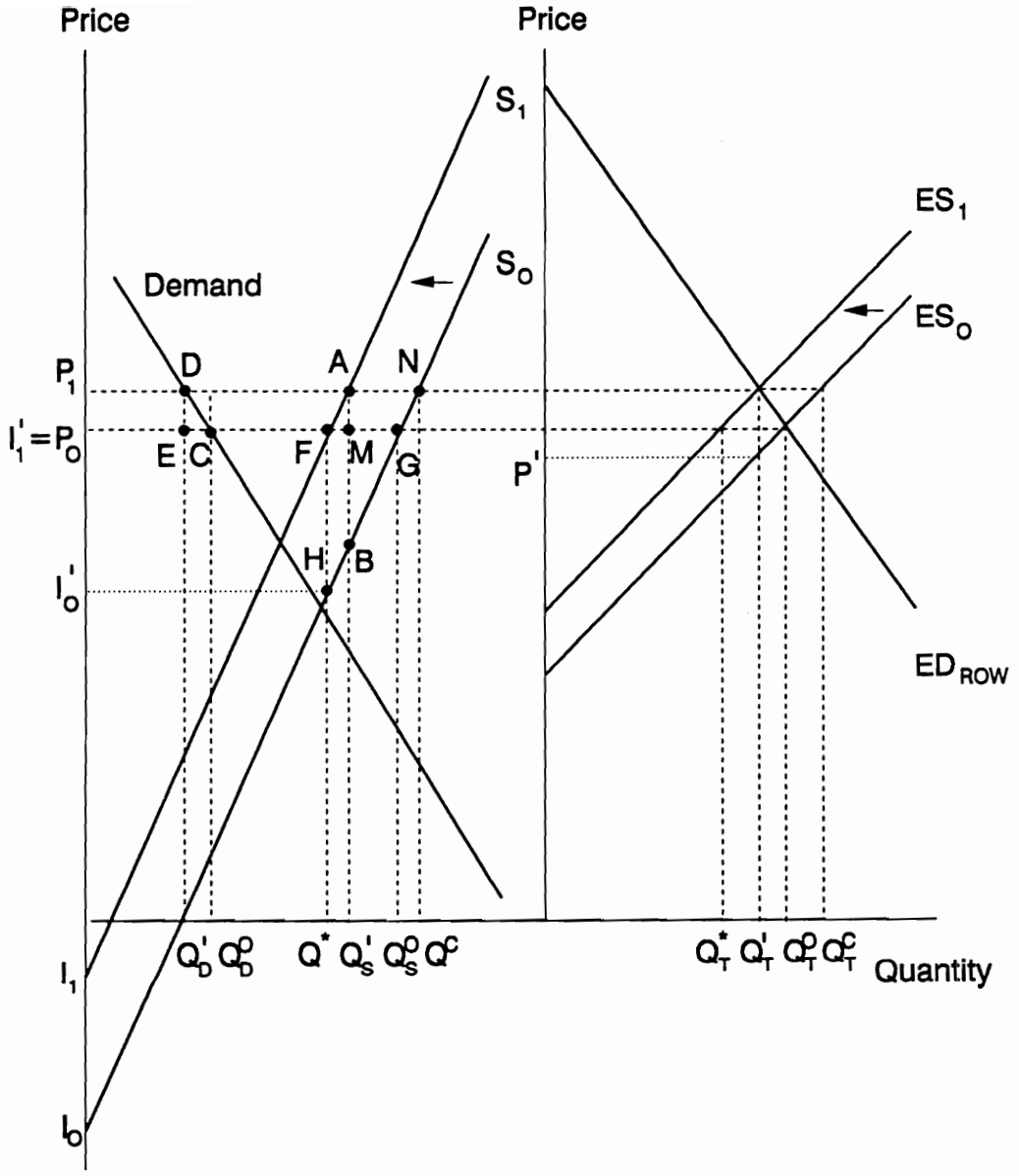

Figure 12. Surplus Changes in a Large-Country Exporter Model with Backward Supply Shifts.

$|\eta|$ = absolute demand price elasticity in the exporting country;

ρ = excess supply price elasticity for the exporting country; and

$|\gamma|$ = absolute excess demand price elasticity in the rest-of-the-world.

Producer surplus can then be graphically described as

$$\Delta PS = I_o fgI_1 - P_0 af P_1 = I'_1 hf P_1 + fgh - (P_0 amP_1 - afm)$$

and calculated from the expression

$$\Delta PS = (P_0 Q_S^0 K - P_0 Q_S^0 g)(1 + \frac{1}{2} K\varepsilon) + \frac{1}{2} P_0 Q_S^0 \varepsilon g(g - K) \quad .$$

This leads to a change in total economic surplus of

$$\Delta TS = \Delta PS + \Delta CS$$

$$= (P_0 Q_S^0 K - P_0 Q_S^0 g)(1 + \frac{1}{2} K\varepsilon) + \frac{1}{2} P_0 Q_S^0 \varepsilon g(g - K) + P_0 Q_D^0 g(1 + \frac{1}{2} |\eta| g) \quad .$$

For backward supply shifts (Figure 12), the change in consumer surplus is geometrically defined as the area

$$\Delta CS = P_0 EDP_1 + CED$$

and calculated as

$$\Delta CS = - P_0 Q_D^0 g(1 - \frac{1}{2} |\eta| g) \quad .$$

Producer surplus is then defined and calculated as

$$\Delta PS = P_0 FAP_1 - I_0 GFP_1$$

$$= (P_0MAP_1 - AMF) - (I'_0HFP_0 + FHG)$$

$$= (P_0Q_S^0g - P_0Q_S^0K)(1 + \frac{1}{2}K\varepsilon) + \frac{1}{2}P_0Q_S^0\varepsilon g(g + K) \quad ,$$

with total economic surplus becoming

$$\Delta TS = \Delta PS + \Delta CS$$

$$= (P_0Q_S^0g - P_0Q_S^0K)(1 + \frac{1}{2}K\varepsilon) + \frac{1}{2}P_0Q_S^0\varepsilon g(g + K) - P_0Q_D^0g(1 - \frac{1}{2}|\eta|g) \quad .$$

Open-Economy Importer Relationships

The import market model used to represent the processed apple market in the U.S. is graphically described in Figure 13 and Figure 14. For outward supply shifts (Figure 13), the change in consumer surplus is geometrically defined as the area

$$\Delta CS = P_0cdP_1 = P_0ceP_1 + ced$$

and can be calculated from

$$\Delta CS = P_0Q_D^0I(1 + \frac{1}{2}I|\eta|) \quad ,$$

where:

$$I = \frac{H\rho}{(\rho + |\gamma|)} \quad ;$$

$$H = \frac{Q_S^0K\varepsilon}{|\gamma|(Q_S^0 - Q_D^0)} \quad ;$$

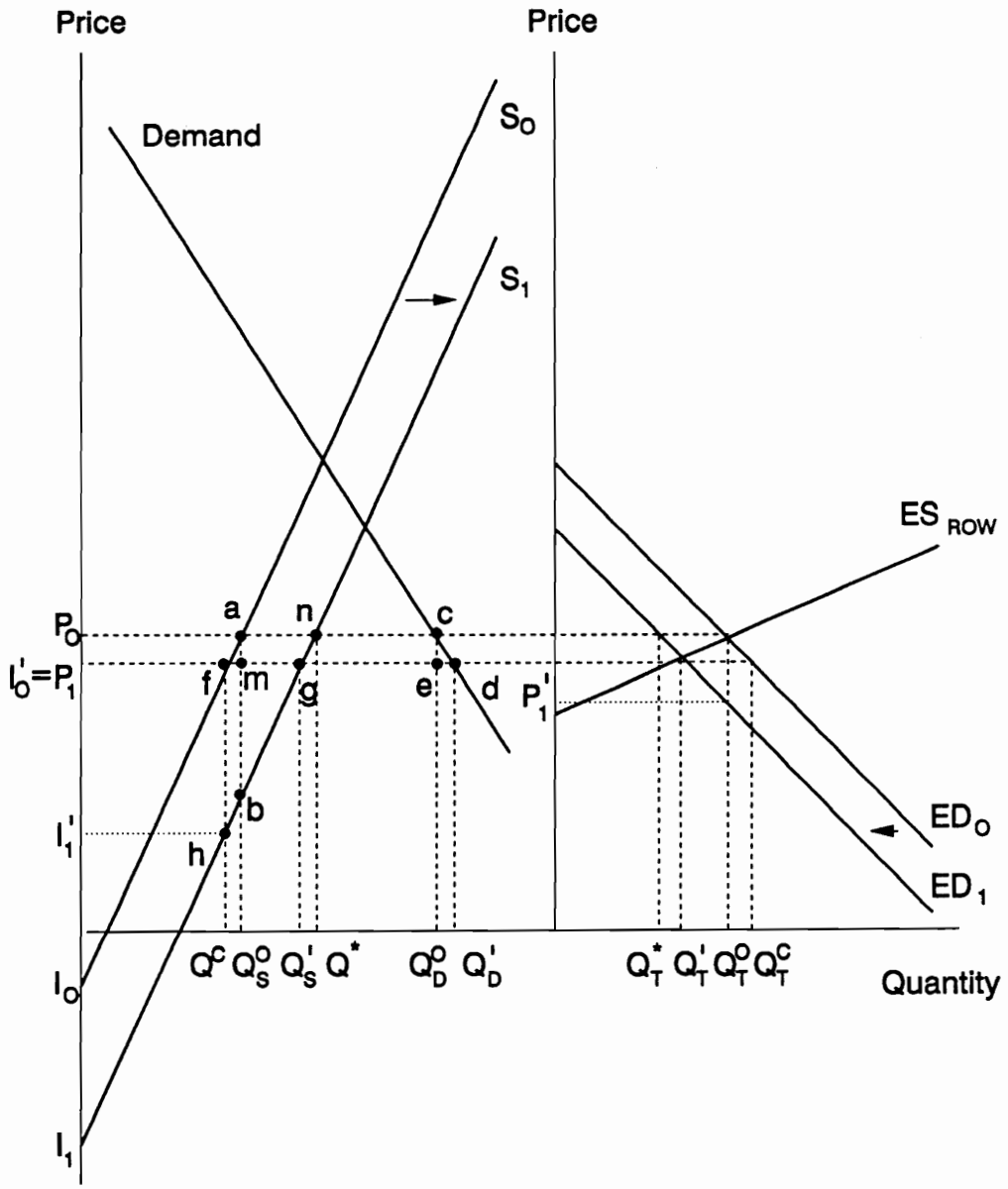

Figure 13. Surplus Changes in a Large-Country Importer Model with Outward Supply Shifts.

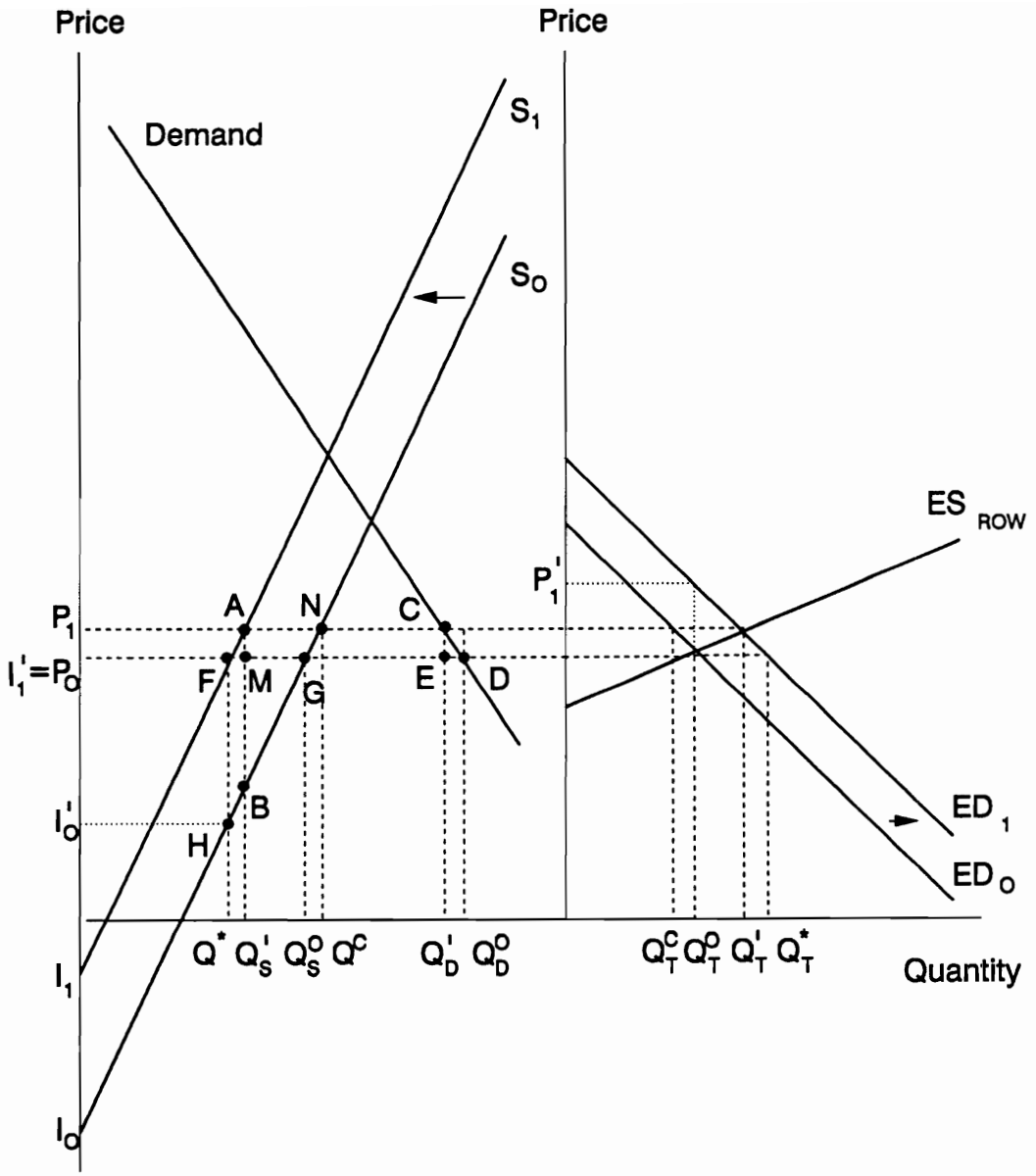

Figure 14. Surplus Changes in a Large-Country Importer Model with Backward Supply Shifts.

$$K = \frac{J}{\varepsilon} \quad ;$$

ε = supply price elasticity in the importing country;

$|\eta|$ = absolute demand price elasticity in the importing country;

ρ = excess supply price elasticity in the rest-of-the-world; and

$|\gamma|$ = absolute excess demand price elasticity in the importing country.

The change in producer surplus is geometrically described by the area

$$\Delta PS = I_0 fgI_1 - P_0 af P_1 = (I'_1 hf P_1 + fgh) - (P_0 amP_1 - afm)$$

and calculated as

$$\Delta PS = (P_0 Q_S^0 K - P_0 Q_S^0 I)(1 + \frac{1}{2} K\varepsilon) + \frac{1}{2} P_0 Q_S^0 \varepsilon I(I - K) \quad ,$$

which leads to a change in total economic surplus of

$$\Delta TS = \Delta PS + \Delta CS$$

$$= (P_0 Q_S^0 K - P_0 Q_S^0 I)(1 + \frac{1}{2} K\varepsilon) + \frac{1}{2} P_0 Q_S^0 \varepsilon I(I - K) + P_0 Q_D^0 I(1 + \frac{1}{2} I|\eta|) \quad .$$

For backward supply shifts (Figure 14), the change in consumer surplus is geometrically defined as the area

$$\Delta CS = P_0 ECP_1 + CED$$

and calculated as

$$\Delta CS = - P_0 Q_D^0 I(1 - \frac{1}{2} |\eta| I) \quad .$$

Producer surplus changes arising from a backward supply shift can be described by the area

$$\Delta PS = P_0FAP_1 - I_0GFI_1 = (P_0MAP_1 - AMF) - (I'_0HFP_0 + FHG)$$

and calculated as

$$\Delta PS = (P_0Q_S^0I - P_0Q_S^0K)(1 + \frac{1}{2}K\varepsilon) + \frac{1}{2}P_0Q_S^0\varepsilon I(I + K) \quad .$$

Total change in economic surplus can then be expressed as the sum of changes in producer and consumer surplus:

$$\Delta TS = \Delta PS + \Delta CS$$

$$= (P_0Q_S^0I - P_0Q_S^0K)(1 + \frac{1}{2}K\varepsilon) + \frac{1}{2}P_0Q_S^0\varepsilon I(I + K) - P_0Q_D^0I(1 - \frac{1}{2}|\eta|I) \quad .$$

Given the relationships for changes in producer, consumer, and total economic surplus, the calculation of the economic impacts arising from supply schedule shifts is a relatively straightforward operation. However, before these impacts can be estimated, specific measures of market structures need to be determined. This will be the focus of the remaining sections in this chapter.

The Apple Supply/Demand Model

The desire to employ expected-surplus as a welfare measure of shifts in market structure requires that each sub-market be described in terms of: 1) domestic supply and demand elasticities, 2) excess supply or demand elasticities for the country being examined, 3) excess supply or demand elasticities for the rest-of-the-world, 4) the expected changes in quantities supplied to the domestic market, and 5) the initial prices and quantities in the domestic market. While the calculation of domestic prices, quantities, and expected quantity changes will

be examined in the next chapter, this section details the specification and estimation of an apple market model that provided the structural information for determining all relevant elasticities.

Model Specification

In order to operationalize the conceptual model of the U.S. apple market as being composed of two distinct, yet interrelated, sub-markets, a mathematical description of the system must not only account for the interactions within a sub-market, but also for the potential allocation of supplies across sub-markets. This suggests that the aggregate U.S. apple market should be empirically modelled as a simultaneous system. Although the U.S. apple market has been modelled as a simultaneous system in other studies (Tomek; Baumes and Conway; Kazmierczak and Norton), it has usually been done within the conceptual context of a closed domestic market. Combined with the assumption of price dependent retail-level demand because farm-level utilization was essentially fixed at harvest, the closed-economy formulation of these previous studies meant that simultaneity arose from the inability to identify supply and demand schedule data that were independent of each other. This study, however, assumes an open-market scenario, with the U.S. being a net exporter of fresh apples and a net importer of processed apples. Given the year-round production capabilities of the world as a whole, open-markets imply that quantity available at retail in the domestic market does not need to be fixed at harvest. So, instead of a price dependent demand system, this study postulates quantity dependency for both supply and demand. Of course, this also implies that retail prices are not directly determined within the country being examined, but instead are determined on the world market, perhaps with some influence from the country's excess supply or demand schedule. Overall, the simultaneity in this study's apple market model primarily arises from the potential movement of inferior fresh-targeted apples into the processed market.

The supply/demand model, presented in Table 3, was specified in a way that attempts to accurately represent the salient structural features of the U.S. apple market with the minimum number of parameters requiring estimation. This latter condition was especially important given that complete data could only be obtained on a yearly basis, and that problems with reporting procedures on earlier series limited the usable data to post-1960. Given the relatively sparse apple market data and the potential estimation problems experienced under these conditions, substitute and complementary relationships with other fruits were not included in the model. By implication, the production and consumption of other fruits was assumed constant. Thus, the market model was specified as a four equation simultaneous system with one identifying relationship. Details of the model specification include:

- **Fresh Supply to the Retail Market.** The total amount of domestically produced fresh apples supplied to the retail market (FS) was assumed to be a function of the real retail price of fresh apples (FP) and the total level of domestic apple production (PROD). FS was hypothesized to vary positively with both FP and PROD.

- **Processed Supply to the Retail Market.** The total amount of domestically produced processed apples supplied to the retail market (PS) was assumed to be a function of the real retail price of processed apples (PP) and the total amount of domestically produced fresh apples supplied to the retail market (FS). FS was included as an explanatory variable in an effort to model the potential flows of fresh-targeted apples to the processed market, with the assumption that as fresh supplies increase, rejection on grading standards also increase in the fresh market, resulting in some redirection to supplies in the processed market. PS was hypothesized to vary positively with both FS and PP.

- **Retail Demand for Fresh Apple Products.** Underlying the specification of demand in this study is utility maximization (Russell and Wilkinson), the assumption of quantity dependency, and the recognition that imports and exports have the potential to play a significant role in satisfying demand. Thus, retail demand for fresh apple products (FC) was as-

Table 3. Specification of the Apple Market Model.

$$FS = \alpha_{FS} + \beta_1 PROD + \beta_2 FP$$

$$PS = \alpha_{PS} + \beta_3 FS + \beta_4 PP$$

$$FC = \alpha_{FC} + \beta_5 FP + \beta_6 NFEXP + \beta_7 PERY$$

$$PC = \alpha_{PC} + \beta_8 PP + \beta_9 NPIMP + \beta_{10} STOCKS + \beta_{11} PERY$$

$$TDS = FS + PS$$

where:

FS = fresh domestic supply to retail;
PS = processed domestic supply to retail;
FC = fresh domestic demand;
PC = processed domestic demand;
FP = real fresh apple retail price;
PP = real processed apple retail price;
TDS = total domestic apple supply to retail;
$PROD$ = total domestic apple production;
$PERY$ = real domestic per capita disposable income;
$NFEXP$ = net export of fresh apples;
$NPIMP$ = net import of processed apples (juice); and
$STOCKS$ = domestic inventory of processed apples.

sumed to be a function of real fresh retail price (FP), net fresh exports (NFEXP), and the yearly real per capita disposable income in the U.S. (PERY). In accordance with utility theory and the assumption of fresh apples as a normal good, it was hypothesized that FC would vary positively with income, but negatively with own price.

- **Retail Demand for Processed Apple Products.** In a specification similar to that for fresh demand, retail demand for processed apple products (PC) was assumed to be a function of real processed retail price (PP), net processed imports (NPIMP), current stock of processed products in storage (STOCKS), and yearly real per capita disposable income in the U.S. (PERY). Again, it was hypothesized that PC would vary positively with income and import levels, but negatively with own price.

- **Total Domestic Production Identity Relationship**. Total domestic production of both fresh and processed apples (TDS) was defined as the sum of PS and FS.

Data Sources

Data used to estimate the apple market model were obtained and/or constructed from published secondary statistical sources for the years 1960 to 1987. Details of the series formation are outlined below, along with an evaluation of the information's appropriateness given the objectives of the model. Specifics concerning the estimation procedure then follows. Note that, where appropriate, data were detrended following standard procedures (Maddala; Nelson and Plosser). The actual data series used in the estimation can be found in Appendix G.

- **Fresh (FS) and processed (PS) domestic supply to the retail market.** The total quantity of fresh and processed apples supplied to the retail market from domestic sources was defined as yearly farm-level domestic fresh and processed utilization. Farm-level utilization

data was obtained from yearly summaries of "Non-citrus Fruits and Nuts" and reported in millions of pounds.

- **Total domestic production (TDS).** Total domestic production levels, of both fresh and processed apple combined, was calculated as the sum of FS and PS.

- **Fresh (FC) and processed (PC) domestic retail demand.** Given the exporting nature of the U.S. fresh apple market, total quantity of fresh apples consumed in the domestic retail market was calculated as fresh domestic supply utilization minus the net level of fresh exports. Fresh consumption information was obtained from various issues of "Agricultural Statistics" and the "Statistical Abstract of the United States," while the net level of fresh exports was calculated as described below. Given the importing nature of the U.S. processed apple market, the total quantity of processed apples consumed in the domestic retail market was calculated as the sum of domestic processed utilization and the net level of processed imports. Processed utilization was obtained from yearly summaries of "Non-citrus Fruits and Nuts." Net processed imports were calculated as described below. All data were converted to millions of pounds.

- **Net fresh apple exports (NFEXP) and net processed apple imports (NPIMP).** The net level of fresh exports and processed imports were calculated from import and export data reported in "Foreign Agricultural Trade in the U.S." (FATUS). The information was reported in millions of pounds.

- **Real fresh apple retail prices (FP).** Real fresh prices were calculated as the nominal yearly fresh retail prices deflated to 1967 levels using the U.S. consumer price index for all food. Nominal information was obtained from various issues of "Food, Consumption, Prices, and Expenditures." The consumer price index was obtained from various issues of "Agricultural Statistics." All data were converted to cents per pound.

- **Real processed apple retail prices (PP).** Real processed prices were calculated as the nominal yearly processed prices deflated to 1967 levels using the U.S. consumer price index for all food. Nominal prices were found in various issues of "The Almanac of the Canning, Freezing, and Preserving Industries" and aggregated by total yearly sales weight for each processed category. The consumer price index was obtained from various issues of "Agricultural Statistics." All data were converted to cents per pound.

- **Real per capita disposable income.** Nominal per capita disposable income was calculated from data in various issues of "Agricultural Statistics" and deflated to 1967 levels using the consumer price index for all goods as reported in "Statistical Abstract of the United States". The information was reported in dollars per individual.

- **Inventory of processed apple products (STOCKS).** The inventory of processed products at the beginning of each year was constructed from data contained in various issues of "Agricultural Statistics" and "The Almanac of the Canning, Freezing, and Preserving Industries." The information was presented in thousands of pounds.

Estimation Procedures

Given the simultaneous nature of the model specification, two-stage-least-squares (2SLS) was used for estimation (Kmenta). As the name implies, 2SLS proceeds in two stages. First, the reduced form equations, containing only predetermined variables on the right-hand side, are estimated individually using ordinary least squares (OLS). This step generates predictions of the left-hand side variables. In the second stage, the right-hand side "endogenous" variables in the structural equations are replaced by the predictions from stage one, and each equation is individually estimated using OLS. Note that, given the over-identification of each equation in the system, 2SLS will produce estimates that are unique only for a given normalization that specifies the presumed "dependent" variable in each equation.

While this tends to run counter to the spirit of simultaneous equations, where it is assumed that all left-hand side variables in structural equations are jointly determined and thus not "dependent" if other endogenous variables are present in an equation, economic supply and demand theory was employed to specify the normalization pattern used in this study (Maddala).

In addition to the potential ambiguities associated with normalization, 2SLS and its equation-by-equation estimation procedure ignores any information that may be contained in non-zero covariances between equation error-terms. Because of this characteristic, it belongs to the class of limited-information (LIML) methods and is not fully efficient in the statistical sense. Its main advantage is that every system equation need not be estimated, as the method operates by replacing the endogenous variables on the right-hand side of each equation with their predicted values from the reduced form. It is only critical that all the predetermined variables are identified so that the reduced form system can be obtained (Spanos).

Actual 2SLS estimation of the system in Table 3 was accomplished using the Statistical Analysis System computer package (SAS). Because standard errors calculated in the second-stage of estimation will generally be incorrect (Maddala), it is important to note that the SAS package calculates adjusted standard errors when using 2SLS estimation.

Model Evaluation

Econometric models can be evaluated on at least two different levels: with respect to the appropriateness of the data used in estimation, and with respect to the conformance of the estimated model to economic theory. Overall, the data series used in this study exhibited the desired characteristics. Tests for normality and heteroscedasticity on each series failed to reject the null hypothesis in all but the net fresh export variable, which proved to be non-normal until square-root transformed. Durbin-Watson tests for first-order autocorrelation were

generally inconclusive, with the estimated degree of autocorrelation being quite small for all equations (Table 4). The exception was the equation for fresh consumption, where the test failed to reject the null hypothesis of no autocorrelation. Other tests for autocorrelated errors were also conducted (Spanos) and yielded similar results. Thus, the serious correlation difficulties normally encountered when using time-series data for estimating supply and demand schedules were avoided.

Another important measure of how appropriate the data was for estimation involves collinearity between regressors. Extensive collinearity can be a symptom of problems with system specification, but some collinearity will always be present (Spanos). For the specification used in this study, collinearity tended to be minor. High R^2s were accompanied by a number of low standard errors in each equation (Table 5), the exact opposite of what might be expected if collinearity were a significant problem. Variance inflation factors and condition indices (Table 4) were generally below the levels that would cause concern (Maddala). Where the condition numbers were elevated, examination of variance proportions suggested that there were no serious interrelationships among regressors. This information, along with the fact that system residuals showed relatively little correlation among themselves (Table 6), suggests that collinearity was not a major impediment to system estimation.

The second level of evaluation used in econometric applications involves the adherence of the estimated coefficients to the signs predicted by economic theory. As shown in Table 5, the goodness-of-fit for the estimated system was relatively high. The most important coefficients with respect to elasticity calculations, those on own-price, were estimated with the hypothesized signs (positive in the supply equations and negative in the demand equations). Note, however, that there was only borderline significance of the price coefficients in the processed supply and fresh demand equations. For processed supply, the magnitude of fresh supply appeared to be more important than price, lending credence to the assumption of supply dependency between the fresh and processed sub-markets. The most significant variable determining fresh demand was net fresh exports. Net processed imports proved to be significant in the processed demand equation. Income levels were insignificant in both fresh

Table 4. Data Diagnostics for the Apple Market Model.

Fresh Domestic Supply Equation

Durbin-Watson D = 1.276 > D_L = 1.16
Estimated 1st Order Autocorr. = 0.250

Collinearity Diagnostics:

Number	Eigenvalue	Condition Number	Var. Prop. Intercept	Var. Prop. FP	Var. Prop. NFEXP
1	2.9753	1.0000	0.0009	0.0030	0.0011
2	0.0200	12.1871	0.0300	0.8812	0.1379
3	0.0046	25.3579	0.9692	0.1157	0.8610
Varience Inflation Factors			0.0000	1.0010	1.0010

Processed Domestic Supply Equation

Durbin-Watson D = 1.202 > D_L = 1.16
Estimated 1st Order Autocorr. = 0.311

Collinearity Diagnostics:

Number	Eigenvalue	Condition Number	Var. Prop. Intercept	Var. Prop. FS	Var. Prop. PP
1	2.9676	1.0000	0.0005	0.0014	0.0017
2	0.0293	10.0620	0.0001	0.5105	0.2642
3	0.0030	31.2054	0.9994	0.4880	0.7342
Varience Inflation Factors			0.0000	1.3832	1.3832

Fresh Consumption Equation

Durbin-Watson D = 1.919 > D_U = 1.76
Estimated 1st Order Autocorr. = 0.039

Collinearity Diagnostics:

Number	Eigenvalue	Condition Number	Var. Prop. Intercept	Var. Prop. FP	Var. Prop. NFEXP	Var. Prop. PERY
1	3.8747	1.0000	0.0006	0.0005	0.0066	0.0010
2	0.1091	5.9584	0.0059	0.0103	0.7429	0.0045
3	0.0117	18.2056	0.1740	0.0412	0.0833	0.9082
4	0.0044	29.6042	0.8196	0.9480	0.1672	0.0862
Varience Inflation Factors			0.0000	1.3407	1.3074	1.4504

Processed Consumption Equation

Durbin-Watson D = 1.630 > D_L = 1.01
Estimated 1st Order Autocorr. = 0.181

Collinearity Diagnostics:

Number	Eigenvalue	Condition Number	Var. Prop. Intercept	Var. Prop. PP	Var. Prop. STOCKS	Var. Prop. NPIMP	Var. Prop. PERY
1	4.3576	1.0000	0.0003	0.0005	0.0041	0.0040	0.0005
2	0.5589	2.7922	0.0003	0.0015	0.0063	0.2172	0.0000
3	0.0726	7.7463	0.0047	0.0212	0.8500	0.0002	0.0044
4	0.0072	24.6638	0.0005	0.4162	0.0950	0.7621	0.4673
5	0.0037	34.4989	0.9943	0.5607	0.0446	0.0165	0.5278
Varience Inflation Factors			0.0000	2.1841	1.0972	4.1246	2.5489

Table 5. The 2SLS Estimated Apple Market Model with Parameter Standard Errors and Goodness-of-fit.

$$FS = \underset{(399.59)}{-98.24} + \underset{(0.05)}{0.48}\,PROD + \underset{(13.24)}{31.79}\,FP \qquad R^2 = 0.94$$

$$PS = \underset{(698.34)}{472.28} + \underset{(0.18)}{0.79}\,FS + \underset{(27.95)}{49.92}\,PP \qquad R^2 = 0.92$$

$$FC = \underset{(538.25)}{3039.01} - \underset{(15.02)}{24.03}\,FP + \underset{(6.80)}{18.50}\,NFEXP + \underset{(0.24)}{0.04}\,PERY \qquad R^2 = 0.90$$

$$PC = \underset{(764.60)}{3822.82} - \underset{(34.73)}{75.95}\,PP + \underset{(0.12)}{0.84}\,NIMP - \underset{(0.0008)}{0.001}\,STOCKS + \underset{(0.41)}{0.41}\,PERY \qquad R^2 = 0.97$$

where:

FS = fresh domestic supply to retail;

PS = processed domestic supply to retail;

FC = fresh domestic demand;

PC = processed domestic demand;

FP = real fresh apple retail price;

PP = real processed apple retail price;

TDS = total domestic apple supply to retail;

$PROD$ = total domestic apple production;

$PERY$ = real domestic per capita disposable income;

$NFEXP$ = net export of fresh apples;

$NPIMP$ = net import of processed apples (juice); and

$STOCKS$ = domestic inventory of processed apples.

Table 6. Correlation Matrix of Reduced Form Residuals for the 2SLS Estimated Market Model.

	FS	PS	FC	PC	TDS
FS	1.000				
PS	-0.612	1.000			
FC	0.448	-0.199	1.000		
PC	-0.312	0.568	0.133	1.000	
TDS	0.186	0.664	0.176	0.410	1.000

and processed demand equations. Given the type of extended analysis for which the apple market model was to be used, the estimated system was considered acceptable.

Elasticity Calculations

The information summarized in the apple market model was used to calculate point-estimates of the domestic supply and demand elasticities for both fresh and processed sub-markets. Denoting the subscripts *F* as fresh market and *P* as processed market,

$$\varepsilon_F = \frac{\partial FS}{\partial FP}\ \frac{\overline{FP}}{\overline{FS}} = (31.79)\frac{(20.46)}{(3642.75)} = 0.179 \quad ,$$

$$\varepsilon_P = \frac{\partial PS}{\partial PP}\ \frac{\overline{PP}}{\overline{PS}} = (49.92)\frac{(13.43)}{(2954.43)} = 0.227 \quad ,$$

$$\eta_F = \left|\frac{\partial FC}{\partial FP}\right|\ \frac{\overline{FP}}{\overline{FC}} = (24.03)\frac{(20.46)}{(3592.89)} = 0.137 \quad ,$$

and

$$\eta_P = \left|\frac{\partial PC}{\partial PP}\right|\ \frac{\overline{PP}}{\overline{PC}} = (75.95)\frac{(13.43)}{(3667.17)} = 0.278 \quad ,$$

where ε is supply price elasticity; $|\eta|$ is the absolute value demand price elasticity; and it is understood that the elasticities are calculated at the means of prices and quantities. As outlined in Appendix F, the excess supply price elasticity in the fresh market (ρ_F) and the absolute value excess demand price elasticity in the processed market ($|\gamma_P|$) can then be calculated as

$$\rho_F = \varepsilon_F \frac{\overline{FS}}{\overline{NFEXP}} + \eta_F \frac{\overline{FC}}{\overline{NFEXP}} = (0.179)\frac{(3642.75)}{(292.96)} + (0.137)\frac{(3592.89)}{(292.96)} = 3.906 \qquad [26]$$

and

$$\gamma_P = \varepsilon_P \frac{\overline{PS}}{\overline{NPIMP}} + \eta_P \frac{\overline{PC}}{\overline{NPIMP}} = (0.227)\frac{(2954.43)}{(712.74)} + (0.278)\frac{(3667.17)}{(712.74)} = 2.371 \quad , \quad [27]$$

where it is understood that the excess elasticities are also calculated at mean quantities.

The final pieces of market structure information needed to implement the expected-surplus framework are the excess processed supply price elasticity (ρ_P^{ROW}) and the excess fresh demand price elasticity (γ_F^{ROW}) for the rest-of-the-world. Given the regular supply and demand price elasticities for the ROW, ρ_P^{ROW} and γ_F^{ROW} can be calculated in a manner analogous to equations [26] and [27] (see Appendix F). However, the ROW elasticities are not widely available, are difficult to directly estimate, and even when estimated are the subject of considerable debate. As a result, ρ_P^{ROW} and γ_F^{ROW} are often assigned values based on knowledge of the domestic market structure and the size of the domestic country's trade relative to supply and demand in the ROW. For example, the ROW's excess processed supply elasticity can be calculated as

$$\rho_P^{ROW} = \theta \frac{S_{ROW}}{Q_T^0} + \sigma \frac{D_{ROW}}{Q_T^0} \quad ,$$

where: θ is the ROW's supply price elasticity for processed apples; σ is the ROW's demand price elasticity for processed apples; S_{ROW} is the ROW's supply of processed apples; D_{ROW} is the ROW's demand for processed apples; and Q_I^0 is the amount of processed apple products imported into the domestic country (Norton et al.). The data presented in Table 2 suggests that both $\frac{S_{ROW}}{Q_I^0}$ and $\frac{D_{ROW}}{Q_I^0}$ will be approximately one order of magnitude larger than the corresponding ratios in equations [26] and [27]. In addition, economic theory implies that regular supply and demand price elasticity should be more elastic in the ROW than in the domestic country. Taken together, this information suggests that ρ_P^{ROW} and γ_F^{ROW} will be at least one order of magnitude greater that ρ_F and γ_P, respectively. Thus, for this study, the two remaining elasticities were assigned the values ($\rho_P^{ROW} = 39.0$) and ($\gamma_F^{ROW} = 24.0$). In practice, excess supply and demand elasticities greater than 10 have been demonstrated to yield results which

approximate calculations utilizing perfectly elastic excess supply and demand schedules (Norton, personal comm.).

Summary

This chapter described the theoretical basis, limitations, and implementation of an expected-surplus framework that was used to measure the society-level impacts of insecticide use, pest resistance, and regulations. The information obtained from this economic analysis is combined with the optimizer presented in Chapter VIII to identify optimal insecticide use and regulatory patterns given the desire to maximize total social welfare. Because the economic measure is critical to the operation of the overall research model employed in this study, a number of its important characteristics are summarized below:

- The expected-surplus approach to measuring economic impacts is a theoretically imperfect, but empirically practical, method of assigning a money value to potential net changes in social utility or welfare.

- Expected-surplus measures, like all welfare measures that assume ordinal preference structures, can only be used to rank the desirability of potential policy-outcomes regardless of how much cardinality the use of numerical scales appear to convey upon the analysis.

- Given the observed pricing mechanisms, governmental intervention, and links between the farm and retail levels, the U.S. apple market was most appropriately modelled as an open market where price was competitively determined at the world level for each of two related sub-markets; one for fresh apples and one for processed apple products.

- Formulas were developed for the calculation of expected- surplus changes under various potential market scenarios. These relationships are advantageous in that they provide estimates of future welfare changes based solely on information known in the current period, including equilibrium price and quantity, expected proportionate changes in output, and the relevant supply and demand price elasticities.

- An econometric apple market model is presented, estimated, and evaluated. Estimated coefficients and other data are used to calculate relevant supply and demand elasticities, including excess price elasticities for the domestic and world markets. Domestic apple supply and demand proved to be quite inelastic, while excess price relationships were calculated to be very elastic.

CHAPTER VIII: EMPIRICAL MODEL III -- OPTIMIZATION

Studies employing simulation have demonstrated the method's usefulness in modelling the complexities of biological and economic systems. As a result, simulation has been incorporated into a number of economic studies, particularly as a replacement for the small sets of analytic equations that are frequently used to characterize agroecosystems. Similarly, the expected-surplus approach to *ex ante* welfare analysis has also been adopted as a practical method for estimating the social impacts of proposed policy actions. The empirical successes of simulation and expected-surplus analyses have even led researchers to utilize both methods in the same study. This combined approach usually employs simulation to generate the expected price or quantity changes required to calculate the expected-surplus relationships. But, rarely have studies attempted to utilize both simulation and expected-surplus within an overall dynamic optimization framework, and those investigations that have utilized combined simulation/expected-surplus/optimization methods almost invariably resort to problem simplification in order to meet the demands of optimization algorithms that were developed for analytic applications. In contrast, one objective of this study is to model the agroecosystem in ways that avoid stylization as much as possible. This chapter focuses on the procedures by which simulation, expected-surplus, and optimization can be combined to investigate the operation of a realistically portrayed agroecosystem.

Model Linkages

Linking the simulation and expected-surplus models into a single coherent unit amenable to optimization not only requires yield relationships capable of transforming varying pest densities into time-paths for expected-surplus, price, and quantity changes, but also a method by which differing time-paths can be compared for relative performance. While the first part of this linkage process can be accomplished through the use of experimentally derived equations for expected pest damage, the second part involves the somewhat controversial application of present value discounting.

Using published studies and expert opinions, this section begins by detailing the pest density/apple yield relationships employed in the linkage between the expected-surplus and simulation models. This section concludes with a presentation of the potential advantages and problems encountered when using present value discounting, along with a description of how discounting was used in this study.

Pest Density/Apple Yield Relationships

Perennial crops, such as apples, are usually not killed by a single season's pest infestation. However, both tree growth and potential yield can be affected in the current season and for several seasons after an infestation has ceased (Kulman; McKinley). As a result, apparent yield losses may not be strictly proportional to current pest populations, especially when the pest is an indirect feeder and does not physically attack the marketable product. Instead, the relationship between an insect infestation and apple yield is a complex function of tree age, variety, vigor, climate, soil, cultivation practices, and pest levels (Hull, Beers, and Grimm). In addition, the level of pest damage may not be solely related to season-ending pest population levels, but also to the timing of peak population levels and/or the cumulative season-long pest pressure on the crop.

Given the intricate reaction of an apple tree to pest injury, accurate pest density/apple yield relationships have been extremely difficult to develop. And yet, an understanding of how insects affect current and future yields is critical to determining suitable methods and tactics of pest control (Bardner and Fletcher). In this study, the relationship among past pest levels, current pest levels, and changing yields is essential to the linkage between the simulation and expected-surplus models. These linkages require an estimate of year-to-year changes in expected yield, and for this study were based on peak and/or cumulative season-long pest pressure. But in doing so, the method implicitly assumes *ceteris paribus* conditions on all yield factors not directly related to pest populations. This allows model development to concentrate on the main objectives of the study, including the impact of chemical regulations on long-run chemical use. However, it does preclude substitution of non-chemical for chemical control methods, with the major exception of the potential importance of the BLB as a control for ERM. Although outside the domain of this study, the inclusion of non-chemical substitution possibilities are one obvious way in which the model might be expanded in the future if the relevant data becomes available.

TABM Damage Functions

As a direct feeder on apple fruit, TABM yield damage is generally restricted to quantity and quality losses in the year in which the infestation occurs. TABM tends to have little impact on tree physiology, and is usually controlled without regard to its potential effects on yields in subsequent growing seasons. Because of these single season characteristics, total TABM damage can be determined from the population levels in the current year, perhaps making the TABM damage function the simplest to conceptualize. But, even conceptually simple pest damage relationships can be extremely difficult to empirically estimate because of the multiple physical, biological, and management factors that can influence crop yield. The complexity of the estimation problem is often compounded by a lack of data sufficient to statistically estimate a comprehensive yield loss specification. As a result, studies tend to rely

on general theoretical forms for the damage relationships, with relevance to a specific system being obtained from various experimental observations that can be used to calibrate the theoretical form. One way of accomplishing this calibration is to relate peak population levels, cumulative population levels, or both, to total quantity and quality yield losses. Considering that TABM economic thresholds are usually calculated on the population level at a given point in time, this study utilizes peak population levels as an indicator of total damage.

Beginning in 1961 (Tammes), various theoretical forms have been suggested for the relationship between plant yields and stress. Because stress can be defined as any potentially injurious biotic or abiotic environmental factor (Levitt), the impact of insect pests on an apple tree's ability to produce marketable yield would appear to fall within this general framework. While specific theoretical forms vary, they often express loss as a proportion of stress levels. In doing so, attempts are made to include various theoretical considerations, including 1) a pest threshold below which no loss occurs due to a plant's ability to compensate for an infestation by boosting growth rates; 2) a maximum level of stress above which losses are no longer strictly incremental; and 3) an intermediate region where there is some type of positive incremental relationship between increasing stress and crop loss (Madden et al.). A popular and flexible form for incorporating these characteristics is

$$L = 1 - e^{-\left(\frac{P-D}{B}\right)^{S}} \qquad [28]$$

where:

L = proportionate crop loss;

P = a proportionate measure of pest population;

D = initial proportionate pest population at which damage occurs;

S = a shape parameter for the relationship; and

B = a slope for the intermediate region.

This relationship yields a family of curves (Figure 15) that have been used in life-testing, reliability, and plant disease studies (Weibull; Billman, Antle, and Bain; Madden et al.), and pro-

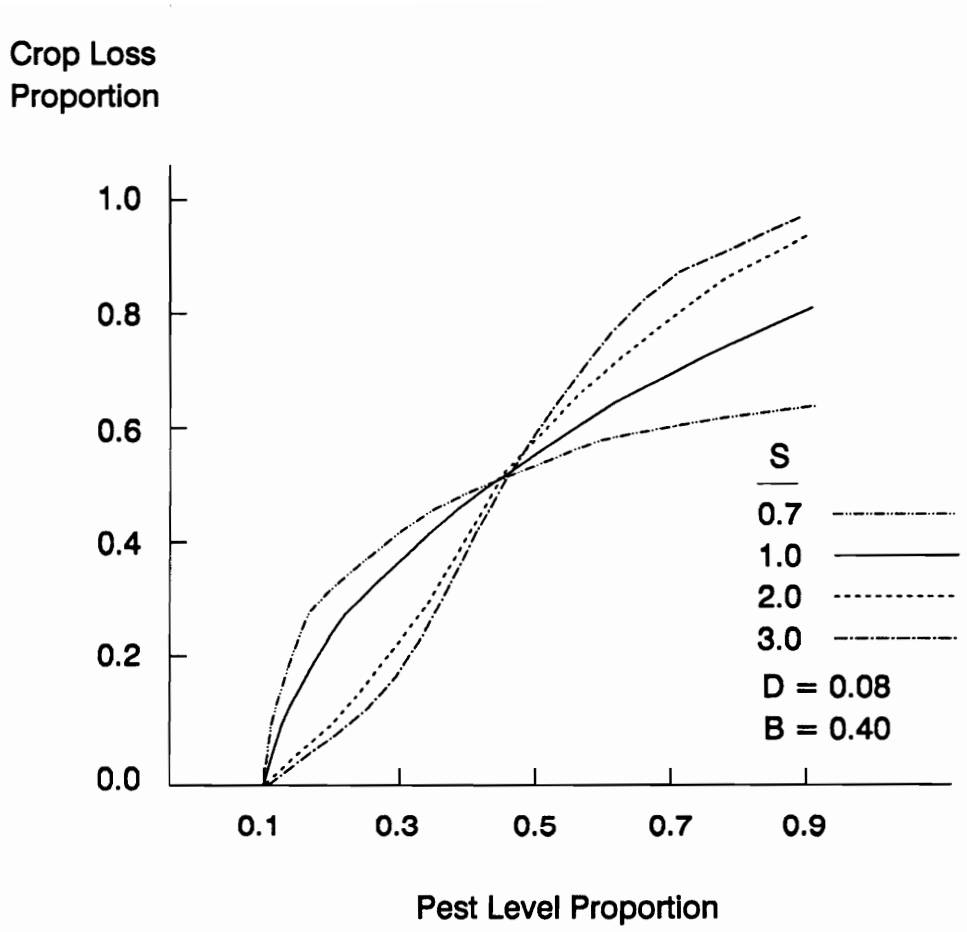

Figure 15. Family of Curves Describing the General Form of the TABM Damage Function: Adapted from Madden et al., p. 686.

vides a reasonable approximation to the interactions between TABM populations and apple losses.

Calibrating equation [28] to the TABM/apple situation required specific information on 1) the minimum TABM population level at which yield damage would be expected; 2) the maximum amount of yield damage expected and the TABM population levels at which it would occur; and 3) some intermediate points that relate various TABM population levels with specific yield damage. The information used in this study was obtained from the literature, proposed economic thresholds, and expert opinion. The theoretical model was calibrated using an iterative least squares procedure, and it resulted in

$$L = 1 - e^{-(\frac{P}{1.1})^{0.9}} \quad . \qquad [29]$$

As can be observed from the graphical depiction of this function (Figure 16), yield damage is assumed to be incurred even at the lowest TABM population levels. Damage increases rapidly, but at a decreasing rate. An economic threshold of 2 percent damage is reached when TABM levels are at 1.5 percent of carrying capacity, while maximum damage of 60 percent is reached when TABM levels are at 100 percent of carrying capacity. Note, however, that the damage levels calculated from this calibrated relationship do not necessarily map one-to-one into marketable yield loss. In fact, Hull and Rajotte demonstrated that as much as 84 percent of TABM damaged apples will still be graded into the top quality classification, at least for processed apples. Thus, for the purposes of this study, the damage levels calculated in equation [29] were used as calculated for the fresh market, but reduced by 84 percent for the processed market. Furthermore, 84 percent of damaged fresh apples were assumed to be marketed through processed channels. While this latter adjustment is likely to overestimate the ability of producers to re-target their damaged fruit, in the absence of better data it provides for a conservative estimate of losses to pest damage and thus a conservative estimate of the value of any given insecticide.

Implementation of the calibrated TABM damage relationship within the overall empirical model initially required, for each simulated year, the calculation of how close peak population

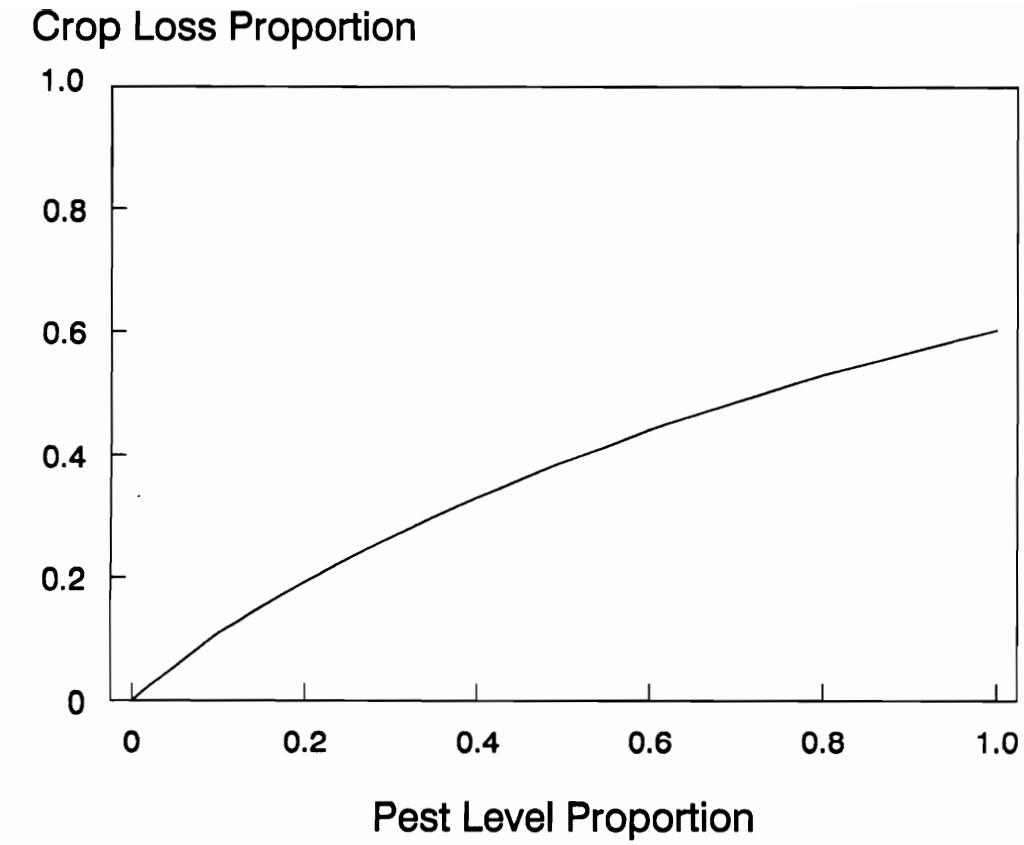

Figure 16. The Calibrated TABM Damage Function.

was to the carrying capacity of the agroecosystem. With this information, a percent loss was obtained from equation [29] and used to determine the quantity change from the original quantities produced in the initial year in each market. This approach implied that the initial year's quantity levels represented a steady-state equilibrium. Quantity change was then used to derive the year-to-year change in economic surplus. In the long-run, increasing pest resistance insures that the average year-to-year quantity change will be negative, making application of the backward-shift surplus formulas appear to be relatively straightforward. However, fluctuating pest population levels and control regimes may result in a positive quantity change in any specific year of the planning horizon. If this occurred, outward-shift surplus formulas were used to calculate the surplus changes.

ERM Damage Functions

Because it is an indirect feeder on apple trees, the yield damage of ERM is not, at least in theory, restricted to quantity and quality losses in the year of infestation. ERM can potentially have multi-season impacts on tree physiology, leading not only to decreased fruit size and yield in the year of infestation (Light and Ludlam; Ames, Johnson, and Rom), but also decreased return bloom, yield, and fruit size in subsequent years (Baker; Coghill). While these multi-season effects are not universally observed following ERM infestations (Zwick, Fields, and Mellenthin; Beers, Hull, and Grimm), the potential for long-term economic damage has been a serious concern to commercial orchardists. This is especially true given ERM's propensity for explosive population growth. As a result, studies have attempted to discern the link between ERM population levels and apple yield/quality (Hull, Beers, and Grimm; Beers, Hull, and Greene; Hull, personal communication). It is from these studies that the data for the following yield relationships were obtained.

Unlike the economic thresholds calculated for TABM, ERM economic thresholds tend to be based on the cumulative population numbers that occur over the course of a growing season. The primary rationale behind not using estimates of population densities at a single

point in time arises from the indirect mode of damage. Because ERM does not directly attack the marketable product, its population level on a particular day is not necessarily related to potential damage on the day after. Instead, a given population level's damage may only become apparent a number of days, weeks, or even seasons after it occurs, and then only if the physiology of the tree is such that it cannot compensate for the indirect feeding that takes place. This time-lag relationship between indirect feeding and potential damage to marketable products is further complicated by the fact that indirect feeding often must take place over a period of time before stress reaches a damaging level. Because instantaneous damage does not occur, cumulative pest pressure has proven to be a more useful indicator of potential damage, and is usually measured in terms of cumulative mite days (CMD).[38]

In addition to the CMD, crop load, timing of stress, tree nutritional status, and weather can potentially play a role in the expression of mite damage (Hull, Beers, and Grimm). These factors may be especially critical in an orchard level model because they tend to vary significantly over relatively small geographic areas. However, given the aggregate nature of this study, the objectives employed, and the observation that regulations and insecticide use only directly affect CMD, most of these additional factors can be held constant in order to examine the impact of induced CMD fluctuations on yield and quality. Of course, in doing so it should be recognized that the potential total short-run impact of insecticide use, and thus regulatory action, may vary as these other factors vary in a particular region. But, over a sufficient planning horizon, the impacts of these other factors might reasonably be expected to converge on long-run averages for a given area.

Even after an effort to focus on the factors directly relevant for this study, the calibration of an ERM/apple damage relationship proved to be non-trivial. Published data on the impact of CMD varied widely with geographic region, apple cultivar, and the year in which the study was executed. General agreement existed as to the importance of CMD and timing of peak populations, but little convergence occurred as to the specific values of critical parameters.

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[38] (CMD) is the sum of the numbers of days that each individual mite is present and feeding in the orchard. In practice, it can be calculated as the average population level over a given time period times the number of days in the time period.

Thus, this study relied heavily on information obtained from the Pennsylvania State University Tree Fruit Experiment Station (Hull, unpublished data). Observations of experimental orchards indicated that a multi-linear relationship could be expressed between the mean weight of an apple in grams (*MWT*), the number of cumulative mite days (*CMD*), and a measure of the crop load (*CL*):

$$MWT = 198.696 - 5.922(CL) - 0.0083(CMD) \quad .$$

Given the decision to concentrate on the effects of *CMD*, a median value was assigned to the remaining independent variable ($CL = 10.0$), yielding a simplified relationship of

$$MWT = 139.476 - 0.0083(CMD) \quad ,$$

which implies that, in the absence of ERM, the average apple will obtain a weight yield of 139.476 grams. Thus, the presence of ERM will lead to a reduction in optimum average weight yield of

$$PWL = \frac{0.0083(CMD)}{139.476} \quad , \qquad [30]$$

where *PWL* is the percent weight loss expressed in decimal form and *CMD* is obtained from the biological simulator.

As may be obvious from the above construction, *PWL* only represents yield loss relative to induced reductions in fruit size. Studies have also shown that significant same-season yield losses might occur due to mite-induced premature fruit drop, especially when the trees are carrying medium fruit loads. Although data documenting this effect is somewhat limited, at least one published study suggests that a 0.013 percent reduction in yield due to premature fruit drop can be expect per CMD (Ames, Johnson, and Rom). Incorporating this information into equation [30] results in a new single-season yield loss equation of

$$PWL = \frac{0.0083(CMD) + 0.0181(CMD)}{139.476} = \frac{0.0264(CMD)}{139.476} \quad . \qquad [31]$$

This indicates a total expected single-season yield loss of 18.9 percent per 1000 CMD, a value that falls into the lower part of the reported range (Baker; Ames, Johnson, and Rom; Beers, Hull, and Grimm). But, as of yet, equation [31] does not incorporate any multi-season effects.

A problem arises with incorporating multi-season ERM effects into yield loss equations for apples because of a relative lack of data. Researchers have had difficulties in separating out the inter-season effects of indirect mite feedings from the impact of early-season weather. ERM effects on return bloom have been documented, with an approximate decrease of 18 percent for every 1000 CMD (Beers and Hull). However, this value cannot be directly related to yield, as it does not account for the relationship between the size of bloom and the chemical thinning that is used to manage crop loads. Other studies suggest that subsequent year yields might decrease as much as 50 percent for CMDs between 500 and 900, while third year yields appear to be unaffected (Baker). Given the lack of a clearly defined multi-season relationship, a conservative approach would seem to be appropriate to restrict second-season effects to at or below the levels of intra-season effects. Thus, for the purposes of this study, second-season effects were initially set at 10.0 percent yield loss per 1000 CMD of the previous season, resulting in (Figure 17)

$$PWL_t = \frac{0.0264(CMD_t) + 0.0139(CMD_{t-1})}{139.476} \quad . \qquad [32]$$

Subsequently, sensitivity analysis was used in order to ascertain the importance of this variable to the optimal depletion paths.

Implementation of the ERM damage relationship within the overall empirical model initially required, for each simulated year, the calculation of the cumulative mite days. With this information, a percent loss was obtained from equation [32] and used to determine the quantity change from the steady-state quantities produced in the initial year in each market. Unlike TABM, however, the ERM yield losses calculated from equation [32] were restricted to the sub-markets in which they occurred. Although ERM damage could potentially lead to a situ-

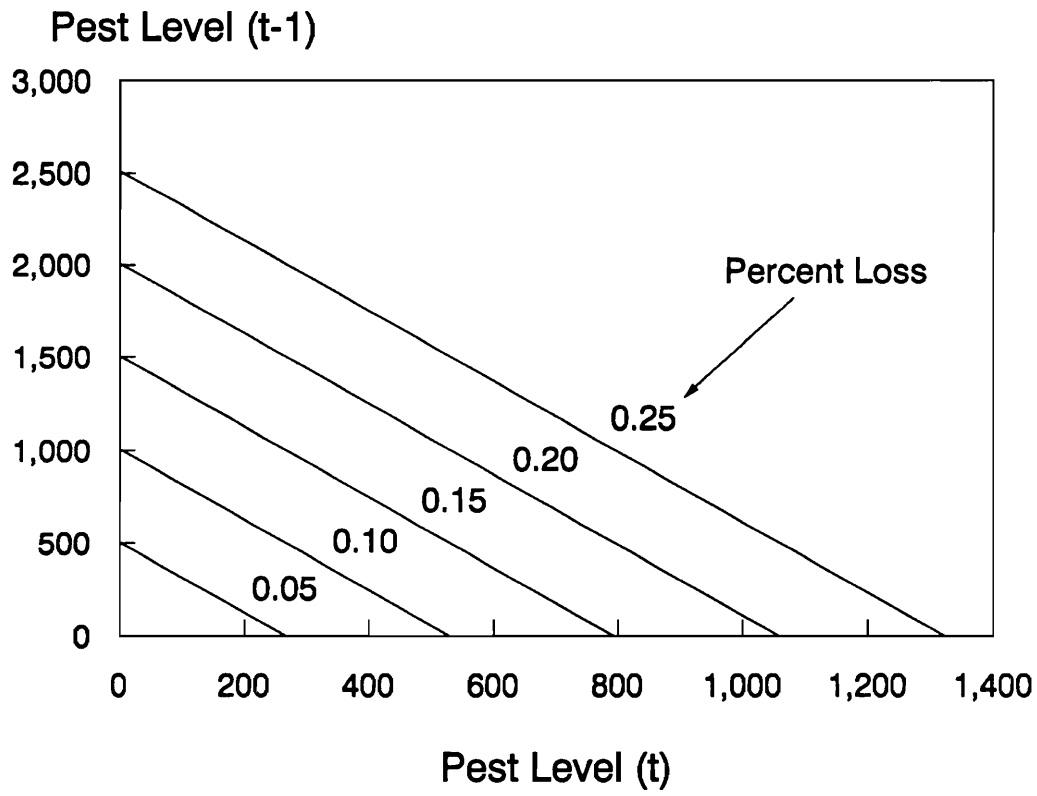

Figure 17. The Calibrated ERM Damage Function.

ation where fresh fruit is re-targeted to the processed market due to its reduced size, the potential extent of this movement has not been studied.

Present Value Discounting

Hotelling's theory of exhaustible resource allocation assumed that price expectations and resource extractions were based on perfect foresight (Knapp). Resource economists also usually assume that a unit of benefits or costs derived from resource extraction are more important if experienced in the near-term, an assertion that has led to the use of positive rates to discount future economic outcomes. Taken together with other standard welfare theory assumptions, perfect foresight and time-preference promote an analysis that calculates the expected future economic outcomes of a current action in nominal terms, and then discounts the outcomes in a way that normalizes them into present values. When these present values are used to determine the welfare implications of potential policy actions, the emphasis is placed on the current period's utility structure. Thus, present value discounting tends to promote near-term consumption of exhaustible resources because positive discount rates implicitly suggest that it is optimal to let the resources decay in the long-run (Dasgupta and Heal). The theoretical and empirical implications of these behavioral assumptions are the next topic for consideration.

Origin and Identification

The present value approach to intertemporal resource allocation is based on two fundamental assumptions (Pearce and Turner): 1) humans are by nature impatient; and 2) capital productivity is generally positive. Impatience suggest that individuals will have a time preference for current consumption over future consumption. If the supremacy of individual preferences in determining social welfare is maintained, then social rates of time preference will

also reflect impatience, with impatience implying a positive discount rate. At the same time, positive rates of capital productivity also suggest the use of a positive discount rate, for it will be rational to divert resources from consumption to investment only when the diverted resources yield higher consumption in the future. Given the simplicity and apparent universality of these two observations, the consensus opinion in economics is that social discount rates should be used, and they should be positive (Mishan).

Discount rates can theoretically be determined from the principle that, at the margin, the value of capital formed by public investment should be equivalent to the value of private capital and the value of foregone consumption (Randall). Because efficiency implies that the market interest rates, the marginal efficiency of investment, and the marginal rate of time preference will all be equal, the choice of an appropriate discount rate under perfect market conditions would be self evident. However, in reality the discount rate will diverge from the market rate of interest, sometimes significantly. This situation arises because, in an imperfect market, market interest rates can be a function of four different values (Randall): 1) the real discount rate; 2) financial transactions costs; 3) anticipated inflation; and 4) risk. This suggests that a real discount rate appropriate for present value calculation will almost always be lower than the market interest rate, although how much lower is a matter for debate. Thus, a social discount rate is usually chosen from a reasonable range, with reasonable being defined on some benchmark studies (for example, see Lind et al.). In general, the real social discount rate is often chosen from the 0 to 6 percent range (Randall).

Empirical Impact

Because the actual social discount rate is usually not known with precision, a general notion has developed in empirical economic studies that lower discount rates should be used when allocating an exhaustible resource. In theory, high discount rates may shift cost burdens forward to later generations while, at the same time, depressing the current level of investment in the development of alternative resources (Pearce and Turner). However, it is not in-

tuitively obvious that these discount rate effects will be significant in an empirical context. In fact, Chapman demonstrated that sharply divergent production time-paths can still yield essentially equivalent levels of return, implying that optimal planning may not always be necessary in order to gain near-optimal value from a system. His study clearly suggested that theory alone was not enough to analyze exhaustible resource allocation problems, with numerical analysis in an optimal control framework being perhaps the only way to examine the specific economic implications of a production path.

Subsequent to Chapman's study, the somewhat paradoxical occurrence of multiple near-optimal depletion paths was shown to arise from the relative flatness of conventional social welfare functions (Rowse 1988). But, given that discounting tends to ignore costs and benefits in the long-run, Rowse's results implied that the only way to choose between the near-optimal depletion paths was by some criteria other than present value. In other words, continued use of conventional social welfare functions suggests that there may only be small near-term opportunity costs associated with non-efficiency goals or decisions based on incorrect information. Expanding this line of inquiry in a later study, Rowse (1990) empirically demonstrated that an incorrect choice of discount rates leads to overall welfare losses of only 0 to 6 percent, even in cases where the chosen rate was different from the true rate by a factor of four. However, an incorrect rate could lead to major shifts in the distribution of economic benefits between consumers and producers, suggesting that issues of current income distribution may be more important than those surrounding intergenerational equity.

Of course, the empirical results from the studies discussed above may be related to the specific problem formulations that were employed. Nonetheless, at least one implication is clear; the theoretical relationship between positive discount rates and near-term consumption do not necessarily lead to a situation where lower discount rates significantly change the amount of total net benefits obtained from a system. Lower discount rates will, however, prolong the availability of an exhaustible resource and shift benefits between producers and consumers. Whether producers or consumers gain from the lower rates depends on the location of the rate change relative to the true discount rate (Rowse 1990). But if a study's ob-

jective is the maximization of overall welfare, then any discount rate within a reasonable range will yield near-optimal depletion paths. Thus, given the objectives of this study, positive present value discounting was adopted with the provision that sensitivity analysis be conducted. To make the analysis roughly comparable to that of Rowse (1990), a real discount rate range of 0 to 12 percent was used.

Time Horizons

The choice of a time horizon for analysis is a decision that must be made in all dynamic investigations. In a broad sense, two alternatives exist: open- and fixed-time (Takayama). With an open-time specification, optimization occurs over the indefinite future, implying that it is technically feasible and desirable to maintain a stream of system benefits. Under this alternative, the value of the resource being exploited may never reach zero, and as a result may always contribute to the value of the objective function. An example of this approach to dynamic investigations was portrayed in the qualitative analysis of Chapter V. The second alternative, fixed-time, assumes that optimization occurs over some well defined time period. This approach implies that it is either technically impossible or undesirable to indefinitely maintain the stream of benefits derived from the system. However, the value of the resource being exploited does not necessarily reach zero at the end of the optimization period. If the final resource value is not desired to be zero, then some specific salvage relationship must be incorporated into the objective function.

The process of choosing between an open- and fixed-time specification for an empirical dynamic analysis is complex and depends on various assumptions concerning the life-cycle of the technology being modelled and the practical limitations of policy-making. In terms of technology, historical experience (for example, see Figure 6) and expert opinion (Rajotte, personal communication) suggests that the research and development behind a new insecticide chemistry occurs over a horizon of 20 to 25 years. At the end of this period, old

insecticides are usually abandoned in favor of the new, more effective controls. In the past, experimentation with potential insecticides was motivated primarily by the desire to diversify company product lines, with the relatively long time periods between significantly different insecticides arising from the random fashion in which research was conducted. The historic constancy of the product life-cycle, and the general elimination of old chemistries from the market, suggests the use of a fixed-time analysis with a horizon of 25 years and no salvage value associated with any remaining susceptibility to the outdated products. However, the expanding ability of resistance to dramatically shorten the marketable life of an insecticide has generated pressure to shorten the product planning horizon, perhaps through a more directed research program involving biotechnology applications. Along with the short planning horizons currently imbedded in the federal pesticide regulatory process, the impact of resistance suggests the use of a fixed-time analysis with a horizon of less that 25 years, although the assumption of no salvage value can still be maintained. Ultimately, the choice of a planning horizon will itself be a policy decision based on expectations for technological innovations. For this investigation, dynamic analyses were conducted over a range of different planning horizons bounded by 1 and 25 years, with susceptibility to the initial technology set assumed to be without value at the end of each planning horizon. By comparing results generated from optimizations over different time horizons, the trade-offs between maintaining and maximizing the flow of benefits from a system over different planning periods will become transparent, as will the time dynamic nature of resistance and its varying importance to system behavior.

Optimizing Simulation Models

Having described the linkages between the simulation and expected-surplus components of the empirical model, it remains to examine how such a combined model can be optimized. For the agricultural economist interested in dynamic systems, the integration of

simulation models with economic theory demands some form of numerical optimization. But most algorithms for empirical dynamic problems require that system objective functions and constraints be analytically expressed (Evtushenko). This is generally true for direct use of control theory and the maximum principle, with their reliance on boundary value, gradient, or math programming solution techniques. In addition, specialized math programming methods are often restricting because they tend to require that problems be formulated in very specific ways due to severe limits on computational ability. This problem has repeatedly arisen in dynamic programming applications, where the "curse of dimensionality" continues to plague complex analyses even though access to high-speed computing has greatly improved.[39] Faced with these difficulties, operations researchers have developed and tested a number of different general methods for optimizing the simulation process itself, thereby eliminating the need to analytically specify the relationships in the system. Although it is not practical to discuss all the different techniques in detail, the remainder of this chapter will include: 1) a general discussion of simulation optimization; 2) a detailed presentation of the specific optimization algorithm used in this study; and 3) a description of how the optimization algorithm was implemented to guide the solution process.

The Simulation Optimization Problem

Consider the general dynamic optimization problem[40]

$$\text{MAX} \quad \int_0^T \phi[U(t), X(t), t]\, e^{-rt}\, dt$$

$$\text{subject to} \quad \dot{X}(t) = f[U(t), X(t), t] \;, \qquad X(0) = X^0 \quad (X^0 \text{ fixed in } R^n)$$

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[39] For an example of how problems often need to be simplified, see Hornbaker and Mapp.

[40] The following discussion was adapted, in part, from Azadivar and Lee.

$$x_i(T) = x_i^T \ , \qquad i = 1, ..., l \quad (x_i^T \text{ all fixed}) \qquad [33]$$

$$x_i(T) \geq x_i^T \ , \qquad i = l+1, ..., m \quad (x_i^T \text{ all fixed}) \qquad [34]$$

$$g_j[X(t), U(t), t] \geq 0 \qquad j = 1, ..., k \qquad [35]$$

$$g_j[X(t), t] \geq 0 \qquad j = k+1, ..., h \qquad [36]$$

$$U(t) \in \overline{U} \ , \text{ a fixed set in } R^w \qquad [37]$$

where $\phi[U(t), X(t), t]$ can be the response of a simulation model at time t for values of w decision variables u_1, u_2, ..., u_w represented by the vector $U(t)$. Let these decision variables be constrained by membership in set $\overline{U}$ and non-negativity ([37] and [35] above), while pure state constraints include non-negativity ([36]) and terminal conditions ([33] and [34]). In addition, let $\dot{X}(t)$ denote the rate change in state variable levels, or the equations of motion. Some or all of these constraints may be deterministic or stochastic functions represented by other responses of the same simulation model that may be generating $\phi[U(t), X(t), t]$. This formulation is completely general in that it contains both mixed and pure state constraints.

There are two main problems immediately encountered when trying to proceed towards an empirical solution to a problem of this form:

1. If $\phi[U(t), X(t), t]$, $f[U(t), X(t), t]$, $g_j[U(t), X(t), t]$, and/or $g_j[X(t), t]$ represent simulation response functions, then they cannot necessarily be expressed explicitly in terms of $\{U(t), X(t), t\}$. This may make the problem unsuitable for standard solution techniques, instead suggesting the need for some type of search routine through the feasible solution space; and
2. Although $\phi[U(t), X(t), t]$ can potentially be evaluated for any given $\{U(t), X(t), t\}$, even under condition (1), it may not be possible to optimize by ordinary search techniques. Most search methods are adaptive in that they proceed sequentially towards a solution

by comparing system response criteria for two or more points in the feasible region. But if $\phi[U(t), X(t), t]$ is a stochastically simulated function, comparing its mean response based on one observation at each point in the feasible region may result in the selection of a wrong direction for the next search step. Even the average of several replications at each point may be insufficient to offset stochastic characteristics in the simulated response.

From an economist's viewpoint, the ideal solution procedure for this type of problem would be one that is capable of guiding the search process by incorporating some theoretical optimization conditions along with explicit use of simulation for representing constraints, objective functions, and stochastic behavior. The specifics of how this search might be accomplished will be discussed after a general description of simulation optimization techniques.

General Approaches to Optimization

There are two general categories into which all simulation optimization techniques can be classified; internal and external methods. Internal optimization methods are used within the actual development and construction of the simulation model, in essence modifying the mathematical expression of the relationships between system components so as to generate optimal responses. These approaches are problem specific, inflexible, and tend to redefine the basic nature of a simulation model by making it dependent on a pre-specified state of nature. External methods, on the other hand, are designed for use after the simulation model has been completely developed. They do not directly affect the representation of the modelled system, instead treating the simulation as an experiment in which the responses produced by the model may be observed, with prescribed decision rules subsequently used to select optimal values for controllable inputs to the system (Smith 1973a). This separation between simulation development and problem analysis through optimization allows the system and its processes to be characterized in line with observation and not as mandated by a solution

procedure. In addition, because they operate on completed simulation models, external methods tend to be applicable over a wide range of problems.

Included in the category of external optimization methods are response surface techniques (Myers), pattern searches (Hooke and Jeeves), and random searches (Smith 1973b). Although well developed, these techniques have found limited use in analyzing complex problems because they are based on one or more of the following assumptions (Smith 1973a):

1. The optimization search involves a function with a known algebraic form;
2. All responses are observed without error or statistical variation;
3. The number of available computer runs is essentially unlimited; and
4. The number of controllable inputs under investigation is relatively small.

In general, none of these assumptions are valid for complex simulations with large numbers of controllable inputs and stochastic variation. Also, because they are often slow to converge on the neighborhood of the optimal solution, the above techniques can require many more simulations than resources permit. These deficiencies have generated research efforts to find a truly general and powerful external optimizer. Much of this work has centered on modification to the Simplex Method (Nelder and Mead), which itself arose out of the idea Spendley et al. presented for optimizing either physical processes or mathematical functions by application of simplexes.

The Simplex Method

As originally envisioned, the Simplex Method was an external optimization procedure that sought to incorporate the results of previous simulations into the process of searching for an optimum. As a "direct" procedure, the Simplex Method does not require derivatives of the objective function. Instead, it operates with information on the relative response rank of parameter levels. Thus, the method is readily applicable to situations where analytic an math-

ematical representation of the system is difficult to obtain but it is possible to order the results arising from different input levels.

A simplex can be geometrically defined as a convex hull of $n + 1$ points in general position[41] in R^n, where n denotes the number of variables over which optimization is to take place. Thus, the procedure derives its name from the geometric figure that is moved through R^n in search of an optimum. What makes the Simplex Method attractive is its adaptive features. In particular, a simplex is moved through solution space in such a way that allows it to reflect, extend, contract, and/or shrink to conform to the characteristics of the response surface.[42] The simplex can elongate down long, inclined planes, change direction upon encountering a valley at an angle, or contract in the neighborhood of the optimum, with the aim of eventually including the optimum within the bounds of the simplex. This is in contrast to other external optimization procedures, most of which try to move along a line towards the optimum.

Using the notation from the general dynamic optimization problem, the Simplex method is initiated by defining a starting set of $w + 1$ controllable simulation input levels in the solution space R^w.[43] Simulation responses are then generated for each group of initial input levels. The algorithm operates by identifying the input levels that produce the worst simulation response and moving the simplex away from this worst vertex by replacing it with a new group of input levels. The new vertex is calculated by projecting the inferior vertex through the centroid of the w remaining vertices and simulating its associated system response. As this process repeats, the simplex can either grow or shrink in size depending on whether the response value at each new vertex is an improvement over the response associated with the last inferior vertex. The Simplex Method is completed when no more improvements can be

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[41] General position indicates that no three points defining the simplex are collinear.

[42] Note that the relationship between the Nelder-Mead Simplex Method and the simplex method of linear programming algorithms is in name only. Where as the simplex of linear programming is a permanently rigid geometric figure defined by constraint boundaries, the Nelder-Mead simplex is a movable, flexible geometric figure in hyperspace.

[43] Each $w + 1$ setting is called a vertex and will generally consist of specific levels for multiple input variables. The $w + 1$ settings as a group are termed a simplex.

made by eliminating the worst vertex (Jacobson and Schruben), when the simplex has collapsed to a single vertex, or, more likely, when the simplex reduces to some predetermined small neighborhood around the optimum.[44]

Among the external optimization procedures, the basic Simplex Method has produced some of the most promising techniques for analyzing simulated systems. The advantages of the method and its variants include:

1. Beginning at a point far from the optimum, a simplex search approaches the neighborhood of the optimum rapidly compared to other techniques (Suk and Mitra);
2. In general, a simplex search results in only a small fraction of the feasible solution space being simulated, thereby conserving computing resources (Lee);
3. The Simplex Method can be easily modified to accommodate various special characteristic of the simulated system, including stochasticity (Lee);
4. The Simplex Method is completely generalizable and can be used on both continuous and discrete simulations (Beveridge and Schechter);
5. A constrained version of the Simplex Method (dubbed the Complex Method) has been developed for application to optimization problems with all types of constraints (Box; Lee); and
6. The Simplex Method does not require an explicit expression of the objective or constraint functions.

Because of its flexibility, efficiency, and relative ease of implementation, the Complex Method variation of the simplex search was used in this study. But before describing the Complex Method, it will be useful to detail the specifics of the simplex search algorithm and provide an assertion of its convergence properties.[45]

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[44] In this case, the solution obtained can only be considered approximately optimal.

[45] A preliminary proof of convergence under certain conditions is provided in Appendix H.

The Specific Algorithm

The Nelder-Mead Simplex algorithm can be broken down into five basic operations: ordering, reflection, expansion, contraction, and shrinkage. In the description that follows,[46] v_i indicates a current simplex vertex with the rank of i, where $i = 0$ is the best system response and $i = w$ is the worst system response out of all the simplex's vertices. A vertex denoted by v indicates that the vertex is either unranked or not a member of the current simplex. Angled brackets, as in $< v_0, v_1, \ldots, v_w >$, indicate that the included vertices are members of the same simplex.

To begin a simplex search, a set of feasible vertices must be identified and used to define the initial simplex. A number of identification schemes have been developed, ranging from those that generate the initial vertices randomly to those that attempt to distribute the initial vertices uniformly throughout the solution space (Mitchell and Kaplan; Sargent). However, convergence difficulties tend to arise in bounded problems if the initial simplex lies close to the edge of the feasible region (Lee). For this reason, non-random selection methods have proven to be the most useful and are employed in this study to: 1) keep the initial simplex away from the constraint boundaries; and 2) provide the largest initial coverage of the feasible region. This latter objective is desirable because convergence speed tends to be highest when the initial simplex contains the final optimum. To accomplish this, the first vertex of the initial simplex is defined as the central point of the feasible region, and includes the elements

$$u_w = \frac{(c_w - a_w)}{2} \quad ,$$

where c_w is the upper and a_w is the lower constraint boundary on the control variable u_w. Other potential vertices are identified by dividing the range of each control variable into two equal sub-ranges and taking the center of each sub-region as a potential vertex. This second op-

[46] This discussion of the Simplex method is adapted from Nelder and Mead, Box, and Woods.

eration yields $2w$ potential points in hyperspace, of which the outermost w feasible vertices are included in the initial simplex.

Once an initial set of vertices are chosen, ordering occurs by whatever method is appropriate given the structure of the research model. Ordering in the context of the current study was conducted by simulating the system response for each individual vertex, and then ranking the responses, and thus the vertices, by comparison. The initial simulations and ordering is often the most time and computer intensive part of the simplex search algorithm, requiring $w + 1$ initial vertices for peak algorithm efficiency.

After the initial ordering, the algorithm proceeds by moving the simplex away from the worst vertex v_w. To do this, the centroid $\bar{v}$ of the current vertices, excluding v_w, is calculated. This centroid essentially serves as the hyperspace center of the non-worst vertices, and provides a focal point through which a potentially superior vertex can be found. Calculating the centroid as

$$\bar{v} = \frac{1}{w} \sum_{i=0}^{w-1} v_i \quad ,$$

a reflected vertex v_r is obtained from

$$v_r = (1 + \alpha)\,\bar{v} - \alpha v_w \quad , \qquad [38]$$

where $\alpha > 0$ is a reflection coefficient that essentially determines how far away from the inferior vertex the new vertex will be located. If the simulation response of the new vertex v_r is superior to the next-to-worst vertex v_{w-1}, then v_r replaces the worst vertex v_w as a member of the simplex. Stopping criteria are then tested, and if not satisfied, the vertices are reranked and the process begins again.

As might be expected, it is possible for the process of reflection to yield a vertex v_r that is superior to the best vertex v_0. If this occurs, then it is often advantageous to continue the

search in the direction of the original reflection. This is accomplished by calculating an expanded vertex

$$v_e = \gamma v_r + (1 - \gamma)\bar{v} \quad ,$$

where $\gamma > 1$ is the expansion coefficient. For v_e superior to v_r, the new simplex is defined by replacing v_w with v_e. If, however, v_e is not superior to v_r, then v_r replaces v_w and a new v_r is calculated after reordering.

But what happens if the v_r calculated in equation [38] is actually inferior to v_{w-1}, thereby making its replacement of v_w an act of futility? When this occurs, a contraction process is used to move the reflected vertex back along the projection path towards v_w. Letting v_t be the better of v_w and v_r, in terms of system response, then the contracted vertex is given by

$$v_c = \beta v_t + (1 - \beta)\bar{v} \quad ,$$

where $0 < \beta < 1$ is the contraction coefficient. If v_c is superior to v_{w-1}, then the new simplex is formed by replacing v_w with v_c. Stopping criteria are again checked, and if not satisfied, a new ranking and reflection initiated. If v_c is still inferior to v_{w-1}, then a shrinkage process is used to reduce the size of the simplex by moving all but the best vertex towards the best vertex:

$$v_i = \frac{1}{2}(v_0 + v_i) \quad i = 1, 2, \ldots, w.$$

A completely new simulation response must now be obtained for all but v_0 of the simplex, and re-ranking determined before proceeding on to a new reflection. This makes the shrinkage operation expensive, in terms of computing resources, relative to the other operations of the simplex method because order information for a new vertex is usually obtained incrementally in the decision-making on whether to accept or reject a new vertex. Thus, shrinkage has been a frequent focus of attempts to improve the Simplex Method.

The Nelder-Mead Simplex algorithm is illustrated for a response function defined over R^2 in Figure 18. Level sets of the function are also included in the illustration to demonstrate how the algorithm proceeds. In general, however, these level sets are unknown when optimizing a simulation, and information on them can only be obtained through the process of optimization itself. Figure 19 illustrates the potential relationships among various reflection, expansion, contraction, and shrinkage steps that may need to be taken when beginning with a simplex $< v_0, v_1, v_2 > \epsilon R^2$.

Although only alluded to in the above discussion, stopping criteria are critical to the operation of the Simplex Method. Assuming the existence of a simplex $< v_0, v_1, \ldots, v_w >$, where response values at each vertex are denoted by $\phi(v_0)$, $\phi(v_1)$, ... , $\phi(v_w)$, then the algorithm as proposed by Nelder and Mead will have found an optimizer $\phi(v^*)$ when

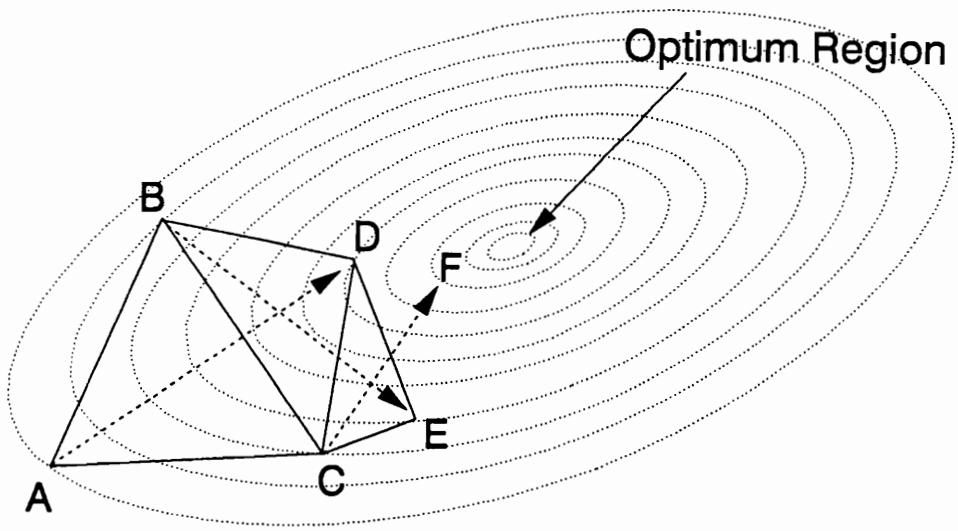
$$\frac{1}{w} \sum_{i=0}^{w} [\phi(v_i) - \overline{\phi}]^2 < \varepsilon \ ,$$

where

$$\overline{\phi} = \frac{1}{w} \sum_{i=0}^{w} \phi(v_i)$$

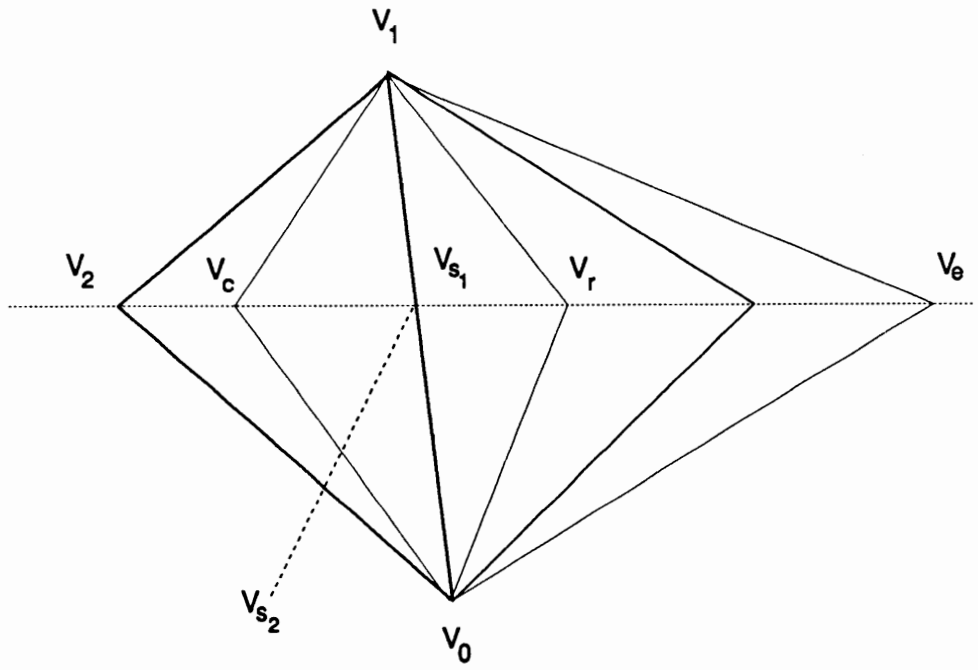
and $\varepsilon > 0$. In other words, the proposed stopping criteria halts the search when the standard error of the response values of all simplex vertices are less than the tolerance level ε, or when the simplex has collapsed to a pre-defined size. Note, however, that this stopping criteria can fail if the initial simplex is too small relative to the tolerance level ε. Thus, care must be taken when choosing the stopping parameter ε, even though the choice of an arbitrarily small ε involves a trade-off with the number of iterations the optimization process must execute.

Careful consideration of the Nelder-Mead Simplex method has led many researchers to label it an important tool for general optimization. Intuitively appealing in its operation, the



Beginning with the initial simplex (ABC), reflection and/or expansion of the inferior vertex (A) leads to the identification of a superior vertex (D) and a new simplex (BCD). As this process continues, the simplex moves towards the optimum. Note that the level sets, represented by the dotted lines, will generally not be known *a priori*, especially in simulation studies. They are included here only for illustration.

Figure 18. Graphic demonstration of the Simplex method in the solution space R^2: Adapted From Woods, p. 22.



Where the following definitions hold: $< V_0, V_1, V_2 >$ is the initial simplex; V_r is the reflection vertex; V_e is the expansion vertex; V_{s_1} and V_{s_2} are shrinkage vertices; V_c is the contraction vertex from V_n; and $\hat{V}_c$ is the contraction vertex from V_r.

Figure 19. A Comparison of the Simplices Generated by Reflection, Expansion, Contraction, and Shrinkage: Adapted From Woods, p. 22.

Simplex method appears to be robust and applicable to a wide range of simulation problems. And yet, little theoretical study has been given to the method's claim of convergence properties, even though the nature of the stopping criteria does not logically imply convergence to anything other than a local approximate optimizer. In an effort to support convergence properties of the simplex method, the following section will present some preliminary theoretical results developed by Woods.

Assertion of Convergence

In an early work on the nature of iterative solution procedures, Ortega and Rheinboldt examined methods of the form

$$v_{k+1} = v_k - \omega_k \alpha_k p_k \quad k = 0, 1, \ldots.$$

for finding the critical points of a function ϕ: $R^n \rightarrow R^1$, where $\alpha \in R^1$ is a step-length parameter and $\omega \in R^1$ is a relaxation parameter. The authors demonstrated that an iterative maximizer must have property

$$\phi(v_{k+1}) \geq \phi(v_k) \quad k = 0, 1, \ldots. \qquad [39]$$

where ϕ is bounded from above. Wolfe extended their analysis to show that the optimizer should also be able to satisfy

$$\lim_{k \rightarrow \infty} \frac{\nabla\phi(v_k)^T P_k}{\|P_k\|} = 0 \qquad [40]$$

and

$$\lim_{k \rightarrow \infty} \nabla\phi(v_k) = 0 \qquad [41]$$

where $\nabla\phi$ denotes the gradient vector of ϕ and $\|P_k\|$ denotes the Euclidean norm of P_k. This section will sketch the sufficient conditions associated with equations [39] through [41] and present a convergence theorem for the Simplex Method. A preliminary proof of the theorem is relegated to Appendix H.

To begin, consider the fact that the Nelder-Mead algorithm yields at least three distinct sequences of vertices that can be examined. These include the best approximate solutions $\{v_b^k\}$, the worst approximate solutions $\{v_w^k\}$, and the centroids of all the simplexes, $\{\bar{v}^k\}$. The sequence $\{v_b^k\}$ trivially satisfies condition [39] because v_b^k is replaced by $\hat{v}$ only if $\phi(\hat{v}) > \phi(v_b^k)$. Demonstrating condition [40] or [41] for sequence $\{v_w^k\}$, however, requires a concavity assumption on ϕ such that

$$\phi[\alpha v_1 + (1-\alpha)v_2] > \alpha\phi(v_1) + (1-\alpha)\phi(v_2) \quad \alpha \in [0,1] \ ,$$

where concavity also suggests the inequality

$$\phi\left[\sum_{i=1}^{w} \alpha_i v_i\right] > \sum_{i=1}^{w} \alpha_i \phi(v_i)$$

when $\alpha_i > 0 \ \ \forall\, i$ and $\sum_{i=1}^{w} \alpha_i = 1$. With these additional assumptions, the vertices of any simplex S_k will have response values greater than $\phi(v_w^k)$ such that, if a reflected vertex is accepted,

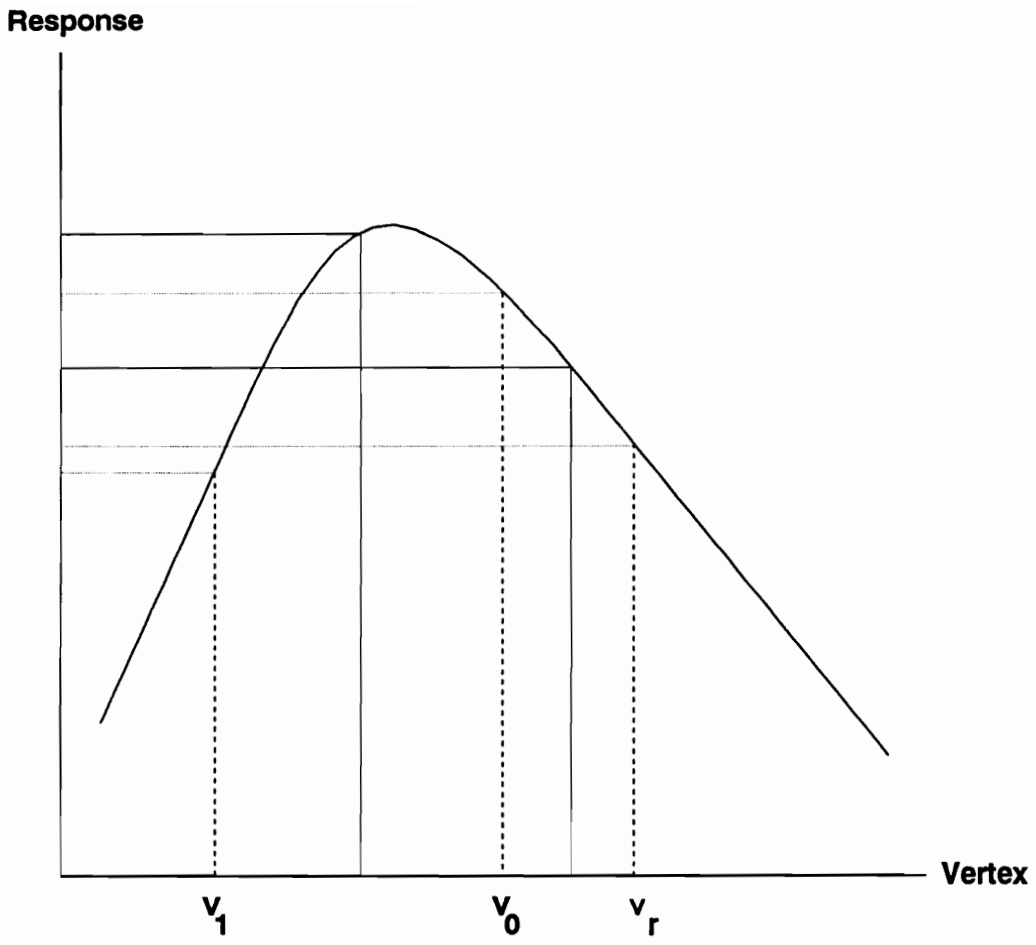
$$\phi(v_r^k) \geq \phi(v_{w-1}^k) > \phi(v_w^k) \quad .$$

If, however, a contraction vertex is accepted or the simplex is shrunk, then

$$\phi(v_w^{k+1}) > \phi(v_w^k)$$

and condition [40] or [41] holds for all $\{v_w^k\}$. In fact, this line of reasoning suggests that every sequence $\{v_i^k\}$ has the non-decreasing property.

Having demonstrated the potential viability of sequences $\{v_b^k\}$ and $\{v_w^k\}$, the sequence $\{\bar{v}^k\}$ remains to be examined. Consider the simple example in Figure 20, where the vertices



Given the initial simplex $<V_0, V_1>$, v_r would be accepted by the Nelder-Mead algorithm. However, it is obvious that $\phi\left[\frac{1}{2}(V_0 + V_1)\right] > \phi\left[\frac{1}{2}(V_0 + v_r)\right]$, demonstrating the inferiority of the sequence $\{\bar{v}^k\}$.

Figure 20. Demonstrating the Inferiority of the Centroid as a Potential Optimal Solution: Adapted From Woods, p. 30.

of a simplex S_k are v_0 and v_1, with v_r having just been accepted by the Nelder-Mead algorithm. From this example, it can be observed that

$$\phi\left[\frac{1}{2}(v_0+v_1)\right] > \phi\left[\frac{1}{2}(v_0+v_r)\right] \quad ,$$

thereby violating condition [39]. Thus, the sequence $\{\bar{v}^k\}$ is shown to be unacceptable, and only the sequences $\{v_0^k\}$ and $\{v_w^k\}$ can be considered viable alternatives. It is with these two sequences that the following convergence theorem was postulated by Woods:[47]

THEOREM: If the set $L(v_w^0) = \{v\colon \phi(v) \geq \phi(v_w^0)\}$ *is bounded, where* v_w^0 *is the worst vertex of the initial simplex, then a subsequence of the sequence of simplexes converges to some* $S^* = <\hat{v}_0, \hat{v}_1, \ldots, \hat{v}_w>$. *Additionally, let the Nelder-Mead algorithm be modified such that, at the* k^{th} *iteration, the expansion step* v_e^k *is accepted only if*

$$\phi(v_e^k) > \phi(v_r^k) > \phi(v_0^k) \quad ,$$

and the reflection step v_r^k *is accepted only if*

$$\phi(v_r^k) > \max\left\{\phi(v_{w-1}^k),\ \phi(v_w^k)(1+\text{sign}\left[(\phi(v_w^k))\right]\varepsilon)\right\} \quad ,$$

for some $\varepsilon > 0$ *and* $\phi(v_w^k) \neq 0$. *If* $\phi(v_w^k) = 0$, *then* v_r *is accepted only if*

$$\phi(v_r) > \varepsilon > 0 \quad .$$

Then, for any $\varepsilon > 0$ *and strictly concave* $\phi\colon R^n \rightarrow R^1$, *each convergent subsequence of the sequence* $\{S_k\}$ *generated by the algorithm converges to some degenerate simplex* $\hat{S}_i = <\hat{v}_{0,i}, \hat{v}_{0,i}, \ldots, \hat{v}_{0,i}>$. *Moreover, the function values at all limit points are equal, and the set of limit points is connected, so that the sequence either converges to a point, or else there are infinitely many limit points.*

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[47] Woods' theorem was set in the context of a minimization problem. The theorem presented here has been set in the context of maximization.

Furthermore, a corollary near the end of the above theorem's proof (see Appendix H) establishes that the Nelder-Mead algorithm will converge to a connected set of limit points even when the calculations are implemented on a finite precision, discrete computer.

As may have been noticed, the formal arguments for convergence hinge, to a large degree, on the concavity of the response function. In reality, however, few complex simulations will yield globally concave response surfaces. In these cases, the above discussion would apply to locally concave regions and would establish convergence to a local optimizer. As a practical matter, the difficulties associated with non-global concavity can be overcome by initiating the Simplex Method in different regions of the feasible space and comparing the resulting optimizers.

Following the introduction of the Simplex Method, attempts to employ it in large scale research tended to highlight some of the method's inherent weaknesses. As a result, numerous improvements and modifications have been suggested. While each of these brought some extension of the method's domain of applicability or greater efficiency to its operation, none changed the basic way in which the algorithm operated. Thus, the following discussion of the Complex Method variant used in this study should be considered as building on the basic simplex search already presented.

Modified Complex Method

The modified complex procedure (Azadivar and Lee) proposes to solve the problem

$$\text{MAX} \quad E[\,\phi(U, X)\,] = Y(U, X) \ ,$$

$$\text{subject to} \quad f_j(U, X) \begin{matrix} > \\ = \\ < \end{matrix} C_j \quad j = 1, 2, \ldots, b \ ,$$

where: U is a vector of discrete-valued decision variables; $\phi(U, X)$ is the random variable corresponding to observations on the simulated system; $Y(U, X)$ is the theoretical $\phi(U, X)$; and $f_j(U, X)$ is a set of b constraints. The algorithm makes the following assumptions:

1. The theoretical regression function $Y(U, X)$ is a real-valued, but perhaps unknown, function; and
2. There exists a finite constant M such that

$$\text{VAR}[\,\phi(U, X)\,] < M \quad \forall\, U\ .$$

Implementation

Implementing the Complex Method for optimizing the combined bioeconomic model involved a number of specific steps.[48] These steps, which will rely on the operational background presented in the discussion of the simplex search algorithm, include:

1. **Generate the initial simplex.** As mentioned earlier, this process entails the creation of a set of initial vertices, each composed of w elements representing the possible pest control actions that can be taken. These w elements are the variables over which optimization takes place. Given the nature of the problem being addressed and the desire to make the simulation portion of the solution procedure as realistic as possible, it might seem reasonable to define each of the w elements as corresponding to a simulation time-step in which some level of control action, including zero, could potentially occur. However, six different control chemicals were available for use in each time-step, and it was not obvious how they could be combined into a single measure of control while still maintaining the simulation's realism. Thus, each w element of a vertex was defined as being the level of control taken using a particular control chemical in a particular time-

[48] These steps are further detailed in the annotated optimizer program code contained in Appendix I.

step. Taking into consideration the six possible chemicals and 40 simulation time-steps per year, this construction resulted in 240 control variables per year of planning horizon. The levels obtainable by each control variable were determined by labelled application rates, and ranged from zero to 25 percent above the recommended application rate for each chemical in each time period. If re-entry and/or day-to-harvest restrictions were also part of the label, then appropriate constraints were placed on the time-steps in which a particular control chemical was available for use.

2. **Order the vertices of the initial simplex by response.** Each vertex selected in step (1) yields a response, in terms of total economic surplus, from the system being simulated. These responses, which are not necessarily unique, are then used to arrange the vertices in descending order relative to the amount of surplus each generates.

3. **Determine the worst performing vertex from step (2).** If there is more than one worst performing vertex because of non-unique simulated responses, each is operated on sequentially in the following steps.

4. **Calculate the centroid of all the vertices except the worst performer.** This calculation may include the other worst performing vertices if they exist.

5. **Calculate the reflection vector.** This serves to point the search away from a poorly performing region of the solution space and towards the general region in which lie all the other vertices. After some experimentation, a reflection coefficient of 1.5 for each chemical control was used.

6. **Project a new vertex.** This operation is accomplished by using the reflection vector to project the worst performing vertex through the calculated centroid into a new region of the solution space. Similar to the reflection coefficient, the proportional distance covered by the projection was defined, after some experimentation, as 3. The new vertex was then

checked for adherence to any active constraint boundaries. If any of the elements of a vertex violated a constraint boundary, they were set equal to the value of the constraint boundary. This latter, somewhat obvious step, was an important addition that greatly improved the efficiency of the original Simplex method.

7. **Calculate the response of the newly created vertex.** If the new vertex elicits a system response that is better than the worst and second-to-worst vertex, and if the system response does not violate any explicit or implicit constraints on the state variables, then the projection is considered successful and the process iterates back to step (2) after testing for stopping conditions. Again, the comparison of system responses and potential state variables to constraints was an efficiency improvement over the original Simplex method.

8. **If projection failed, then contract.** If the new vertex is not better, or if it leads to explicit or implicit constraint violations, then the new vertex is sequentially contracted back towards the original inferior vertex until a better vertex is found. If the new vertex never improves, then the next-to-worst vertex is selected and the process restarted at step (4).

9. **Terminate the simplex search.** The search is considered successful when the simplex collapses to a single vertex, or the simplex reaches some small, pre-defined size in the neighborhood around the optimum. For this study, an epsilon of 5 percent of the maximum application rate was used.

Improvements Over the Simplex Method

The complex search procedure, especially when applied to a deterministic simulation as in this study, is very similar to the Simplex Method in that movement occurs through the feasible solution space as simplex vertices, each composed of a specific vector U containing decision variable levels from the set $\overline{U}$, get closer to each other and converge towards the system optimum. The simplex search is transformed into the Complex Method by making

provisions for preventing any projected vertex of the simplex from leaving the feasible region. This is generally accomplished by testing each projected vertex for adherence to the constraint set. If the vertex falls outside the constraints, then it is either set to the constraint limit (for input variable bound constraints) or systematically retracted until it enters back into the feasible region (for explicit and implicit simulated function constraints).

The procedure outlined above is directly applicable to problems involving deterministic simulation. But the method is also extendable to stochastic systems, even though stochasticity results in a situation where there is no guarantee that a vertex rejected on the basis of comparing single simulations is indeed the worst point. In fact, the probability of an erroneous rejection increases as the variance in responses increases, and can only be ameliorated by incorporating multiple observations on the simulated response for any given vertex. One way of doing this is to calculate confidence intervals on the mean responses for a vertex. Because the confidence interval lengths will shorten as the number of simulation observations on any given vertex increases, a vertex can be rejected (with a chosen level of probability) as soon as its confidence interval is distinct and its mean is worse than the other vertices of the simplex. If confidence interval calculations and comparisons are sequentially made after small numbers of simulation runs on a set of vertices, then the total number of simulation runs for each search step can be kept to a minimum.

Stochasticity in the simulation constraint set can also lead to difficulties in directly applying the complex search method. This arises because stochastic variation implies that the constraints may never be satisfied on anything other than a probabilistic level. Thus, attaining a solution would require that the constraints be stated in terms of the maximum acceptable risk of violation. For example, a stochastic constraint might be expressed as

$$P[\phi_1(U, X) \leq C_1] \geq 1 - \alpha_1 \ ,$$

where $0 < \alpha_1 < 1$ and α_1 is the maximum acceptable risk of violating the constraint. This representation can lead to constraints formulated in terms of upper or lower confidence limits of the type

$$HU_1(U, X) \leq C_1 \quad for \quad P[\phi_1(U, X) \leq C_1] \geq 1 - \alpha_1 \ ,$$

where $HU_1(U, X)$ represents the upper confidence limit on the response $\phi_1(U, X)$.

Empirical Model Summary

In a broad sense, the overall empirical model was formulated as three distinct components; biological, economic, and optimization. Each component represents a more or less self contained research effort, capable of being updated or replaced independent of the remaining parts of the model. And yet, the three model components are intimately linked through a series of operational relationships. A brief description of these relationships concludes this chapter and provides a context within which the overall model can be viewed.

As shown in Figure 21, the three model components interrelate in a circular fashion. In particular:

- Using an initial time-path of insecticide inputs, the biological simulator generates the progression of pest populations and genetic resistance through and across growing seasons. From this information, an estimate of the time-path for apple yield and quality is calculated for an entire planning horizon;

- Yield and quality effects resulting from the insecticide use time-paths are incorporated into the economic surplus model, which then produces an estimate of the year-to-year changes in economic surplus generated by the production system. Total consumer, producer, and combined economic surplus changes are then generated, for the entire planning horizon, from the yearly surplus changes. This information is subsequently used as an indicator of the welfare impacts induced by a specific input set.

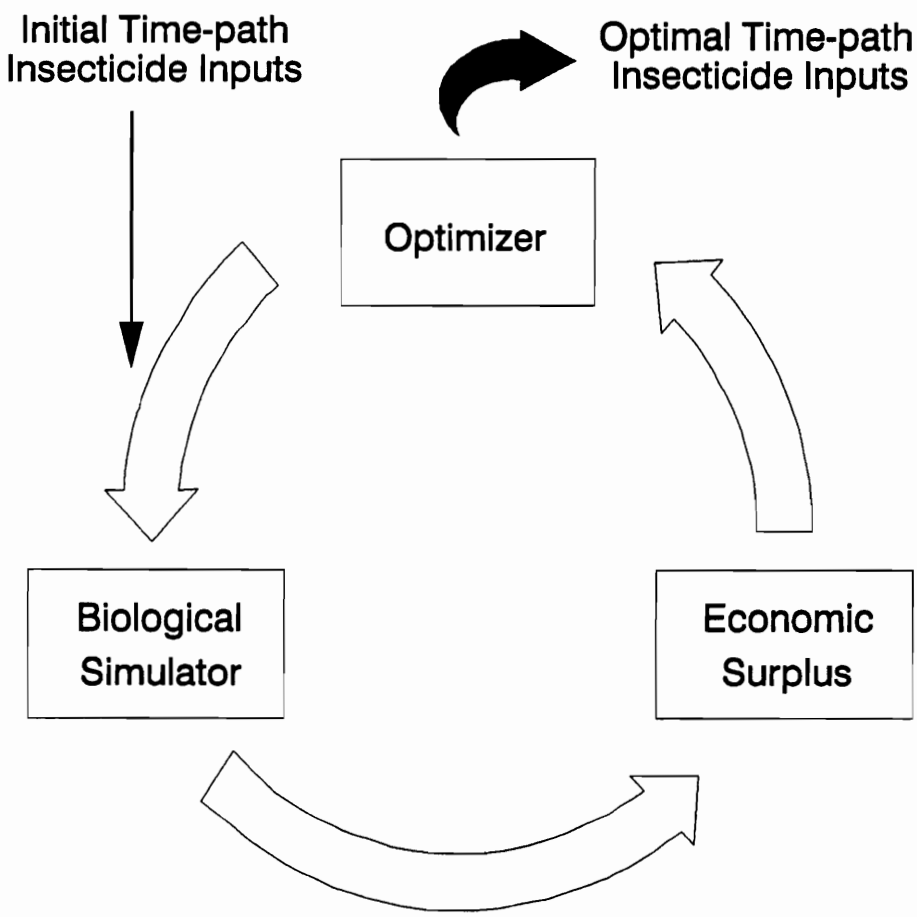

Figure 21. An Overview of the Empirical Model and Its Operation.

- Estimated aggregate welfare impacts are used by the optimizer to evaluate the performance, relative to an objective, of specific inputs, and to guide the generation of new, potentially superior input levels. As a time-path, the newly generated input levels consisted of potential control actions in each simulated time-step. Thus, the optimization occurs across all time-steps, both within and between years, and constitutes a truly dynamic optimization. These new input levels are then utilized by the biological simulator, repeating the above process.

Given an initial technology set, the model continues in this iterative fashion until an optimum control use policy (based on some pre-specified stopping criteria) is found. The impact of a proposed regulatory action can then be evaluated by changing the initial technology set to reflect the regulations and beginning the process over again. In the end, this modelling approach yields the optimum time-paths for input use, economic surplus, and susceptibility depletion.

CHAPTER IX: RESULTS AND DISCUSSION

The negative externalities associated with the interactions between pest resistance and insecticide regulations can have severe impacts on both commercial agriculture and the environment. Consequently, the management of the resistance/regulation nexus has become an important policy issue for private and public institutions, especially the U.S. Environmental Protection Agency. But, in order to mitigate the detrimental impacts of regulatory policy, quantitative models are required to analyze the dynamic economic consequences of actions that affect the use of insecticides in production systems. The objectives of this study included the development of an empirical procedure capable of incorporating the time-dynamic impacts of pest resistance into the evaluation of potential federal insecticide regulations. Following a brief restatement of the problem and an overview of this chapter's organization, the discussion focuses on the results generated from this modelling effort.

Resistance can be defined as the genetic changes that occur in a pest population in response to the selection pressure generated by repeated control actions (Sawicki). These genetic changes confer on pest organisms the ability to survive exposure to the insecticide being used. The emergence of pest control failures due to resistance, along with the common-property characteristics of pest susceptibility, point to the need for a nationwide policy addressing the pest resistance problem. But, while the EPA continues to search for a way to include resistance considerations into the static procedures for analyzing insecticide benefits

and costs, insecticide regulatory actions themselves may hasten the onset of pest resistance. Because the time-path of susceptibility depletion can be directly influenced by the numerical and technical characteristics of the control technology set, decisions about insecticide availability and use will affect future susceptibility changes in ways that are currently unpredictable. The solution to this problem requires that pest susceptibility, insecticide use, and regulatory actions be dynamically modelled in order to address the issues behind the simultaneous management of pests, pest resistance, and insecticide use. To implement this approach to the problem, this study combines a dynamic biological simulator (see Chapter VI), an economic market model (see Chapter VII), and an efficient search optimizer (see Chapter VIII). The study extends previous work by formulating a dynamic bioeconomic model that is not only economically realistic, but that also incorporates a high degree of biological realism. In addition, the optimization procedures employed do not require that the system be analytically specified, thus relieving the pressure to make unrealistic assumptions about system behavior in order to reduce the dimensionality of the problem.

The empirical model developed in this study is capable of providing voluminous and detailed information concerning the impact of resistance and regulation on the biological and economic evolution of the mid-Atlantic apple production system. While detail is highly desirable within a research context, it also makes the coherent presentation of results somewhat challenging. To address the key points that motivated this study and examine the specific working hypotheses identified in Chapter I, the remainder of this chapter is structured as follows:

1. **Optimality Characteristics Without Regulation.** This first section examines the effects of resistance under a no-regulation scenario by detailing the total and distributional economic impacts of susceptibility depletion when producers have a hypothetically full complement of chemical controls available for use. As a result, this section develops a baseline against which the economic impacts of regulatory withdrawals are compared.

The complete control technology set was defined to contain six insecticides[49] from three major chemical classes; organophosphates, carbamates, and synthetic pyrethroids. Being the least expensive and most widely used control alternatives in the study system, organophosphates composed the bulk of the initial set and included azinphosmethyl (Guthion), chlorpyrifos (Lorsban), phosmet (Imidan), and microencapsulated methyl parathion (PennCap-M). The carbamates were represented by methomyl (Lannate), while esfenvalerate (Asana) was designated as the synthetic pyrethroid. All six of the insecticides are currently registered for use in the mid-Atlantic apple production system. The results generated with this no-regulation, initial control set will subsequently be referred to as the base-case.

Using the initial control technology set, the empirical model was optimized twice for each planning horizon and discount rate combination; once with no potential for resistance development, and once with the potential for resistance development. The difference between these two optimizations isolated and described the effects of resistance on the production system. In general, susceptibility depletion was shown to cause only minor losses in total economic surplus for planning horizons of less than 10 years. Resistance-induced surplus losses accelerated as longer planning horizons were employed, although the maximum surplus losses were less than 10 percent of the estimated total economic surplus obtainable from a system without the potential for resistance. As expected, discounting tended to reduce the overall impact of resistance, with the dampening effect increasing as the discount rate increased.

2. **Regulatory Impacts.** Given the base-case developed in the previous section, the second section describes the additional economic impacts generated by selectively withdrawing insecticides from the initial technology set. It is in this section that most of Chapter I's hypotheses are examined, and an effort made to determine which insecticides are most

[49] For ease of exposition, the term insecticide is used in this chapter to refer to any chemical in the initial technology set, including those that primarily have acaricidal properties.

important to the long-run performance of the production system. Using an initial control technology set that contains fewer than the full compliment of insecticides, the empirical model was again optimized twice for each planning horizon and discount rate combination; once with no potential for resistance development, and once with the potential for resistance development. The difference between these two optimizations isolated and described the effects of resistance on the production system when there were less than the full compliment of chemical controls available to the producer. In general, the withdrawal of any chemical from the initial technology set resulted in larger resistance-induced surplus losses compared to the base-case, with the effects being especially pronounced for shorter planning horizons. However, total insecticide use either increased or decreased, depending on the specific chemical withdrawn.

3. **Comparative Analysis.** In this final section, some of the dynamically-optimal results presented in the first two sections are compared to the results generated when producers are assumed to employ statically optimal (myopic) decision-making or when they use currently recommended control schedules and strategies. Although the dynamically-optimal control paths would be expected to out-perform any other control strategy, the objective of this section is to examine whether the difference in performance is large enough to warrant efforts to include the dynamic information into the current regulatory process. Consideration is also given to comprehensive versus chemical-by-chemical assessments of entire control technology sets.

Overall, both myopic and currently recommended control strategies were severely sub-optimal relative to the dynamically optimal controls, with total economic surplus losses increasing more than 10-fold for some planning horizons. However, the myopic and currently recommended control strategies did tend to be superior in terms of reduced total insecticide use, especially for shorter planning horizons. In terms of regulatory benefit assessments, these results indicate the need to incorporate dynamic analyses into the regulatory process in order to fully account for the benefits associated with continued

registration of a chemical. But, in terms of regulatory risk analysis,[50] it is also clear that the use of dynamic analyses requires an accounting for any additional costs, in terms of human health and environmental contamination, that may arise due to increased insecticide use. Comprehensive evaluation of technology sets was demonstrated to be preferred to chemical-by-chemical withdrawals, especially when the planning horizon exceeded 5 years.

Each of the above sections is organized into two more or less distinct parts. First, the main results generated by the model are presented in tabular and/or graphical form, with the text identifying the important system features described by the information. Although somewhat lengthy, this first part is necessary to form a basis for discussion and explanation of the results, a task which constitutes the second part of each section. A synthesis of all three sections, along with some policy and research recommendations, are reserved for Chapter X.

Before proceeding, note that the empirical model employed in this study was parameterized to evaluate a specific production system. Given that the biological simulator reflects the mid-Atlantic pest complex, global inferences about the nature of the resistance/regulation nexus need to be cautiously drawn. However, the results discussed in this chapter should be at least broadly applicable to the evaluation of regulatory strategies in other perennial crop systems, and can be used to indicate the potential impacts that might arise in production systems containing similar pest complexes.

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[50] Within the federal regulatory process, the term risk analysis denotes the analysis of an insecticide's use in terms of potential costs to human health and environmental contamination.

Optimality Characteristics Without Regulation

Given a complete control technology set and no regulatory effort, the empirical model was optimized over a number of different planning horizons and social discount rates in order to generate the time-paths of insecticide use that would minimize the economic surplus lost due to resistance. As discussed in Chapter VIII, a range of discount rates were used in order to examine the impact of this variable on potential decision-making. A maximum planning horizon of 25 years was employed, and it represented the expected product life-cycle length, based on historical evidence, for a particular insecticide chemistry. Intermediate horizons were then based on even intervals that would likely be used by federal regulators.

Changes in Economic Surplus

The total changes in economic surplus, for 42 combinations of planning horizon and discount rate, are detailed in Table 7. Because the impact of discounting is typically experienced only in multi-year calculations, no systematic variation in economic surplus changes was observed for a one-year planning horizon. However, non-systematic variation did occur, with total surplus changes for a one-year horizon varying from -$8,000 to -$11,000. For the most part, this variation was a manifestation of the tolerance levels used to determine the stopping criterion for the optimization process. Thus, values presented in Table 7, and all subsequent economic surplus tables, are best described as being optimal to the nearest ten-thousand dollars.

Relative to the longer planning horizons, the impact of resistance is modest across all discount rates for planning periods of 5 and 10 years (Table 7). For horizons of 15 years or more, economic surplus losses build rapidly as susceptibility depletion accelerates, with the impacts of resistance being distinctly more pronounced at lower discount rates. For example, the present value surplus losses for a 5 year planning horizon varied by discount rate over a

Table 7. Total Economic Surplus Changes Due to Resistance: By Planning Horizon and Discount Rate for the Unregulated Base Case.

Discount Rate	Planning Horizon (Years)					
	1	5	10	15	20	25
	Million Dollars (Present Value)					
0.00	-0.011	-1.379	-14.509	-469.732	-5,360.519	-7,968.838
0.02	-0.009	-1.090	-11.445	-301.969	-3,468.722	-3,748.405
0.04	-0.011	-1.092	-9.633	-227.009	-2,639.510	-2,868.559
0.06	-0.011	-1.090	-8.849	-146.997	-1,029.858	-1,385.430
0.08	-0.009	-1.003	-7.921	-99.382	-787.611	-909.811
0.10	-0.008	-0.956	-6.854	-31.644	-602.639	-706.134
0.12	-0.009	-0.876	-6.409	-14.691	-499.871	-604.063

relatively narrow range, from -$0.876 million at a 12 percent discount rate to -$1.379 million at a 0 percent discount rate. A similar situation arose for the 10 year horizon, where the range of economic surplus losses varied from -$6.409 million for a 12 percent discount rate to -$14.509 million for a 0 percent discount rate. But even a change of -$14.509 million was relatively insignificant, amounting to less than a 0.05 percent decrease in the estimated total economic surplus that would have been generated by a resistance-free apple production system in the U.S.[51]

Substantial losses from resistance began to emerge when using a 15 year planning horizon, and they accelerated rapidly for planning horizons between 15 and 20 years (Table 7). In addition, 15 and 20 year planning horizons also experienced significant variation in the impact of different discount rates. Twenty-five year planning horizons, which captured the obsolescence of the control technology set, essentially complete susceptibility depletion, and therefore most of the impacts of resistance, had the highest levels of economic surplus losses at all discount rates. Total present value economic surplus losses for a 25 year horizon at a 0 percent discount rate amounted to over -$7.9 billion, or an 8.9 percent loss in the total surplus that could have been generated by the system if the potential for resistance was not present. Evaluated at a 12 percent discount rate, the surplus losses over a 25 year horizon exceeded -$604 million, or 2.2 percent lower than the total surplus possible without resistance.

As might be expected, the components of total economic surplus change, namely variation in producer and consumer surplus, both followed the same pattern of increasing present value losses as planning horizons lengthened and discount rates decreased. Producer surplus losses ranged from a low of -$7,000, or $0.08 per acre,[52] for a 1 year planning horizon at a number of different discount rates, to a present value high of -$6.7 billion, or -$3,080 per acre

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[51] Assuming steady-state at the initial conditions used to parameterize the model, and an absence of resistance, the estimated total economic surplus generated by the U.S. apple production system was $3.6 billion per year.

[52] Because the pest complex being modelled only occurs in the mid-Atlantic system, production related impacts are confined to the producers in this region. Thus, all per acre calculations in this chapter are based on the approximately 87,000 producing acres in the study region.

per year,[53] for a 25 year planning horizon with a 0 percent discount rate (Table 8). Consumer surplus losses ranged from a low of -$2,000 for a 1 year horizon to a high of -$1.27 billion for a 25 year planning horizon with a 0 percent discount rate (Table 9). As with total economic surplus losses, both producer and consumer surplus losses grew rapidly for planning horizons exceeding 10 years. In addition, the majority of the impact of resistance was borne by producers, who accounted for 78 to 84 percent of the total economic surplus losses, depending on the planning horizon and discount rate combination.

The results presented in Table 7 through Table 9 demonstrate the impact of resistance on the final amount of surplus that can be generated by the U.S. apple production system. In terms of planning, the specific surplus-loss time-paths that arise from different horizons and discount rates are also of interest. Cumulative surplus-loss time-paths varied little by discount rate for short horizons, but diverged by discount rate as horizons expanded (Figure 22). For example, the time-paths for present value surplus losses over a 5 year planning horizon are nearly identical during the first 2 years regardless of the discount rate. Between years 2 and 5, however, the effects of varying discount rates begin to emerge, with significant time-path divergence arising as the planning horizon is expanded to 15 years (Figure 23). By year 10 of the 15 year horizon, susceptibility depletion became severe enough to seriously hamper the effectiveness of the initial control technology set, leading to the acceleration of yearly surplus losses. As might be expected, the present value of the losses decreased as the discount rate increased.

Extending the planning horizon to 25 years resulted in time-paths of cumulative surplus loss that exhibited a couple of new features. Rapid increases in cumulative losses still developed after year 10, but these losses stabilized for a period of time around year 15, with the period of stability being longer for higher discount rates (Figure 24). Cumulative surplus losses renewed after the period of stability, and eventually leveled out as year 25 approached.

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[53] Per acre net revenue to a producer can be as high as $4,000, depending on the percent of production allocated to the fresh market, the fresh market price, and the level of pest pressure in a given season. Thus, the magnitude of the calculated losses associated with susceptibility depletion were within reasonable bounds.

Table 8. Producer Surplus Changes Due to Resistance: By Planning Horizon and Discount Rate for the Unregulated Base Case.

Discount Rate	Planning Horizon (Years)					
	1	5	10	15	20	25
	Million Dollars (Present Value)					
0.00	-0.009	-1.155	-12.147	-393.503	-4,516.237	-6,700.371
0.02	-0.007	-0.913	-9.582	-252.965	-2,922.398	-3,151.740
0.04	-0.009	-0.918	-8.028	-190.631	-2,208.188	-2,419.056
0.06	-0.009	-0.913	-7.407	-123.164	-865.141	-1,164.500
0.08	-0.007	-0.842	-6.662	-83.531	-665.295	-761.421
0.10	-0.007	-0.804	-5.761	-26.282	-501.637	-591.670
0.12	-0.008	-0.734	-5.364	-12.451	-421.016	-507.772

Table 9. Consumer Surplus Changes Due to Resistance: By Planning Horizon and Discount Rate for the Unregulated Base Case.

Discount Rate	Planning Horizon (Years)					
	1	5	10	15	20	25
	Million Dollars (Present Value)					
0.00	-0.002	-0.224	-2.362	-76.229	-844.282	-1,268.467
0.02	-0.002	-0.176	-1.863	-49.001	-546.324	-596.660
0.04	-0.002	-0.174	-1.605	-36.378	-431.322	-449.503
0.06	-0.002	-0.176	-1.443	-23.831	-164.714	-220.924
0.08	-0.002	-0.161	-1.259	-15.851	-122.316	-148.390
0.10	-0.001	-0.152	-1.093	-5.362	-101.002	-114.464
0.12	-0.001	-0.142	-1.045	-2.237	-78.855	-96.292

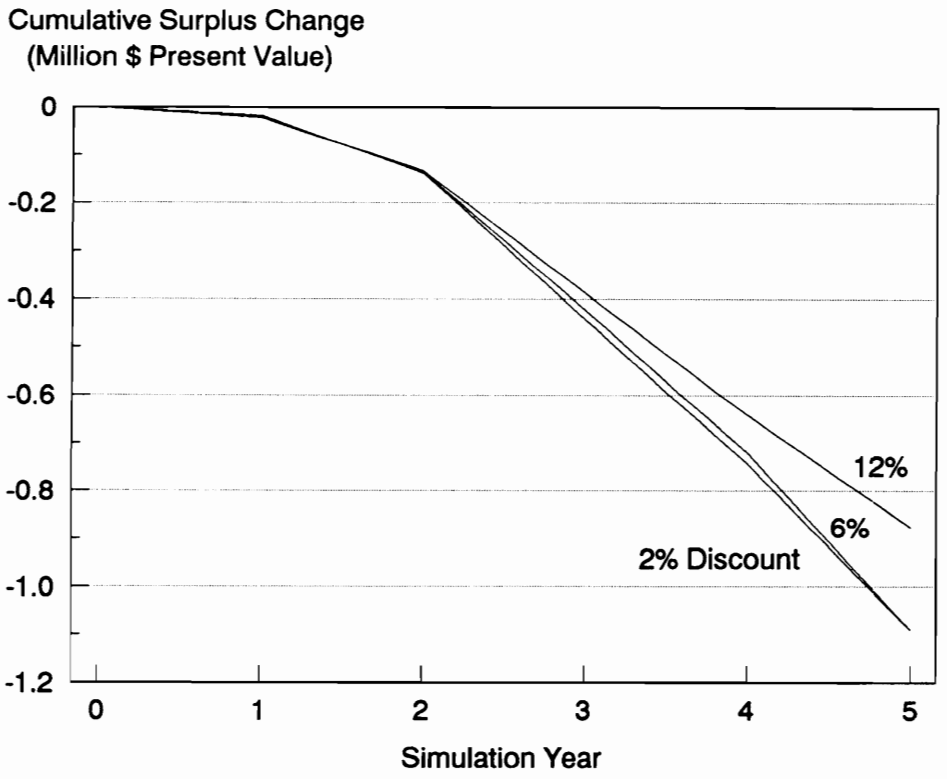

Figure 22. Optimal Cumulative Economic Surplus Changes for the Unregulated Base Case: By Discount Rate for a 5 Year Planning Horizon.

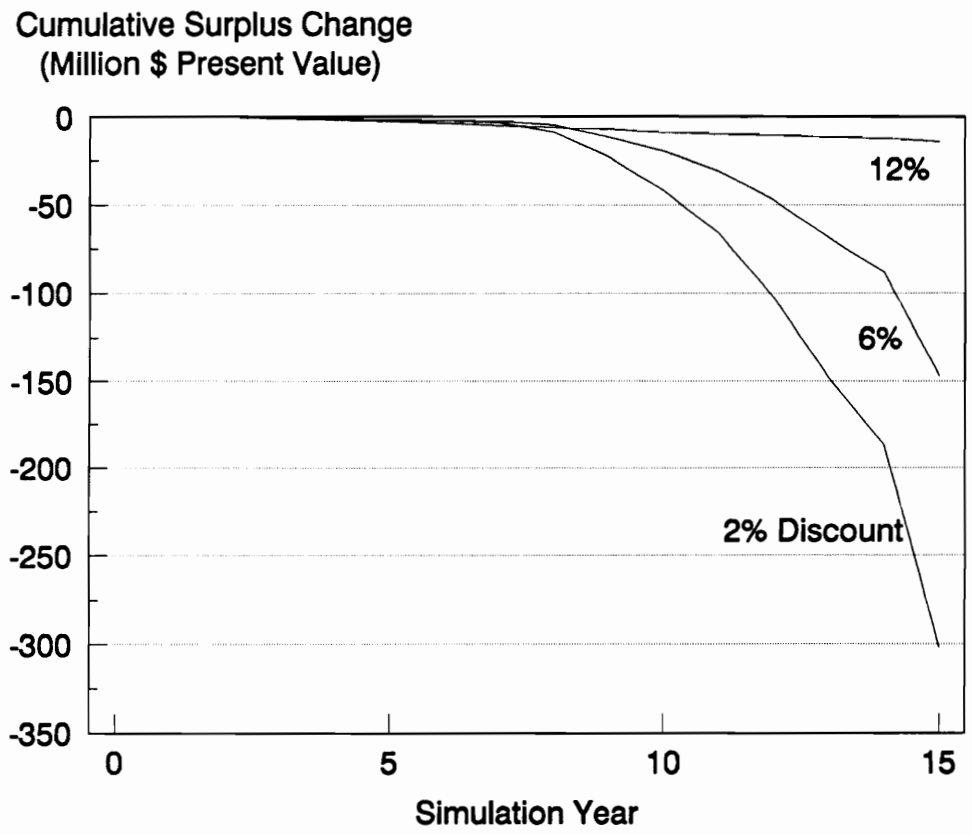

Figure 23. Optimal Cumulative Economic Surplus Changes for the Unregulated Base Case: By Discount Rate for a 15 Year Planning Horizon.

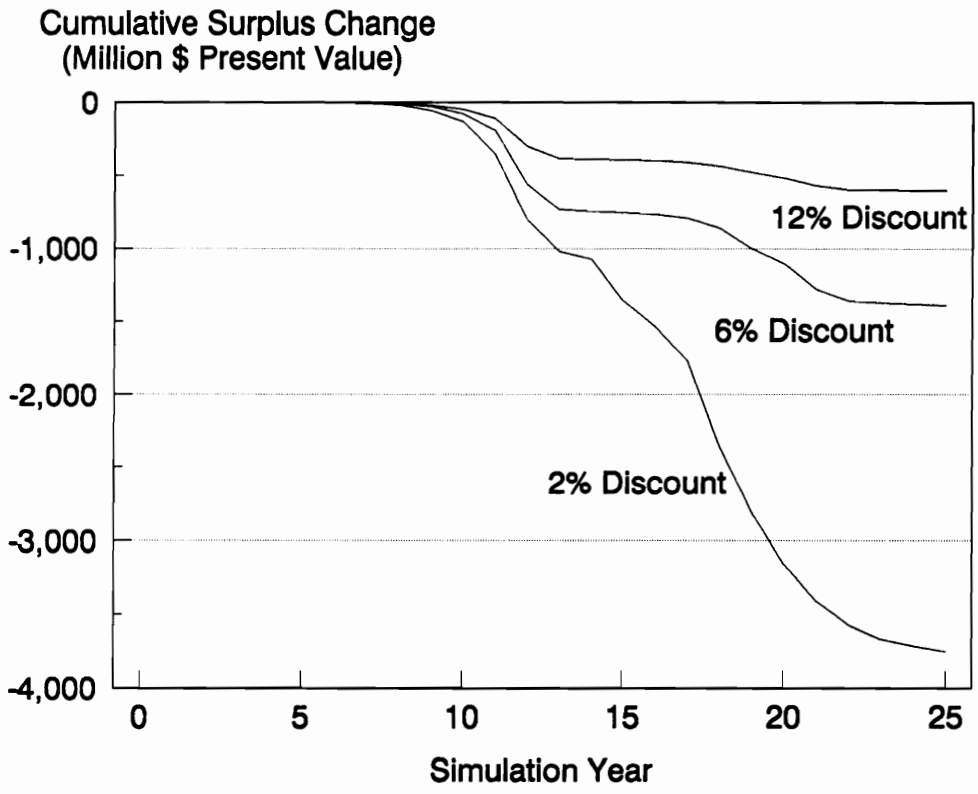

Figure 24. Optimal Cumulative Economic Surplus Changes for the Unregulated Base Case: By Discount Rate for a 25 Year Planning Horizon.

This final period of relative stability coincided with near complete susceptibility depletion, extensive crop damage, and heavy discounting. Cumulative present-value surplus losses continued to rise only for a 0 percent discount rate (graph not shown), and it did so at a yearly increment that represented the maximum possible crop loss. In fact, optimization with longer planning horizons resulted in very little change to the time-paths generated for a 25 year horizon, at least for the range of discount rates used in this study. Also notice that, as the planning horizon increased, cumulative surplus losses in any given year also increased. Using a discount rate of 2 percent as an example, surplus losses at year 15 of a 15 year planning horizon amounted to nearly -$301 million (Figure 23, Table 7). But, when the horizon was extended to 25 years, cumulative surplus losses at year 15 increased to over -$1.4 billion (Figure 24).

The temporal shifts in cumulative surplus losses that arise from changes in planning horizons can be best illustrated by focusing on the early years of a simulation. Given a 2 percent discount rate, the time-paths of surplus losses for different horizons began to separate almost immediately, and by year 5 had established the pattern of increasing divergence that would continue throughout the planning period (Figure 25). A similar situation was observed when using a 12 percent discount rate, but the magnitude and acceleration of the divergence was considerably higher (Figure 26). Overall, the cumulative surplus losses generated at any given year increased as the planning horizon increased, even for the earliest years of a simulation, with the separation between the 5 year trajectory and other planning horizons expanding as the discount rate increased.

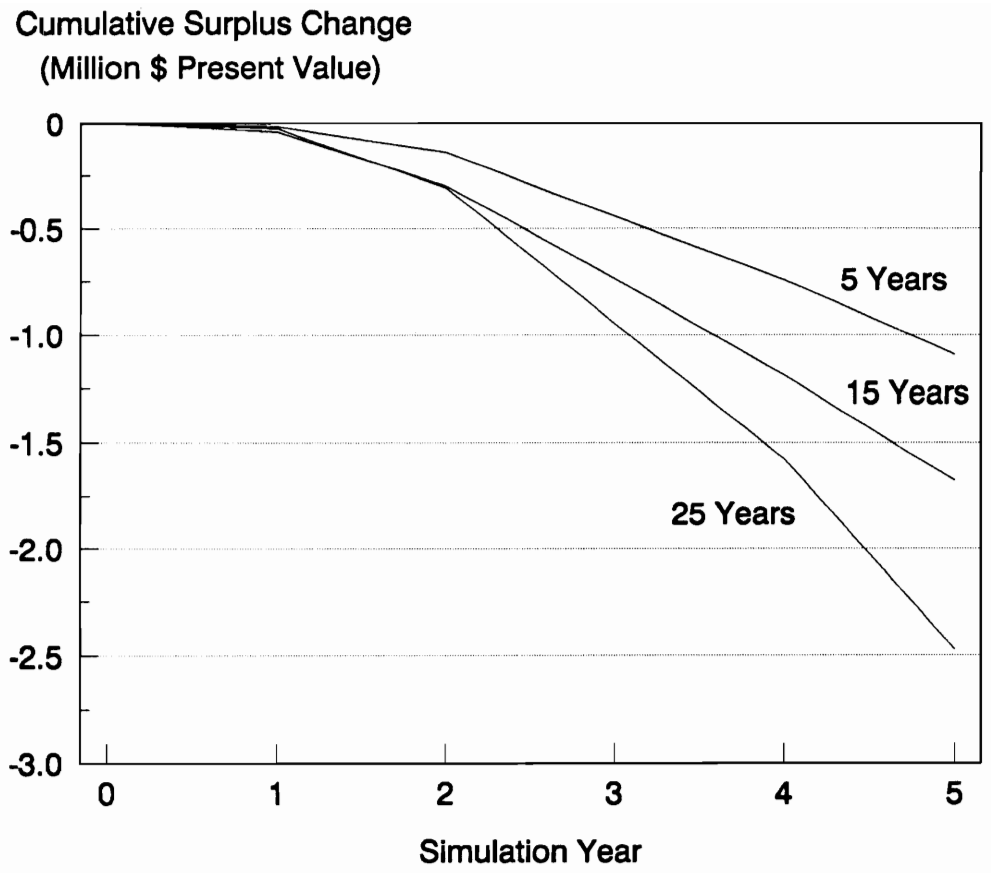

Figure 25. Optimal Cumulative Economic Surplus Changes for the Unregulated Base Case: By Planning Horizon for a 2 Percent Discount.

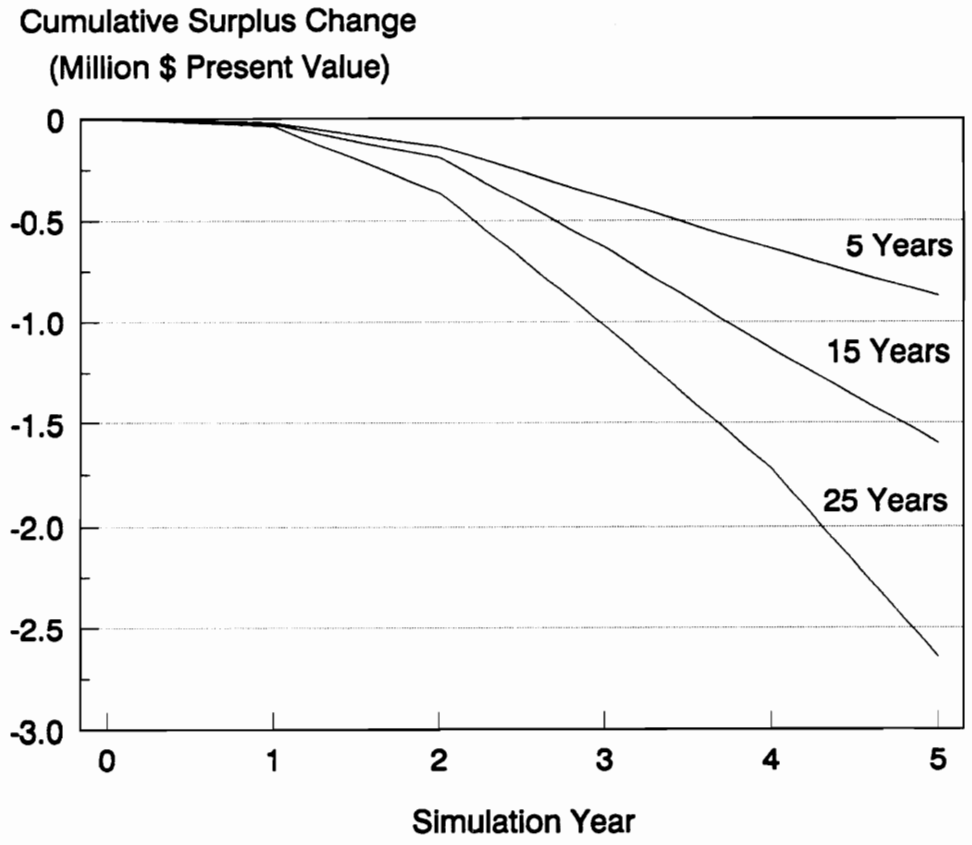

Figure 26. Optimal Cumulative Economic Surplus Changes for the Unregulated Base Case: By Planning Horizon for a 12 Percent Discount.

Changes in Insecticide Use

While the optimal time-paths of cumulative surplus losses are of direct interest in examining the impacts of resistance, the time-paths of cumulative insecticide use also have an important role in analyzing the dynamic impacts of susceptibility depletion. The optimal cumulative insecticide use, in terms of total pounds of active ingredients (a.i.) applied in the mid-Atlantic region, are detailed for 42 combinations of planning horizon and discount rate in Table 10. Unlike the pattern observed with respect to cumulative surplus changes, discount rates did not have a uniform effect on the total use of insecticides. For planning horizons of 5 and 20 years, total insecticide use decreased as the discount rate increased. For a 10 year planning horizon, total insecticide use increased as the discount rate increased. In general, however, the magnitude of these trends were relatively small, with the maximum change amounting to an increase of 2.8 pounds a.i. per acre per year between 0 and 12 percent discounts in the 10 year planning horizon.

The organophosphate Imidan was the most intensely used chemical in all of the no-regulation simulations, accounting for up to 32 percent of total insecticide use. The other organophosphates were also heavily represented in the optimal solution, but their relative importance varied somewhat by planning horizon. For example, Guthion became less important, and Lorsban more important, as the length of the planning horizon increased. On the other hand, the use of PennCap-M held fairly steady at about 14 percent of total insecticide use. The carbamate Lannate was the most extensively used non-organophosphate, with its levels of application approaching those attained by some of the organophosphates. In general, the synthetic pyrethroid Asana was lightly used relative to all other chemicals, with applications primarily occurring during longer planning horizons.

Although discount rate did not significantly effect the total use of insecticides, the length of the planning horizon contributed directly to cumulative insecticide use and the time-paths of that use. Insecticide use increased from a low of 632,900 pounds a.i., or 7.3 pounds a.i. per acre, for a 1 year horizon with a 2 percent discount rate, to a high of 25.4 million pounds a.i.,

Table 10. Total Use of Insecticides By Planning Horizon and Discount Rate for the Unregulated Base Case.

Discount Rate	Planning Horizon (Years)					
	1	5	10	15	20	25
	1,000 Pounds A.I.					
0.00	632.9	4,605.8	7,051.9	11,986.8	18,536.5	25,399.2
0.02	781.0	4,783.4	8,036.1	11,591.4	18,033.1	25,062.7
0.04	907.2	4,779.6	8,359.3	11,554.3	18,005.7	25,211.8
0.06	907.2	4,783.4	8,517.7	11,481.6	17,859.3	25,366.2
0.08	802.8	4,695.8	8,868.1	11,399.7	17,762.9	25,398.6
0.10	777.3	4,631.0	9,213.9	11,286.9	17,433.5	25,401.3
0.12	795.3	4,565.6	9,485.8	11,128.6	16,957.4	25,366.2

or 11.7 pounds a.i. per acre per year, for a 25 year horizon with a 10 percent discount rate (Table 10). At all discount rates, the largest increase in insecticide use occurred as the planning horizon expanded from 20 to 25 years. Similar to the time-paths for cumulative surplus change, the trajectories of insecticide use tended to diverge by planning horizon early in the simulations, with the extent of the divergence increasing slightly as the discount rate increased. Cumulative use to any given year increased as the length of the planning horizon decreased, with the pattern of separation being fairly constant across discount rates (Figure 27, Figure 28, Figure 29). In general, trajectory characteristics that were established by year 5 remained stable for the duration of the longer planning horizons.

Discussion

The information generated by base-case, no-regulation simulations highlight some important features concerning the reaction of the production system to susceptibility depletion. As might be expected given its evolutionary nature, pest resistance became an increasingly important economic problem as the decision-making time horizon lengthened (Table 7). For most of the optimal solutions, the effects of resistance were minimal for 1, 5, and 10 year planning horizons. As a result, optimal insecticide use within this range of horizons was primarily based on the need to reduce current-year surplus losses by suppressing current-year pest populations. The effects of insecticide use on current and future susceptibility depletion was not an important factor in determining the optimal solution. The choice of discount rate did have some effect on the magnitude of surplus losses in the shorter time horizons (Figure 22). However, the maximum impact, which occurred for a 10 year horizon with a 0 percent discount rate, amounted to only a 0.05 percent reduction in the estimated total surplus generated by the system. Thus, it is not surprising that resistance has not been incorporated into private or public decision-making during the early years of an insecticide's use, especially given short planning horizons and high discount rates.

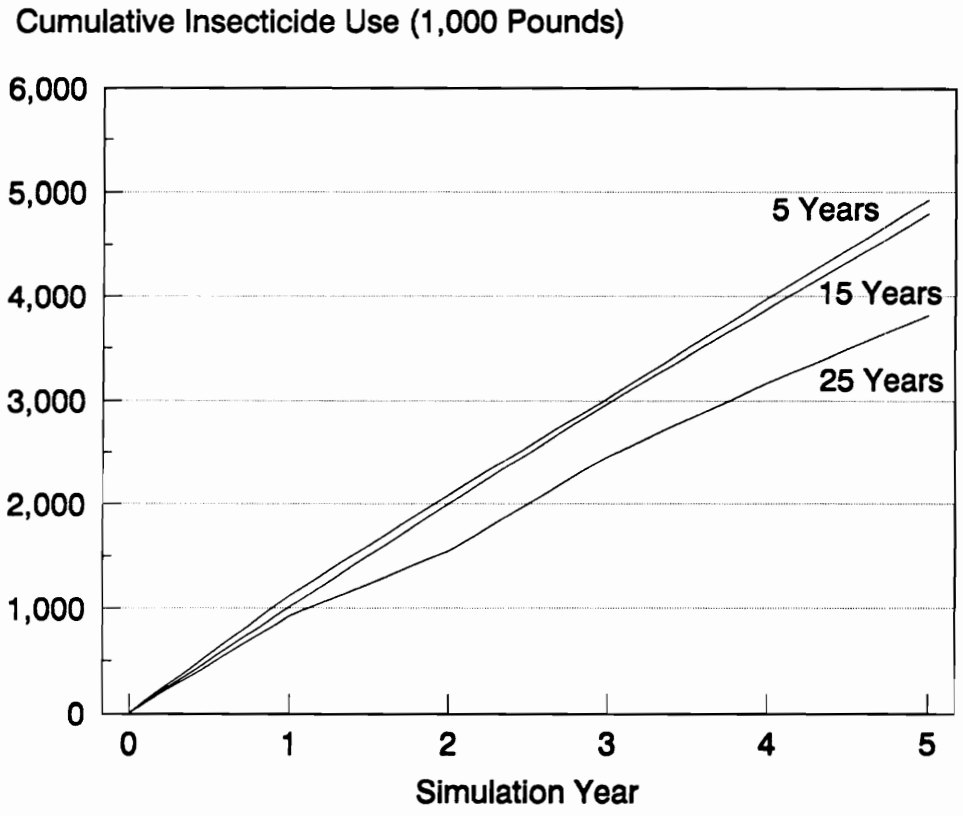

Figure 27. Optimal Cumulative Insecticide Use for the Unregulated Base Case: By Planning Horizon for a 2 Percent Discount Rate.

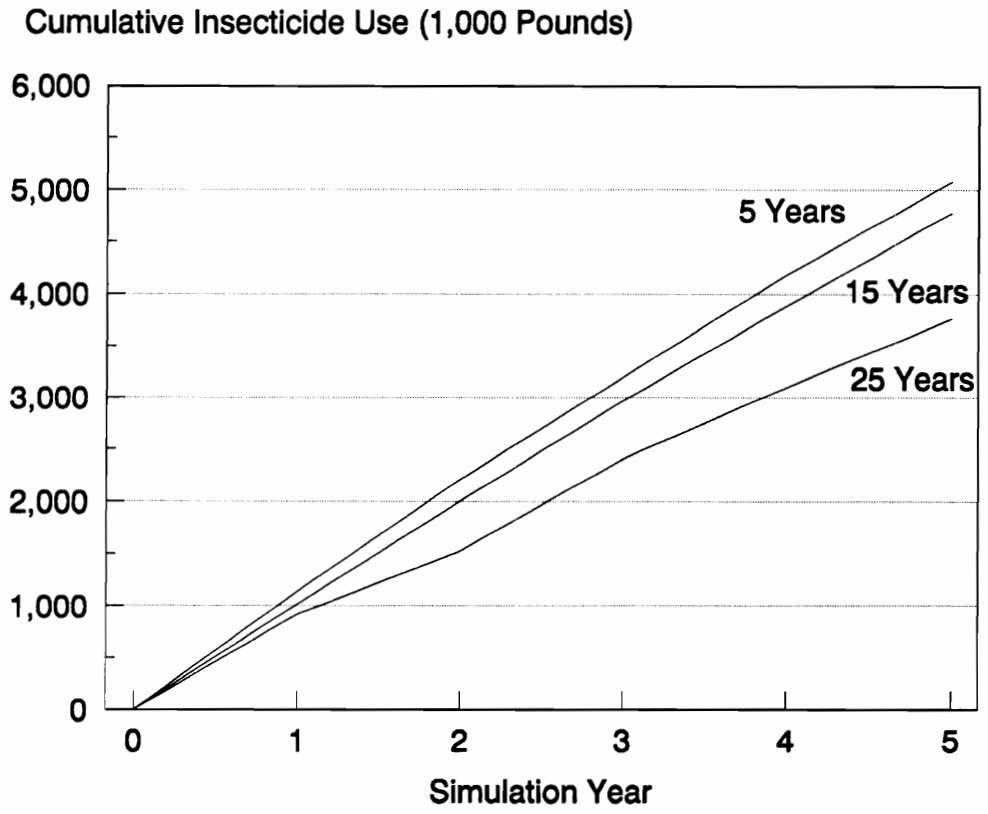

Figure 28. Optimal Cumulative Insecticide Use for the Unregulated Base Case: By Planning Horizon for a 6 Percent Discount Rate.

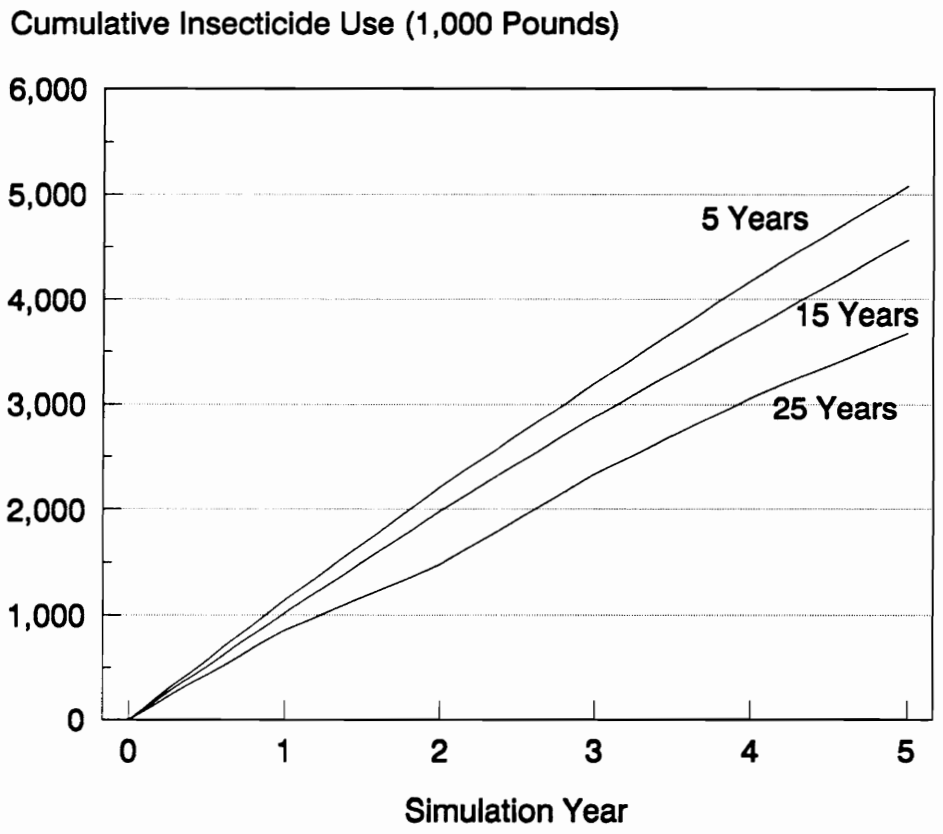

Figure 29. Optimal Cumulative Insecticide Use for the 6 Chemical Base Case: By Planning Horizon for a 12 Percent Discount Rate.

By the time susceptibility depletion enters the final phase of its critical second-stage,[54] its effect on the economic surplus generated by a production system accelerates rapidly. For example, expanding the planning horizon from 10 to 15 years allowed the dynamic analysis to incorporate the end of stage 2 and the beginning of stage 3 resistance. Even when discounted by a median rate of 6 percent, the 50 percent increase in the planning horizon led to a more than 15-fold increase in economic surplus losses (Table 7). Meanwhile, a change from a 10 to 20 year horizon, or a 100 percent increase in the planning period, resulted in a more than 115-fold increase in economic surplus losses. At its extreme, and using a 0 percent discount rate, losses to the estimated total economic surplus generated by the apple production system rapidly rose from less than 0.05 percent for a 10 year planning horizon, to nearly 9 percent for a 25 year planning horizon. Even with heavy discounting at 12 percent, surplus losses exceeded 2.2 percent in the long-run. So although the general effect of discounting over long time horizons is to reduce the importance of surplus flows in later years, this study demonstrated that susceptibility depletion, and the pest control failures it implies, can be damaging enough so as to overcome discounting's current-period biases. This suggests that decision-makers should be concerned about the long-run impacts of their regulatory actions on resistance development, particularly if the real social discount rate is low. These results also indicate that the choice of planning horizon and discount rate will largely determine the perceived importance of susceptibility depletion.

Incorporating extended planning horizons into the federal regulatory process involves some trade-offs in terms of the temporal flow of economic surplus. When planning horizons are short, and especially when they terminate before resistant genes can spread, optimal insecticide use in based solely on the need to reduce contemporaneous pest populations and economic surplus losses. But, because resistance leads to substantial future production damage through the progressive inability to control pests, optimal solutions for horizons that are long enough to incorporate susceptibility depletion tended to have reduced reduced

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[54] A discussion covering the stages of susceptibility depletion, or resistance development, can be found in Chapter II, beginning on p. 23.

insecticide use early in the planning period. By reducing insecticide use, the rate of susceptibility depletion was lowered, thereby enabling the production system to generate surplus flows for a longer period of time. However, decreased insecticide use also tended to result in higher pest populations, thereby elevating the economic surplus losses that were experienced early in a horizon (Figure 25). The trade-off consists of balancing the need to extend the economic life of the production system while at the same time limiting the surplus losses that must shifted closer to the beginning of the planning horizon. As might be expected, this trade-off not only depends on the length of the chosen planning period, but also on the discount rate chosen for the evaluation. As the discount rate increases, the importance of surplus losses in later years, and thus susceptibility depletion, decreases, thereby reducing the attractiveness of postponing the use of insecticides.

Another important result generated by the base-case simulations concerns the temporal changes in total insecticide use. Although not directly the object of optimization, insecticide use was the proximate force causing susceptibility depletion and economic surplus losses. But, as was discussed in Chapter III, the common-property characteristics of susceptibility and the dynamics of resistance development make it impossible for producers to incorporate potential changes in the effectiveness of the control technology set into current insecticide-use decision-making. As a result, the total use of insecticides and susceptibility depletion would be expected to simultaneously increase under static optimization as producers attempted to maintain the levels of crop output obtainable without resistance. On the other hand, a dynamically optimal solution might be expected to reduce the insecticide use response to susceptibility depletion in favor of various chemical mixture or switching strategies.[55] If this were the case, then the resistance problem might best be controlled by active, multiple-period management at an aggregate level, perhaps through the regulation of both the specific chemicals in the control technology set and times at which they could be applied.

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[55] For a review of the various pest control strategies in the presence of resistance, see Chapter II, p. 27.

Although the dynamically-optimal time-paths of insecticide use were expected to be unlike those that might arise if producers alone made the application decisions, this proved not to be the case. Results generated by the bioeconomic model actually contained some of the theoretical aspects of both static and dynamic solutions. For all planning horizons less than 10 years, a high-dose strategy was optimal, thereby suggesting that resistance was not an important factor and that a static optimization might be sufficient. For planning horizons of 15 years or longer, optimal insecticide use involved not only high- doses, but also complex combinations of insecticide mixtures and switching. While the dynamic optimality of mixtures and switching was predictable, the persistance of high-dose strategies in the optimal solutions for extended planning horizons was unexpected, even though it has been vigorously debated as one method to combat resistance in some pest complexes (see Chapter II). Its inclusion in the optimal solutions for the apple production system may have been possible because of the reversion rates implied by the specific parameterization of the biological model. Thus, if the parameterization was sufficiently precise, then the results lend support to the potential viability of high-dose strategies as a resistance management tool.

A final result generated by the no-regulation scenarios concerns the distribution of surplus changes. As with other studies that have included changes in the biological operation of a production system, the majority of the economic surplus losses were found to accrue to producers, even though these losses were managed in a dynamically optimal manner. The distributional concentration of surplus losses can be at least partially attributed to the structure of the U.S. apple market, and thus is outside the direct control of both producers and regulators. However, calculated distributional effects are also largely determined by the various assumption that form the market model and its hypothesized changes due to resistance development. Theoretically, the parallel supply curve shifts employed in this study tend to emphasize changes in producer surplus. As long as supply shifts actually are parallel, then the reported results should be good estimates of the expected surplus changes. But if the supply shifts are pivotal, then the reported results may overestimate total surplus losses, and

producer surplus changes, by as much as 100 percent.[56] Because there is no practical way to determine the expected nature of a supply shift, the reported results can be considered as an upper bound on producer surplus changes and a lower bound on consumer surplus changes. Nonetheless, the skewness of the distributional impacts suggest that it would be to a producer's advantage to support the optimal, long-run management of resistance, even if this required some increases in the surplus losses experienced early in a planning horizon.

Regulatory Impacts

Using the results for the no-regulation simulations as a benchmark, the analysis now focuses on the impacts of insecticide regulatory actions on the performance of the apple production system. For the purposes of this section, potential withdrawals are investigated on a chemical-by-chemical basis. The effects of the withdrawals are expressed in terms of changes from the levels of surplus loss and insecticide use observed under the base-case scenarios. This section concludes with a discussion that examines the relative importance of each insecticide to the dynamic viability of the apple system.

Optimal Results Under Withdrawal Scenarios

The additional economic surplus change following the removal of each chemical from the initial control technology set was calculated for 42 combinations of planning horizon and discount rate. In general, withdrawal of a chemical led to multi-fold increases in amount of total economic surplus lost due to resistance, especially for planning horizons up to 15 years.

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[56] This arises directly from the geometry of the surplus calculation. In essence, a pivotal supply shift would exactly bisect the area presumed lost under a parallel supply shift, thereby resulting in a surplus loss that was 50 percent lower. As a result, surplus changes from parallel supply shifts tend to be twice as large as those from pivotal shifts.

Impacts over longer horizons were somewhat muted, and resulted in a doubling, or less, of the surplus lost to resistance. For example, the withdrawal of Guthion caused present value surplus losses for a 6 percent discount rate to increase -$5.857 million over a 5 year planning horizon, and a -$1.604 billion over a 15 year horizon (Table 11). These changes represented a 537 percent increase in resistance impacts over a 5 year horizon, and a 1,091 percent increase in resistance impacts over a 15 year horizon. Losses over a 25 year horizon at a 6 percent discount rate increased -$1.517 billion, or an additional 109 percent. As with the total surplus losses observed for the base-case scenarios, additional losses due to Guthion withdrawal varied by discount rate, especially for the longer planning horizons. The largest percentage increases in surplus loss emerged over 10 year horizons, while the smallest percentage increases were calculated over 1 and 25 year horizons.

In addition to the aggregate surplus losses caused by the withdrawal of Guthion from the control technology set, the time-path of cumulative surplus changes also differed significantly from those observed under the no-regulation scenarios. Trajectories of surplus losses over a 5 year horizon at a 6 percent discount rate indicated that the effects of Guthion withdrawal strongly emerged after the third year of simulation and continued to the end of the planning period (Figure 30). This pattern of divergence between the cumulative surplus losses experienced under no-regulation and Guthion withdrawal scenarios also occurred over a 15 year planning horizon. However, the acceleration of the divergence began to slow after year 7, even though the gap between the no-regulation and Guthion withdrawal scenarios continued to expand (Figure 31).

Similar to the base-case scenarios, increasing the length of the horizon resulted in an increase in surplus losses in the early years of the planning period (Figure 32). The magnitude of this effect was amplified when Guthion was eliminated from the control technology set, with an expansion of the planning horizon from 5 to 15 years causing a doubling in the cumulative surplus lost by year 5. In addition, the discount rate also affected the time-path of cumulative surplus losses. For example, over a 15 year horizon, lower discount rates led to an increase in cumulative surplus losses early in the planning period (Figure 33). But, while

Table 11. Total Economic Surplus Changes From the Base Case Due to the Withdrawal of Guthion: By Planning Horizon and Discount Rate.

Discount Rate	Planning Horizon (Years)					
	1	5	10	15	20	25
	Million Dollars (Present Value)					
0.00	-0.006	-9.431	-1,364.872	-4,005.991	-3,987.628	-2,709.531
0.02	-0.007	-7.211	-1,055.631	-2,634.862	-2,016.531	-1,249.107
0.04	-0.005	-6.392	-987.622	-2,243.197	-1,982.778	-1,448.722
0.06	-0.005	-5.857	-938.095	-1,604.362	-1,596.312	-1,516.705
0.08	-0.005	-5.368	-861.131	-1,462.183	-1,466.995	-1,473.776
0.10	-0.004	-5.013	-796.683	-1,262.778	-1,200.403	-1,198.001
0.12	-0.006	-4.584	-663.981	-1,003.993	-996.181	-904.612

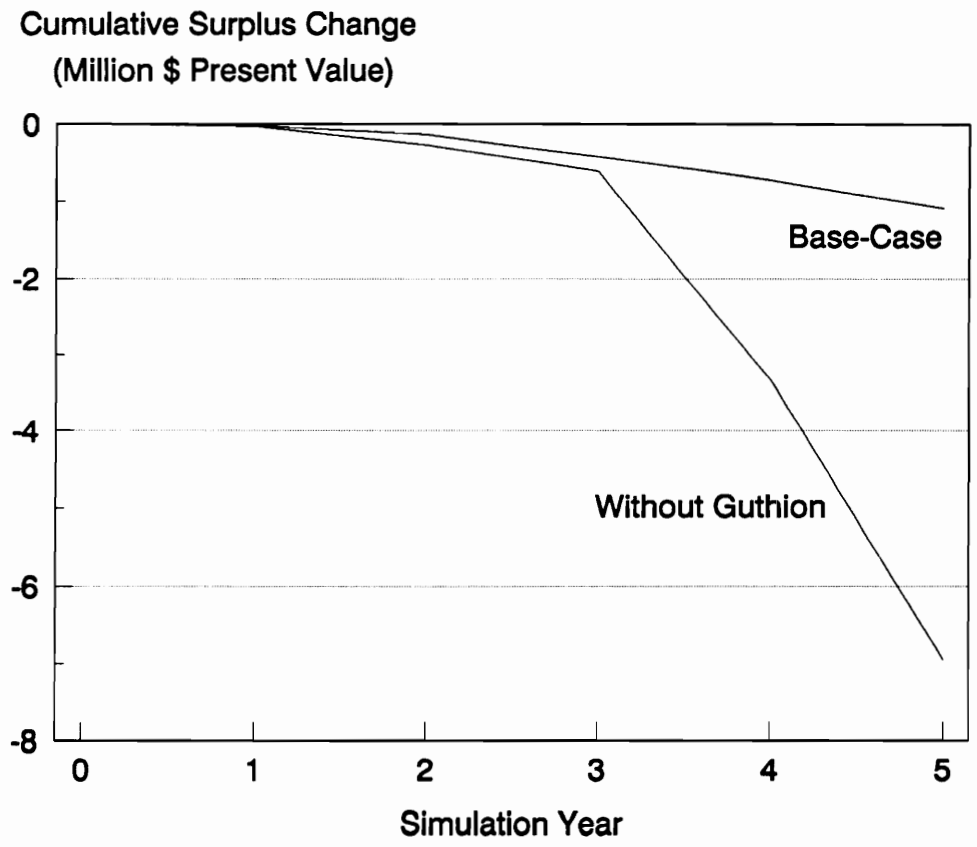

Figure 30. Cumulative Surplus Changes from the Base Case When Guthion is Withdrawn: For a 6 Percent Discount Rate and 5 Year Horizon.

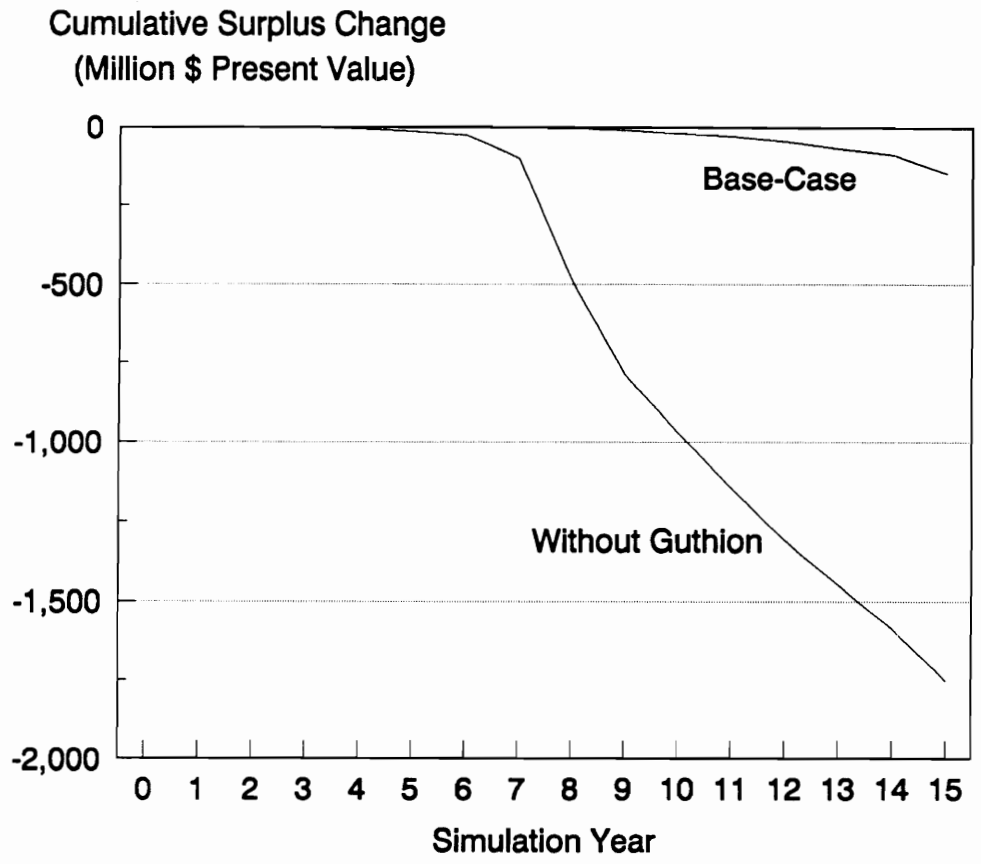

Figure 31. Cumulative Surplus Changes from the Base Case When Guthion is Withdrawn: For a 6 Percent Discount Rate and 15 Year Horizon.

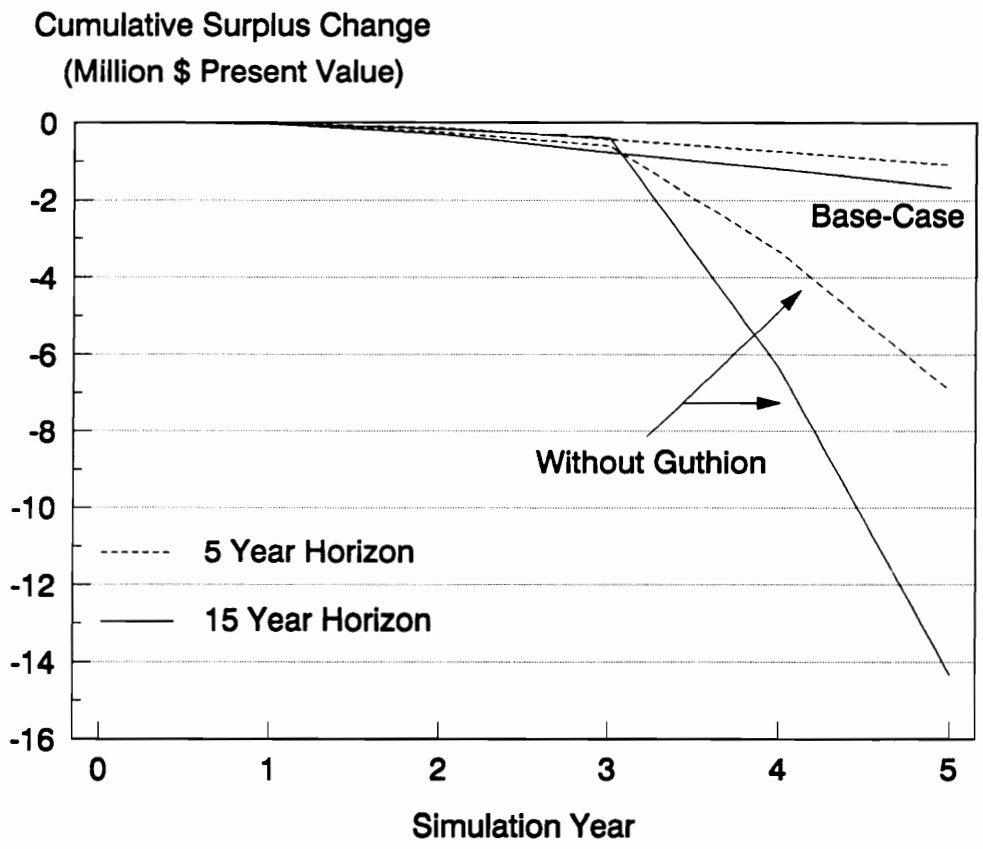

Figure 32. Cumulative Surplus Changes from the Base Case When Guthion is Withdrawn: Comparison of 5 and 15 Year Planning Horizons.

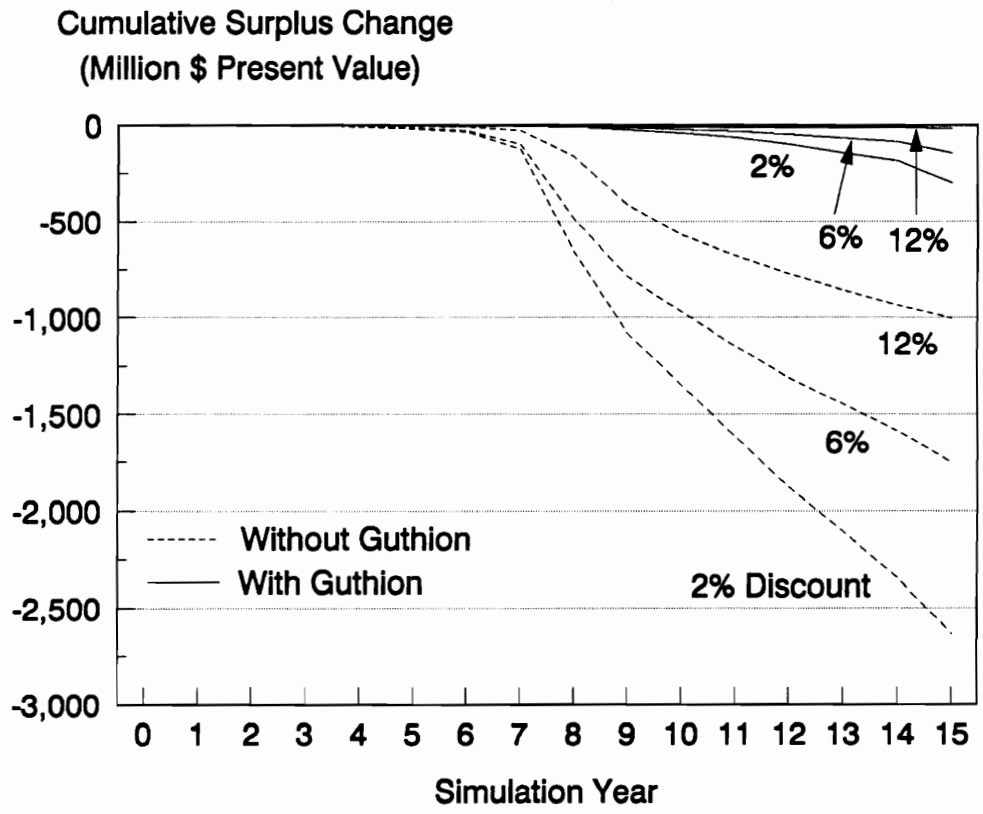

Figure 33. Cumulative Surplus Changes from the Base Case When Guthion is Withdrawn: By Discount Rates for a 15 Year Horizon.

the withdrawal of Guthion coincided with higher surplus losses at all discount rates, the accelerated divergence of these additional losses tended to cease between years 8 and 9. This is in contrast to the 15 year base-case simulation, where cumulative surplus losses continued accelerate throughout the planning period irrespective of the discount rate.

The withdrawal of Guthion from the control technology set not only led to changes in the total economic surplus loss due to resistance, but to an average reduction in total insecticide use over all planning horizons (Table 12). However, Guthion withdrawal did lead to an increase in insecticide use at high discount rates for horizons of 5 to 25 years. For the planning horizon/discount rate combinations at which total chemical use declined, the percentage decrease were in the -10 to -20 percent range, with the effect becoming more pronounced as the horizon expanded and the discount rate decreased. For those instances where total chemical use increased, the effect was most pronounced at longer horizons and high discount rates. The largest increase on a percentage basis occurred with a 15 year planning horizon at a 12 percent discount rate. At this combination, chemical use expanded over 3.3 million pounds a.i., or 2.58 pounds a.i. per acre per year, representing a 30.2 percent increase.

Single withdrawals of the other organophosphate insecticides originally in the base-case control technology set led to optimal results that were broadly similar to those seen for the withdrawal of Guthion. In general, cumulative surplus losses increased and total insecticide use decreased over the levels calculated for the base-case. However, each chemical scenario exhibited some unique features. For example, the withdrawal of Lorsban led to increased surplus losses that were almost uniformly higher than those experienced when Guthion was withdrawn (Table 13). Surplus losses, following Lorsban withdrawal, for a 5 year planning horizon at a 6 percent discount rate were -$6.886 million higher than the base-case, an increase of 632 percent and 17.6 percent higher than the losses from Guthion withdrawal. For a 15 year horizon and a 6 percent discount rate, losses associated with a Lorsban withdrawal were -$1.984 billion, or 1,350 percent above the base-case results and 23.7 percent above levels experienced after Guthion withdrawal. This pattern of higher resistance-induced losses for Lorsban versus Guthion withdrawal was reversed only for a 15 year, 12 percent planning

Table 12. Change in the Total Use of Insecticides Following the Withdrawal of Guthion: By Planning Horizon and Discount Rate.

Discount Rate	Planning Horizon (Years)					
	1	5	10	15	20	25
	----------	----------	1,000 Pounds A.I.	----------	----------	----------
0.00	-106.5	-832.5	-1,605.1	-1,888.2	-4,654.8	-6,487.0
0.02	-94.1	-685.3	-1,200.9	-1,497.3	-3,871.2	-5,188.6
0.04	-186.5	-228.5	-987.6	-1,275.6	-2,261.1	-3,986.1
0.06	-217.8	-106.5	-716.2	-1,387.5	-1,995.4	-2,439.6
0.08	-176.3	-53.2	-288.1	-643.1	-1,007.9	-2,315.7
0.10	-118.5	6.4	634.4	1,085.4	1,056.2	1,683.5
0.12	-68.1	111.9	1,055.9	3,361.6	2,987.7	2,088.9

Table 13. Total Economic Surplus Changes From the Base Case Due to the Withdrawal of Lorsban: By Planning Horizon and Discount Rate.

Discount Rate	Planning Horizon (Years)					
	1	5	10	15	20	25
	Million Dollars (Present Value)					
0.00	-0.010	-8.937	-2,105.811	-4,869.123	-4,505.014	-4,173.910
0.02	-0.011	-8.428	-1,788.913	-2,738.581	-2,548.312	-2,005.538
0.04	-0.010	-7.531	-1,621.682	-2,279.512	-2,469.881	-1,906.981
0.06	-0.009	-6.886	-1,568.273	-1,984.186	-2,009.773	-1,884.482
0.08	-0.011	-5.997	-1,435.531	-1,666.091	-1,768.938	-1,699.300
0.10	-0.011	-5.486	-862.581	-1,287.620	-1,322.901	-1,466.988
0.12	-0.012	-5.327	-411.962	-987.699	-1,119.622	-1,137.707

horizon/discount rate combination. The time-paths of cumulative surplus losses for Lorsban withdrawal were nearly identical in form, although higher in magnitude, to those seen for Guthion, and longer planning horizons again led to higher levels of surplus loss in the early years of any given simulation.

Although the surplus loss results for Lorsban and Guthion withdrawals were similar, the two scenarios did differ in terms of the effects on optimal total insecticide use. Lorsban withdrawal consistently led to larger reductions in the amount of insecticides used than that experienced after Guthion withdrawal, with the effect increasing as the discount rate increased (Table 14). For example, the decrease in chemical use for 5 year horizon at a 6 percent discount rate was -1.305 million pounds a.i., or 3.0 pounds a.i. per acre per year. This represented a decrease of 27.3 percent over the base-case, compared to a 2.2 percent decrease for the Guthion withdrawal scenario. Overall, the largest percentage decreases in chemical use following Lorsban withdrawal occurred for any discount rate within the 5 year planning horizon. The decreases in insecticide use tended to moderate as the planning horizon increased, with some of the smallest decreases occurring for 25 year horizons. In only 5 cases (20 year horizon, 0 and 2 percent discounts; 25 year horizon, 0, 2 and 4 percent discounts) were the declines in total chemical use following Guthion withdrawal higher than declines following Lorsban withdrawal.

Given the intensity of Imidan use in the base-case, its withdrawal might have been expected to cause extreme increases in cumulative surplus losses. However, surplus losses following Imidan withdrawal were mixed, falling below those experienced after Guthion and Lorsban withdrawal for 1 and 5 year horizons; between Guthion and Lorsban withdrawal scenarios for 10 and 15 year scenarios; and above all other insecticide withdrawal scenarios only for the 20 and 25 year planning horizons (Table 15). Because of this pattern, the range of percentage surplus effects due to Imidan withdrawal varied widely. For example, additional cumulative surplus losses for a 5 year planning horizon at a 6 percent discount rate were -$3.640 million, or 334 percent above the base-case simulation, while additional losses for a 15 year horizon, averaged over all discount rates, were -$2.6 billion, or 1,759 percent above

Table 14. Change in the Total Use of Insecticides Following the Withdrawal of Lorsban: By Planning Horizon and Discount Rate.

Discount Rate	Planning Horizon (Years)					
	1	5	10	15	20	25
	1,000 Pounds					
0.00	-205.6	-1,405.7	-1,862.1	-2,137.0	-2,567.9	-3,184.2
0.02	-150.9	-1,304.8	-2,005.7	-2,441.1	-2,662.5	-3,011.8
0.04	-206.9	-1,383.1	-2,686.7	-2,866.9	-3,050.8	-3,009.7
0.06	-277.1	-1,304.8	-2,817.9	-3,006.1	-3,115.9	-3,115.8
0.08	-211.3	-1,295.7	-3,065.8	-3,285.0	-3,000.3	-3,121.6
0.10	-198.6	-1,152.5	-3,110.7	-3,363.9	-3,106.2	-3,199.9
0.12	-165.2	-1,086.4	-3,097.3	-3,401.7	-3,029.7	-3,084.3

Table 15. Total Economic Surplus Changes From the Base Case Due to the Withdrawal of Imidan: By Planning Horizon and Discount Rate.

Discount Rate	Planning Horizon (Years)					
	1	5	10	15	20	25
	Million Dollars (Present Value)					
0.00	-0.005	-6.179	-2,017.532	-5,862.101	-6,419.301	-6,783.534
0.02	-0.003	-4.551	-1,462.724	-2,892.715	-3,093.651	-3,186.928
0.04	-0.005	-4.048	-1,244.918	-2,665.523	-2,962.533	-2,875.622
0.06	-0.004	-3.640	-1,022.423	-2,586.931	-2,682.955	-2,771.631
0.08	-0.004	-3.562	-984.622	-2,367.582	-2,578.917	-2,662.851
0.10	-0.007	-3.219	-900.533	-2,294.781	-2,445.510	-2,593.213
0.12	-0.006	-2.808	-620.687	-2,186.511	-2,331.719	-2,500.901

the base-case scenario. Imidan withdrawal also resulted in the largest long-run impacts on surplus loss. For a 25 year planning horizon, additional surplus declines over the base-case ranged from 85 percent for a 0 percent discount rate to 414 percent for a 12 percent discount rate. This compares to range of 34 percent to 150 percent for Guthion and 52 percent to 188 percent for Lorsban (Table 11 and Table 13). The general time-path of cumulative surplus following Imidan withdrawal was similar to the ones observed for Guthion and Lorsban.

The withdrawal of Imidan not only led to declines in economic surplus, but also to a pattern of decreased chemical use that generally exceeded the one seen for Lorsban. While the use of a 1 year horizon resulted in the most extreme percentage declines in total insecticide use, applications were down substantially at all planning horizons following Imidan withdrawal (Table 16). The decline in chemical use for a 1 year horizon, 6 percent discount rate was 0.757 million pounds a.i., or over 83 percent less than under than under the base-case scenario. For a 15 year horizon, 6 percent discount rate, chemical use fell over 3.6 million pounds a.i., or 2.76 pounds a.i. per acre per year, while for a 25 year horizon the decline was 5.0 million pounds a.i., or 2.30 pounds a.i. per acre per year. In fact, a number of planning horizon/discount rate combinations were associated with a decline in total insecticide use that actually exceeded the total amount of Imidan employed in the optimal base-case solutions.

The withdrawal of the last organophosphate in the initial control technology set, PennCap-M, yielded surplus losses that were comparable in magnitude and time-path to those seen for Imidan, with the exception of results for 10 and 15 year planning horizons (Table 17). For these intermediate periods, cumulative surplus losses following PennCap-M withdrawal were less than those observed for the withdrawal of any other organophosphate. This was especially true for the 10 year horizon at a 6 percent discount rate, where additional surplus losses were only -$299 million, compared to losses of -$938 million for Guthion withdrawal, -$1.6 billion for Lorsban withdrawal, and -$1.0 billion for Imidan withdrawal. Perhaps the most significant difference between the withdrawal of PennCap-M and the other organophosphates concerned the effects on total insecticide use. For all but 3 planning

Table 16. Change in the Total Use of Insecticides Following the Withdrawal of Imidan: By Planning Horizon and Discount Rate.

Discount Rate	Planning Horizon (Years)					
	1	5	10	15	20	25
	1,000 Pounds A.I.					
0.00	-684.1	-1,297.6	-2,884.1	-4,236.7	-4,287.9	-4,283.6
0.02	-679.5	-1,230.0	-2,786.5	-3,994.0	-4,153.0	-4,528.9
0.04	-700.3	-1,231.6	-2,694.3	-3,879.6	-4,099.7	-4,762.5
0.06	-756.9	-1,230.1	-2,651.4	-3,601.2	-4,079.6	-5,003.7
0.08	-695.1	-1,195.8	-2,493.1	-3,600.5	-3,977.1	-5,011.3
0.10	-705.2	-1,162.1	-2,348.7	-3,497.6	-3,821.6	-4,962.7
0.12	-687.5	-1,012.2	-2,011.2	-3,018.2	-3,608.3	-4,985.9

Table 17. Total Economic Surplus Changes From the Base Case Due to Withdrawal of PennCap-M: By Planning Horizon and Discount Rate.

Discount Rate	Planning Horizon (Years)					
	1	5	10	15	20	25
	Million Dollars (Present Value)					
0.00	-0.035	-5.954	-362.851	-5,009.052	-6,178.932	-6,769.118
0.02	-0.033	-3.156	-148.638	-2,353.622	-2,915.613	-3,005.984
0.04	-0.031	-3.985	-200.855	-2,461.583	-2,561.331	-2,860.114
0.06	-0.034	-4.043	-298.631	-2,261.051	-2,297.703	-2,629.031
0.08	-0.030	-4.256	-310.094	-2,119.623	-2,090.101	-2,407.214
0.10	-0.027	-5.914	-387.622	-2,018.012	-1,960.842	-2,261.005
0.12	-0.029	-5.876	-389.090	-1,962.723	-1,883.812	-2,032.257

horizon/discount rate combinations, the withdrawal of PennCap-M resulted in substantial increases in the total amount of insecticides used (Table 18). Given a 6 percent discount rate, this effect ranged from an average increase in use of 3.4 percent, or 0.33 pounds a.i. per acre per year, for a 10 year horizon, to an average 11.9 percent increase, or 1.04 pounds a.i. per acre per year, for a 15 year horizon. In fact, the effects of a withdrawal of PennCap-M proved to be quite similar to those seen for the withdrawal of the carbamate Lannate and the synthetic pyrethroid Asana. In Lannate's case, a withdrawal resulted in additional surplus losses at a 6 percent discount rate that increased from 172 percent over base for a 1 year horizon to 2,369 percent for a 10 year horizon, thereafter falling back to 215 percent for a 25 year horizon (Table 19). Total insecticide use after Lannate withdrawal increased for all planning horizon/discount rate combinations, reaching a maximum increase of 12.2 percent for 15 years at a 6 percent discount rate (Table 20). While the changes in economic surplus following Asana's withdrawal were much the same as those seen for Lannate withdrawal (Table 21), increases in total insecticide use were substantially less (Table 22).

Discussion

The withdrawal scenarios addressed a number of issues that were originally raised in Chapter I. In particular, the simulated withdrawals generated information that was used as evidence to evaluate the maintained hypotheses that usually accompany policy deliberations concerning the resistance/regulation nexus. These hypotheses have generally been developed from short-run observations of the impacts of regulation on resistance, and have included the conjecture that decreases in the size of the control technology set will always:

1. hasten the development of resistance;

2. decrease the amount of economic surplus obtainable from the system;

Table 18. Change in the Total Use of Insecticides Following the Withdrawal of PennCap-M: By Planning Horizon and Discount Rate.

Discount Rate	Planning Horizon (Years)					
	1	5	10	15	20	25
	----------	----------	1,000 Pounds A.I.	----------	----------	----------
0.00	97.6	-631.9	448.7	1,867.9	1,941.7	2,084.6
0.02	94.1	-373.8	362.9	1,532.2	1,638.7	1,716.0
0.04	90.5	-305.6	329.4	1,487.1	1,594.2	1,688.6
0.06	86.3	-100.1	289.7	1,362.3	1,565.9	1,609.3
0.08	83.9	200.8	264.3	1,300.0	1,400.8	1,595.1
0.10	78.1	196.3	201.4	1,281.2	1,409.7	1,562.0
0.12	79.8	189.9	128.9	1,017.3	1,394.1	1,499.1

Table 19. Total Economic Surplus Changes From the Base Case Due to the Withdrawal of Lannate: By Planning Horizon and Discount Rate.

Discount Rate	Planning Horizon (Years)					
	1	5	10	15	20	25
	Million Dollars (Present Value)					
0.00	-0.020	-3.051	-105.838	-2,165.719	-3,562.841	-6,009.782
0.02	-0.017	-2.956	-136.791	-2,013.770	-3,005.688	-3,184.661
0.04	-0.021	-3.486	-187.770	-2,152.883	-2,841.121	-3,094.811
0.06	-0.019	-3.879	-209.634	-2,206.578	-2,387.888	-2,976.401
0.08	-0.018	-3.951	-284.043	-2,295.721	-2,113.344	-2,762.996
0.10	-0.020	-4.362	-327.661	-2,367.005	-2,005.013	-2,489.733
0.12	-0.019	-5.003	-354.989	-2,448.721	-2,000.048	-2,305.015

Table 20. Change in the Total Use of Insecticides Following the Withdrawal of Lannate: By Planning Horizon and Discount Rate.

Discount Rate	Planning Horizon (Years)					
	1	5	10	15	20	25
	----------	----------	1,000 Pounds A.I.	----------	----------	----------
0.00	60.7	293.8	405.1	1,891.0	1,786.5	2,019.3
0.02	62.9	285.6	356.7	1,499.7	1,684.1	1,853.0
0.04	58.4	296.5	303.8	1,483.2	1,605.3	1,701.9
0.06	57.6	228.1	272.1	1,400.8	1,572.0	1,623.3
0.08	52.9	200.9	209.3	1,324.1	1,436.7	1,602.6
0.10	50.4	152.7	191.0	1,250.0	1,401.9	1,581.7
0.12	51.9	143.9	172.5	1,098.4	1,381.8	1,533.3

Table 21. Total Economic Surplus Changes From the Base Case Due to the Withdrawal of Asana: By Planning Horizon and Discount Rate.

Discount Rate	Planning Horizon (Years)					
	1	5	10	15	20	25
	Million Dollars (Present Value)					
0.00	-0.038	-0.361	-14.051	-1,595.622	-3,785.997	-5,215.033
0.02	-0.040	-0.258	-15.063	-1,489.762	-2,874.211	-3,287.665
0.04	-0.038	-0.621	-19.872	-1,501.531	-3,005.962	-2,289.210
0.06	-0.036	-0.551	-21.201	-1,472.044	-2,985.762	-3,181.674
0.08	-0.033	-0.262	-28.673	-1,615.583	-2,431.014	-2,881.533
0.10	-0.035	-0.973	-32.476	-1,372.938	-2,382.911	-2,767.012
0.12	-0.037	-0.410	-33.200	-1,200.910	-2,101.378	-2,462.119

Table 22. Change in the Total Use of Insecticides Following the Withdrawal of Asana: By Planning Horizon and Discount Rate.

Discount Rate	Planning Horizon (Years)					
	1	5	10	15	20	25
	----------	----------	1,000 Pounds A.I.	----------	----------	----------
0.00	20.8	27.1	159.8	842.9	1,700.3	1,894.6
0.02	21.7	29.8	153.0	783.3	1,583.7	1,760.8
0.04	20.9	30.2	146.5	842.6	1,601.9	1,693.1
0.06	25.2	35.7	148.9	772.0	1,534.0	1,605.3
0.08	26.7	33.1	151.0	753.2	1,490.7	1,534.2
0.10	29.8	38.6	142.7	702.1	1,409.8	1,486.7
0.12	25.4	37.5	140.9	684.3	1,362.5	1,500.9

3. increase the long-run total use of insecticides; and

4. in the case of organophosphate withdrawals, lead to larger surplus losses than those caused by withdrawal of non-organophosphate control alternatives.

Each of these hypotheses are discussed below, with the remaining questions from Chapter I relegated to the final section.

Optimal solutions generated from the simulation model suggested that any reduction in the size of the control technology set leads to accelerated susceptibility depletion and increased resistance-induced surplus losses. However, the rate change in susceptibility depletion varied substantially with the choice of time horizon and discount rate. Because resistance is essentially an evolutionary phenomenon, withdrawal of an insecticide would be expected to have a relatively small short-run impact on economic surplus. For a 1 year planning horizon, additional surplus losses due to a withdrawal averaged less than one-tenth percent of total economic surplus for every insecticide. These negligible short-run impacts indicated why resistance effects are currently excluded from the benefits assessment process, which almost totally concentrates on the near-term effects of a withdrawal. But although the short-run impacts may be small as a percentage of total surplus, on average they doubled the magnitude of resistance effects compared to the base-case, and in the cases of PennCap-M and Asana tripled the economic loss (Table 7, Table 17, and Table 21). So, even for a 1 year horizon, the results suggested that the interaction of susceptibility depletion and regulation has the potential to significantly alter the long-run economic performance of the system. Discount rate had little effect on the additional surplus losses generated over a 1 year planning horizon.

As the length of the planning horizon increased, the economic impact of a regulatory withdrawal accelerates rapidly, with the effect being more pronounced at lower discount rates. Examination of the surplus loss time-paths suggested that substantial impacts began to occur after year 3 for all planning horizon/discount rate combination. For example, the withdrawal of Guthion resulted in economic surplus losses that were 537 percent higher than the base-

case over a 5 year horizon, and over 1091 percent higher over a 15 year horizon (Table 7 and Table 11). Interestingly, the impacts of accelerated susceptibility depletion moderated quickly for planning horizons longer than 15 years, and generally resulted in less than a 100 percent increase in surplus loss over the base-case by year 25. This dampening influence of the planning horizon suggested that the important interactions between resistance and regulation may occur in the intermediate-term, or as resistance builds in the system. Thus, the argument for including dynamic resistance considerations in insecticide benefit assessments can be directly related to the choice of a time horizon. Given the magnitude of the additional surplus losses, static benefit assessments seriously underestimated the impacts of an insecticide withdrawal when resistance is present in the system.

While the length of the planning horizon can help determine whether or not the impact of susceptibility depletion on economic surplus is large enough to justify incorporating resistance considerations into regulatory decision-making, the use and magnitude of present value discounting also had a critical role. However, the interaction is complex because resistance guarantees that present value calculations are not only a function of the discount rate. As the discount rate increased, the importance of future surplus streams declined. Within the context of the optimization model, high discount rates promoted increased, prophylactic use of insecticides to control pest population early in the planning horizon, thereby minimizing early-horizon surplus losses. For example, the additional economic surplus losses caused by withdrawal of Guthion under a 15 year planning horizon were, by year 10, over two and one-half times larger for a 2 percent discount rate compared to a 12 percent discount rate (Figure 33). Although this type of control strategy led to accelerated susceptibility depletion, by the time resistance became firmly entrenched surplus losses were heavily discounted and no longer contributed significantly to the present value calculations. Overall, the model results suggested that shrinkage in the control technology set hastens the development of resistance, thereby increasing resistance-induced economic surplus losses and decreasing the amount of economic surplus obtainable from the production system.

Even though the size of the control technology set influenced the economic surplus losses attributable to resistance, questions still remain concerning the potential magnitude of the surplus losses. As it turns out, it was not only the length of the planning horizon and the discount rate that determined the extent of surplus losses due to resistance, but also the nature of the specific chemical chosen for withdrawal. Optimal economic surplus losses following the withdrawal of an organophosphate tended to be many times larger than those calculated for a complete technology set at 10, 15, and 20 year planning horizons. The withdrawal of Imidan and PennCap-M resulted in the largest surplus losses over these intermediate horizons, and for some horizon/discount rate combinations exceeded the losses from withdrawal of Guthion and Lorsban by over 100 percent (Table 11, Table 13, Table 15, and Table 17). The relative long-run importance of Imidan and PennCap-M to the amount of economic surplus lost from the apple production system also held for a 25 year planning horizon, where the present value surplus losses due to the withdrawal of Guthion and Lorsban fell relative to levels for a 20 year horizon. Only for short-term planning horizons did this relationship change. For example, the withdrawal of Guthion and Lorsban led to economic surplus losses over a 5 year horizon that were 30 to 60 percent higher than those calculated following Imidan and PennCap-M withdrawals. Surplus losses over a 1 year horizon were highest for the withdrawal of PennCap-M and Lorsban, with the losses due to withdrawal of Imidan and Guthion being nearly identical. Taken together, these results suggested that, in the short-run, Guthion, Lorsban, and PennCap-M are the most important organophosphates in the control technology set. In the long-run, Imidan and PennCap-M assumed a critical role.

When considering the non-organophosphates in the initial control set, it was the withdrawal of Lannate that had the biggest effect on additional economic surplus loss. The surplus losses associated with Lannate withdrawal at 20 and 25 year time horizons approached the levels experienced following Imidan and PennCap-M withdrawals (Table 19, Table 15, and Table 17). While the loss of Asana from the control set led to extremely small additional surplus losses for time horizons of less than 15 years, losses at longer time horizons tended to exceed those calculated for Guthion and Lorsban (Table 21, Table 11, and Table 13). Thus,

the non-organophosphates were important to the optimal operation of apple production system, especially in the long-run. As pests developed resistance to the organophosphates registered for use in the mid-Atlantic apple system, the optimal solution switched to using mixtures, eventually followed by sprays of the available carbamates and synthetic pyrethroids. Somewhat surprising was the result indicating that the optimal use of Lannate required its application in a switching strategy even in the earliest years of a planning horizon. Problems of toxicity to the predator in the system were mitigated by applying Lannate only early or late in the growing season. Asana was the chemical control of last resort, as evidenced by the relatively low short-term, high long-term magnitude of surplus losses following its withdrawal.

Considering the large surplus losses associated with reductions in the initial control technology set, especially in the long-run, the model results indicated that:

1. insecticide regulatory decision-making needs to be conducted within a dynamic context in order to avoid grossly underestimating the long-run benefits associated with a particular chemical control; and

2. except in cases where the long-run environmental and human health risks associated with a chemical were large enough to outweigh potential, but unmeasured, dynamic benefits, past withdrawals of insecticides may have led to sub-optimal system performance, even though they appeared to be expendable from a short-run perspective.

However, another facet of regulatory withdrawal needs to be included in the discussion, namely its impact on the total use of insecticides.

Given the optimal results generated from the simulation model, total long-run insecticide use does not necessarily have to increase following a reduction in the size of the control technology set. Instead, changes in total chemical use were related to the characteristics of the specific insecticide withdrawn. For example, withdrawal of Lorsban and Imidan resulted in decreased insecticide use for all planning horizon/discount rate combinations, a situation that was repeated for Guthion at all but the higher discount rates (Table 12, Table 14,

Table 16). But, withdrawal of PennCap-M, Lannate, and Asana led to increases in total insecticide use for all planning horizon/discount rate combinations (Table 18, Table 20, Table 22). These results suggested that the long-run changes in chemical use would also be important when examining the resistance/regulation interaction and its impact on policy-making. From a comprehensive regulatory perspective, the additional economic losses associated with the withdrawal of Guthion, Lorsban, and Imidan, to some degree, offset by corresponding declines in the total amount of insecticides used in the production system. While the reduction in chemical use was incorporated into the present value calculations, it would also figure prominently in the environmental and human health risk analysis of the regulatory process. Given a sufficient unit-cost in terms of environmental contamination and/or human health, the benefits of a decrease in total insecticide use following a withdrawal of Guthion, Lorsban, and Imidan could offset the social impacts of a decline in economic surplus, thereby leading to a net increase in social welfare from a withdrawal. On the other hand, the increased total insecticide use associated with withdrawal of PennCap-M, Lannate, and Asana indicated that even a dynamic benefits assessment would alone underestimate the value of continued registration, given that the increased use of other chemicals following withdrawal would need to be incorporated into the environmental and human health risk analysis. Lacking a dynamic approach, the static assessment cannot take into account the changes in insecticide use that accompany the development of resistance.

Comparative Analysis

The preceding sections described the potential impact of resistance on the total economic surplus obtainable from a production system, and how the economic surplus might change given various regulatory actions affecting the pest control technology set. Evidence generated from the simulation model suggested that resistance alone can lead to nearly a 9 percent loss in economic surplus over a 25 year period. Regulatory pressure, combined with

the presence of resistance, can potentially increase surplus losses more than 13-fold over the losses experience under no-regulation scenarios, with the ultimate effects of regulation dependent on the specific insecticides involved. However, these results arise within an optimization framework. Still needing investigation, under both non-regulatory and regulatory scenarios, are the relationships between dynamically-optimal solutions, extant recommended solutions, and statically-optimal solutions. In addition to these issues, this final section examines the potential benefits of comprehensive versus chemical-by-chemical regulatory decision-making.

Non-regulatory Resistance Impacts

Results have demonstrated the importance of conducting benefit assessment with a planning period that is of sufficient length to allow the decision-making process to incorporate the long-run impacts of resistance. But, it has not been demonstrated that dynamically-optimal solutions are superior enough to simple multi-period optimal solutions or current practices to warrant the effort a dynamic solution requires. To investigate the importance of dynamic solutions, comparisons were made with the impacts of myopic optimization and currently recommended insecticide application regimes on resistance-induced total economic surplus losses. Myopic solutions consisted of sequential yearly optimizations over planning horizon/discount rate combinations, with the levels of state variables updated before each year's optimization. Current application regimes consisted of single chemicals used at labelled levels until susceptibility decreased 50 percent, after which a another chemical from the initial control technology set was applied in the following order: Guthion, Imidan, Lorsban, PennCap-M, Lannate, and Asana. In practice, no simulation progressed beyond the application of Lannate.

Given a complete control technology set, the dynamically-optimal, myopic, and current recommendation impacts of resistance were simulated at various planning horizons for a 6

Table 23. Economic Surplus Changes For Dynamic Optimal, Myopic, and Currently Recommended Control Actions at a 6 Percent Discount.

Discount Rate	Planning Horizon (Years)					
	1	5	10	15	20	25
	Million Dollars (Present Value)					
Dynamic	-0.011	-1.090	-8.849	-146.997	-1,029.858	-1,385.430
Myopic	-0.010	-1.105	-10.628	-581.972	-2,078.622	-2,678.933
Current	-0.359	-10.966	-49.098	-617.244	-2,651.997	-2,877.486

percent discount rate (Table 23). Total economic surplus losses were generally higher for both statically-optimal and currently recommended regimes than the losses experienced under dynamic optimization. Because the effects of resistance primarily emerged in the long-run, myopic optimization was unable to incorporate future information into the current year's decision-making, thereby limiting the ability to mitigate susceptibility depletion through managed insecticide use. Currently recommended regimes had almost no ability to proactively mitigate susceptibility depletion, and this management approach led to large declines in economic surplus.

While the general results from the above comparisons were anticipated, a few anomalies did arise. Most obvious was that total economic surplus losses for 1 and 5 year myopic optimizations were slightly lower than the losses calculated for dynamic optimization and currently recommended applications (Table 23). This results can be explained by observing that as the discount rate decreased for a sufficiently long horizon under dynamic optimization, surplus losses shifted forward in time due to a postponement in both insecticide use and susceptibility depletion. Myopic optimization, by not incorporating information about future susceptibility depletion, led to increased use of insecticides early in any planning horizon. This initially lowered pest populations below levels achieved under dynamic optimization and, because susceptibility was not yet depleted, lowered total economic surplus losses. However, the increased use of insecticides eventually increased the rate of susceptibility depletion, with resistance emerging for planning horizons of 10 years or more. The importance of increased early insecticide use to susceptibility depletion was demonstrated by the substantially higher surplus losses suffered under myopic versus dynamic optimization for 15, 20, and 25 year planning horizons.

A second interesting feature of Table 23 concerns the surplus losses associated with currently recommended application regimes. These losses were substantially higher than those experienced under myopic and dynamic optimization for horizons of 10 years or less, primarily because current recommendations resulted in lower total insecticide use and inferior pest control. For horizons longer than 10 years, surplus losses under current recomm-

endations were still higher than those for dynamic optimization, but they rapidly converged to the levels calculated for myopic optimization. Convergence over longer planning horizons was due to equivalent effects on long-run surplus losses from increased susceptibility depletion (myopic optimization) and increased pest population levels (currently recommended applications).

Regulation/Resistance Impacts

Examined under regulatory withdrawal scenarios, dynamically-optimal, myopic, and current recommendation simulations each yielded changes in total economic surplus that tended to repeat the patterns established for the complete technology set. Dynamically optimal decision-making resulted in the lowest surplus losses for all except 1 and 5 year planning horizons, where myopic decision-making led to slightly lower surplus losses (Table 24). In fact, surplus losses under dynamic decision-making were substantially less than either of the other two alternatives, especially for longer planning horizons. Proportional changes in surplus loss between resistance-only and withdrawal scenarios for myopic and current recommendation simulations also greatly exceeded those experienced under dynamic optimization. At all planning horizons, current recommendations were severely sub-optimal to both myopic and dynamic optimization, indicating the inability to incorporate any future susceptibility information into current decision-making. Overall, the results in Table 24 suggest that, while in some instances modest in the short- run, there are severe long-run impacts associated with not incorporating dynamically-optimal information into the regulatory decision-making process.

Table 24. Total Economic Surplus Changes Following Single Withdrawals For Dynamic Optimal, Myopic, and Currently Recommended Actions.

Chemical Withdrawn	Planning Horizon (Years)					
	1	5	10	15	20	25
	Million Dollars (Present Value)					
Dynamic						
Guthion	-0.005	-5.857	-938.095	-1,604.362	-1,596.312	-1,516.705
Lorsban	-0.009	-6.886	-1,568.273	-1,984.186	-2,009.773	-1,884.482
Imidan	-0.004	-3.640	-1,022.423	-2,586.931	-2,682.955	-2,771.631
PennCap-M	-0.034	-4.043	-298.631	-2,261.051	-2,297.703	-2,629.031
Lannate	-0.019	-3.879	-209.634	-2,206.578	-2,387.888	-2,976.401
Asana	-0.036	-0.551	-21.201	-1,472.044	-2,985.762	-3,181.674
Myopic						
Guthion	-0.003	-5.217	-1,053.684	-2,293.736	-5,981.761	-5,889.317
Lorsban	-0.006	-5.936	-1,691.335	-2,196.003	-6,238.052	-5,993.621
Imidan	-0.004	-3.001	-1,175.641	-3,391.991	-7,664.931	-7,835.327
PennCap-M	-0.030	-3.855	-334.048	-2,471.663	-6,876.800	-7,641.329
Lannate	-0.018	-3.271	-276.994	-2,581.022	-7,225.525	-8,560.038
Asana	-0.029	-0.499	-25.638	-1,669.132	-8,005.614	-8,796.941
Current						
Guthion	-0.398	-15.608	-1,305.977	-3,019.668	-5,843.691	-5,639.481
Lorsban	-0.457	-16.215	-1,761.009	-2,985.056	-6,203.442	-5,932.062
Imidan	-0.362	-13.372	-1,438.726	-3,884.931	-7,804.644	-7,201.353
PennCap-M	-0.495	-14.992	-441.233	-2,281.831	-7,450.123	-8,091.685
Lannate	-0.400	-13.793	-227.483	-2,137.209	-7,309.486	-8,664.831
Asana	-0.513	-4.366	-86.982	-2,088.651	-8,766.101	-8,994.738

Comprehensive Regulation

All the results of regulation scenarios have dealt with the impacts of single- chemical withdrawals. Given the relatively long maximum planning horizons examined in this study, it is reasonable to assume that a second insecticide from the initial technology set may come under regulatory scrutiny after the original withdrawal. If the second chemical is eventually withdrawn, then the time-paths associated with the single chemical withdrawal may change. While comprehensively evaluating the initial withdrawal of both insecticides might be optimal to a sequential withdrawal, it is not clear *a priori* whether the gains in optimality will be enough to offset the effort involved in a comprehensive analysis.

To illustrate the multiple withdrawal problem, Imidan and Lannate were designated as the insecticides subject to regulatory action. Two scenarios were then developed. In the first scenario, representing comprehensive evaluation, both insecticides were withdrawn from the technology set at the beginning of the planning horizon. The results were then compared to a second scenario, where only Imidan was initially withdrawn and Lannate was withdrawn in year 11 of all planning horizons greater than 10 years. In the latter scenario, dynamic optimization was first conducted with a 5 chemical control technology set, the reoptimized for the remaining time beyond 10 years with a 4 chemical technology set. A comparison of the results is given in Table 25.

Considering the results generated for single chemical withdrawals, it might be expected that shrinking the initial technology set from five to four insecticides would lead to higher total economic surplus losses for all relevant planning horizon/discount rate combinations. Indeed, this did occur for planning horizons of 1, 5, and 10 years (Table 25). During this time, the absence of Lannate from the 4 insecticide set resulted in at least a doubling of the surplus lost due to resistance. But, the proportional change these losses represented was restrained compared to results seen for many single insecticide withdrawals. The somewhat moderate *reaction* following Lannate withdrawal for time horizons under 15 years can be traced to the chemical's relatively light optimal use early in most planning horizons. Thus, it appeared that

Table 25. Total Economic Surplus Changes Following a Double Initial and Spaced Withdrawals of Imidan and Lannate.

Discount Rate	Planning Horizon (Years)					
	1	5	10	15	20	25
	Million Dollars (Present Value)					
Double Initial						
0.00	-0.061	-18.409	-3,894.012	-10,493.621	-12,687.118	-19,377.911
0.06	-0.057	-17.644	-2,034.363	-5,817.044	-5,837.014	-5,974.922
0.12	-0.055	-16.553	-1,736.494	-5,224.738	-5,481.336	-5,593.718
Spaced						
0.00	-0.006	-6.284	-2,019.811	-12,413.728	-16,995.837	-24,344.010
0.06	-0.005	-3.495	-1,020.881	-6,478.311	-6,552.983	-6,664.318
0.12	-0.005	-2.815	-621.787	-6,249.923	-6,499.381	-6,731.005

single chemical withdrawals were preferable to multiple withdrawals, at least in the short-run. However, the results changed dramatically for longer planning horizons.

Once Lannate was withdrawn from the 5 chemical technology set in year 11 and dynamically optimal time-paths reestimated for the remaining portion of planning horizons over 10 years, it became apparent that the initial withdrawal of both chemicals was superior in terms of minimizing surplus loss in the long-run (Table 25). Examination of the insecticide use time-paths suggested a reason for this result. When Lannate was initially withdrawn with Imidan, the optimal use of some remaining insecticides fell substantially relative to the 6 chemical base-case, apparently in an effort to delay susceptibility depletion. But for the scenario in which Lannate was not withdrawn with Imidan, the optimal use of the remaining insecticides did not decline nearly as much, apparently because resistance to them could be accepted knowing that Lannate was available as a control of last resort. However, Lannate was withdrawn in year 11, and control of the pest populations declined to levels below those being attained by the initial 4 chemical technology set. While this result arose due to the characteristics of the particular chemicals chosen for withdrawal, it does demonstrate that there are situations where it might be long-run optimal to comprehensively regulate control technology sets and not individual chemicals on a random basis, even if comprehensive regulation requires a more severe initial shrinkage in the number of insecticides available for use.

CHAPTER X: SUMMARY AND CONCLUSION

The economic problems associated with pest resistance have been examined in a number of studies over the years. Given the common-property and dynamic characteristic of pest susceptibility to chemical controls, it has been demonstrated that the susceptibility resource will be inefficiently utilized by individual producers reacting to free-market price signals. In addition, the current pesticide regulatory process is not structured in a way that allows the quantitative incorporation of resistance considerations. Instead, regulatory planning has been based on the assumption that the control technology set, and the biological system's response to it, remains unchanged following the contemplated regulatory action. This inability to extend regulatory analyses into dynamic scenarios may, in fact, inadvertently promote the depletion of susceptibility.

In an attempt to solve the combined resistance/regulatory problem, researchers have sought a mechanism by which resistance could be mitigated by explicitly including it in the regulatory process. However, most previous studies have resorted to simplification of either the biological or economic system in order to make their empirical models numerically tractable. As a result, published resistance models tend to be highly stylized representations of reality, making them unsuitable for policy-making purposes. In addition, few researchers have actually included potential regulatory actions into their modelling efforts, thereby limiting the ability to engage in the kind of "what if" investigations needed to support the pesticide regu-

latory process. This study overcame these difficulties by forming a dynamic bioeconomic model that was not only economically feasible, but also highly detailed and realistic in its portrayal of the biological components affected by pesticide use in a production system. This chapter will summarize the results obtained from the empirical model, detail their implications for the pesticide regulatory process, and offer a few recommendations concerning future studies.

Summary of Results

The main results generated from the empirical optimization model broadly supported the maintained hypotheses that have often been employed during regulatory policy-making. In particular, the potential for resistance development, in conjunction with a static control technology set, led to long-run losses in the economic surplus that was generated from an apple production system containing the Tufted Apple Bud Moth (TABM), European Red Mite (ERM), and Black Ladybird Beetle (BLB) complex. The economic surplus losses occurred even when insecticides were used in a dynamically-optimal manner, although under optimal application regimes susceptibility depletion emerged slowly over the first ten years of a simulation. It was only after 10 years or more that resistance began to cause substantial economic damage, even when optimal decision-making was not based on present-value discounting. Once resistance did fully emerge, the cumulative losses accelerated rapidly and only leveled off between 20 and 25 years. Thus, the choice of a planning horizon greatly influenced how potential resistance-related problems were perceived.

While resistance always led to long-run economic losses in the simulations, the magnitude of the losses depended on how the dynamic decision-making process was influenced by present-value discounting. On a percentage basis, the largest resistance-induced losses were associated with nominal calculations, reaching a maximum loss of nearly 9 percent over a 25 year planning horizon. Although, as expected, the monetary value of these surplus losses

decreased as the discount rate increased, the losses also steadily decreased in percentage terms. These percentage declines in the impact of resistance as the discount rate increased were at least partially due to shifts in the optimal timing of insecticide applications. As the discount rate increased, the importance of future resistance-induced surplus losses to the present-value decision-making decreased, thereby promoting heavier use of insecticides early in the planning horizon. However, total insecticide use did not increase along with the discount rate, as higher early-horizon applications were followed by reduced insecticide use later in the planning horizon.

After investigating the impact of susceptibility depletion on economic surplus when producers had access to a complete control technology set, insecticide withdrawal scenarios were simulated in order to measure any additional resistance-induced surplus losses that might arise due to regulatory actions. Optimal solutions demonstrated that reductions in the size of the control set leads to accelerated susceptibility depletion and increased economic surplus losses. The magnitude of these additional losses, although sometimes small in absolute terms, were almost always substantially higher than the losses experienced with a complete control set. Even over a 1 year planning horizon, surplus losses from resistance increased by an average of 100 percent due to the withdrawal of a single insecticide, and for intermediate horizons reached as high as an additional 1,350 percent. Only for the longest planning horizons were the effects of insecticide withdrawals muted, increasing resistance-induced losses by less than 100 percent. Meanwhile, discounting played a role similar to that observed for the complete control technology set simulations.

When examined on a chemical specific basis, the impact of regulatory action varied. In general, the largest additional economic surplus losses were simulated following the withdrawal of any organophosphate insecticide, with azinphosmethyl (Guthion) and chorpyrifos (Lorsban) being the most important chemicals for short-run (5 years or less) containment of economic surplus losses due to resistance. In the intermediate and long-run, phosmet (Imidan) and microencapsulated methylparathion (PennCap-M) were the organophosphates most critical in terms of maintaining the surplus obtained from the system. Because the use

of all four organophosphates figured prominently in the optimal solutions for no-regulation scenarios, the economic surplus losses associated with their withdrawal were not unexpected. Somewhat surprising, however, were the effects of non-organophosphate withdrawals. For longer horizons, the economic impact brought about by withdrawing the carbamate methomyl (Lannate) and the synthetic pyrethroid esfenvalerate (Asana) approached the levels seen for some organophosphates, with the importance of the Asana accelerating rapidly as horizons exceeded 20 years. Because of their toxicity to the predator in the system, the optimal use of these non-organophosphate insecticides tended to be restricted in the no-regulation scenarios to early-season periods when the orchard populations of BLB were low. Under organophosphate withdrawal scenarios, however, both Lannate and Asana began to be used on a more regular basis early in the planning horizons. In addition, the optimal use of both Lannate and Asana included their use as controls of last resort for longer planning horizons. Thus, the temporal positioning of their importance was clearly demonstrated by the fact that the vast majority of the additional surplus losses associated with their withdrawal occurred for planning horizon longer than 10 years.

In addition to the economic surplus losses engendered by a chemical withdrawal from the initial technology set, regulatory actions also led to large changes in the total amount of insecticides used. But unlike the changes in resistance-induced surplus losses, the direction of the changes depended on the chemical withdrawn. Removal of Imidan and Lorsban from the control set always led to large reductions in the total use of insecticides, a situation that was repeated for Guthion at all but the higher discount rates. However, the withdrawal of PennCap-M, Lannate, and Asana led to increases in total insecticide use. So although a regulatory withdrawal appears to always decrease the direct economic surplus obtained from the apple production system, these losses may, in some cases, be offset by the environmental and/or health benefits that might accrue due to reduced chemical use.

The final part of the study examined the long-run impacts associated with myopic and currently recommended strategies for pest control in the mid-Atlantic apple production system, and compared them to the dynamically optimal solutions. In addition, the expected dif-

ferences between chemical-by-chemical and comprehensive regulatory evaluations were detailed through the use of an example. Overall, both myopic and currently recommended application regimes were severely sub-optimal in terms of economic surplus losses. While the myopic solutions were near-optimal for planning horizons of less than 10 years, they diverged from optimality sharply for longer horizons. On the other hand, currently recommended regimes were substantially sub-optimal for all planning horizons. However, all the sub-optimal solutions were superior in terms of reduced total insecticide use, especially in the early years of a planning horizon. In fact, it was mainly because of reduced insecticide use that the myopic and recommended applications were sub-optimal. For a positive discount rate, both of these non-optimal regimes retarded susceptibility depletion, but only at the cost of higher pest populations than those experienced with optimal insecticide use. Comprehensive evaluations of a control technology set were generally shown to be preferred to chemical-by-chemical withdrawals, but mainly when planning horizons exceeded 10 years.

Regulatory Implications

Taken together, the information generated by the simulation model about the development of resistance under different scenarios suggested a number of important implications for the insecticide regulatory process. If the structure and function of the bioeconomic model used in this study is a sufficiently good approximation to reality, then the development of pest resistance to chemical controls in an agricultural production system is a complex dynamic phenomenon that leads to long-run economic losses when there is a static control-technology set. While current benefit assessments already make the simplifying assumption that there will be no pest control innovations, the process also assumes that the underlying biological systems in agriculture are at, and will remain in, steady-state. But if there is a potential for resistance in a system, then this latter assumption cannot be maintained. The result is that the static budgeting analysis usually used in the benefits assessment procedure may severely

underestimate the actual benefits associated with a continued chemical registration. When this situation occurs, the regulatory process needs to be restructured so as to incorporate in policy-making any dynamic changes that might occur. Thus, the concerns that originally motivated this study appear to be valid and potentially important to the continued viability of at least the mid-Atlantic apple production system.

Once it is recognized that time-dynamic impacts need to be evaluated as part of the insecticide regulatory process, difficulties arise concerning the choice of an appropriate planning horizon over which to examine the system. This study suggested that the negative economic impacts associated with resistance are relatively minor in the short-run, accelerate rapidly in both absolute and proportional terms over intermediate time-horizons of 10 to 20 years, and then tend to level off in absolute terms and decline in proportional terms over the long-run. If regulatory planning horizons of 1, 5, or 10 years are chosen, then the long-run benefits associated with the continued availability of a particular control technology set will be underestimated. But if intermediate horizons are chosen, then the long-run benefits associated with a technology set's continued use will be overestimated. Thus, the appropriate planning horizon might be one that encompasses the emergence of new control technology and the abandonment of the old control technology set. In some ways, this approach would be analogous to the time-to-elimination horizons calculated for exhaustible resource problems. For the mid-Atlantic apple production system, the appropriate planning period under single insecticide withdrawal scenarios appeared to be approximately 25 years. However, the exact length of the time-horizon may vary depending on the specific biology underlying the system and the point in the control technology's life-cycle at which the evaluation takes place. This suggests that when time-dynamic factors are important, regulatory procedures need to be based on detailed biological and chemical technology models capable of generating close approximations to agricultural system behavior. Ultimately, the desire to regulate insecticide use when there is a potential for pest resistance requires that the time-dynamics of system biology play a prominent role, along with current economic data, in the benefits assessment

process. Of course, this can only be accomplished if a significant investment is made in the necessary basic research and model development.

Along with the critical nature of the choice of planning horizon comes the need to choose between nominal and present-value benefits calculations. Although the current insecticide regulatory process is essentially based on static information, planning and regulation associated with many government-sponsored projects already mandate discounting. As a result, present-value discounting would likely be necessary if the benefits assessment process were to incorporate the dynamic effects of resistance. But, given discounting, the need arises to choose an appropriate discount rate. Results of this study suggest that the choice of a discount rate is important, for as discount rates increased, not only did the present-value of future economic surplus losses decline, but so did the proportional impact of those losses. In addition, the use of a higher discount rates was accompanied by temporal shifts in the use of insecticides, in essence leading to heavier chemical use early in each planning horizon. In general, perhaps the best approach would be to calculate potential benefits over a reasonable range of discount rates, thereby obtaining information on how sensitivity the benefit/risk regulatory trade-off is to discount rates for the particular system being analyzed. This procedure would tend to provide confidence in a particular regulatory action if the same decision arises over a variety of discount rates.

To this point, the discussion has concentrated on the implications of resistance for calculating the losses in benefits associated with a particular control technology set. However, the regulatory process usually focuses on what happens when the size of the control technology set changes. This study demonstrated that even single-chemical regulatory withdrawals can lead to large absolute changes in the expected economic surplus losses due to resistance. On a proportional basis, the additional losses fluctuated by planning horizon in much the same way as the losses experienced with a complete technology set. But, in the long-run, withdrawal of a single insecticide can nearly double the resistance-induced economic surplus losses. These results suggest that as the size of the control set decreases due to regulation, the time-dynamic effects of resistance will play an increasing important role in

a dynamic benefits assessment. In fact, not only does the withdrawal of an insecticide lead to higher resistance induced losses in the intermediate- and long-run, but it also increases short-run losses as early-horizon chemical use is curtailed in an apparent effort to mitigate some of the effects of resistance in intermediate-run. So, regardless of the chosen time-horizon, additional resistance-induced losses and temporal shifts in total chemical use following an insecticide withdrawal can be important factors on both sides of the benefit/risk calculation.

While the calculated impact of a general insecticide withdrawal appeared to be dynamically important enough for inclusion in a benefits assessment, the impact of any particular regulatory action was demonstrated to vary among and within chemical classes. For the mid-Atlantic apple production system, organophosphates were the most important insecticides to the short- and intermediate-run maintenance of economic surplus. For short horizons, the potential loss of azinphosmethyl or chlorpyrifos resulted in the highest levels of economic surplus loss due to resistance. Over intermediate horizons, loss of phosmet or microencapsulated methylparathion tended to yield the largest surplus losses. But in the long-run, the two non-organophosphate controls, methomyl and esfenvalerate, also emerged as important to the control of pest populations and thus resistance-induced surplus losses. The fact that the importance of a particular insecticide can vary with the chosen planning horizon, and also as a time progresses through a long planning horizon, implies that a benefits assessment cannot be accurately conducted on a chemical-by-chemical basis, isolated from long-run fluctuations in the use of insecticides not currently under scrutiny. This was further demonstrated by comparing the optimal solution associated with a simultaneous initial withdrawal of two insecticides to the optimal solution that arose when the same two insecticides were withdrawn, one at a time, over a 10 year interval. In the example generated by the simulation model, long-run resistance induced losses were substantially lower for the initial two-chemical withdrawal.

Research Recommendations

The research described in this dissertation, and the approach that was used to obtain empirical information, could be improved and expanded in a number of different ways. Perhaps the most obvious extension would be to examine other production systems to see if the results and implications generated by this study are widely applicable. Another improvement would be to move from deterministic simulations to stochastic ones. In doing so, it might be possible to obtain a more comprehensive estimate of how resistance and regulation interact when there are other short-term selection pressures, such as weather, acting on the pest and predator organisms. Of course, the size of the control technology set, and the type of controls that it includes, could be varied. Particularly interesting would be the inclusion of biological or cultural pest controls into the technology set. Unfortunately, the detailed information required for this improvement is only now being collected, and it may be a number of years before it is widely available.

The objective used for optimization in this study, namely total expected economic surplus, was relatively simple and easy to identify. However, the results subsequently obtained from model simulations suggested that total insecticide use might also be an important objective, particularly if the modelled regulatory process is expanded to encompass both benefits assessment and risk analysis. In addition, because insecticide use is not directly affected by discounting, it might be interesting to include the temporal allocation of insecticides as a part of the objective, especially if its optimal allocation is expected to conflict with the discounted parts of the objective, or if there are reasons to favor, but not require, the use of specific insecticides during particular time periods. These additions would change the problem into a multi-objective optimization, which, in theory, could still be handled by the simulation optimization technique that was presented.

With respect to the simulation and optimization procedure, various modifications might be incorporated to enhance speed and convergence properties. In this study, there generally

was a rapid movement of the simplex to the near-region in which the optimal solution resided. However, collapse of the simplex to the optimal solution itself was relatively slow process. It might be possible to program variable expansion, retraction, and reflection coefficients into the search procedure so that the process becomes more efficient as the relative movement of the simplex through the solution space slows. Preliminary attempts to make this kind of modification were not successful. But, perhaps the biggest factor determining the speed of convergence was the size of the biological simulator. Although the optimization process itself is quick relative to other search algorithms, it still depends on the generation of system responses by the simulator for each attempted simplex move. Any improvements on the operation of the simulator would greatly enhance overall performance.

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Appendix A. TABM SERA PROGRAM CODE

The following program code simulates the population dynamics and genetic evolution of the Tufted Apple Bud Moth, the primary pest in the mid-Atlantic apple production system, in response to environmental and chemical control factors. This section of code was structured as a sub-program and incorporated into the main optimization program contained in Appendix I. All simulations reported in this dissertation were executed on an IBM mainframe computer using the VSPASCAL programming language. Because of site-specific computing constraints, a number of the arrays in this program were disaggregated. Thus, while loosely conforming to structured programming conventions, the listed code contains some anomalies that could be rectified for use on other computing systems. Before listing the TABM program code, the important variables, data structures, and sub-procedures are defined. More extensive explanation and documentation can be obtained from the author.

Arrays

TOTRF[1..6]: Tracking the total number of reproductive females in the orchard, the array can be used in determining immigration from woods, other fruits, and other apple orchards.

RSDIS[1..6], RRDIS[1..6]: Defines the reproductive disadvantage associated with the resistant R gene.

THLIF[1..6]: Defines the half-life of each potential chemical control.

SS1[1..6], RS1[1..6], RR1[1..6]: Tracks the number of eggs produced per day for each of the genotypes.

WIMIG[1..6], FIMIG[1..6], AIMIG[1..6]: Tracks the immigration rates from woods, other fruits, and other apple orchards, respectively.

BEGN[1..20]: The initial distribution of overwintering populations.

FRACIN[1..20]: The fraction of each lifestage residing within the orchard and exposed to potential chemical controls.

PROP[1..10]: The normal distribution of 10 susceptibility classes for each genotype. Class 11 tracks the individuals in refugia during any time-step.

REFINIT[1..20]: Proportion of each life-stage that was initially in refugia.

SURV[1..19]: Defines the daily survival rates between substages.

RR[1..11,1..20,1..6], RS[1..11,1..20,1..6], SS[1..11,1..20,1..6]: Tracks the genotypic arrays by substage and potential chemical control.

STAREF[1..6]: Proportion of each lifestage within a stage-long refugia.

CRESIS[1..6,1..6]: Defines the relative ratio of cross-resistance among the potential chemical controls.

TOTAL[1..6,1..20]: Tracks the total number of individuals of all genotypes that are within each substage.

RGENE[1..6,1..20]: Tracks the number of resistant R genes within each substage.

RGENEFREQ[1..6,1..20]: Tracks the frequency of the resistant R gene within each substage.

WOODS[1..40]: Tracks the number of adults that emerge for each time-step of the simulation. The measure is based on the initial density and step mortality rates as specified in the input data files, and can be used to calculate the immigration rates from woods.

SS50[1..6,1..6], RS50[1..6,1..6], RR50[1..6,1..6], SS95[1..6,1..6], RS95[1..6,1..6], RR95[1..6,1..6]: Defines the lethal dose 50 and 95 levels for each potential chemical. The values are calculated from regression of log dose-probit curves.

WSURV[1..20]: Defines the overwintering survival rate of each substage.

Non-Array Variables

LE: Defines the last egg substage.

ISL: Defines the initial small larva substage.

LSL: Defines the last small larva substage.

IML: Defines the initial medium larva substage.

LML: Defines the last medium larva substage

ILL: Defines the initial large larva substage.

LLL: Defines the last large larva substage.

IP: Defines the initial pupa substage.

LP: Defines the last pupa substage.

IA: Defines the initial adult substage.

IRF: Defines the initial reproductive adult substage.

SXRAT: Defines the sex ratios for the adult population.

NCC1: Defines the first substage to be used in calculating the carrying capacity.

NCC2: Defines the last substage to be used in calculating the carrying capacity.

NUMYEARS: The number of years over which the simulation takes place.

NGENS: The number of species generations per simulation year.

NUMDAYSPERYEAR: The number of simulated days per simulation year, where 1 simulated day is equivalent to 4 real days.

FEC: The number of eggs laid per female per time-step.

CC: The carrying capacity of the environment, it is used to calculate density dependent mortality.

PINIT: The initial frequency of the R-gene in the population.

T: The total number of individuals in the population.

COUNTER: Tracks the index of the last chemical control to be used.

J: A counter variable usually employed to designate a substage.

K: A counter usually employed to designate a potential chemical control.

P: A counter variable also employed to designate a potential chemical control.

I: A counter usually employed to designate a susceptibility class.

Procedures

DATAINITIALIZE: Specifies the distribution of individuals into the 10 susceptibility classes. In addition, total R gene frequency, number of reproductive females, and the genotype arrays are zeroed.

GETLIBPARAMS: Reads, from the input data set, the parameters used for defining the species being simulated.

INIT: Initializes the genotype arrays and the R gene frequencies.

SUM: Summarizes the values of each life-stage and R gene frequencies over all potential chemical controls.

THRESH: Used to determine if population totals have exceeded the density dependent levels.

NEXT: Determines how much mating and oviposition occurs by genotype. Updates the numbers in the genotype arrays based on inter-substage survivorship and density dependent mortality.

REINIT: Uses Hardy-Weinberg equilibrium to redistribute individuals into the 3 genotype arrays based on the R gene frequencies.

KILL: Removes individuals from the genotype arrays based on susceptibility class, current control dosage, and the lethal dose 50 and 95 values.

UPDATE: Determines the new R gene frequencies by chemical following the KILL procedure.

WINTER: Removes individuals from genotype arrays based on winter survivorship rates.

MIGRATE: Determines the immigration of individuals by genotype.

EGGS: Adds the eggs from SS1, RS1, and RR1 to the first substage of the genotype arrays.

REFUGE: Assigns a portion of each lifestage into a stage-long refugia.

MAIN: Calls all the initializing, input, and output procedures, then sequentially executes two nested loops; one for the number of days in a simulation year, and one for the number of simulation years. For each simulated day, the program tests to see if a control application is to be made. If so, then the environmental dose of the control is updated, and the current residue used to calculate the control-induced mortality. After the mortality is calculated, all arrays are updated. If more than one chemical is used, arrays are also reinitialized using the Hardy-Weinberg formulas. Next, oviposition is determined and individuals are incremented one substage in age. Immigrants and their eggs are then added. Lastly, individuals are assigned to refugia, and numbers and gene frequencies updated. After a single years simulation is completed, overwintering mortality is calculated and all variables adjusted for the initial run of any subsequent year.

```
PROCEDURE TABMSIMULATOR;

 TYPE

 CHEMAGE   = @CA;
 CA        = RECORD
             CONTROLARRAY: ARRAY [1..NUMPESTICIDES, 1..20] OF REAL;
             END;

 A6BY6OFREAL  = @FIRST;
 FIRST        = RECORD
                ARRAY6X6: ARRAY[1..6, 1..6] OF REAL;
                END;

 A11BY20BY6OFREAL = @SECOND;
 SECOND           = RECORD
                    ARRAY11X20X6: ARRAY[1..11, 1..20, 1..6] OF REAL;
                    END;

 A1BY6OFREAL   = @THIRD;
 THIRD         = RECORD
                 ARRAY1X6: ARRAY[1..6] OF REAL;
                 END;

 A1BY20OFREAL  = @FOURTH;
 FOURTH        = RECORD
                 ARRAY1X20: ARRAY[1..20] OF REAL;
                 END;

 A6BY20OFREAL  = @FIFTH;
 FIFTH         = RECORD
                 ARRAY6X20: ARRAY[1..6, 1..20] OF REAL;
                 END;

 A1BYNUMCHEMOFREAL = @SIXTH;
 SIXTH             = RECORD
                     ARRAY1XNUMCHEM: ARRAY[1..NUMPESTICIDES] OF REAL;
                     END;

 A1BYNUMDAYSPERYEAROFREAL = @SEVENTH;
 SEVENTH                  = RECORD
                            ARRAY1XNUMDAYSPERYEAR:
                            ARRAY[1..NUMDAYSPERYEAR] OF REAL;
                            END;

VAR

 I, IA, ISL, IP, IRF, LE, LSL, LP, IML, BEFORE,
 LML, ILL, LLL, NCC1, NCC2, KORIG, IYR, IDAY,
 NUMRUNS, SIMNUMYEARS                    : INTEGER;

 ADUL, CC, DINC, EGG, FEC, PINIT, PUP, Q,
 TNOEGGS, SXRAT, T, TLARV, TIMIG, MAXT,
 TABMTOTALS                              : REAL;

 TOTRF, RSDIS, RRDIS, CD, RFREQ, THLIF,
 SS1, RS1, RR1, TOTRFREQ, WIMIG, FIMIG, AIMIG,
 STAREF                                  : A1BY6OFREAL;

 BEGN, FRACIN, REFINIT, SURV             : A1BY20OFREAL;

 PROP                                    : ARRAY [1..10] OF REAL;

 RR, RS, SS                              : A11BY20BY6OFREAL;

 CRESIS                                  : A6BY6OFREAL;

 TOTAL, RGENE, RGENEFREQ                 : A6BY20OFREAL;

 WOODS                                   : A1BYNUMDAYSPERYEAROFREAL;
```

```
 IGD, IGEN, INTRVL, L, K, M, N, NDAYS, NGENS,
 NTHR1, NTHR2, NYRS, INITIAL, B, COUNTER,
 NEWYR, NEWIDAY                          : INTEGER;

 ETHRESH, STEP, TIMMGEN, TEST, IMIGFREQ      : REAL;

 EARLY, OW, SIGNAL                       : BOOLEAN;

 SSD50, RSD50, RRD50, SSD95, RSD95, RRD95    : A6BY6OFREAL;

 WSURV                                   : A1BY20OFREAL;

 NAME                                    : PACKED ARRAY [1..20] OF CHAR;

LABEL 187, 240, 250;


PROCEDURE DATAINITIALIZE;

 VAR

  I, J, P: INTEGER;

 BEGIN

  IGEN : = 1;
  IDAY : = 0;
  IYR : = 1;
  PROP[1] : = 0.001;
  PROP[2] : = 0.012;
  PROP[3] : = 0.054;
  PROP[4] : = 0.16;
  PROP[5] : = 0.273;
  PROP[6] : = 0.273;
  PROP[7] : = 0.16;
  PROP[8] : = 0.054;
  PROP[9] : = 0.012;
  PROP[10] : = 0.001;

  FOR I : = 1 TO 6 DO
   BEGIN
    CD@.ARRAY1X6[I] : = 0.0;
    TOTRFREQ@.ARRAY1X6[I] : = 0.0;
   END;
  FOR I : =1 TO CURRENTNUMCHEMS DO TOTRF@.ARRAY1X6[I] : = 0.0;
  FOR P:= 1 TO CURRENTNUMCHEMS DO
   FOR J : = 1 TO 20 DO
    FOR I : = 1 TO 11 DO
     BEGIN
      SS@.ARRAY11X20X6[I,J,P] : = 0.0;
      RS@.ARRAY11X20X6[I,J,P] : = 0.0;
      RR@.ARRAY11X20X6[I,J,P] : = 0.0;
     END;

 END; {Procedure DATAINITIALIZE}


PROCEDURE GETLIBPARAMS;

 VAR

  NUM, I, J, CHOICE, SELECTION: INTEGER;

  PROCEDURE INA(VAR A: A1BY20OFREAL);

  VAR

   I, J: INTEGER;

  BEGIN

   FOR I : = 1 TO 20 DO READ(PARAMS1, A@.ARRAY1X20[I]);
```

```
  READLN(PARAMS1);

 END;  {Procedure INA}

BEGIN  {GETLIBPARAMS}

 IF NUMRUNS = 1
  THEN RESET(PARAMS1,'NAME = SERA1.DAT.B')
  ELSE RESET(PARAMS1,'NAME = SERA1SS.DAT.B');

 READLN(PARAMS1, LE, ISL, LSL, IML, LML, ILL, LLL, IP, LP, IA, IRF,
   SXRAT, NCC1, NCC2, NTHR1, NTHR2, IMIGFREQ, NYRS, NGENS, NDAYS);
 INA(BEGN);
 INA(REFINIT);
 FOR I := 1 TO 19 DO  READ(PARAMS1, SURV@.ARRAY1X20[I]);
 READLN(PARAMS1);
 INA(WSURV);
 INA(FRACIN);
 FOR I := 1 TO 40 DO  READ (PARAMS1,WOODS@.ARRAY1XNUMDAYSPERYEAR[I]);
 READLN (PARAMS1);
 FOR I := 1 TO 6 DO
  BEGIN
   FOR J := 1 TO NUMPESTICIDES DO
    READ(PARAMS1, CRESIS@.ARRAY6X6[I,J]);
   READLN(PARAMS1);
  END;
 FOR I := 1 TO 6 DO  READ(PARAMS1, STAREF@.ARRAY1X6[I]);
 READLN(PARAMS1);
 READLN(PARAMS1, DINC, FEC,INTRVL,CC, ETHRESH, PINIT, T);
 FOR I := 1 TO 6 DO  READ(PARAMS1, THLIF@.ARRAY1X6[I]);
 READLN(PARAMS1);
 FOR I := 1 TO 6 DO
  READ(PARAMS1, RSDIS@.ARRAY1X6[I], RRDIS@.ARRAY1X6[I]);
 READLN(PARAMS1);
 FOR I := 1 TO 6 DO
  BEGIN
   READ(PARAMS1, WIMIG@.ARRAY1X6[I], FIMIG@.ARRAY1X6[I]);
   READ(PARAMS1, AIMIG@.ARRAY1X6[I]);
  END;
 READLN(PARAMS1);
 FOR I := 1 TO 6 DO
  BEGIN
   FOR J := 1 TO 3 DO
    BEGIN
     READ(PARAMS1, SSD50@.ARRAY6X6[J,I], RSD50@.ARRAY6X6[J,I]);
     READ(PARAMS1, RRD50@.ARRAY6X6[J,I]);
    END;
   READLN(PARAMS1);
   FOR J := 4 TO 6 DO
    BEGIN
     READ(PARAMS1, SSD50@.ARRAY6X6[J,I], RSD50@.ARRAY6X6[J,I]);
     READ(PARAMS1, RRD50@.ARRAY6X6[J,I]);
    END;
   READLN(PARAMS1);
  END;
 FOR I := 1 TO 6 DO
  BEGIN
   FOR J := 1 TO 3 DO
    BEGIN
     READ(PARAMS1, SSD95@.ARRAY6X6[J,I], RSD95@.ARRAY6X6[J,I]);
     READ(PARAMS1, RRD95@.ARRAY6X6[J,I]);
    END;
   READLN(PARAMS1);
   FOR J := 4 TO 6 DO
    BEGIN
     READ(PARAMS1, SSD95@.ARRAY6X6[J,I], RSD95@.ARRAY6X6[J,I]);
     READ(PARAMS1, RRD95@.ARRAY6X6[J,I]);
    END;
   READLN(PARAMS1);
  END;

END;  {GETLIBPARAMS}
```

```
PROCEDURE INIT;

VAR

 TEMPSS, TEMPRS, TEMPRR   : A1BYNUMCHEMOFREAL;
 T1, T2                   : REAL;
 I, J, P, N1              : INTEGER;

BEGIN

 NEW(TEMPSS);
 NEW(TEMPRS);
 NEW(TEMPRR);
 EARLY : = TRUE;

 FOR P : = 1 TO CURRENTNUMCHEMS DO
  BEGIN
   FOR J : = 1 TO 20 DO
    BEGIN
     TOTAL@.ARRAY6X20[P,J] : = 0.0;
     RGENE@.ARRAY6X20[P,J] : = 0.0;
     RGENEFREQ@.ARRAY6X20[P,J] : = PINIT;
    END;
   TOTRFREQ@.ARRAY1X6[P] : = PINIT;
  END;

 Q : = 1.0 - PINIT;
 T1 : = Q * Q * T;
 T2 : = PINIT * PINIT;
 TIMIG : = 0.0;
 IF T1 < 0.00000000000000000001 THEN T1 : = 0.0;

 FOR P : = 1 TO CURRENTNUMCHEMS DO
  FOR J : = 1 TO 20 DO
   BEGIN
    TEMPSS@.ARRAY1XNUMCHEM[P] : = T1*BEGN@.ARRAY1X20[J]
      *(1.0-REFINIT@.ARRAY1X20[J]);
    TEMPRS@.ARRAY1XNUMCHEM[P] : = (2.0*PINIT*Q)*T
      *BEGN@.ARRAY1X20[J]*(1.0-REFINIT@.ARRAY1X20[J]);
    TEMPRR@.ARRAY1XNUMCHEM[P] : = T2*T
      *BEGN@.ARRAY1X20[J]*(1.0-REFINIT@.ARRAY1X20[J]);

    SS@.ARRAY11X20X6[1,J,P] : =
      TEMPSS@.ARRAY1XNUMCHEM[P]*PROP[1];
    RS@.ARRAY11X20X6[1,J,P] : =
      TEMPRS@.ARRAY1XNUMCHEM[P]*PROP[1];
    RR@.ARRAY11X20X6[1,J,P] : =
      TEMPRR@.ARRAY1XNUMCHEM[P]*PROP[1];
    SS@.ARRAY11X20X6[2,J,P] : =
      TEMPSS@.ARRAY1XNUMCHEM[P]*PROP[2];
    RS@.ARRAY11X20X6[2,J,P] : =
      TEMPRS@.ARRAY1XNUMCHEM[P]*PROP[2];
    RR@.ARRAY11X20X6[2,J,P] : =
      TEMPRR@.ARRAY1XNUMCHEM[P]*PROP[2];
    SS@.ARRAY11X20X6[3,J,P] : =
      TEMPSS@.ARRAY1XNUMCHEM[P]*PROP[3];
    RS@.ARRAY11X20X6[3,J,P] : =
      TEMPRS@.ARRAY1XNUMCHEM[P]*PROP[3];
    RR@.ARRAY11X20X6[3,J,P] : =
      TEMPRR@.ARRAY1XNUMCHEM[P]*PROP[3];
    SS@.ARRAY11X20X6[4,J,P] : =
      TEMPSS@.ARRAY1XNUMCHEM[P]*PROP[4];
    RS@.ARRAY11X20X6[4,J,P] : =
      TEMPRS@.ARRAY1XNUMCHEM[P]*PROP[4];
    RR@.ARRAY11X20X6[4,J,P] : =
      TEMPRR@.ARRAY1XNUMCHEM[P]*PROP[4];
    SS@.ARRAY11X20X6[5,J,P] : =
      TEMPSS@.ARRAY1XNUMCHEM[P]*PROP[5];
    RS@.ARRAY11X20X6[5,J,P] : =
      TEMPRS@.ARRAY1XNUMCHEM[P]*PROP[5];
```

```
    RR@.ARRAY11X20X6[5,J,P] : =
      TEMPRR@.ARRAY1XNUMCHEM[P]*PROP[5];
    SS@.ARRAY11X20X6[6,J,P] : =
      TEMPSS@.ARRAY1XNUMCHEM[P]*PROP[6];
    RS@.ARRAY11X20X6[6,J,P] : =
      TEMPRS@.ARRAY1XNUMCHEM[P]*PROP[6];
    RR@.ARRAY11X20X6[6,J,P] : =
      TEMPRR@.ARRAY1XNUMCHEM[P]*PROP[6];
    SS@.ARRAY11X20X6[7,J,P] : =
      TEMPSS@.ARRAY1XNUMCHEM[P]*PROP[7];
    RS@.ARRAY11X20X6[7,J,P] : =
      TEMPRS@.ARRAY1XNUMCHEM[P]*PROP[7];
    RR@.ARRAY11X20X6[7,J,P] : =
      TEMPRR@.ARRAY1XNUMCHEM[P]*PROP[7];
    SS@.ARRAY11X20X6[8,J,P] : =
      TEMPSS@.ARRAY1XNUMCHEM[P]*PROP[8];
    RS@.ARRAY11X20X6[8,J,P] : =
      TEMPRS@.ARRAY1XNUMCHEM[P]*PROP[8];
    RR@.ARRAY11X20X6[8,J,P] : =
      TEMPRR@.ARRAY1XNUMCHEM[P]*PROP[8];
    SS@.ARRAY11X20X6[9,J,P] : =
      TEMPSS@.ARRAY1XNUMCHEM[P]*PROP[9];
    RS@.ARRAY11X20X6[9,J,P] : =
      TEMPRS@.ARRAY1XNUMCHEM[P]*PROP[9];
    RR@.ARRAY11X20X6[9,J,P] : =
      TEMPRR@.ARRAY1XNUMCHEM[P]*PROP[9];
    SS@.ARRAY11X20X6[10,J,P] : =
      TEMPSS@.ARRAY1XNUMCHEM[P]*PROP[10];
    RS@.ARRAY11X20X6[10,J,P] : =
      TEMPRS@.ARRAY1XNUMCHEM[P]*PROP[10];
    RR@.ARRAY11X20X6[10,J,P] : =
      TEMPRR@.ARRAY1XNUMCHEM[P]*PROP[10];

   END;
  FOR P:= 1 TO CURRENTNUMCHEMS DO
   FOR J : = 1 TO 20 DO
    BEGIN
     SS@.ARRAY11X20X6[11,J,P] : = T1*BEGN@.ARRAY1X20[J]
       *REFINIT@.ARRAY1X20[J];
     RS@.ARRAY11X20X6[11,J,P] : = 2.0*PINIT*Q*T
       *BEGN@.ARRAY1X20[J]*REFINIT@.ARRAY1X20[J];
     RR@.ARRAY11X20X6[11,J,P] : = T2*T
       *BEGN@.ARRAY1X20[J]*REFINIT@.ARRAY1X20[J];
    END;
  FOR P : = 1 TO CURRENTNUMCHEMS DO
   FOR J : = 1 TO 20 DO
    BEGIN
     FOR I : = 1 TO 11 DO
      BEGIN
       TOTAL@.ARRAY6X20[P,J] : = TOTAL@.ARRAY6X20[P,J]
         +SS@.ARRAY11X20X6[I,J,P]+RS@.ARRAY11X20X6[I,J,P]
         +RR@.ARRAY11X20X6[I,J,P];
       RGENE@.ARRAY6X20[P,J] : = RGENE@.ARRAY6X20[P,J]
         +RR@.ARRAY11X20X6[I,J,P]+(0.5*RS@.ARRAY11X20X6[I,J,P]);
      END; {I Loop}
     IF (TOTAL@.ARRAY6X20[P,J] > 0.0)
      THEN RGENEFREQ@.ARRAY6X20[P,J] : = RGENE@.ARRAY6X20[P,J]
        /TOTAL@.ARRAY6X20[P,J]
      ELSE RGENEFREQ@.ARRAY6X20[P,J] : = 0.0;
    END; {J and P Loop}

  DISPOSE(TEMPRR);
  DISPOSE(TEMPRS);
  DISPOSE(TEMPSS);

 END; {Procedure INIT}


PROCEDURE SUM;

 VAR
```

```
 I, J, P, J1                    : INTEGER;

 TOT, TOTRGENE, TSS, TRS, TRR, A      : A1BYNUMCHEMOFREAL;

 DIFF, AGENEFREQ, TEMPRGENE, TEMPRFREQ  : CHEMAGE;

BEGIN

 NEW(DIFF);
 NEW(AGENEFREQ);
 NEW(TEMPRGENE);
 NEW(TEMPRFREQ);
 NEW(TOT);
 NEW(TOTRGENE);
 NEW(TSS);
 NEW(TRS);
 NEW(TRR);
 NEW(A);

 FOR P : = 1 TO CURRENTNUMCHEMS DO
  BEGIN
   TSS@.ARRAY1XNUMCHEM[P] : = 0.0;
   TRS@.ARRAY1XNUMCHEM[P] : = 0.0;
   TRR@.ARRAY1XNUMCHEM[P] : = 0.0;
  END;
IF (EARLY = TRUE) THEN COUNTER : = 1;
EGG : = 0.0;
TLARV : = 0.0;
PUP : = 0.0;
ADUL : = 0.0;

 FOR I : = 1 TO 11 DO
  BEGIN
   FOR J : = 1 TO LE DO
    EGG : = EGG + SS@.ARRAY11X20X6[I,J,COUNTER]
      + RS@.ARRAY11X20X6[I,J,COUNTER]
      + RR@.ARRAY11X20X6[I,J,COUNTER];
   FOR J : = ISL TO LLL DO
    TLARV : = TLARV + SS@.ARRAY11X20X6[I,J,COUNTER]
      + RS@.ARRAY11X20X6[I,J,COUNTER]
      + RR@.ARRAY11X20X6[I,J,COUNTER];
   FOR J : = IP TO LP DO
    PUP : = PUP + SS@.ARRAY11X20X6[I,J,COUNTER]
      + RS@.ARRAY11X20X6[I,J,COUNTER]
      + RR@.ARRAY11X20X6[I,J,COUNTER];
   FOR J : = IA TO 20 DO
    ADUL : = ADUL + SS@.ARRAY11X20X6[I,J,COUNTER]
      + RS@.ARRAY11X20X6[I,J,COUNTER]
      + RR@.ARRAY11X20X6[I,J,COUNTER];
  END; {I Loop}

FOR P : = 1 TO CURRENTNUMCHEMS DO
 FOR J : = 1 TO 20 DO
  FOR I : =1 TO 11 DO
   BEGIN
    TSS@.ARRAY1XNUMCHEM[P] : = TSS@.ARRAY1XNUMCHEM[P]
      + SS@.ARRAY11X20X6[I,J,P];
    TRS@.ARRAY1XNUMCHEM[P] : = TRS@.ARRAY1XNUMCHEM[P]
      + RS@.ARRAY11X20X6[I,J,P];
    TRR@.ARRAY1XNUMCHEM[P] : = TRR@.ARRAY1XNUMCHEM[P]
      + RR@.ARRAY11X20X6[I,J,P];
   END;
T : = TSS@.ARRAY1XNUMCHEM[COUNTER] + TRS@.ARRAY1XNUMCHEM[COUNTER]
  + TRR@.ARRAY1XNUMCHEM[COUNTER];

IF TLARV > MAXT THEN MAXT : = TLARV;
IF ((EARLY = FALSE) AND (OW = FALSE)) THEN
 BEGIN
  FOR J : = 1 TO 20 DO
   BEGIN
    TOTAL@.ARRAY6X20[COUNTER,J] : = 0.0;
    TEMPRGENE@.CONTROLARRAY[COUNTER,J] : = 0.0;
```

```
    END;
   FOR J:= 1 TO 20 DO
    BEGIN
     FOR I : = 1 TO 11 DO
      BEGIN
       TOTAL@.ARRAY6X20[COUNTER,J] : = TOTAL@.ARRAY6X20[COUNTER,J]
         + SS@.ARRAY11X20X6[I,J,COUNTER]
         + RS@.ARRAY11X20X6[I,J,COUNTER]
         + RR@.ARRAY11X20X6[I,J,COUNTER];
       TEMPRGENE@.CONTROLARRAY[COUNTER,J] :=
         TEMPRGENE@.CONTROLARRAY[COUNTER,J]
         + RR@.ARRAY11X20X6[I,J,COUNTER]
         + (0.5*RS@.ARRAY11X20X6[I,J,COUNTER]);
      END: {I Loop}
     IF (TOTAL@.ARRAY6X20[COUNTER,J] = 0.0)
      THEN TEMPRFREQ@.CONTROLARRAY[COUNTER,J] : = 0.0
      ELSE TEMPRFREQ@.CONTROLARRAY[COUNTER,J] : =
        TEMPRGENE@.CONTROLARRAY[COUNTER,J]
        / TOTAL@.ARRAY6X20[COUNTER,J];
    END: {J Loop}
   FOR P : = 1 TO CURRENTNUMCHEMS DO
    BEGIN
     A@.ARRAY1XNUMCHEM[P] : = 0.0;
     AGENEFREQ@.CONTROLARRAY[P,1] : = 0.0;
     FOR J : = IRF TO 20 DO
      BEGIN
       IF (RGENEFREQ@.ARRAY6X20[P,J] > 0.0) THEN
        BEGIN
         AGENEFREQ@.CONTROLARRAY[P,1] : =
           AGENEFREQ@.CONTROLARRAY[P,1]
           + RGENEFREQ@.ARRAY6X20[P,J];
         A@.ARRAY1XNUMCHEM[P] : = A@.ARRAY1XNUMCHEM[P] + 1.0;
        END;
      END;
     FOR J1 : = 1 TO 19 DO
      BEGIN
       J : = 21 -J1;
       DIFF@.CONTROLARRAY[P,J] : =
         TEMPRFREQ@.CONTROLARRAY[COUNTER,J]
         - RGENEFREQ@.ARRAY6X20[COUNTER,J-1];
      END: {J1 Loop}
    END: {P Loop}
   FOR P : = 1 TO CURRENTNUMCHEMS DO
    BEGIN
     FOR J1 : = 1 TO 19 DO
      BEGIN
       J : = 21-J1;
       RGENEFREQ@.ARRAY6X20[P,J] : = RGENEFREQ@.ARRAY6X20[P,J-1]
         + DIFF@.CONTROLARRAY[P,J];
       IF (RGENEFREQ@.ARRAY6X20[P,J] < 0.0) THEN
         RGENEFREQ@.ARRAY6X20[P,J] : = 0.0;
       IF (RGENEFREQ@.ARRAY6X20[P,J] > 1.0) THEN
         RGENEFREQ@.ARRAY6X20[P,J] : = 1.0;
      END;
     IF (A@.ARRAY1XNUMCHEM[P] > 0.0)
      THEN RGENEFREQ@.ARRAY6X20[P,1] : =
        AGENEFREQ@.CONTROLARRAY[P,1]/A@.ARRAY1XNUMCHEM[P]
      ELSE RGENEFREQ@.ARRAY6X20[P,1] : = 0.0;
       IF (RGENEFREQ@.ARRAY6X20[P,J] > 1.0) THEN
         RGENEFREQ@.ARRAY6X20[P,J] : = 1.0;
    END: {P Loop}
   FOR P : = 1 TO CURRENTNUMCHEMS DO
    FOR J : = 1 TO 20 DO
     IF (TOTAL@.ARRAY6X20[COUNTER,J] = 0.0)
      THEN RGENE@.ARRAY6X20[P,J] : = 0.0
      ELSE RGENE@.ARRAY6X20[P,J] : = RGENEFREQ@.ARRAY6X20[P,J]
        *TOTAL@.ARRAY6X20[COUNTER,J];
  END: {Early loop}

IF (OW = TRUE) THEN
 BEGIN
  FOR J : = 1 TO 20 DO
```

```
  BEGIN
   TOTAL@.ARRAY6X20[COUNTER,J] : = 0.0;
   TEMPRGENE@.CONTROLARRAY[COUNTER,J] : = 0.0;
  END;
 FOR J:= 1 TO 20 DO
  BEGIN
   FOR I : = 1 TO 11 DO
    BEGIN
     TOTAL@.ARRAY6X20[COUNTER,J] : = TOTAL@.ARRAY6X20[COUNTER,J]
       + SS@.ARRAY11X20X6[I,J,COUNTER]
       + RS@.ARRAY11X20X6[I,J,COUNTER]
       + RR@.ARRAY11X20X6[I,J,COUNTER];
     TEMPRGENE@.CONTROLARRAY[COUNTER,J] :=
       TEMPRGENE@.CONTROLARRAY[COUNTER, J]
       + RR@.ARRAY11X20X6[I,J,COUNTER]
       + (0.5 * RS@.ARRAY11X20X6[I,J,COUNTER]);
    END: {I Loop}
   IF (TOTAL@.ARRAY6X20[COUNTER,J] = 0.0)
    THEN TEMPRFREQ@.CONTROLARRAY[COUNTER,J] : = 0.0
    ELSE TEMPRFREQ@.CONTROLARRAY[COUNTER,J] : =
      TEMPRGENE@.CONTROLARRAY[COUNTER,J]
      /TOTAL@.ARRAY6X20[COUNTER,J];
  END: {J Loop}
 FOR P : = 1 TO CURRENTNUMCHEMS DO
  BEGIN
   FOR J : = 1 TO 20 DO
    BEGIN
     DIFF@.CONTROLARRAY[P,J] : =
       TEMPRFREQ@.CONTROLARRAY[COUNTER,J]
       - RGENEFREQ@.ARRAY6X20[COUNTER,J];
     RGENEFREQ@.ARRAY6X20[P,J] : = RGENEFREQ@.ARRAY6X20[P,J]
       + DIFF@.CONTROLARRAY[P,J];
     IF (RGENEFREQ@.ARRAY6X20[P,J] < 0.0) THEN
       RGENEFREQ@.ARRAY6X20[P,J] : = 0.0;
     IF (RGENEFREQ@.ARRAY6X20[P,J] > 1.0) THEN
       RGENEFREQ@.ARRAY6X20[P,J] : = 1.0;
    END;
  END;
 FOR P : = 1 TO CURRENTNUMCHEMS DO
  FOR J : = 1 TO 20 DO
   IF (TOTAL@.ARRAY6X20[COUNTER,J] = 0.0)
    THEN RGENE@.ARRAY6X20[P,J] : = 0.0
    ELSE RGENE@.ARRAY6X20[P,J] : = RGENEFREQ@.ARRAY6X20[P,J]
      *TOTAL@.ARRAY6X20[COUNTER,J];
END: {OW Loop}

FOR P : = 1 TO CURRENTNUMCHEMS DO
 BEGIN
  TOT@.ARRAY1XNUMCHEM[P] : = 0.0;
  TOTRGENE@.ARRAY1XNUMCHEM[P] : = 0.0;
  FOR J : = 1 TO 20 DO
   BEGIN
    TOT@.ARRAY1XNUMCHEM[P] : = TOT@.ARRAY1XNUMCHEM[P]
      + TOTAL@.ARRAY6X20[COUNTER,J];
    TOTRGENE@.ARRAY1XNUMCHEM[P] : = TOTRGENE@.ARRAY1XNUMCHEM[P]
      + RGENE@.ARRAY6X20[P,J];
   END;
  IF (TOT@.ARRAY1XNUMCHEM[P] > 0.0)
   THEN TOTRFREQ@.ARRAY1X6[P] : = TOTRGENE@.ARRAY1XNUMCHEM[P]
     /TOT@.ARRAY1XNUMCHEM[P]
   ELSE TOTRFREQ@.ARRAY1X6[P] : = PINIT;
  IF (TOTRFREQ@.ARRAY1X6[P] < PINIT)
   THEN TOTRFREQ@.ARRAY1X6[P] : = PINIT;
 END;

 IF (IYR MOD 50) = 0 THEN
 BEGIN
  WRITE(FIRSTPOP,VERTEXNUMBER:4,IDAY:8);
  WRITE(FIRSTPOP,EGG:12:2,TLARV:11:2,PUP:11:2);
  WRITELN(FIRSTPOP,ADUL:11:2,T:11:2);
 END;
```

```
 IF IDAY = 40 THEN
  BEGIN
   WRITE(RESIST,'  ',VERTEXNUMBER:2,'      ',IYR:2,'   ');
   CHEMNUMBER : = 0;
   FOR P : = 1 TO NUMPESTICIDES DO
    IF P IN CURRENTCHEMS
     THEN
      BEGIN
       CHEMNUMBER : = CHEMNUMBER + 1;
       WRITE(RESIST,' ',TOTRFREQ@.ARRAY1X6[CHEMNUMBER]:8:6);
      END
     ELSE
      WRITE(RESIST,'         ');
   WRITELN(RESIST);
   WRITE(POPS,VERTEXNUMBER:4,IYR:8);
   WRITE(POPS,EGG:12:2,TLARV:11:2,PUP:11:2);
   WRITELN(POPS,ADUL:11:2,T:11:2,MAXT:11:2);
  END;

 DISPOSE(DIFF);
 DISPOSE(AGENEFREQ);
 DISPOSE(TEMPRGENE);
 DISPOSE(TEMPRFREQ);
 DISPOSE(TOT);
 DISPOSE(TOTRGENE);
 DISPOSE(TSS);
 DISPOSE(TRS);
 DISPOSE(TRR);
 DISPOSE(A);

END;  {Procedure SUM}


PROCEDURE THRESH(N1, N2, P: INTEGER; VAR TEST: REAL);

 VAR

  I, J  : INTEGER;

 BEGIN

  FOR I : = 1 TO 11 DO
   FOR J : = N1 TO N2 DO
    TEST : = TEST+SS@.ARRAY11X20X6[I,J,P]+RS@.ARRAY11X20X6[I,J,P]
      +RR@.ARRAY11X20X6[I,J,P];

 END;  {Procedure THRESH}


PROCEDURE NEXT;

 VAR

  MID, T1, T2, T3, TEMP, CCTEST, DDS, T4, T5, T6  : REAL;

  TOTM, SSF, RSF, RRF, SSM, RSM, RRM              : A1BYNUMCHEMOFREAL;

  I, J, J1, P                                     : INTEGER;

  TEMPSS, TEMPRS, TEMPRR                          : CHEMAGE;

 BEGIN

  NEW(TEMPSS);
  NEW(TEMPRS);
  NEW(TEMPRR);
  NEW(TOTM);
  NEW(SSF);
  NEW(RSF);
  NEW(RRF);
  NEW(SSM);
  NEW(RSM);
```

```
NEW(RRM);

FOR P : = 1 TO CURRENTNUMCHEMS DO
 BEGIN                    {Begin re-init proc}
  FOR J : = IRF TO 20 DO
   BEGIN
    TOTAL@.ARRAY6X20[P,J] : = 0.0;
    FOR I : = 1 TO 11 DO
     TOTAL@.ARRAY6X20[P,J] : = TOTAL@.ARRAY6X20[P,J]
       + SS@.ARRAY11X20X6[I,J,P] + RS@.ARRAY11X20X6[I,J,P]
       + RR@.ARRAY11X20X6[I,J,P];
   END;  {J Loop}
  FOR J : = IRF TO 20 DO
   BEGIN
    Q : = 1.0 - RGENEFREQ@.ARRAY6X20[P,J];
    T4 : = Q * Q;
    T5 : = RGENEFREQ@.ARRAY6X20[P,J] * RGENEFREQ@.ARRAY6X20[P,J];
    T6 : = 2 * RGENEFREQ@.ARRAY6X20[P,J] * Q;
    IF T4 < 0.00000000000000000001 THEN T4 : = 0.0;
    IF T5 < 0.00000000000000000001 THEN T5 : = 0.0;
    IF T6 < 0.00000000000000000001 THEN T6 : = 0.0;
    TEMPSS@.CONTROLARRAY[P,J] : = T4*TOTAL@.ARRAY6X20[P,J]
      *(1.0-STAREF@.ARRAY1X6[6]);
    TEMPRS@.CONTROLARRAY[P,J] : = T6*TOTAL@.ARRAY6X20[P,J]
      *(1.0-STAREF@.ARRAY1X6[6]);
    TEMPRR@.CONTROLARRAY[P,J] : = T5*TOTAL@.ARRAY6X20[P,J]
      *(1.0-STAREF@.ARRAY1X6[6]);
    SS@.ARRAY11X20X6[11,J,P] : = T4*TOTAL@.ARRAY6X20[P,J]
      *STAREF@.ARRAY1X6[6];
    RS@.ARRAY11X20X6[11,J,P] : = T6*TOTAL@.ARRAY6X20[P,J]
      *STAREF@.ARRAY1X6[6];
    RR@.ARRAY11X20X6[11,J,P] : = T5*TOTAL@.ARRAY6X20[P,J]
      *STAREF@.ARRAY1X6[6];

    SS@.ARRAY11X20X6[1,J,P] : = TEMPSS@.CONTROLARRAY[P,J]
      *PROP[1];
    RS@.ARRAY11X20X6[1,J,P] : = TEMPRS@.CONTROLARRAY[P,J]
      *PROP[1];
    RR@.ARRAY11X20X6[1,J,P] : = TEMPRR@.CONTROLARRAY[P,J]
      *PROP[1];
    SS@.ARRAY11X20X6[2,J,P] : = TEMPSS@.CONTROLARRAY[P,J]
      *PROP[2];
    RS@.ARRAY11X20X6[2,J,P] : = TEMPRS@.CONTROLARRAY[P,J]
      *PROP[2];
    RR@.ARRAY11X20X6[2,J,P] : = TEMPRR@.CONTROLARRAY[P,J]
      *PROP[2];
    SS@.ARRAY11X20X6[3,J,P] : = TEMPSS@.CONTROLARRAY[P,J]
      *PROP[3];
    RS@.ARRAY11X20X6[3,J,P] : = TEMPRS@.CONTROLARRAY[P,J]
      *PROP[3];
    RR@.ARRAY11X20X6[3,J,P] : = TEMPRR@.CONTROLARRAY[P,J]
      *PROP[3];
    SS@.ARRAY11X20X6[4,J,P] : = TEMPSS@.CONTROLARRAY[P,J]
      *PROP[4];
    RS@.ARRAY11X20X6[4,J,P] : = TEMPRS@.CONTROLARRAY[P,J]
      *PROP[4];
    RR@.ARRAY11X20X6[4,J,P] : = TEMPRR@.CONTROLARRAY[P,J]
      *PROP[4];
    SS@.ARRAY11X20X6[5,J,P] : = TEMPSS@.CONTROLARRAY[P,J]
      *PROP[5];
    RS@.ARRAY11X20X6[5,J,P] : = TEMPRS@.CONTROLARRAY[P,J]
      *PROP[5];
    RR@.ARRAY11X20X6[5,J,P] : = TEMPRR@.CONTROLARRAY[P,J]
      *PROP[5];
    SS@.ARRAY11X20X6[6,J,P] : = TEMPSS@.CONTROLARRAY[P,J]
      *PROP[6];
    RS@.ARRAY11X20X6[6,J,P] : = TEMPRS@.CONTROLARRAY[P,J]
      *PROP[6];
    RR@.ARRAY11X20X6[6,J,P] : = TEMPRR@.CONTROLARRAY[P,J]
      *PROP[6];
    SS@.ARRAY11X20X6[7,J,P] : = TEMPSS@.CONTROLARRAY[P,J]
      *PROP[7];
```

```
   RS@.ARRAY11X20X6[7,J,P] : = TEMPRS@.CONTROLARRAY[P,J]
     *PROP[7]:
   RR@.ARRAY11X20X6[7,J,P] : = TEMPRR@.CONTROLARRAY[P,J]
     *PROP[7]:
   SS@.ARRAY11X20X6[8,J,P] : = TEMPSS@.CONTROLARRAY[P,J]
     *PROP[8]:
   RS@.ARRAY11X20X6[8,J,P] : = TEMPRS@.CONTROLARRAY[P,J]
     *PROP[8]:
   RR@.ARRAY11X20X6[8,J,P] : = TEMPRR@.CONTROLARRAY[P,J]
     *PROP[8]:
   SS@.ARRAY11X20X6[9,J,P] : = TEMPSS@.CONTROLARRAY[P,J]
     *PROP[9]:
   RS@.ARRAY11X20X6[9,J,P] : = TEMPRS@.CONTROLARRAY[P,J]
     *PROP[9]:
   RR@.ARRAY11X20X6[9,J,P] : = TEMPRR@.CONTROLARRAY[P,J]
     *PROP[9]:
   SS@.ARRAY11X20X6[10,J,P] : = TEMPSS@.CONTROLARRAY[P,J]
     *PROP[10]:
   RS@.ARRAY11X20X6[10,J,P] : = TEMPRS@.CONTROLARRAY[P,J]
     *PROP[10]:
   RR@.ARRAY11X20X6[10,J,P] : = TEMPRR@.CONTROLARRAY[P,J]
     *PROP[10]:

  END:   {J Loop}
 END: {P Loop}

FOR P : = 1 TO CURRENTNUMCHEMS DO
 BEGIN
  TOTRF@.ARRAY1X6[P] : = 0.0:
  SSF@.ARRAY1XNUMCHEM[P] : = 0.0:
  RSF@.ARRAY1XNUMCHEM[P] : = 0.0:
  RRF@.ARRAY1XNUMCHEM[P] : = 0.0:
  SSM@.ARRAY1XNUMCHEM[P] : = 0.0:
  RSM@.ARRAY1XNUMCHEM[P] : = 0.0:
  RRM@.ARRAY1XNUMCHEM[P] : = 0.0:
  FOR J : = IRF TO 20 DO
   FOR I : = 1 TO 11 DO
    TOTRF@.ARRAY1X6[P] : = TOTRF@.ARRAY1X6[P]
      +SS@.ARRAY11X20X6[I,J,P]+RS@.ARRAY11X20X6[I,J,P]
      +RR@.ARRAY11X20X6[I,J,P]:
    IF (TOTRF@.ARRAY1X6[P] < > 0.0) THEN
     BEGIN
      TEMP : = SXRAT + 1:
      FOR I : = 1 TO 11 DO
       FOR J : = IRF TO 20 DO
        BEGIN
         SSF@.ARRAY1XNUMCHEM[P] : = SSF@.ARRAY1XNUMCHEM[P]
           +SS@.ARRAY11X20X6[I,J,P]*SXRAT/TEMP:
         RSF@.ARRAY1XNUMCHEM[P] : = RSF@.ARRAY1XNUMCHEM[P]
           +RS@.ARRAY11X20X6[I,J,P]*SXRAT/TEMP:
         RRF@.ARRAY1XNUMCHEM[P] : = RRF@.ARRAY1XNUMCHEM[P]
           +RR@.ARRAY11X20X6[I,J,P]*SXRAT/TEMP:
         SSM@.ARRAY1XNUMCHEM[P] : = SSM@.ARRAY1XNUMCHEM[P]
           +SS@.ARRAY11X20X6[I,J,P]/TEMP:
         RSM@.ARRAY1XNUMCHEM[P] : = RSM@.ARRAY1XNUMCHEM[P]
           +RS@.ARRAY11X20X6[I,J,P]/TEMP:
         RRM@.ARRAY1XNUMCHEM[P] : = RRM@.ARRAY1XNUMCHEM[P]
           +RR@.ARRAY11X20X6[I,J,P]/TEMP:
        END:  {J Loop}

     TOTRF@.ARRAY1X6[P] : = SSF@.ARRAY1XNUMCHEM[P]
       +RSF@.ARRAY1XNUMCHEM[P]+RRF@.ARRAY1XNUMCHEM[P]:
     TOTM@.ARRAY1XNUMCHEM[P] : = SSM@.ARRAY1XNUMCHEM[P]
       +RSM@.ARRAY1XNUMCHEM[P]+RRM@.ARRAY1XNUMCHEM[P]:
     RSF@.ARRAY1XNUMCHEM[P] : = RSF@.ARRAY1XNUMCHEM[P]
       *RSDIS@.ARRAY1X6[P]:
     RRF@.ARRAY1XNUMCHEM[P] : = RRF@.ARRAY1XNUMCHEM[P]
       *RRDIS@.ARRAY1X6[P]:

     MID : = SSF@.ARRAY1XNUMCHEM[P]*(SSM@.ARRAY1XNUMCHEM[P]
       +RSM@.ARRAY1XNUMCHEM[P]/2.0)+RSF@.ARRAY1XNUMCHEM[P]
       *(SSM@.ARRAY1XNUMCHEM[P]/2.0+RSM@.ARRAY1XNUMCHEM[P]
```

```
      /4.0);
     IF TOTM@.ARRAY1XNUMCHEM[P] = 0
      THEN TOTM@.ARRAY1XNUMCHEM[P] : = 0.000001;
     SS1@.ARRAY1X6[P] : = FRACIN@.ARRAY1X20[IRF]*MID*FEC
       /TOTM@.ARRAY1XNUMCHEM[P];
     T1 : = RSM@.ARRAY1XNUMCHEM[P]/2.0+RRM@.ARRAY1XNUMCHEM[P];
     T2 : = SSM@.ARRAY1XNUMCHEM[P]+RSM@.ARRAY1XNUMCHEM[P]
       +RRM@.ARRAY1XNUMCHEM[P];
     T3 : = RSM@.ARRAY1XNUMCHEM[P]/2.0+SSM@.ARRAY1XNUMCHEM[P];
     IF T1 < 0.00000000000000000001 THEN T1 : = 0.0;
     IF T2 < 0.00000000000000000001 THEN T2 : = 0.0;
     IF T3 < 0.00000000000000000001 THEN T3 : = 0.0;
     MID : = SSF@.ARRAY1XNUMCHEM[P]*T1+(RSF@.ARRAY1XNUMCHEM[P]
       /2.0)*T2+RRF@.ARRAY1XNUMCHEM[P]*T3;
     RS1@.ARRAY1X6[P] : = FRACIN@.ARRAY1X20[IRF]*MID*FEC
       /TOTM@.ARRAY1XNUMCHEM[P];
     T1 : = RSM@.ARRAY1XNUMCHEM[P]/4.0;
     T2 : = RRM@.ARRAY1XNUMCHEM[P]/2.0;
     T3 : = T1 + T2;
     IF T3 < 0.00000000000000000001 THEN T3 : = 0.0;
     IF RRF@.ARRAY1XNUMCHEM[P] < 0.00000000000000000001
      THEN RRF@.ARRAY1XNUMCHEM[P] : = 0.0;
     IF RRM@.ARRAY1XNUMCHEM[P] < 0.00000000000000000001
      THEN RRM@.ARRAY1XNUMCHEM[P] : = 0.0;
     MID : = RSF@.ARRAY1XNUMCHEM[P]*T3+RRF@.ARRAY1XNUMCHEM[P]
       *(RSM@.ARRAY1XNUMCHEM[P]/2.0+RRM@.ARRAY1XNUMCHEM[P]);
     RR1@.ARRAY1X6[P] : = FRACIN@.ARRAY1X20[IRF]*MID*FEC
       /TOTM@.ARRAY1XNUMCHEM[P];
    END; {TOTRF> LOOP}

   FOR I : = 1 TO 11 DO
    FOR J1 : = 1 TO 19 DO
     BEGIN
     J : = 21 - J1;
     IF SS@.ARRAY11X20X6[I,J-1,P] < 0.00000000000000000001
      THEN SS@.ARRAY11X20X6[I,J-1,P] : = 0.0;
     IF RS@.ARRAY11X20X6[I,J-1,P] < 0.00000000000000000001
      THEN RS@.ARRAY11X20X6[I,J-1,P] : = 0.0;
     IF RR@.ARRAY11X20X6[I,J-1,P] < 0.00000000000000000001
      THEN RR@.ARRAY11X20X6[I,J-1,P] : = 0.0;
     SS@.ARRAY11X20X6[I,J,P] : = SS@.ARRAY11X20X6[I,J-1,P]
       *SURV@.ARRAY1X20[J-1];
     RS@.ARRAY11X20X6[I,J,P] : = RS@.ARRAY11X20X6[I,J-1,P]
       *SURV@.ARRAY1X20[J-1];
     RR@.ARRAY11X20X6[I,J,P] : = RR@.ARRAY11X20X6[I,J-1,P]
       *SURV@.ARRAY1X20[J-1];
     END; {J1 Loop}

   IF (T > = (0.1 * CC)) THEN
    BEGIN
    CCTEST : = 0.0;
    THRESH(NCC1, NCC2, P, CCTEST);
    IF (CCTEST > = (0.1 * CC)) THEN
     BEGIN
     DDS : = 1 - CCTEST / CC;
     IF DDS < 0.1 THEN DDS : = 0.1;
     FOR I : = 1 TO 11 DO
      FOR J : = NCC1 TO NCC2 DO
       BEGIN
        SS@.ARRAY11X20X6[I,J,P] : =
          SS@.ARRAY11X20X6[I,J,P] * DDS;
        RS@.ARRAY11X20X6[I,J,P] : =
          RS@.ARRAY11X20X6[I,J,P] * DDS;
        RR@.ARRAY11X20X6[I,J,P] : =
          RR@.ARRAY11X20X6[I,J,P] * DDS;
       END;
     END; {CCTEST Loop}
    END; {T > Loop}
 END; {P Loop}

DISPOSE(TEMPSS);
DISPOSE(TEMPRS);
```

```
 DISPOSE(TEMPRR);
 DISPOSE(TOTM);
 DISPOSE(SSF);
 DISPOSE(RSF);
 DISPOSE(RRF);
 DISPOSE(SSM);
 DISPOSE(RSM);
 DISPOSE(RRM);

END; {Procedure NEXT}


PROCEDURE REINIT;

VAR

 TEMPSS, TEMPRS, TEMPRR, SAFE : CHEMAGE;

 T4, T5, T6              : REAL;

 I, J, P, N1             : INTEGER;

BEGIN

 NEW(TEMPSS);
 NEW(TEMPRS);
 NEW(TEMPRR);
 NEW(SAFE);

 FOR J := 1 TO 20 DO
  TOTAL@.ARRAY6X20[COUNTER,J] := 0.0;
 FOR J := 1 TO 20 DO
  BEGIN

    TOTAL@.ARRAY6X20[COUNTER,J] := TOTAL@.ARRAY6X20[COUNTER,J]
      + SS@.ARRAY11X20X6[1,J,COUNTER]
      + RS@.ARRAY11X20X6[1,J,COUNTER]
      + RR@.ARRAY11X20X6[1,J,COUNTER];
    TOTAL@.ARRAY6X20[COUNTER,J] := TOTAL@.ARRAY6X20[COUNTER,J]
      + SS@.ARRAY11X20X6[2,J,COUNTER]
      + RS@.ARRAY11X20X6[2,J,COUNTER]
      + RR@.ARRAY11X20X6[2,J,COUNTER];
    TOTAL@.ARRAY6X20[COUNTER,J] := TOTAL@.ARRAY6X20[COUNTER,J]
      + SS@.ARRAY11X20X6[3,J,COUNTER]
      + RS@.ARRAY11X20X6[3,J,COUNTER]
      + RR@.ARRAY11X20X6[3,J,COUNTER];
    TOTAL@.ARRAY6X20[COUNTER,J] := TOTAL@.ARRAY6X20[COUNTER,J]
      + SS@.ARRAY11X20X6[4,J,COUNTER]
      + RS@.ARRAY11X20X6[4,J,COUNTER]
      + RR@.ARRAY11X20X6[4,J,COUNTER];
    TOTAL@.ARRAY6X20[COUNTER,J] := TOTAL@.ARRAY6X20[COUNTER,J]
      + SS@.ARRAY11X20X6[5,J,COUNTER]
      + RS@.ARRAY11X20X6[5,J,COUNTER]
      + RR@.ARRAY11X20X6[5,J,COUNTER];
    TOTAL@.ARRAY6X20[COUNTER,J] := TOTAL@.ARRAY6X20[COUNTER,J]
      + SS@.ARRAY11X20X6[6,J,COUNTER]
      + RS@.ARRAY11X20X6[6,J,COUNTER]
      + RR@.ARRAY11X20X6[6,J,COUNTER];
    TOTAL@.ARRAY6X20[COUNTER,J] := TOTAL@.ARRAY6X20[COUNTER,J]
      + SS@.ARRAY11X20X6[7,J,COUNTER]
      + RS@.ARRAY11X20X6[7,J,COUNTER]
      + RR@.ARRAY11X20X6[7,J,COUNTER];
    TOTAL@.ARRAY6X20[COUNTER,J] := TOTAL@.ARRAY6X20[COUNTER,J]
      + SS@.ARRAY11X20X6[8,J,COUNTER]
      + RS@.ARRAY11X20X6[8,J,COUNTER]
      + RR@.ARRAY11X20X6[8,J,COUNTER];
    TOTAL@.ARRAY6X20[COUNTER,J] := TOTAL@.ARRAY6X20[COUNTER,J]
      + SS@.ARRAY11X20X6[9,J,COUNTER]
      + RS@.ARRAY11X20X6[9,J,COUNTER]
      + RR@.ARRAY11X20X6[9,J,COUNTER];
    TOTAL@.ARRAY6X20[COUNTER,J] := TOTAL@.ARRAY6X20[COUNTER,J]
      + SS@.ARRAY11X20X6[10,J,COUNTER]
```

```
      + RS@.ARRAY11X20X6[10,J,COUNTER]
      + RR@.ARRAY11X20X6[10,J,COUNTER];
    TOTAL@.ARRAY6X20[COUNTER,J] : = TOTAL@.ARRAY6X20[COUNTER,J]
      + SS@.ARRAY11X20X6[11,J,COUNTER]
      + RS@.ARRAY11X20X6[11,J,COUNTER]
      + RR@.ARRAY11X20X6[11,J,COUNTER];

   IF TOTAL@.ARRAY6X20[COUNTER,J] < 0.0000000001
    THEN TOTAL@.ARRAY6X20[COUNTER,J] : = 0.0;
   SAFE@.CONTROLARRAY[COUNTER,J] : = SS@.ARRAY11X20X6[11,J,COUNTER]
     + RS@.ARRAY11X20X6[11,J,COUNTER]
     + RR@.ARRAY11X20X6[11,J,COUNTER];
   IF (TOTAL@.ARRAY6X20[COUNTER,J] = 0.0)
    THEN SAFE@.CONTROLARRAY[COUNTER,J] : = 0.0
    ELSE SAFE@.CONTROLARRAY[COUNTER,J] : =
      SAFE@.CONTROLARRAY[COUNTER,J]/TOTAL@.ARRAY6X20[COUNTER,J];
  END;
  FOR J : = 1 TO 20 DO
  BEGIN
   Q : = 1.0 - RGENEFREQ@.ARRAY6X20[K,J];
   T4 : = Q * Q;
   T5 : = RGENEFREQ@.ARRAY6X20[K,J] * RGENEFREQ@.ARRAY6X20[K,J];
   T6 : = 2 * RGENEFREQ@.ARRAY6X20[K,J] * Q;
   IF T4 < 0.00000000000000000001 THEN T4 : = 0.0;
   IF T5 < 0.00000000000000000001 THEN T5 : = 0.0;
   IF T6 < 0.00000000000000000001 THEN T6 : = 0.0;
   TEMPSS@.CONTROLARRAY[K,J] : = T4*TOTAL@.ARRAY6X20[COUNTER,J]
     *(1.0-SAFE@.CONTROLARRAY[COUNTER,J]);
   TEMPRS@.CONTROLARRAY[K,J] : = T6*TOTAL@.ARRAY6X20[COUNTER,J]
     *(1.0-SAFE@.CONTROLARRAY[COUNTER,J]);
   TEMPRR@.CONTROLARRAY[K,J] : = T5*TOTAL@.ARRAY6X20[COUNTER,J]
     *(1.0-SAFE@.CONTROLARRAY[COUNTER,J]);
   SS@.ARRAY11X20X6[11,J,K] : = T4*TOTAL@.ARRAY6X20[COUNTER,J]
     *SAFE@.CONTROLARRAY[COUNTER,J];
   RS@.ARRAY11X20X6[11,J,K] : = T6*TOTAL@.ARRAY6X20[COUNTER,J]
     *SAFE@.CONTROLARRAY[COUNTER,J];
   RR@.ARRAY11X20X6[11,J,K] : = T5*TOTAL@.ARRAY6X20[COUNTER,J]
     *SAFE@.CONTROLARRAY[COUNTER,J];
   FOR I : = 1 TO 10 DO
    BEGIN
     SS@.ARRAY11X20X6[I,J,K] : = TEMPSS@.CONTROLARRAY[K,J]
       *PROP[I];
     RS@.ARRAY11X20X6[I,J,K] : = TEMPRS@.CONTROLARRAY[K,J]
       *PROP[I];
     RR@.ARRAY11X20X6[I,J,K] : = TEMPRR@.CONTROLARRAY[K,J]
       *PROP[I];
    END;
  END;

  DISPOSE(TEMPSS);
  DISPOSE(TEMPRS);
  DISPOSE(TEMPRR);
  DISPOSE(SAFE);

 END; {Procedure REINIT}


PROCEDURE KILL(D50, D95: A6BY6OFREAL; VAR XX: A11BY20BY6OFREAL);

 VAR

  STDEV, Z                  : REAL;

  I, J, P, INITSTAG, LASTSTAG   : INTEGER;

  N                         : ARRAY [1..6] OF INTEGER;

 BEGIN

  FOR J : = 1 TO 6 DO
   BEGIN
    STDEV : = (LN(D95@.ARRAY6X6[J,KORIG]/D50@.ARRAY6X6[J,KORIG])
```

```
    /LN(10.0))/1.6449;
  Z : = (LN(CD@.ARRAY1X6[K]/D50@.ARRAY6X6[J,KORIG])
    /LN(10.0))/STDEV;
  IF (Z > = ( - 3.75)) THEN
   BEGIN
    IF (Z < = 0.0) THEN N[J] : = TRUNC(Z / 0.75) + 5;
    IF (Z > 0.0) THEN N[J] : = TRUNC(Z / 0.75) + 6;
    IF (Z > 3.0) THEN N[J] : = 10;
    CASE J OF
     1: BEGIN
        INITSTAG : = 1;
        LASTSTAG : = LE;
       END;
     2: BEGIN
        INITSTAG : = ISL;
        LASTSTAG : = LSL;
       END;
     3: BEGIN
        INITSTAG : = IML;
        LASTSTAG : = LML;
       END;
     4: BEGIN
        INITSTAG : = ILL;
        LASTSTAG : = LLL;
       END;
     5: BEGIN
        INITSTAG : = IP;
        LASTSTAG : = LP;
       END;
     6: BEGIN
        INITSTAG : = IA;
        LASTSTAG : = 20;
       END;
     OTHERWISE CASECHECK : = 0;
    END; {Case}
    FOR P : = INITSTAG TO LASTSTAG DO
     BEGIN
      IF ((1.0 - FRACIN@.ARRAY1X20[P]) < > 1.0) THEN
       FOR I : = 1 TO N[J] DO
        XX@.ARRAY11X20X6[I,P,K] : = XX@.ARRAY11X20X6[I,P,K]
          *(1.0-FRACIN@.ARRAY1X20[P]);
     END;
   END;
  END;  {For J loop}

END;  {Procedure KILL}


PROCEDURE UPDATER;  {Allows cross resistance}

VAR

 I, J, P, CHEMNUMHOLD  : INTEGER;

 TEMPRGENE, TEMPRFREQ  : CHEMAGE;

 DIFF                  : ARRAY [1..20] OF REAL;

BEGIN

 NEW(TEMPRGENE);
 NEW(TEMPRFREQ);

 FOR J : = 1 TO 20 DO
  BEGIN
   TOTAL@.ARRAY6X20[K,J] : = 0.0;
   TEMPRGENE@.CONTROLARRAY[K,J] : = 0.0;

    TOTAL@.ARRAY6X20[K,J] : = TOTAL@.ARRAY6X20[K,J]
      + SS@.ARRAY11X20X6[1,J,K] + RS@.ARRAY11X20X6[1,J,K]
      + RR@.ARRAY11X20X6[1,J,K];
    TEMPRGENE@.CONTROLARRAY[K,J] : = TEMPRGENE@.CONTROLARRAY[K,J]
```

```
    + RR@.ARRAY11X20X6[1,J,K] + (0.5 * RS@.ARRAY11X20X6[1,J,K]);
  TOTAL@.ARRAY6X20[K,J] : = TOTAL@.ARRAY6X20[K,J]
    + SS@.ARRAY11X20X6[2,J,K] + RS@.ARRAY11X20X6[2,J,K]
    + RR@.ARRAY11X20X6[2,J,K];
  TEMPRGENE@.CONTROLARRAY[K,J] : = TEMPRGENE@.CONTROLARRAY[K,J]
    + RR@.ARRAY11X20X6[2,J,K] + (0.5 * RS@.ARRAY11X20X6[2,J,K]);
  TOTAL@.ARRAY6X20[K,J] : = TOTAL@.ARRAY6X20[K,J]
    + SS@.ARRAY11X20X6[3,J,K] + RS@.ARRAY11X20X6[3,J,K]
    + RR@.ARRAY11X20X6[3,J,K];
  TEMPRGENE@.CONTROLARRAY[K,J] : = TEMPRGENE@.CONTROLARRAY[K,J]
    + RR@.ARRAY11X20X6[3,J,K] + (0.5 * RS@.ARRAY11X20X6[3,J,K]);
  TOTAL@.ARRAY6X20[K,J] : = TOTAL@.ARRAY6X20[K,J]
    + SS@.ARRAY11X20X6[4,J,K] + RS@.ARRAY11X20X6[4,J,K]
    + RR@.ARRAY11X20X6[4,J,K];
  TEMPRGENE@.CONTROLARRAY[K,J] : = TEMPRGENE@.CONTROLARRAY[K,J]
    + RR@.ARRAY11X20X6[4,J,K] + (0.5 * RS@.ARRAY11X20X6[4,J,K]);
  TOTAL@.ARRAY6X20[K,J] : = TOTAL@.ARRAY6X20[K,J]
    + SS@.ARRAY11X20X6[5,J,K] + RS@.ARRAY11X20X6[5,J,K]
    + RR@.ARRAY11X20X6[5,J,K];
  TEMPRGENE@.CONTROLARRAY[K,J] : = TEMPRGENE@.CONTROLARRAY[K,J]
    + RR@.ARRAY11X20X6[5,J,K] + (0.5 * RS@.ARRAY11X20X6[5,J,K]);
  TOTAL@.ARRAY6X20[K,J] : = TOTAL@.ARRAY6X20[K,J]
    + SS@.ARRAY11X20X6[6,J,K] + RS@.ARRAY11X20X6[6,J,K]
    + RR@.ARRAY11X20X6[6,J,K];
  TEMPRGENE@.CONTROLARRAY[K,J] : = TEMPRGENE@.CONTROLARRAY[K,J]
    + RR@.ARRAY11X20X6[6,J,K] + (0.5 * RS@.ARRAY11X20X6[6,J,K]);
  TOTAL@.ARRAY6X20[K,J] : = TOTAL@.ARRAY6X20[K,J]
    + SS@.ARRAY11X20X6[7,J,K] + RS@.ARRAY11X20X6[7,J,K]
    + RR@.ARRAY11X20X6[7,J,K];
  TEMPRGENE@.CONTROLARRAY[K,J] : = TEMPRGENE@.CONTROLARRAY[K,J]
    + RR@.ARRAY11X20X6[7,J,K] + (0.5 * RS@.ARRAY11X20X6[7,J,K]);
  TOTAL@.ARRAY6X20[K,J] : = TOTAL@.ARRAY6X20[K,J]
    + SS@.ARRAY11X20X6[8,J,K] + RS@.ARRAY11X20X6[8,J,K]
    + RR@.ARRAY11X20X6[8,J,K];
  TEMPRGENE@.CONTROLARRAY[K,J] : = TEMPRGENE@.CONTROLARRAY[K,J]
    + RR@.ARRAY11X20X6[8,J,K] + (0.5 * RS@.ARRAY11X20X6[8,J,K]);
  TOTAL@.ARRAY6X20[K,J] : = TOTAL@.ARRAY6X20[K,J]
    + SS@.ARRAY11X20X6[9,J,K] + RS@.ARRAY11X20X6[9,J,K]
    + RR@.ARRAY11X20X6[9,J,K];
  TEMPRGENE@.CONTROLARRAY[K,J] : = TEMPRGENE@.CONTROLARRAY[K,J]
    + RR@.ARRAY11X20X6[9,J,K] + (0.5 * RS@.ARRAY11X20X6[9,J,K]);
  TOTAL@.ARRAY6X20[K,J] : = TOTAL@.ARRAY6X20[K,J]
    + SS@.ARRAY11X20X6[10,J,K] + RS@.ARRAY11X20X6[10,J,K]
    + RR@.ARRAY11X20X6[10,J,K];
  TEMPRGENE@.CONTROLARRAY[K,J] : = TEMPRGENE@.CONTROLARRAY[K,J]
    + RR@.ARRAY11X20X6[10,J,K] + (0.5 * RS@.ARRAY11X20X6[10,J,K]);
  TOTAL@.ARRAY6X20[K,J] : = TOTAL@.ARRAY6X20[K,J]
    + SS@.ARRAY11X20X6[11,J,K] + RS@.ARRAY11X20X6[11,J,K]
    + RR@.ARRAY11X20X6[11,J,K];
  TEMPRGENE@.CONTROLARRAY[K,J] : = TEMPRGENE@.CONTROLARRAY[K,J]
    + RR@.ARRAY11X20X6[11,J,K] + (0.5 * RS@.ARRAY11X20X6[11,J,K]);

 IF (TOTAL@.ARRAY6X20[K,J] = 0.0)
  THEN TEMPRFREQ@.CONTROLARRAY[K,J] : = 0.0
  ELSE TEMPRFREQ@.CONTROLARRAY[K,J] :=
    TEMPRGENE@.CONTROLARRAY[K,J] / TOTAL@.ARRAY6X20[K,J];
 DIFF[J] : = TEMPRFREQ@.CONTROLARRAY[K,J]
   - RGENEFREQ@.ARRAY6X20[K,J];
 RGENEFREQ@.ARRAY6X20[K,J] : = TEMPRFREQ@.CONTROLARRAY[K,J];
 IF RGENEFREQ@.ARRAY6X20[K,J] > 1.0
  THEN RGENEFREQ@.ARRAY6X20[K,J] : = 1.0;
END; {J Loop}

CHEMNUMHOLD : = CHEMNUMBER;
CHEMNUMBER : = 0;
FOR P : = 1 TO NUMPESTICIDES DO
 IF P IN CURRENTCHEMS THEN
  BEGIN
  CHEMNUMBER : = CHEMNUMBER + 1;
  IF (CHEMNUMBER < > K) THEN
   FOR J : = 1 TO 20 DO
    BEGIN
```

```
      RGENEFREQ@.ARRAY6X20[CHEMNUMBER,J] : =
        RGENEFREQ@.ARRAY6X20[CHEMNUMBER,J]
        + (CRESIS@.ARRAY6X6[KORIG,P] * DIFF[J]);
      IF (RGENEFREQ@.ARRAY6X20[CHEMNUMBER,J] < 0)
       THEN RGENEFREQ@.ARRAY6X20[CHEMNUMBER,J] : = 0.0;
      IF (RGENEFREQ@.ARRAY6X20[CHEMNUMBER,J] > 1.0)
       THEN RGENEFREQ@.ARRAY6X20[CHEMNUMBER,J] : = 1.0;
     END;
  END;
 CHEMNUMBER : = CHEMNUMHOLD;
 FOR P : = 1 TO CURRENTNUMCHEMS DO
  FOR I : = 1 TO 11 DO
   FOR J : = 1 TO 20 DO
    BEGIN
     SS@.ARRAY11X20X6[I,J,P] : = SS@.ARRAY11X20X6[I,J,K];
     RS@.ARRAY11X20X6[I,J,P] : = RS@.ARRAY11X20X6[I,J,K];
     RR@.ARRAY11X20X6[I,J,P] : = RR@.ARRAY11X20X6[I,J,K];
    END;

 DISPOSE(TEMPRGENE);
 DISPOSE(TEMPRFREQ);

END;


PROCEDURE WINTER(WSURV: A1BY20OFREAL);

 VAR

  I, J, P  : INTEGER;

 BEGIN

 FOR P : = 1 TO CURRENTNUMCHEMS DO
  BEGIN
   FOR I : = 1 TO 11 DO
    FOR J : = 1 TO 20 DO
     BEGIN
      SS@.ARRAY11X20X6[I,J,P] : = SS@.ARRAY11X20X6[I,J,P]
        * WSURV@.ARRAY1X20[J];
      RS@.ARRAY11X20X6[I,J,P] : = RS@.ARRAY11X20X6[I,J,P]
        * WSURV@.ARRAY1X20[J];
      RR@.ARRAY11X20X6[I,J,P] : = RR@.ARRAY11X20X6[I,J,P]
        * WSURV@.ARRAY1X20[J];
     END;
   CD@.ARRAY1X6[P] : = 0.0;
  END;

 OW : = TRUE;

END;  {Procedure WINTER}


PROCEDURE MIGRATE;     {Add immigrants}

 VAR

  I, J, P, N        : INTEGER;

  S, T1, T2, T3, T4  : REAL;

 BEGIN

 CHEMNUMBER : = 0;
 FOR P : = 1 TO NUMPESTICIDES DO
 IF P IN CURRENTCHEMS THEN
  BEGIN
   CHEMNUMBER : = CHEMNUMBER + 1;
   FOR N : = 1 TO 3 DO
    BEGIN
     IF (N = 1)
      THEN
```

```
BEGIN
 T1 : = Q * Q;
 T3 : = 2.0 * PINIT * Q;
 T4 : = PINIT * PINIT;
 IF T1 < 0.00000000000000000001 THEN T1 : = 0.0;
 IF T3 < 0.00000000000000000001 THEN T3 : = 0.0;
 IF T4 < 0.00000000000000000001 THEN T4 : = 0.0;
 S : = 0.0;
 FOR J : = IRF TO 20 DO
  IF (RR@.ARRAY11X20X6[10,J,CHEMNUMBER]
     +RR@.ARRAY11X20X6[11,J,CHEMNUMBER]
     > 0.0) THEN S : = S + 1;
 FOR J : = IRF TO 20 DO
  IF (RR@.ARRAY11X20X6[10,J,CHEMNUMBER]
     +RR@.ARRAY11X20X6[11,J,CHEMNUMBER]
     > 0.0) THEN
   BEGIN
    FOR I : = 1 TO 10 DO
     BEGIN
      SS@.ARRAY11X20X6[I,J,CHEMNUMBER] : =
        SS@.ARRAY11X20X6[I,J,CHEMNUMBER]
        +T1*PROP[I]
        *(WIMIG@.ARRAY1X6[P]
        *WOODS@.ARRAY1XNUMDAYSPERYEAR[IDAY])/S;
      RS@.ARRAY11X20X6[I,J,CHEMNUMBER] : =
        RS@.ARRAY11X20X6[I,J,CHEMNUMBER]
        +T3*PROP[I]
        *(WIMIG@.ARRAY1X6[P]
        *WOODS@.ARRAY1XNUMDAYSPERYEAR[IDAY])/S;
      RR@.ARRAY11X20X6[I,J,CHEMNUMBER] : =
        RR@.ARRAY11X20X6[I,J,CHEMNUMBER]
        +T4*PROP[I]
        *(WIMIG@.ARRAY1X6[P]
        *WOODS@.ARRAY1XNUMDAYSPERYEAR[IDAY])/S;
     END;
   END;  {IF Loop}

          {Add immigrant's eggs}

 SS1@.ARRAY1X6[CHEMNUMBER] : = SS1@.ARRAY1X6[CHEMNUMBER]
   +(WIMIG@.ARRAY1X6[P]
   *WOODS@.ARRAY1XNUMDAYSPERYEAR[IDAY])
   *(SXRAT/(SXRAT+1))*FEC*T1*DINC;
 RS1@.ARRAY1X6[CHEMNUMBER] : = RS1@.ARRAY1X6[CHEMNUMBER]
   +(WIMIG@.ARRAY1X6[P]
   *WOODS@.ARRAY1XNUMDAYSPERYEAR[IDAY])
   *(SXRAT/(SXRAT+1))*FEC*T3*DINC;
 RR1@.ARRAY1X6[CHEMNUMBER] : = RR1@.ARRAY1X6[CHEMNUMBER]
   +(WIMIG@.ARRAY1X6[P]
   *WOODS@.ARRAY1XNUMDAYSPERYEAR[IDAY])
   *(SXRAT/(SXRAT+1))*FEC*T4*DINC;
END
ELSE
IF (N = 2)
 THEN
  BEGIN
   T4 : = (IMIGFREQ*TOTRFREQ@.ARRAY1X6[CHEMNUMBER])
     *(IMIGFREQ*TOTRFREQ@.ARRAY1X6[CHEMNUMBER]);
   T1 : = (1.0-SQRT(T4))*(1.0-SQRT(T4));
   T3 : = 2.0*SQRT(T1)*SQRT(T4);
   IF T1 < 0.00000000000000000001 THEN T1 : = 0.0;
   IF T3 < 0.00000000000000000001 THEN T3 : = 0.0;
   S : = 0.0;
   FOR J : = IRF TO 20 DO
    IF (RR@.ARRAY11X20X6[10,J,CHEMNUMBER]
     +RR@.ARRAY11X20X6[11,J,CHEMNUMBER] > 0.0)
     THEN S : = S+1;
   FOR J : = IRF TO 20 DO
    IF (RR@.ARRAY11X20X6[10,J,CHEMNUMBER]
     +RR@.ARRAY11X20X6[11,J,CHEMNUMBER] > 0.0) THEN
     BEGIN
      FOR I : = 1 TO 10 DO
```

```
      BEGIN
       SS@.ARRAY11X20X6[I,J,CHEMNUMBER] : =
         SS@.ARRAY11X20X6[I,J,CHEMNUMBER]
         +T1*PROP[I]
         *(FIMIG@.ARRAY1X6[P]
         * WOODS@.ARRAY1XNUMDAYSPERYEAR[IDAY])
         /S:
       RS@.ARRAY11X20X6[I,J,CHEMNUMBER] : =
         RS@.ARRAY11X20X6[I,J,CHEMNUMBER]
         +T3*PROP[I]
         *(FIMIG@.ARRAY1X6[P]
         *WOODS@.ARRAY1XNUMDAYSPERYEAR[IDAY])
         /S:
       RR@.ARRAY11X20X6[I,J,CHEMNUMBER] : =
         RR@.ARRAY11X20X6[I,J,CHEMNUMBER]
         +T4*PROP[I]
         *(FIMIG@.ARRAY1X6[P]
         *WOODS@.ARRAY1XNUMDAYSPERYEAR[IDAY])
         /S:
      END:
    END:  {IF Loop}

            {Add immigrant's eggs}

  SS1@.ARRAY1X6[CHEMNUMBER] : =
    SS1@.ARRAY1X6[CHEMNUMBER]
    +(FIMIG@.ARRAY1X6[P]
    *WOODS@.ARRAY1XNUMDAYSPERYEAR[IDAY])
    *(SXRAT/(SXRAT+1))*FEC*T1*DINC:
  RS1@.ARRAY1X6[CHEMNUMBER] : =
    RS1@.ARRAY1X6[CHEMNUMBER]
    +(FIMIG@.ARRAY1X6[P]
    *WOODS@.ARRAY1XNUMDAYSPERYEAR[IDAY])
    *(SXRAT/(SXRAT+1))*FEC*T3*DINC:
  RR1@.ARRAY1X6[CHEMNUMBER] : =
    RR1@.ARRAY1X6[CHEMNUMBER]
    +(FIMIG@.ARRAY1X6[P]
    *WOODS@.ARRAY1XNUMDAYSPERYEAR[IDAY])
    *(SXRAT/(SXRAT+1))*FEC*T4*DINC:
 END
ELSE
 BEGIN
 T1 : = (1.0-TOTRFREQ@.ARRAY1X6[CHEMNUMBER])
    *(1.0-TOTRFREQ@.ARRAY1X6[CHEMNUMBER]):
 T3 : = 2.0*TOTRFREQ@.ARRAY1X6[CHEMNUMBER]
    *(1.0-TOTRFREQ@.ARRAY1X6[CHEMNUMBER]);
 T4 : = TOTRFREQ@.ARRAY1X6[CHEMNUMBER]
    *TOTRFREQ@.ARRAY1X6[CHEMNUMBER]:
 IF T1 < 0.00000000000000000001 THEN T1 : = 0.0;
 IF T3 < 0.00000000000000000001 THEN T3 : = 0.0;
 IF T4 < 0.00000000000000000001 THEN T4 : = 0.0;
 S : = 0.0:
 FOR J : = IRF TO 20 DO
  IF (RR@.ARRAY11X20X6[10,J,CHEMNUMBER]
   +RR@.ARRAY11X20X6[11,J,CHEMNUMBER] > 0.0)
   THEN S : = S+1:
 FOR J : = IRF TO 20 DO
  IF (RR@.ARRAY11X20X6[10,J,CHEMNUMBER]
   +RR@.ARRAY11X20X6[11,J,CHEMNUMBER] > 0.0) THEN
   BEGIN
    FOR I : = 1 TO 10 DO
     BEGIN
      SS@.ARRAY11X20X6[I,J,CHEMNUMBER] : =
        SS@.ARRAY11X20X6[I,J,CHEMNUMBER]
        +T1*PROP[I]
        *(AIMIG@.ARRAY1X6[P]
        *WOODS@.ARRAY1XNUMDAYSPERYEAR[IDAY])
        /S:
      RS@.ARRAY11X20X6[I,J,CHEMNUMBER] : =
        RS@.ARRAY11X20X6[I,J,CHEMNUMBER]
        +T3*PROP[I]
        *(AIMIG@.ARRAY1X6[P]
```

```
            *WOODS@.ARRAY1XNUMDAYSPERYEAR[IDAY])
            /S:
          RR@.ARRAY11X20X6[I,J,CHEMNUMBER] : =
            RR@.ARRAY11X20X6[I,J,CHEMNUMBER]
            +T4*PROP[I]
            *(AIMIG@.ARRAY1X6[P]
            *WOODS@.ARRAY1XNUMDAYSPERYEAR[IDAY])
            /S:
         END;
       END; {IF Loop}

            {Add immigrant's eggs}

      SS1@.ARRAY1X6[CHEMNUMBER] : =
        SS1@.ARRAY1X6[CHEMNUMBER]
        +(AIMIG@.ARRAY1X6[P]
        *WOODS@.ARRAY1XNUMDAYSPERYEAR[IDAY])
        *(SXRAT/(SXRAT+1))*FEC*T1*DINC;
      RS1@.ARRAY1X6[CHEMNUMBER] : =
        RS1@.ARRAY1X6[CHEMNUMBER]
        +(AIMIG@.ARRAY1X6[P]
        *WOODS@.ARRAY1XNUMDAYSPERYEAR[IDAY])
        *(SXRAT/(SXRAT+1))*FEC*T3*DINC;
      RR1@.ARRAY1X6[CHEMNUMBER] : =
        RR1@.ARRAY1X6[CHEMNUMBER]
        +(AIMIG@.ARRAY1X6[P]
        *WOODS@.ARRAY1XNUMDAYSPERYEAR[IDAY])
        *(SXRAT/(SXRAT+1))*FEC*T4*DINC;
     END; {Else}
  END; {N Loop}

  TIMIG : = (WIMIG@.ARRAY1X6[P]*WOODS@.ARRAY1XNUMDAYSPERYEAR[IDAY])
    +(FIMIG@.ARRAY1X6[P]*WOODS@.ARRAY1XNUMDAYSPERYEAR[IDAY])
    +(AIMIG@.ARRAY1X6[P]*WOODS@.ARRAY1XNUMDAYSPERYEAR[IDAY]);

 END; {P Loop}

END; {Procedure MIGRATE}


PROCEDURE EGGGS; {Put new eggs in substage 1, suscept. categor. 1-11}

 VAR

 I, P : INTEGER;

 BEGIN

 FOR P : = 1 TO CURRENTNUMCHEMS DO
  BEGIN
   FOR I : = 1 TO 10 DO
    BEGIN
     SS@.ARRAY11X20X6[I,1,P] : = SS1@.ARRAY1X6[P]*PROP[I]
       *(1-STAREF@.ARRAY1X6[1]);
     RS@.ARRAY11X20X6[I,1,P] : = RS1@.ARRAY1X6[P]*PROP[I]
       *(1-STAREF@.ARRAY1X6[1]);
     RR@.ARRAY11X20X6[I,1,P] : = RR1@.ARRAY1X6[P]*PROP[I]
       *(1-STAREF@.ARRAY1X6[1]);
    END;
   SS@.ARRAY11X20X6[11,1,P] : = SS1@.ARRAY1X6[P]*STAREF@.ARRAY1X6[1];
   RS@.ARRAY11X20X6[11,1,P] : = RS1@.ARRAY1X6[P]*STAREF@.ARRAY1X6[1];
   RR@.ARRAY11X20X6[11,1,P] : = RR1@.ARRAY1X6[P]*STAREF@.ARRAY1X6[1];
  END;

END; {Procedure EGGS}


PROCEDURE REFUGE(IX, P: INTEGER);

VAR

 I, G                    : INTEGER;
```

```
 SSHOLD, RSHOLD, RRHOLD, TEMP, TMP  : REAL;

BEGIN

 FOR G : = 1 TO CURRENTNUMCHEMS DO
  BEGIN
   SSHOLD : = 0.0;
   RSHOLD : = 0.0;
   RRHOLD : = 0.0;

        {Compute totals in refugia, store in holders}

   FOR I : = 1 TO 11 DO
    BEGIN
     SSHOLD : = SSHOLD+SS@.ARRAY11X20X6[I,IX,G]*STAREF@.ARRAY1X6[P];
     RSHOLD : = RSHOLD+RS@.ARRAY11X20X6[I,IX,G]*STAREF@.ARRAY1X6[P];
     RRHOLD : = RRHOLD+RR@.ARRAY11X20X6[I,IX,G]*STAREF@.ARRAY1X6[P];
    END;

        {Put those not entering refugia in SUSCATS 1-10}

   TEMP : = 1.0 - STAREF@.ARRAY1X6[P];
   FOR I : = 1 TO 10 DO
    BEGIN
     TMP : = SS@.ARRAY11X20X6[I,IX,G];
     SS@.ARRAY11X20X6[I,IX,G] : = 0.0;
     SS@.ARRAY11X20X6[I,IX,G] : = (TMP+SS@.ARRAY11X20X6[11,IX,G]
       *PROP[I])*TEMP;
    END;
   FOR I : = 1 TO 10 DO
    BEGIN
     TMP : = RS@.ARRAY11X20X6[I,IX,G];
     RS@.ARRAY11X20X6[I,IX,G] : = 0.0;
     RS@.ARRAY11X20X6[I,IX,G] : = (TMP+RS@.ARRAY11X20X6[11,IX,G]
       *PROP[I])*TEMP;
    END;
   FOR I : = 1 TO 10 DO
    BEGIN
     TMP : = RR@.ARRAY11X20X6[I,IX,G];
     RR@.ARRAY11X20X6[I,IX,G] : = 0.0;
     RR@.ARRAY11X20X6[I,IX,G] : = (TMP+RR@.ARRAY11X20X6[11,IX,G]
       *PROP[I])*TEMP;
    END;

        {Put those entering refugia into SUSCAT 11}

   SS@.ARRAY11X20X6[11,IX,G] : = SSHOLD;
   RS@.ARRAY11X20X6[11,IX,G] : = RSHOLD;
   RR@.ARRAY11X20X6[11,IX,G] : = RRHOLD;

  END;

END;  {Procedure REFUGE}


BEGIN  {Main program TABMSIMULATOR}

 REWRITE(RTABMTOTS);
 REWRITE(STABMTOTS);


 FOR NUMRUNS : = 1 TO 2 DO
 BEGIN
  IF NUMRUNS = 1
    THEN SIMNUMYEARS : = NUMYEARS
    ELSE SIMNUMYEARS : = TABMYRHOLD;

NEW(SSD50);
NEW(RSD50);
NEW(RRD50);
NEW(SSD95);
```

```
NEW(RSD95);
NEW(RRD95);
NEW(RR);
NEW(RS);
NEW(SS);
NEW(TOTRF);
NEW(RSDIS);
NEW(RRDIS);
NEW(CD);
NEW(RFREQ);
NEW(THLIF);
NEW(SS1);
NEW(RS1);
NEW(RR1);
NEW(TOTRFREQ);
NEW(WIMIG);
NEW(FIMIG);
NEW(AIMIG);
NEW(BEGN);
NEW(FRACIN);
NEW(REFINIT);
NEW(SURV);
NEW(WSURV);
NEW(STAREF);
NEW(TOTAL);
NEW(RGENE);
NEW(RGENEFREQ);
NEW(WOODS);
NEW(CRESIS);

DATAINITIALIZE;
GETLIBPARAMS;
INIT;
SUM;
EARLY : = FALSE;
WRITELN;
NEWYR : = 0;

REPEAT
 NEWYR : = NEWYR + 1;
 SIGNAL : = TRUE;
 IYR : = NEWYR;
 MAXT : = 0.0;
 OW : = FALSE;
 TIMMGEN : = 0.0;
 IGEN : = 1;
 IGD : = 0;
 STEP : = 0.0;
 K : = 1;
 COUNTER : = 1;

 WRITE('CURRENTLY SIMULATING TABM DYNAMICS, YEAR ',IYR:2,' DAY ');

 FOR NEWIDAY : = 1 TO NUMDAYSPERYEAR DO
  BEGIN
   IDAY : = NEWIDAY;
   CASE IDAY OF
    1, 10, 20, 30, 40: WRITE(IDAY:2,' ');
    OTHERWISE CASECHECK : = 0;
   END; {CASE}

   IF (NEWYR MOD 50) = 0 THEN
    WRITE(CDDAY,VERTEXNUMBER:4,NEWIDAY:8,'  ');
   IF NEWIDAY = 40 THEN
    WRITE(CDYEAR,VERTEXNUMBER:4,NEWYR:8,'  ');
   CHEMNUMBER : = 0;
   FOR L : = 1 TO NUMPESTICIDES DO
    IF L IN CURRENTCHEMS
     THEN
      BEGIN
       CHEMNUMBER : = CHEMNUMBER + 1;
       CD@.ARRAY1X6[CHEMNUMBER] : = (CD@.ARRAY1X6[CHEMNUMBER]
```

```
          * EXP(LN(0.5)/THLIF@.ARRAY1X6[L]))
          +VERTEX@.ACTIONS[CHEMNUMBER,IYR,IDAY];
        IF (NEWYR MOD 50 ) = 0 THEN
         WRITE(CDDAY,CD@.ARRAY1X6[CHEMNUMBER]:9:3);
        IF NEWIDAY = 40 THEN
         WRITE(CDYEAR,CD@.ARRAY1X6[CHEMNUMBER]:9:3);
       END
       ELSE
        BEGIN
        IF (NEWYR MOD 50) = 0 THEN WRITE(CDDAY,'        ');
        IF NEWIDAY = 40 THEN WRITE(CDYEAR,'        ');
       END;
    IF (NEWYR MOD 50) = 0 THEN WRITELN(CDDAY);
    IF NEWIDAY = 40 THEN WRITELN(CDYEAR);

    IF CD@.ARRAY1X6[COUNTER] > 0.0001 THEN
     BEGIN
      KORIG : = COUNTER;
      K : = COUNTER;
      BEFORE : = COUNTER;
      KILL(SSD50, SSD95, SS);
      KILL(RSD50, RSD95, RS);
      KILL(RRD50, RRD95, RR);
      UPDATER;
     END;
    CHEMNUMBER : = 0;
    FOR M : = 1 TO NUMPESTICIDES DO
     IF M IN CURRENTCHEMS THEN
      BEGIN
       CHEMNUMBER : = CHEMNUMBER + 1;
       IF (CHEMNUMBER < > BEFORE)
         AND (CD@.ARRAY1X6[CHEMNUMBER] > 0.0001) THEN
        BEGIN
         K : = CHEMNUMBER;
         REINIT;
         COUNTER : = CHEMNUMBER;
         KORIG : = M;
         KILL(SSD50, SSD95, SS);
         KILL(RSD50, RSD95, RS);
         KILL(RRD50, RRD95, RR);
         UPDATER;
        END;
      END;

    STEP : = STEP + DINC;
    IF (STEP < 1) THEN GOTO 240;
    STEP : = STEP - 1.0;
    IGD : = IGD + 1;
    IF (IGD < = 20) THEN GOTO 187;
    IGEN : = IGEN + 1;
    IGD : = 0;
    TIMMGEN : = 0.0;
    IF (IGEN-1 = NGENS) THEN GOTO 250;

187: FOR K : = 1 TO CURRENTNUMCHEMS DO
     BEGIN
      SS1@.ARRAY1X6[K] : = 0.0;
      RS1@.ARRAY1X6[K] : = 0.0;
      RR1@.ARRAY1X6[K] : = 0.0;
     END;

    NEXT;
    MIGRATE;
    TIMMGEN : = TIMMGEN + TIMIG;
    EGGGS;
    REFUGE(ISL, 2);
    REFUGE(IML, 3);
    REFUGE(ILL, 4);
    REFUGE(IP, 5);
    REFUGE(IA, 6);
    SUM;
```

```
240:

   END; { FOR NEWIDAY }

    WRITELN;

    {The assignment of total population number (T) to the response
    variable occurs here in order to get the pre-winter population
    levels -- this is arbitrary and will disappear in the final
    version}

250:TABMTOTALS : = MAXT;

    IF NUMRUNS = 1
     THEN WRITELN(RTABMTOTS,TABMTOTALS:10)
     ELSE WRITELN(STABMTOTS,TABMTOTALS:10);

    WINTER(WSURV);
    IGEN:=1;
    IDAY:=0;
    IYR : = IYR + 1;
    SUM;
    FOR K : = 1 TO CURRENTNUMCHEMS DO
     IF (TOTRFREQ@.ARRAY1X6[K] < 0.5) OR (NEWYR < MINIMUMYR)
     THEN SIGNAL : = FALSE;

{ UNTIL((NEWYR = SIMNUMYEARS) OR (SIGNAL));
 } UNTIL(NEWYR=SIMNUMYEARS);
   TABMYRHOLD : = NEWYR;


  DISPOSE(SSD50);
  DISPOSE(RSD50);
  DISPOSE(RRD50);
  DISPOSE(SSD95);
  DISPOSE(RSD95);
  DISPOSE(RRD95);
  DISPOSE(RR);
  DISPOSE(RS);
  DISPOSE(SS);
  DISPOSE(TOTRF);
  DISPOSE(RSDIS);
  DISPOSE(RRDIS);
  DISPOSE(CD);
  DISPOSE(RFREQ);
  DISPOSE(THLIF);
  DISPOSE(SS1);
  DISPOSE(RS1);
  DISPOSE(RR1);
  DISPOSE(TOTRFREQ);
  DISPOSE(WIMIG);
  DISPOSE(FIMIG);
  DISPOSE(AIMIG);
  DISPOSE(BEGN);
  DISPOSE(FRACIN);
  DISPOSE(REFINIT);
  DISPOSE(SURV);
  DISPOSE(WSURV);
  DISPOSE(STAREF);
  DISPOSE(TOTAL);
  DISPOSE(RGENE);
  DISPOSE(RGENEFREQ);
  DISPOSE(WOODS);
  DISPOSE(CRESIS);

  END; {NUMRUNS}

END; {Procedure TABMSIMULATOR}
```

Appendix B. TABM LIFE-HISTORY PARAMETERS

The TABM model is run for two generations, with each generation consisting of 20 substages. Each simulation time-step is equivalent to 4 days, with a simulation year being equivalent to 160 days. As currently parameterized, the model simulates the apple growing season from April 23 to September 30.

The age profile of the simulated TABM population has been distributed to fit field data for initial and peak adult emergence and egg mass deposition. A four day protandry was included between the capture of males in traps and female emergence. For brood 1, the simulation was adjusted to yield: first adults on May 13; peak adults on June 2; first eggs from brood 1 on May 21; and peak egg deposition from brood 1 on June 14. For brood 2, the simulation was adjusted to yield: first adults on July 24; first eggs from brood 2 on August 1; peak adults on August 21; and peak egg deposition on August 29.

The entire initial TABM population at the beginning of a simulation year was placed into refugia because the larva, which are only life-stage to overwinter, are usually only found in the ground cover around the trees at the start of the growing season, and not in the trees themselves. Daily (4 day) survival rates were estimated to generate a four-fold population increase during the season for unsprayed orchards without immigration. The highest natural mortality levels were experienced by young larvae: 73 percent of the eggs hatched; 12 percent became small larvae; 8 percent became medium larvae; 7 percent became large larvae; and 6 percent

reached adulthood. Winter mortality was adjusted to produce an approximate 20 percent survival with the same relative population age structure each simulation year.

The fraction of non-adult TABM populations in the orchard were set at 100 percent at the beginning of each simulation. The adult fraction in the orchard was set at a relatively high, but arbitrary, level. Absence of some adults from the orchard primarily served as a mechanism to avoid an unrealistic, chemical-induced extinction. Immigration from the areas surrounding the orchard was adjusted so as to generate fluctuations in population levels that approximated those observed in experimental studies.

Appendix C. ERM/BLB SERA PROGRAM CODE

The following program code simulates the population dynamics and genetic evolution, in response to environmental and chemical control factors, of the European Red Mite/Black Ladybird Beetle pest-predator complex in the mid-Atlantic apple production system. This section of code was structured as a sub-program and incorporated into the main optimization program contained in Appendix I. All simulations reported in this dissertation were executed on an IBM mainframe computer using the VSPASCAL programming language. Because of site-specific computing constraints, a number of the arrays in this program were disaggregated. Thus, while loosely conforming to structured programming conventions, the listed code contains some anomalies that could be rectified for use on other computing systems. Before listing the ERM/BLB program code, the important variables, data structures, and sub-procedures are defined. More extensive explanation and documentation can be obtained from the author.

Arrays

TOTRF[1..6,1..SPECIES]: Tracking the total number of reproductive females in the orchard, the array can be used in determining immigration from woods, other fruits, and other apple orchards.

RSDIS[1..6,1..SPECIES], RRDIS[1..6,1..SPECIES]: Defines the reproductive disadvantage associated with the resistant R gene.

THLIF[1..6]: Defines the half-life of each potential chemical control.

SS1[1..6,1..SPECIES], RS1[1..6,1..SPECIES], RR1[1..6,1..SPECIES]: Tracks the number of eggs produced per day for each of the genotypes.

BEGN[1..20,1..SPECIES]: The initial distribution of overwintering populations.

FRACIN[1..20,1..SPECIES]: The fraction of each lifestage residing within the orchard and exposed to potential chemical controls.

PROP[1..10]: The normal distribution of 10 susceptibility classes for each genotype. Class 11 tracks the individuals in refugia during any time-step.

REFINIT[1..20,1..SPECIES]: Proportion of each life-stage that was initially in refugia.

SURV[1..19,1..SPECIES]: Defines the daily survival rates between substages.

RR[1..11,1..20,1..6,1..SPECIES], RS[1..11,1..20,1..6,1..SPECIES], SS[1..11,1..20,1..6,1..SPECIES]: Tracks the genotypic arrays by substage and potential chemical control.

STAREF[1..6,1..SPECIES]: Proportion of each lifestage within a stage-long refugia.

CRESIS[1..6,1..6,1..SPECIES]: Defines the relative ratio of cross-resistance among the potential chemical controls.

TOTAL[1..6,1..20,1..SPECIES]: Tracks the total number of individuals of all genotypes that are within each substage.

RGENE[1..6,1..20,1..SPECIES]: Tracks the number of resistant R genes within each substage.

RGENEFREQ[1..6,1..20,1..SPECIES]: Tracks the frequency of the resistant R gene within each substage.

SS50[1..6,1..6,1..SPECIES], RS50[1..6,1..6,1..SPECIES], RR50[1..6,1..6,1..SPECIES], SS95[1..6,1..6,1..SPECIES], RS95[1..6,1..6,1..SPECIES], RR95[1..6,1..6,1..SPECIES]: Defines the lethal dose 50 and 95 levels for each potential chemical. The values are calculated from regression of log dose-probit curves.

WSURV[1..20,1..SPECIES]: Defines the overwintering survival rate of each substage.

Arrays of [1..SPECIES]

LE: Defines the last egg substage.

ISL: Defines the initial small larva substage.

LSL: Defines the last small larva substage.

IML: Defines the initial medium larva substage.

LML: Defines the last medium larva substage

ILL: Defines the initial large larva substage.

LLL: Defines the last large larva substage.

IP: Defines the initial pupa substage.

LP: Defines the last pupa substage.

IA: Defines the initial adult substage.

IRF: Defines the initial reproductive adult substage.

SXRAT: Defines the sex ratios for the adult population.

NCC1: Defines the first substage to be used in calculating the carrying capacity.

NCC2: Defines the last substage to be used in calculating the carrying capacity.

FEC: The number of eggs laid per female per time-step.

CC: The carrying capacity of the environment, it is used to calculate density dependent mortality.

PINIT: The initial frequency of the R-gene in the population.

T: The total number of individuals in the population.

IMIGFREQ: A fraction of RGENEFREQ used for immigrants from other fruits.

WIMIG, FIMIG, AIMIG: Indices of immigration rates from woods, other fruits, and other apple orchards.

Non-Array Variables

NUMYEARS: The number of years over which the simulation takes place.

NGENS: The number of species generations per simulation year.

NUMDAYSPERYEAR: The number of simulated days per simulation year, where 1 simulated day is equivalent to 4 real days.

C: A clumping parameter for the negative binomial distribution of the predator.

SMNCON: The predator consumption rate at which predator survival is at or below the minimum.

SRMIN: The predator survival multiplier when predator consumption is less than SMNCON.

FULLMAX: Consumption rate at which fecundity and survival are at a maximum.

CONMAX: Maximum consumption rate per predator.

FMNCON: Predator consumption rate at which fecundity is at or below a minimum.

FECMIN: The predator fecundity multiplier when predator consumption is less than FMNCON.

F: Predator searching efficiency from Royama's equation.

COUNTER: Tracks the index of the last chemical control to be used.

J: A counter variable usually employed to designate a substage.

K: A counter usually employed to designate a potential chemical control.

P: A counter variable also employed to designate a potential chemical control.

I: A counter usually employed to designate a susceptibility class.

W: A counter usually employed to designate a species.

Procedures

DATAINITIALIZE: Specifies the distribution of individuals into the 10 susceptibility classes. In addition, total R gene frequency, number of reproductive females, and the genotype arrays are zeroed.

GETLIBPARAMS: Reads, from the input data set, the parameters used for defining the species being simulated.

INIT: Initializes the genotype arrays and the R gene frequencies.

SUM: Summarizes the values of each life-stage and R gene frequencies over all potential chemical controls.

PRED: Takes all non-egg stages of the predator and distributes them according to the negative binomial distribution on up to 300 leaves. Based on the predator searching efficiency, maximum consumption, and prey density, the number of prey consumed per predator is determined. Finally, predator survival and fecundity multipliers are calculated.

THRESH: Used to determine if population totals have exceeded the density dependent levels.

NEXT: Determines how much mating and oviposition occurs by genotype. Updates the numbers in the genotype arrays based on inter-substage survivorship and density dependent mortality.

REINIT: Uses Hardy-Weinberg equilibrium to redistribute individuals into the 3 genotype arrays based on the R gene frequencies.

KILL: Removes individuals from the genotype arrays based on susceptibility class, current control dosage, and the lethal dose 50 and 95 values.

UPDATE: Determines the new R gene frequencies by chemical following the KILL procedure.

WINTER: Removes individuals from genotype arrays based on winter survivorship rates.

MIGRATE: Determines the immigration of individuals by genotype.

EGGS: Adds the eggs from SS1, RS1, and RR1 to the first substage of the genotype arrays.

REFUGE: Assigns a portion of each lifestage into a stage-long refugia.

MAIN: Calls all the initializing, input, and output procedures, then sequentially executes two nested loops; one for the number of days in a simulation year, and one for the number of simulation years. For each simulated day, the program tests to see if a control application is to be made. If so, then the environmental dose of the control is updated, and the current residue used to calculate the control-induced mortality. After the mortality is calculated, all arrays are updated. If more than one chemical is used, arrays are also reinitialized using the Hardy-Weinberg formulas. Next, oviposition is determined and individuals are incremented one substage in age. Immigrants and their eggs are then added. Lastly, individuals are assigned to refugia, and numbers and gene frequencies updated. After a single years simulation is completed, overwintering mortality is calculated and all variables adjusted for the initial run of any subsequent year.

```
PROCEDURE ERMSIMULATOR;

CONST

 SPECIES = 2;

TYPE

 ARY  = ARRAY [1..20, 1..SPECIES] OF REAL;
 ARRY = ARRAY [1..11, 1..20, 1..6, 1..SPECIES] OF REAL;
 ROSS = ARRAY [1..6, 1..SPECIES] OF REAL;

 COMPLEXTEST = @FIRST;
 FIRST       = RECORD
               COMPLEX: ARRAY [1..6, 1..6, 1..SPECIES] OF REAL;
              END;

 VAR

 ERMIMMIG, CMD1, ERM, ERMTOTALS,
 DINC, BLBIMM                        : REAL;

 IDAY, IYR, BEFORE, NUMRUNS, SIMNUMYEARS  : INTEGER;

 IA, ISL, IP, IRF, LE, LSL, LP,
 IML, LML, ILL, LLL, NCC1, NCC2     : ARRAY [1..SPECIES] OF INTEGER;

 ADUL, CC, EGG, FEC, PINIT, PUP,
 Q, SXRAT, T, TLARV, TIMIG ,
 TIMMGEN, ETHRESH, TNOEGGS          : ARRAY [1..SPECIES] OF REAL;

 CD                          : ARRAY [1..6] OF REAL;

 THLIF                       : ARRAY [1..NUMPESTICIDES] OF REAL;

 TOTRF, RSDIS, RRDIS, SS1, RS1,
 RR1, TOTRFREQ, IMIGFREQ            : CROSS;

 WIMIG, FIMIG, AIMIG                : ARRAY [1..SPECIES] OF REAL;

 BEGN, FRACIN, REFINIT, SURV        : ARY;

 PROP                        : ARRAY [1..10] OF REAL;

 RR, RS, SS                  : ARRY;

 STAREF                      : ARRAY [1..6, 1..SPECIES] OF REAL;

 CRESIS                   : ARRAY [1..6, 1..6, 1..SPECIES] OF REAL;

 TOTAL, RGENE, RGENEFREQ     : ARRAY [1..6, 1..20, 1..SPECIES] OF REAL;


 IGD, IGEN, INTRVL, I, K, M, N,
 W, NDAYS, NGENS, NYRS, NEWIDAY,
 NEWYR, INITIAL, LAST, B,
 COUNTER, CHOICE, KORIG      : INTEGER;

 NTHR1, NTHR2                : ARRAY [1..SPECIES] OF INTEGER;

 STEP, TEST, PREDSR, PREYNC,
 PREDFC, C, SRMIN, SMNCON,
 FECMOD, CONMAX, FULLMAX,
 FECMIN, FMNCON, F           : REAL;

 PREDAB                   : ARRAY [1..300] OF REAL;

 EARLY, OW, SIGNAL           : BOOLEAN;

 SSD50, RSD50, RRD50, SSD95,
 RSD95, RRD95                : COMPLEXTEST;
```

```
 ISPCAL, ISPCALHOLD          : ARRAY [1..20] OF INTEGER;

 WSURV                       : ARY;

 NAME                        : PACKED ARRAY [1..20, 1..SPECIES] OF CHAR;

LABEL  187, 240, 250;


PROCEDURE DATAINITIALIZE;

 VAR

  I, J, K, L, P   : INTEGER;

 BEGIN

  IGEN : = 1;
  IYR : = 1;
  IDAY : = 0;
  PROP[1] : = 0.001;
  PROP[2] : = 0.012;
  PROP[3] : = 0.054;
  PROP[4] : = 0.16;
  PROP[5] : = 0.273;
  PROP[6] : = 0.273;
  PROP[7] : = 0.16;
  PROP[8] : = 0.054;
  PROP[9] : = 0.012;
  PROP[10] : = 0.001;
    FOR I : = 1 TO 6 DO CD[I] : = 0;
    FOR I : = 1 TO 6 DO
   FOR J : = 1 TO SPECIES DO
    BEGIN
     TOTRFREQ[I,J] : = 0;
     TOTRF[I,J] : = 0;
    END;
     FOR J : = 1 TO SPECIES DO TNOEGGS[J] : = 0;
     FOR I : = 1 TO 11 DO
       FOR J : = 1 TO 20 DO
         FOR K : = 1 TO 6 DO
           FOR L : = 1 TO SPECIES DO
             BEGIN
               SS[I,J,K,L] : = 0;
               RS[I,J,K,L] : = 0;
               RR[I,J,K,L] : = 0;
             END;

END {Procedure DATAINITIALIZE};


PROCEDURE GETLIBPARAMS;

 VAR

  NUM, I, W, J, SELECTION  : INTEGER;

   PROCEDURE INA(VAR A: ARY);

  VAR

   I, J  : INTEGER;

  BEGIN

   FOR I : = 1 TO 20 DO READ(PARAMS2, A[I, W]);
   READLN(PARAMS2);

  END  {Procedure INA};

BEGIN  {Procedure GETLIBPARAMS}
```

```
  IF NUMRUNS = 1
    THEN RESET(PARAMS2,'NAME = SERA2.DAT.B')
    ELSE RESET(PARAMS2,'NAME = SERA2SS.DAT.B');

  FOR W : = 1 TO SPECIES DO
   BEGIN
    READLN(PARAMS2, LE[W], ISL[W], LSL[W], IML[W], LML[W], ILL[W],
      LLL[W], IP[W], LP[W], IA[W]);
    READLN(PARAMS2, IRF[W], SXRAT[W], NCC1[W], NCC2[W], NTHR1[W],
           NTHR2[W]);
    INA(BEGN);
    INA(REFINIT);
    FOR I : = 1 TO 19 DO READ(PARAMS2, SURV[I,W]);
    READLN(PARAMS2);
    INA(WSURV);
    INA(FRACIN);
    FOR I : = 1 TO 6 DO
     BEGIN     {CROSS-RESISTANCE}
      FOR J : = 1 TO NUMPESTICIDES DO READ(PARAMS2, CRESIS[I,J,W]);
      READLN(PARAMS2);
     END;
    FOR I : = 1 TO 6 DO READ(PARAMS2, STAREF[I,W]);
    READLN(PARAMS2);
    READLN(PARAMS2, FEC[W], CC[W], ETHRESH[W], PINIT[W], T[W]);
    FOR I : = 1 TO 6 DO READ(PARAMS2, RSDIS[I,W], RRDIS[I,W]);
    READLN(PARAMS2);
    FOR I : = 1 TO 6 DO READ(PARAMS2, IMIGFREQ[I,W]);
    READ(PARAMS2, WIMIG[W], FIMIG[W], AIMIG[W]);
    READLN(PARAMS2);
    FOR I : = 1 TO 6 DO
     BEGIN
      FOR J : = 1 TO 3 DO
        READ(PARAMS2, SSD50@.COMPLEX[J,I,W],
        RSD50@.COMPLEX[J,I,W], RRD50@.COMPLEX[J,I,W]);
      READLN(PARAMS2);
      FOR J : = 4 TO 6 DO
        READ(PARAMS2, SSD50@.COMPLEX[J,I,W],
        RSD50@.COMPLEX[J,I,W], RRD50@.COMPLEX[J,I,W]);
      READLN(PARAMS2);
     END;
    FOR I : = 1 TO 6 DO
     BEGIN
      FOR J : = 1 TO 3 DO
       READ(PARAMS2, SSD95@.COMPLEX[J,I,W], RSD95@.COMPLEX[J,I,W],
         RRD95@.COMPLEX[J,I,W]);
      READLN(PARAMS2);
      FOR J : = 4 TO 6 DO
       READ(PARAMS2, SSD95@.COMPLEX[J,I,W], RSD95@.COMPLEX[J,I,W],
         RRD95@.COMPLEX[J,I,W]);
      READLN(PARAMS2);
     END;
   END; {W LOOP}
  READLN(PARAMS2, NYRS, NGENS, NDAYS, DINC, INTRVL);
  FOR I : = 1 TO 20 DO
   BEGIN
    READ(PARAMS2, ISPCAL[I]);
    ISPCALHOLD[I] : = ISPCAL[I];
   END;
  READLN(PARAMS2);
  FOR J : = 1 TO 6 DO READLN(PARAMS2);
  FOR I : = 1 TO 6 DO READ(PARAMS2, THLIF[I]);
  READLN(PARAMS2);
  READLN(PARAMS2, C, SMNCON, SRMIN, FULLMAX, CONMAX, FECMIN,
    FMNCON, F);

END; {Procedure GETLIBPARAMS}


PROCEDURE INIT;

  VAR
```

```
TEMPSS, TEMPRS, TEMPRR  : ARRAY [1..NUMPESTICIDES] OF REAL;

T1, T2                  : REAL;

I, J, W, P, N1          : INTEGER;

 BEGIN

EARLY : = TRUE;
FOR W : = 1 TO SPECIES DO
 BEGIN
  FOR P : = 1 TO CURRENTNUMCHEMS DO
   BEGIN
    FOR J : = 1 TO 20 DO
     BEGIN
      TOTAL[P,J, W] : = 0.0;
      RGENE[P,J, W] : = 0.0;
      IF (TOTAL[P,J, W] > 0.0)
       THEN RGENEFREQ[P,J,W] : = PINIT[W]
       ELSE RGENEFREQ[P,J,W] : = 0.0;
     END;
    TOTRFREQ[P,W] : = PINIT[W];
   END;
  Q[W] : = 1.0 - PINIT[W];
  T1 : = Q[W] * Q[W] * T[W];
  T2 : = PINIT[W] * PINIT[W];
  IF T1 < 0.00000000000000000001 THEN T1 : = 0.0;
  IF T2 < 0.00000000000000000001 THEN T2 : = 0.0;
  TIMIG[W] : = 0.0;
  FOR P : = 1 TO CURRENTNUMCHEMS DO
   BEGIN
    FOR J : = 1 TO 20 DO
     BEGIN
      TEMPSS[P] : = T1*BEGN[J,W]*(1.0-REFINIT[J,W]);
      TEMPRS[P] : = (2.0*PINIT[W]*Q[W])*T[W]*BEGN[J,W]
        *(1.0-REFINIT[J,W]);
      TEMPRR[P] : = T2*T[W]*BEGN[J,W]*(1.0-REFINIT[J,W]);

      SS[1,J,P,W] : = TEMPSS[P]*PROP[1];
      RS[1,J,P,W] : = TEMPRS[P]*PROP[1];
      RR[1,J,P,W] : = TEMPRR[P]*PROP[1];
      SS[2,J,P,W] : = TEMPSS[P]*PROP[2];
      RS[2,J,P,W] : = TEMPRS[P]*PROP[2];
      RR[2,J,P,W] : = TEMPRR[P]*PROP[2];
      SS[3,J,P,W] : = TEMPSS[P]*PROP[3];
      RS[3,J,P,W] : = TEMPRS[P]*PROP[3];
      RR[3,J,P,W] : = TEMPRR[P]*PROP[3];
      SS[4,J,P,W] : = TEMPSS[P]*PROP[4];
      RS[4,J,P,W] : = TEMPRS[P]*PROP[4];
      RR[4,J,P,W] : = TEMPRR[P]*PROP[4];
      SS[5,J,P,W] : = TEMPSS[P]*PROP[5];
      RS[5,J,P,W] : = TEMPRS[P]*PROP[5];
      RR[5,J,P,W] : = TEMPRR[P]*PROP[5];
      SS[6,J,P,W] : = TEMPSS[P]*PROP[6];
      RS[6,J,P,W] : = TEMPRS[P]*PROP[6];
      RR[6,J,P,W] : = TEMPRR[P]*PROP[6];
      SS[7,J,P,W] : = TEMPSS[P]*PROP[7];
      RS[7,J,P,W] : = TEMPRS[P]*PROP[7];
      RR[7,J,P,W] : = TEMPRR[P]*PROP[7];
      SS[8,J,P,W] : = TEMPSS[P]*PROP[8];
      RS[8,J,P,W] : = TEMPRS[P]*PROP[8];
      RR[8,J,P,W] : = TEMPRR[P]*PROP[8];
      SS[9,J,P,W] : = TEMPSS[P]*PROP[9];
      RS[9,J,P,W] : = TEMPRS[P]*PROP[9];
      RR[9,J,P,W] : = TEMPRR[P]*PROP[9];
      SS[10,J,P,W] : = TEMPSS[P]*PROP[10];
      RS[10,J,P,W] : = TEMPRS[P]*PROP[10];
      RR[10,J,P,W] : = TEMPRR[P]*PROP[10];

     END;
   END;
  FOR P:= 1 TO CURRENTNUMCHEMS DO
```

```
    FOR J : = 1 TO 20 DO
     BEGIN
      SS[11,J,P,W] : = T1*BEGN[J,W]*REFINIT[J,W];
      RS[11,J,P,W] : = 2.0*PINIT[W]*Q[W]*T[W]*BEGN[J,W]
        *REFINIT[J,W];
      RR[11,J,P,W] : = T2*T[W]*BEGN[J,W]*REFINIT[J,W];
     END;
   FOR P : = 1 TO CURRENTNUMCHEMS DO
    BEGIN
     FOR J : = 1 TO 20 DO
      BEGIN

       TOTAL[P,J,W] : = TOTAL[P,J,W] + SS[1,J,P,W]
         + RS[1,J,P,W] + RR[1,J,P,W];
       RGENE[P,J,W] : = RGENE[P,J,W] + RR[1,J,P,W]
         + (0.5*RS[1,J,P,W]);
       TOTAL[P,J,W] : = TOTAL[P,J,W] + SS[2,J,P,W]
         + RS[2,J,P,W] + RR[2,J,P,W];
       RGENE[P,J,W] : = RGENE[P,J,W] + RR[2,J,P,W]
         + (0.5*RS[2,J,P,W]);
       TOTAL[P,J,W] : = TOTAL[P,J,W] + SS[3,J,P,W]
         + RS[3,J,P,W] + RR[3,J,P,W];
       RGENE[P,J,W] : = RGENE[P,J,W] + RR[3,J,P,W]
         + (0.5*RS[3,J,P,W]);
       TOTAL[P,J,W] : = TOTAL[P,J,W] + SS[4,J,P,W]
         + RS[4,J,P,W] + RR[4,J,P,W];
       RGENE[P,J,W] : = RGENE[P,J,W] + RR[4,J,P,W]
         + (0.5*RS[4,J,P,W]);
       TOTAL[P,J,W] : = TOTAL[P,J,W] + SS[5,J,P,W]
         + RS[5,J,P,W] + RR[5,J,P,W];
       RGENE[P,J,W] : = RGENE[P,J,W] + RR[5,J,P,W]
         + (0.5*RS[5,J,P,W]);
       TOTAL[P,J,W] : = TOTAL[P,J,W] + SS[6,J,P,W]
         + RS[6,J,P,W] + RR[6,J,P,W];
       RGENE[P,J,W] : = RGENE[P,J,W] + RR[6,J,P,W]
         + (0.5*RS[6,J,P,W]);
       TOTAL[P,J,W] : = TOTAL[P,J,W] + SS[7,J,P,W]
         + RS[7,J,P,W] + RR[7,J,P,W];
       RGENE[P,J,W] : = RGENE[P,J,W] + RR[7,J,P,W]
         + (0.5*RS[7,J,P,W]);
       TOTAL[P,J,W] : = TOTAL[P,J,W] + SS[8,J,P,W]
         + RS[8,J,P,W] + RR[8,J,P,W];
       RGENE[P,J,W] : = RGENE[P,J,W] + RR[8,J,P,W]
         + (0.5*RS[8,J,P,W]);
       TOTAL[P,J,W] : = TOTAL[P,J,W] + SS[9,J,P,W]
         + RS[9,J,P,W] + RR[9,J,P,W];
       RGENE[P,J,W] : = RGENE[P,J,W] + RR[9,J,P,W]
         + (0.5*RS[9,J,P,W]);
       TOTAL[P,J,W] : = TOTAL[P,J,W] + SS[10,J,P,W]
         + RS[10,J,P,W] + RR[10,J,P,W];
       RGENE[P,J,W] : = RGENE[P,J,W] + RR[10,J,P,W]
         + (0.5*RS[10,J,P,W]);
       TOTAL[P,J,W] : = TOTAL[P,J,W] + SS[11,J,P,W]
         + RS[11,J,P,W] + RR[11,J,P,W];
       RGENE[P,J,W] : = RGENE[P,J,W] + RR[11,J,P,W]
         + (0.5*RS[11,J,P,W]);

      IF (TOTAL[P,J,W] > 0.0)
       THEN RGENEFREQ[P,J,W] : = RGENE[P,J,W]/TOTAL[P,J,W]
       ELSE RGENEFREQ[P,J,W] : = 0.0;

      IF RGENEFREQ[P,J,W] > 1.0 THEN RGENEFREQ[P,J,W] : = 1.0;
     END; {J LOOP}
    END; {P LOOP}
  END; {W LOOP}
END; {Procedure INIT}


PROCEDURE SUM;

TYPE
```

```
 CHEMAGE   = @CA;
 CA        = RECORD
             CONTROLARRAY : ARRAY [1..NUMPESTICIDES, 1..20] OF REAL;
             END;

VAR

 AGENEFREQ, TEMPRGENE, TEMPRFREQ  : CHEMAGE;

PROCEDURE SUMP1;

 BEGIN

  NEW(AGENEFREQ);
  NEW(TEMPRGENE);
  NEW(TEMPRFREQ);

 END;

PROCEDURE SUMP2;

 VAR

  TOT, TOTRGENE, TN, TSS, TRS,
  TRR, ALAN, A             : ARRAY [1..NUMPESTICIDES] OF REAL;

  DIFF                     : ARRAY [1..NUMPESTICIDES,1..20] OF REAL;

 VAR

  I, W, J, P, J1  : INTEGER;

 PROCEDURE SUMP22(W:INTEGER);

  VAR

   I, J, P, J1  : INTEGER;

  BEGIN

   FOR I : = 1 TO CURRENTNUMCHEMS DO
    BEGIN
     TSS[I] : = 0;
     TRS[I] : = 0;
     TRR[I] : = 0;
    END;
   IF (EARLY = TRUE) THEN COUNTER : = 1;
   EGG[W] : = 0.0;
   TLARV[W] : = 0.0;
   PUP[W] : = 0.0;
   ADUL[W] : = 0.0;
   FOR I : = 1 TO 11 DO
    BEGIN
     FOR J : = 1 TO LE[W] DO
       EGG[W] : = EGG[W]+SS[I,J,COUNTER,W]+RS[I,J,COUNTER,W]
       +RR[I,J,COUNTER,W];
     FOR J : = ISL[W] TO LLL[W] DO
       TLARV[W] : = TLARV[W]+SS[I,J,COUNTER,W]+RS[I,J,COUNTER,W]
       +RR[I,J,COUNTER,W];
     FOR J : = IP[W] TO LP[W] DO
       PUP[W] : = PUP[W]+SS[I,J,COUNTER,W]+RS[I,J,COUNTER,W]
       +RR[I,J,COUNTER,W];
     FOR J : = IA[W] TO 20 DO
       ADUL[W] : = ADUL[W]+SS[I,J,COUNTER,W]+RS[I,J,COUNTER,W]
       +RR[I,J,COUNTER,W];
    END;
   FOR P : = 1 TO CURRENTNUMCHEMS DO
    FOR J : = 1 TO 20 DO
     FOR I : =1 TO 11 DO
      BEGIN
       TSS[P] : = TSS[P] + SS[I,J,P,W];
       TRS[P] : = TRS[P] + RS[I,J,P,W];
```

```
    TRR[P] : = TRR[P] + RR[I,J,P,W];
   END;
 T[W] : = TSS[COUNTER] + TRS[COUNTER] + TRR[COUNTER];

END; {Procedure SUMP22}

PROCEDURE SUMP23(W:INTEGER);

VAR

 I, J, P, J1   : INTEGER;

BEGIN

   FOR J:= 1 TO 20 DO
    BEGIN

      TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
        + SS[1,J,COUNTER,W] + RS[1,J,COUNTER,W]
        + RR[1,J,COUNTER,W];
      TEMPRGENE@.CONTROLARRAY[COUNTER,J] : =
        TEMPRGENE@.CONTROLARRAY[COUNTER, J]
        + RR[1,J,COUNTER,W] + (0.5*RS[1,J,COUNTER,W]);
      TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
        + SS[2,J,COUNTER,W] + RS[2,J,COUNTER,W]
        + RR[2,J,COUNTER,W];
      TEMPRGENE@.CONTROLARRAY[COUNTER,J] : =
        TEMPRGENE@.CONTROLARRAY[COUNTER, J]
        + RR[2,J,COUNTER,W] + (0.5*RS[2,J,COUNTER,W]);
      TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
        + SS[3,J,COUNTER,W] + RS[3,J,COUNTER,W]
        + RR[3,J,COUNTER,W];
      TEMPRGENE@.CONTROLARRAY[COUNTER,J] : =
        TEMPRGENE@.CONTROLARRAY[COUNTER, J]
        + RR[3,J,COUNTER,W] + (0.5*RS[3,J,COUNTER,W]);
      TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
        + SS[4,J,COUNTER,W] + RS[4,J,COUNTER,W]
        + RR[4,J,COUNTER,W];
      TEMPRGENE@.CONTROLARRAY[COUNTER,J] : =
        TEMPRGENE@.CONTROLARRAY[COUNTER, J]
        + RR[4,J,COUNTER,W] + (0.5*RS[4,J,COUNTER,W]);
      TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
        + SS[5,J,COUNTER,W] + RS[5,J,COUNTER,W]
        + RR[5,J,COUNTER,W];
      TEMPRGENE@.CONTROLARRAY[COUNTER,J] : =
        TEMPRGENE@.CONTROLARRAY[COUNTER, J]
        + RR[5,J,COUNTER,W] + (0.5*RS[5,J,COUNTER,W]);
      TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
        + SS[6,J,COUNTER,W] + RS[6,J,COUNTER,W]
        + RR[6,J,COUNTER,W];
      TEMPRGENE@.CONTROLARRAY[COUNTER,J] : =
        TEMPRGENE@.CONTROLARRAY[COUNTER, J]
        + RR[6,J,COUNTER,W] + (0.5*RS[6,J,COUNTER,W]);
      TOTAL[COUNTER.J.W] : = TOTAL[COUNTER,J,W]
        + SS[7,J,COUNTER,W] + RS[7,J,COUNTER,W]
        + RR[7,J,COUNTER,W];
      TEMPRGENE@.CONTROLARRAY[COUNTER,J] : =
        TEMPRGENE@.CONTROLARRAY[COUNTER, J]
        + RR[7,J,COUNTER,W] + (0.5*RS[7,J,COUNTER,W]);
      TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
        + SS[8,J,COUNTER,W] + RS[8,J,COUNTER,W]
        + RR[8,J,COUNTER,W];
      TEMPRGENE@.CONTROLARRAY[COUNTER,J] : =
        TEMPRGENE@.CONTROLARRAY[COUNTER, J]
        + RR[8,J,COUNTER,W] + (0.5*RS[8,J,COUNTER,W]);
      TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
        + SS[9,J,COUNTER,W] + RS[9,J,COUNTER,W]
        + RR[9,J,COUNTER,W];
      TEMPRGENE@.CONTROLARRAY[COUNTER,J] : =
        TEMPRGENE@.CONTROLARRAY[COUNTER, J]
        + RR[9,J,COUNTER,W] + (0.5*RS[9,J,COUNTER,W]);
      TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
```

```
        +SS[10,J,COUNTER,W]+RS[10,J,COUNTER,W]
        +RR[10,J,COUNTER,W];
      TEMPRGENE@.CONTROLARRAY[COUNTER,J] : =
        TEMPRGENE@.CONTROLARRAY[COUNTER, J]
        +RR[10,J,COUNTER,W]+(0.5*RS[10,J,COUNTER,W]);
      TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
        +SS[11,J,COUNTER,W]+RS[11,J,COUNTER,W]
        +RR[11,J,COUNTER,W];
      TEMPRGENE@.CONTROLARRAY[COUNTER,J] : =
        TEMPRGENE@.CONTROLARRAY[COUNTER, J]
        +RR[11,J,COUNTER,W]+(0.5*RS[11,J,COUNTER,W]);

     IF (TOTAL[COUNTER,J,W] = 0.0)
     THEN TEMPRFREQ@.CONTROLARRAY[COUNTER,J] : = 0.0
     ELSE TEMPRFREQ@.CONTROLARRAY[COUNTER,J] : =
       TEMPRGENE@.CONTROLARRAY[COUNTER,J]
       /TOTAL[COUNTER,J,W];
    END;

END; {Procedure SUMP23}

BEGIN {Procedure SUMP2}

FOR W : = 1 TO SPECIES DO
 BEGIN
  SUMP22(W);
  IF ((EARLY = FALSE) AND (OW = FALSE)) THEN
   BEGIN
    FOR J : = 1 TO 20 DO
     BEGIN
      TOTAL[COUNTER,J,W] : = 0.0;
      TEMPRGENE@.CONTROLARRAY[COUNTER,J] : = 0.0;
     END;
    SUMP23(W);
    FOR P : = 1 TO CURRENTNUMCHEMS DO
     BEGIN
      A[P] : = 0.0;
      AGENEFREQ@.CONTROLARRAY[P,1] : = 0.0;
      FOR J : = IRF[W] TO 20 DO
       IF (RGENEFREQ[P,J,W] > 0.0) THEN
        BEGIN
         AGENEFREQ@.CONTROLARRAY[P,1] : =
           AGENEFREQ@.CONTROLARRAY[P,1]
           +RGENEFREQ[P,J,W];
         A[P] : = A[P]+1.0;
        END;
      FOR J1 : = 1 TO 19 DO
       BEGIN
        J : = 21 -J1;
        DIFF[P,J] : = TEMPRFREQ@.CONTROLARRAY[COUNTER,J]
          -RGENEFREQ[COUNTER,J-1,W];
       END;
     END;
    FOR P : = 1 TO CURRENTNUMCHEMS DO
     BEGIN
      FOR J1 : = 1 TO 19 DO
       BEGIN
        J : = 21-J1;
        RGENEFREQ[P,J,W] : = RGENEFREQ[P,J-1,W]+DIFF[P,J];
        IF RGENEFREQ[P,J,W] > 1.0
         THEN RGENEFREQ[P,J,W] : = 1.0;
        IF (RGENEFREQ[P,J,W] < 0.0)
         THEN RGENEFREQ[P,J,W] : = 0.0;
       END;
      IF (A[P] > 0.0)
       THEN RGENEFREQ[P,1,W] : = AGENEFREQ@.CONTROLARRAY[P,1]
         /A[P]
       ELSE RGENEFREQ[P,1,W] : = 0.0;
      IF RGENEFREQ[P,1,W] > 1.0 THEN RGENEFREQ[P,1,W] : = 1.0;
     END;
    FOR P : = 1 TO CURRENTNUMCHEMS DO
     BEGIN
```

```
   FOR J : = 1 TO 20 DO
    BEGIN
     IF (TOTAL[COUNTER,J,W] = 0.0)
      THEN RGENE[P,J,W] : = 0.0
      ELSE RGENE[P,J,W] : = RGENEFREQ[P,J,W]
                *TOTAL[COUNTER,J,W];
    END;
   END;
 END;
IF (OW = TRUE) THEN
 BEGIN
  FOR J : = 1 TO 20 DO
   BEGIN
    TOTAL[COUNTER,J,W] : = 0.0;
    TEMPRGENE@.CONTROLARRAY[COUNTER,J] : = 0.0;
   END;
  FOR J : = 1 TO 20 DO
   BEGIN

     TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
       + SS[1,J,COUNTER,W] + RS[1,J,COUNTER,W]
       + RR[1,J,COUNTER,W];
     TEMPRGENE@.CONTROLARRAY[COUNTER,J] : =
       TEMPRGENE@.CONTROLARRAY[COUNTER,J]
       + RR[1,J,COUNTER,W] + (0.5*RS[1,J,COUNTER,W]);
     TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
       + SS[2,J,COUNTER,W] + RS[2,J,COUNTER,W]
       + RR[2,J,COUNTER,W];
     TEMPRGENE@.CONTROLARRAY[COUNTER,J] : =
       TEMPRGENE@.CONTROLARRAY[COUNTER,J]
       + RR[2,J,COUNTER,W] + (0.5*RS[2,J,COUNTER,W]);
     TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
       + SS[3,J,COUNTER,W] + RS[3,J,COUNTER,W]
       + RR[3,J,COUNTER,W];
     TEMPRGENE@.CONTROLARRAY[COUNTER,J] : =
       TEMPRGENE@.CONTROLARRAY[COUNTER,J]
       + RR[3,J,COUNTER,W] + (0.5*RS[3,J,COUNTER,W]);
     TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
       + SS[4,J,COUNTER,W] + RS[4,J,COUNTER,W]
       + RR[4,J,COUNTER,W];
     TEMPRGENE@.CONTROLARRAY[COUNTER,J] : =
       TEMPRGENE@.CONTROLARRAY[COUNTER,J]
       + RR[4,J,COUNTER,W] + (0.5*RS[4,J,COUNTER,W]);
     TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
       + SS[5,J,COUNTER,W] + RS[5,J,COUNTER,W]
       + RR[5,J,COUNTER,W];
     TEMPRGENE@.CONTROLARRAY[COUNTER,J] : =
       TEMPRGENE@.CONTROLARRAY[COUNTER,J]
       + RR[5,J,COUNTER,W] + (0.5*RS[5,J,COUNTER,W]);
     TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
       + SS[6,J,COUNTER,W] + RS[6,J,COUNTER,W]
       + RR[6,J,COUNTER,W];
     TEMPRGENE@.CONTROLARRAY[COUNTER,J] : =
       TEMPRGENE@.CONTROLARRAY[COUNTER,J]
       + RR[6,J,COUNTER,W] + (0.5*RS[6,J,COUNTER,W]);
     TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
       + SS[7,J,COUNTER,W] + RS[7,J,COUNTER,W]
       + RR[7,J,COUNTER,W];
     TEMPRGENE@.CONTROLARRAY[COUNTER,J] : =
       TEMPRGENE@.CONTROLARRAY[COUNTER,J]
       + RR[7,J,COUNTER,W] + (0.5*RS[7,J,COUNTER,W]);
     TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
       + SS[8,J,COUNTER,W] + RS[8,J,COUNTER,W]
       + RR[8,J,COUNTER,W];
     TEMPRGENE@.CONTROLARRAY[COUNTER,J] : =
       TEMPRGENE@.CONTROLARRAY[COUNTER,J]
       + RR[8,J,COUNTER,W] + (0.5*RS[8,J,COUNTER,W]);
     TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
       + SS[9,J,COUNTER,W] + RS[9,J,COUNTER,W]
       + RR[9,J,COUNTER,W];
     TEMPRGENE@.CONTROLARRAY[COUNTER,J] : =
       TEMPRGENE@.CONTROLARRAY[COUNTER,J]
```

```
      +RR[9,J,COUNTER,W]+(0.5*RS[9,J,COUNTER,W]);
    TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
      +SS[10,J,COUNTER,W]+RS[10,J,COUNTER,W]
      +RR[10,J,COUNTER,W];
    TEMPRGENE@.CONTROLARRAY[COUNTER,J] : =
      TEMPRGENE@.CONTROLARRAY[COUNTER,J]
      +RR[10,J,COUNTER,W]+(0.5*RS[10,J,COUNTER,W]);
    TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
      +SS[11,J,COUNTER,W]+RS[11,J,COUNTER,W]
      +RR[11,J,COUNTER,W];
    TEMPRGENE@.CONTROLARRAY[COUNTER,J] : =
      TEMPRGENE@.CONTROLARRAY[COUNTER,J]
      +RR[11,J,COUNTER,W]+(0.5*RS[11,J,COUNTER,W]);

   IF (TOTAL[COUNTER,J,W] = 0.0)
    THEN TEMPRFREQ@.CONTROLARRAY[COUNTER,J] : = 0.0
    ELSE TEMPRFREQ@.CONTROLARRAY[COUNTER,J] : =
      TEMPRGENE@.CONTROLARRAY[COUNTER,J]
            /TOTAL[COUNTER,J,W];
  END;
 FOR P : = 1 TO CURRENTNUMCHEMS DO
  FOR J : = 1 TO 20 DO
   BEGIN
    DIFF[P,J] : = TEMPRFREQ@.CONTROLARRAY[COUNTER,J]
      -RGENEFREQ[COUNTER,J,W];
    RGENEFREQ[P,J,W] : = RGENEFREQ[P,J,W]+DIFF[P,J];
    IF RGENEFREQ[P,J,W] > 1.0
     THEN RGENEFREQ[P,J,W] : = 1.0;
    IF (RGENEFREQ[P,J,W] < 0.0)
     THEN RGENEFREQ[P,J,W] : = 0.0;
   END;
 FOR P : = 1 TO CURRENTNUMCHEMS DO
  FOR J : = 1 TO 20 DO
   BEGIN
    IF (TOTAL[COUNTER,J,W] = 0.0)
     THEN RGENE[P,J,W] : = 0.0
     ELSE RGENE[P,J,W] : = RGENEFREQ[P,J,W]
       *TOTAL[COUNTER,J,W];
   END;
 END;
FOR P : = 1 TO CURRENTNUMCHEMS DO
 BEGIN
  TOT[P] : = 0.0;
  TOTRGENE[P] : = 0.0;
  FOR J : = 1 TO 20 DO
   BEGIN
    TOT[P] : = TOT[P]+TOTAL[COUNTER,J,W];
    TOTRGENE[P] : = TOTRGENE[P]+RGENE[P,J,W];
   END;
  IF (TOT[P] > 0.0)
   THEN TOTRFREQ[P,W] : = TOTRGENE[P]/TOT[P]
   ELSE TOTRFREQ[P,W] : = PINIT[W];
  IF (TOTRFREQ[P,W] < PINIT[W])
   THEN TOTRFREQ[P,W] : = PINIT[W];
 END;
TNOEGGS[W] : = ADUL[W]+TLARV[W]+PUP[W];

 IF (W = 1) AND ((IYR MOD 30) = 0) THEN
  BEGIN
   WRITE(FIRSTERM,VERTEXNUMBER:4,IDAY:8);
   WRITE(FIRSTERM,EGG[1]:12:3,TLARV[1]:11:3);
   WRITE(FIRSTERM,PUP[1]:11:3);
   WRITELN(FIRSTERM,ADUL[1]:11:3,T[1]:11:3);
  END;
 IF (W = 1) AND (IDAY = 40) THEN
  BEGIN
   WRITE(RESISTER,' ',VERTEXNUMBER:2,'     ',IYR:2,'  ');
   CHEMNUMBER : = 0;
   FOR P : = 1 TO NUMPESTICIDES DO
    IF P IN CURRENTCHEMS
     THEN
      BEGIN
```

```
          CHEMNUMBER : = CHEMNUMBER + 1;
          WRITE(RESISTER,' ',TOTRFREQ[CHEMNUMBER,1]:8:6);
         END
        ELSE
         WRITE(RESISTER,'        ');
      WRITELN(RESISTER);
      WRITE(POPSERM,VERTEXNUMBER:4,IYR:8);
      WRITE(POPSERM,EGG[1]:12:3,TLARV[1]:11:3);
      WRITE(POPSERM,PUP[1]:11:3);
      WRITELN(POPSERM,ADUL[1]:11:3,T[1]:11:3,CMD1:11:3);
     END;
    IF (W = 2) AND ((IYR MOD 30) = 0) THEN
    BEGIN
     WRITE(FIRSTBLB,VERTEXNUMBER:4,IDAY:8);
     WRITE(FIRSTBLB,EGG[2]:12:3,TLARV[2]:11:3);
     WRITE(FIRSTBLB,PUP[2]:11:3);
     WRITELN(FIRSTBLB,ADUL[2]:11:3,T[2]:11:3);
    END;
    IF (W = 2) AND (IDAY = 40) THEN
    BEGIN
     WRITE(RESISTBL,' ',VERTEXNUMBER:2,'     ',IYR:2,'  ');
     CHEMNUMBER : = 0;
     FOR P : = 1 TO NUMPESTICIDES DO
      IF P IN CURRENTCHEMS
       THEN
        BEGIN
         CHEMNUMBER : = CHEMNUMBER + 1;
         WRITE(RESISTBL,' ',TOTRFREQ[CHEMNUMBER,2]:8:6);
        END
       ELSE
        WRITE(RESISTBL,'        ');
     WRITELN(RESISTBL);
     WRITE(POPSBLB,VERTEXNUMBER:4,IYR:8);
     WRITE(POPSBLB,EGG[2]:12:3,TLARV[2]:11:3);
     WRITE(POPSBLB,PUP[2]:11:3);
     WRITELN(POPSBLB,ADUL[2]:11:3,T[2]:11:3);
    END;

  END;
 END; {Procedure SUMP2}

PROCEDURE SUMP3;

 BEGIN

  DISPOSE(AGENEFREQ);
  DISPOSE(TEMPRGENE);
  DISPOSE(TEMPRFREQ);

 END; {Procedure SUMP3}

BEGIN  {Procedure SUM}

 SUMP1;
 SUMP2;
 SUMP3;

END; {Procedure SUM}


PROCEDURE PRED; {PREDATOR-PREY INTERACTIONS}

VAR

 PREDCN, PDMEAN, R, ZTOT, ABTOT, X, Y, V, Z  : REAL;

 I, J, L  : INTEGER;

 CONTINUES  : BOOLEAN;

BEGIN
```

```
  {COMPUTE PROPORTION OF PREY NOT CONSUMED -- PREYNC}

  PREDCN : = 0.0;
  PDMEAN : = T[2]-EGG[2];
  IF (PDMEAN< >0.0) THEN
   BEGIN
    R : = PDMEAN/(C+PDMEAN);
    ZTOT : = 0.0;

  {PROPORTION OF LEAVES WITH EACH PREDATOR ABUNDANCE -- PREDAB(I) }

    PREDAB[1] : = EXP(LN(1+PDMEAN/C)*(-1)*C);
    ABTOT : = PREDAB[1];
    I : = 1;
    CONTINUES : = TRUE;
    WHILE ((I< =300) AND CONTINUES) DO
     BEGIN
      J : = I+1;
      PREDAB[J] : = PREDAB[I]*(C+I-1)*R/I;
      IF (PREDAB[J]<0.05)
       THEN CONTINUES : = FALSE
       ELSE
        BEGIN
         ABTOT : = ABTOT+PREDAB[J];
         I : = I+1;
        END: {ELSE}
     END: {WHILE}
    PREDAB[J] : = 1.0-ABTOT;

    {ROYAMA EQUATION PARAMETERS -- FIXED FOR A GIVEN PREY ABUNDANCE}

    IF T[1] = 0  THEN T[1] : = 0.000001;
    X : = T[1];
    Y : = CONMAX;
    FOR L : = 2 TO J DO
     BEGIN
      V : = L-1;
      Z : = (-LN(EXP(-Y*F*V)-EXP(-F*X-F*Y*V)+EXP(-F*X)))/F;
      ZTOT : = ZTOT+Z*PREDAB[L];
     END:
    PREYNC : = (X-ZTOT)/X;

    {COMPUTE PREDATOR'S CONSUMPTION RATE}

    PREDCN : = ZTOT/PDMEAN;
   END;

   {COMPUTE PREDATOR'S SURVIVORSHIP MULTIPLIER}

   IF (PREDCN < SMNCON)
    THEN PREDSR:=(1.0-(SMNCON-PREDCN)/SMNCON)*SRMIN
    ELSE PREDSR:=SRMIN+((1.0-SRMIN)/(FULLMAX-SMNCON))*(PREDCN-SMNCON);
   IF (PREDSR > 1.0) THEN PREDSR : = 1.0;

   {COMPUTE PREDATOR'S FECUNDITY MULTIPLIER}

   IF (PREDCN<FMNCON)
    THEN PREDFC:=(1.0-(FMNCON-PREDCN)/FMNCON)*FECMIN
    ELSE PREDFC:=FECMIN+((1.0-FECMIN)/(FULLMAX-FMNCON))
      *(PREDCN-FMNCON);
   IF (PREDFC > 1.0) THEN PREDFC : = 1.0;
 END;  {Procedure PRED}


PROCEDURE THRESH(W, N1, N2, P: INTEGER; VAR TEST: REAL);

{SUM INDIVIDUALS IN STAGES N1 TO N2 TO TEST AGAINST THRESHOLD
OR CARRYING CAPACITY }

 VAR

  I, J  : INTEGER;
```

```
{ONLY TESTED WITH SUM OF ONE CHEMICAL SINCE THEY SHOULD ALL HAVE
THE SAME NUMBERS}

 BEGIN

 FOR I : = 1 TO 11 DO
     FOR J : = N1 TO N2 DO
   TEST : = TEST+SS[I,J,P,W]+RS[I,J,P,W]+RR[I,J,P,W];

 END; {Procedure THRESH}


 PROCEDURE NEXT;

{ DETERMINE THE ARRAY VALUES FOR NEXT DAY }

 TYPE

 CHEMAGE   = @CA;
 CA        = RECORD
      CONTROLARRAY: ARRAY [1..NUMPESTICIDES, 1..20] OF REAL;
      END;

  VAR

 MID, T1, T2, T3, TEMP, CCTEST, DDS,
 T4, T5, T6              : REAL;

 TOTM, SSF, RSF, RRF, SSM, RSM,
 RRM             : ARRAY [1..NUMPESTICIDES] OF REAL;

 W, I, J, J1, P              : INTEGER;

 TEMPSS, TEMPRS, TEMPRR          : CHEMAGE;

BEGIN

 NEW(TEMPSS);
 NEW(TEMPRS);
 NEW(TEMPRR);

 FOR W : = 1 TO SPECIES DO
  BEGIN
   FOR P : = 1 TO CURRENTNUMCHEMS DO
    BEGIN      {BEGIN RE-INIT PROC}
     FOR J : = IRF[W] TO 20 DO
      BEGIN
       TOTAL[P,J,W] : = 0.0;

        TOTAL[P,J,W] : = TOTAL[P,J,W]+SS[1,J,P,W]+RS[1,J,P,W]
          +RR[1,J,P,W];
        TOTAL[P,J,W] : = TOTAL[P,J,W]+SS[2,J,P,W]+RS[2,J,P,W]
          +RR[2,J,P,W];
        TOTAL[P,J,W] : = TOTAL[P,J,W]+SS[3,J,P,W]+RS[3,J,P,W]
          +RR[3,J,P,W];
        TOTAL[P,J,W] : = TOTAL[P,J,W]+SS[4,J,P,W]+RS[4,J,P,W]
          +RR[4,J,P,W];
        TOTAL[P,J,W] : = TOTAL[P,J,W]+SS[5,J,P,W]+RS[5,J,P,W]
          +RR[5,J,P,W];
        TOTAL[P,J,W] : = TOTAL[P,J,W]+SS[6,J,P,W]+RS[6,J,P,W]
          +RR[6,J,P,W];
        TOTAL[P,J,W] : = TOTAL[P,J,W]+SS[7,J,P,W]+RS[7,J,P,W]
          +RR[7,J,P,W];
        TOTAL[P,J,W] : = TOTAL[P,J,W]+SS[8,J,P,W]+RS[8,J,P,W]
          +RR[8,J,P,W];
        TOTAL[P,J,W] : = TOTAL[P,J,W]+SS[9,J,P,W]+RS[9,J,P,W]
          +RR[9,J,P,W];
        TOTAL[P,J,W] : = TOTAL[P,J,W]+SS[10,J,P,W]+RS[10,J,P,W]
          +RR[10,J,P,W];
        TOTAL[P,J,W] : = TOTAL[P,J,W]+SS[11,J,P,W]+RS[11,J,P,W]
          +RR[11,J,P,W];
```

```
  END;
 FOR J : = IRF[W] TO 20 DO
  BEGIN
   Q[W] : = 1.0-RGENEFREQ[P,J,W];
   T4 : = Q[W]*Q[W];
   T5 : = RGENEFREQ[P,J,W]*RGENEFREQ[P,J,W];
   T6 : = 2*RGENEFREQ[P,J,W]*Q[W];
   IF T4 < 0.00000000000000000001 THEN T4 : = 0.0;
   IF T5 < 0.00000000000000000001 THEN T5 : = 0.0;
   IF T6 < 0.00000000000000000001 THEN T6 : = 0.0;
   TEMPSS@.CONTROLARRAY[P,J] : = T4*TOTAL[P,J,W]
     *(1.0-STAREF[6,W]);
   TEMPRS@.CONTROLARRAY[P,J] : = T6*TOTAL[P,J,W]
     *(1.0-STAREF[6,W]);
   TEMPRR@.CONTROLARRAY[P,J] : = T5*TOTAL[P,J,W]
     *(1.0-STAREF[6,W]);
   SS[11,J,P,W] : = T4*TOTAL[P,J,W]*STAREF[6,W];
   RS[11,J,P,W] : = T6*TOTAL[P,J,W]*STAREF[6,W];
   RR[11,J,P,W] : = T5*TOTAL[P,J,W]*STAREF[6,W];

     SS[1,J,P,W] : = TEMPSS@.CONTROLARRAY[P,J]*PROP[1];
     RS[1,J,P,W] : = TEMPRS@.CONTROLARRAY[P,J]*PROP[1];
     RR[1,J,P,W] : = TEMPRR@.CONTROLARRAY[P,J]*PROP[1];
     SS[2,J,P,W] : = TEMPSS@.CONTROLARRAY[P,J]*PROP[2];
     RS[2,J,P,W] : = TEMPRS@.CONTROLARRAY[P,J]*PROP[2];
     RR[2,J,P,W] : = TEMPRR@.CONTROLARRAY[P,J]*PROP[2];
     SS[3,J,P,W] : = TEMPSS@.CONTROLARRAY[P,J]*PROP[3];
     RS[3,J,P,W] : = TEMPRS@.CONTROLARRAY[P,J]*PROP[3];
     RR[3,J,P,W] : = TEMPRR@.CONTROLARRAY[P,J]*PROP[3];
     SS[4,J,P,W] : = TEMPSS@.CONTROLARRAY[P,J]*PROP[4];
     RS[4,J,P,W] : = TEMPRS@.CONTROLARRAY[P,J]*PROP[4];
     RR[4,J,P,W] : = TEMPRR@.CONTROLARRAY[P,J]*PROP[4];
     SS[5,J,P,W] : = TEMPSS@.CONTROLARRAY[P,J]*PROP[5];
     RS[5,J,P,W] : = TEMPRS@.CONTROLARRAY[P,J]*PROP[5];
     RR[5,J,P,W] : = TEMPRR@.CONTROLARRAY[P,J]*PROP[5];
     SS[6,J,P,W] : = TEMPSS@.CONTROLARRAY[P,J]*PROP[6];
     RS[6,J,P,W] : = TEMPRS@.CONTROLARRAY[P,J]*PROP[6];
     RR[6,J,P,W] : = TEMPRR@.CONTROLARRAY[P,J]*PROP[6];
     SS[7,J,P,W] : = TEMPSS@.CONTROLARRAY[P,J]*PROP[7];
     RS[7,J,P,W] : = TEMPRS@.CONTROLARRAY[P,J]*PROP[7];
     RR[7,J,P,W] : = TEMPRR@.CONTROLARRAY[P,J]*PROP[7];
     SS[8,J,P,W] : = TEMPSS@.CONTROLARRAY[P,J]*PROP[8];
     RS[8,J,P,W] : = TEMPRS@.CONTROLARRAY[P,J]*PROP[8];
     RR[8,J,P,W] : = TEMPRR@.CONTROLARRAY[P,J]*PROP[8];
     SS[9,J,P,W] : = TEMPSS@.CONTROLARRAY[P,J]*PROP[9];
     RS[9,J,P,W] : = TEMPRS@.CONTROLARRAY[P,J]*PROP[9];
     RR[9,J,P,W] : = TEMPRR@.CONTROLARRAY[P,J]*PROP[9];
     SS[10,J,P,W] : = TEMPSS@.CONTROLARRAY[P,J]*PROP[10];
     RS[10,J,P,W] : = TEMPRS@.CONTROLARRAY[P,J]*PROP[10];
     RR[10,J,P,W] : = TEMPRR@.CONTROLARRAY[P,J]*PROP[10];

  END;
 END; {P LOOP}
FOR P : = 1 TO CURRENTNUMCHEMS DO
 BEGIN
 TOTRF[P,W] : = 0.0;
 SSF[P] : = 0.0;
 RSF[P] : = 0.0;
 RRF[P] : = 0.0;
 SSM[P] : = 0.0;
 RSM[P] : = 0.0;
 RRM[P] : = 0.0;
 FOR J : = IRF[W] TO 20 DO
  FOR I : = 1 TO 11 DO
   TOTRF[P,W] : = TOTRF[P,W]+SS[I,J,P,W]+RS[I,J,P,W]
     +RR[I,J,P,W];
 IF (TOTRF[P,W] < > 0.0) THEN
  BEGIN

  { SUM REPRODUCTION MALES AND FEMALES }
```

```
    TEMP : = SXRAT[W]+1;
    FOR I : = 1 TO 11 DO
     FOR J : = IRF[W] TO 20 DO
      BEGIN
       SSF[P] : = SSF[P]+SS[I,J,P,W]*SXRAT[W]/TEMP;
       RSF[P] : = RSF[P]+RS[I,J,P,W]*SXRAT[W]/TEMP;
       RRF[P] : = RRF[P]+RR[I,J,P,W]*SXRAT[W]/TEMP;
       SSM[P] : = SSM[P]+SS[I,J,P,W]/TEMP;
       RSM[P] : = RSM[P]+RS[I,J,P,W]/TEMP;
       RRM[P] : = RRM[P]+RR[I,J,P,W]/TEMP;
      END;
    TOTRF[P,W] : = SSF[P]+RSF[P]+RRF[P];
    TOTM[P] : = SSM[P]+RSM[P]+RRM[P];

{ REPRODUCTIVE DISADVANTAGE (IF DIS = 1.0, THEN NO DISADVANTAGE) }

    RSF[P] : = RSF[P]*RSDIS[P,W];
    RRF[P] : = RRF[P]*RRDIS[P,W];

{ RANDOM MATING AND OVIPOSITION }

    IF (W = 1)
     THEN FECMOD : = PREYNC
     ELSE FECMOD : = PREDFC;
    MID : = SSF[P]*(SSM[P]+RSM[P]/2.0)+RSF[P]
      *(SSM[P]/2.0+RSM[P]/4.0);
    IF TOTM[P] = 0 THEN TOTM[P]:=0.000001;
    SS1[P,W] : =(FRACIN[IRF[W],W]*MID*FEC[W]/TOTM[P])*FECMOD;
    T1 : = RSM[P]/2.0+RRM[P];
    T2 : = SSM[P]+RSM[P]+RRM[P];
    T3 : = RSM[P]/2.0+SSM[P];
    IF T1 < 0.00000000000000000001 THEN T1 : = 0.0;
    IF T2 < 0.00000000000000000001 THEN T2 : = 0.0;
    IF T3 < 0.00000000000000000001 THEN T3 : = 0.0;
    MID : = SSF[P]*T1+(RSF[P]/2.0)*T2+RRF[P]*T3;
    RS1[P,W] : = (FRACIN[IRF[W],W]*MID*FEC[W]/TOTM[P])*FECMOD;
    T1 : = RSM[P]/4.0;
    T2 : = RRM[P]/2.0;
    T3 : = T1+T2;
    IF T3 < 0.0000000000000000001 THEN T3 : = 0.0;
    IF RRF[P] < 0.0000000000000000001 THEN RRF[P] : = 0.0;
    IF RRM[P] < 0.0000000000000000001 THEN RRM[P] : = 0.0;
    MID : = RSF[P]*T3+RRF[P]*(RSM[P]/2.0+RRM[P]);
    RR1[P,W] : = (FRACIN[IRF[W],W]*MID*FEC[W]/TOTM[P])*FECMOD;

   END; {TOTRF > LOOP}

  { MOVE SURVIVORS UP ONE STAGE }

  IF (W = 1)
   THEN
    BEGIN
     FOR I : = 1 TO 11 DO
      FOR J1 : = 1 TO 19 DO
       BEGIN
        J : = 21 - J1;
        IF SS[I,J-1,P,W] < 0.000000000000000001
         THEN SS[I,J-1,P,W] : = 0.0;
        IF RS[I,J-1,P,W] < 0.000000000000000001
         THEN RS[I,J-1,P,W] : = 0.0;
        IF RR[I,J-1,P,W] < 0.000000000000000001
         THEN RR[I,J-1,P,W] : = 0.0;
        SS[I,J,P,W] : = SS[I,J-1,P,W]*SURV[J-1,W]*PREYNC;
        RS[I,J,P,W] : = RS[I,J-1,P,W]*SURV[J-1,W]*PREYNC;
        RR[I,J,P,W] : = RR[I,J-1,P,W]*SURV[J-1,W]*PREYNC;
       END;
    END
   ELSE
    BEGIN
     FOR I : = 1 TO 11 DO
      FOR J1 : = 1 TO 19 DO
       BEGIN
```

```
          J : = 21 - J1;
          SS[I,J,P,W] : = SS[I,J-1,P,W]*SURV[J-1,W]*PREDSR;
          RS[I,J,P,W] : = RS[I,J-1,P,W]*SURV[J-1,W]*PREDSR;
          RR[I,J,P,W] : = RR[I,J-1,P,W]*SURV[J-1,W]*PREDSR;
         END;
       END;

     {DENSITY DEPENDENT SURVIVORSHIP }

     IF (T[W] > = (0.1 * CC[W])) THEN
      BEGIN
       CCTEST : = 0.0;
       THRESH(W, NCC1[W], NCC2[W], P, CCTEST);
       IF (CCTEST > = (0.1 * CC[W])) THEN
        BEGIN
         DDS : = 1 - CCTEST / CC[W];
         IF DDS < 0.1 THEN DDS : = 0.1;
         FOR I : = 1 TO 11 DO
          FOR J : = NCC1[W] TO NCC2[W] DO
           BEGIN
            SS[I,J,P,W] : = SS[I,J,P,W]*DDS;
            RS[I,J,P,W] : = RS[I,J,P,W]*DDS;
            RR[I,J,P,W] : = RR[I,J,P,W]*DDS;
           END;
        END;
      END;
    END;  {P LOOP}
  END;  {W LOOP}

 DISPOSE(TEMPSS);
 DISPOSE(TEMPRS);
 DISPOSE(TEMPRR);

END;  {Procedure NEXT}


PROCEDURE REINIT;

 TYPE

 CHEMAGE   = @CA;
 CA       = RECORD
      CONTROLARRAY: ARRAY [1..NUMPESTICIDES, 1..20] OF REAL;
      END;

 VAR

 TEMPSS, TEMPRS, TEMPRR, SAFE: CHEMAGE;

 T4, T5, T6: REAL;

 I, J, P, N1: INTEGER;

 BEGIN

 NEW(TEMPSS);
 NEW(TEMPRS);
 NEW(TEMPRR);
 NEW(SAFE);

 FOR J : = 1 TO 20 DO TOTAL[COUNTER,J,W] : = 0.0;
 FOR J : = 1 TO 20 DO
  BEGIN

   TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
          + SS[1,J,COUNTER,W]
          + RS[1,J,COUNTER,W]
          + RR[1,J,COUNTER,W];
   TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
          + SS[2,J,COUNTER,W]
          + RS[2,J,COUNTER,W]
          + RR[2,J,COUNTER,W];
```

```
  TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
          +SS[3,J,COUNTER,W]
          +RS[3,J,COUNTER,W]
          +RR[3,J,COUNTER,W];
  TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
          +SS[4,J,COUNTER,W]
          +RS[4,J,COUNTER,W]
          +RR[4,J,COUNTER,W];
  TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
          +SS[5,J,COUNTER,W]
          +RS[5,J,COUNTER,W]
          +RR[5,J,COUNTER,W];
  TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
          +SS[6,J,COUNTER,W]
          +RS[6,J,COUNTER,W]
          +RR[6,J,COUNTER,W];
  TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
          +SS[7,J,COUNTER,W]
          +RS[7,J,COUNTER,W]
          +RR[7,J,COUNTER,W];
  TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
          +SS[8,J,COUNTER,W]
          +RS[8,J,COUNTER,W]
          +RR[8,J,COUNTER,W];
  TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
          +SS[9,J,COUNTER,W]
          +RS[9,J,COUNTER,W]
          +RR[9,J,COUNTER,W];
  TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
          +SS[10,J,COUNTER,W]
          +RS[10,J,COUNTER,W]
          +RR[10,J,COUNTER,W];
  TOTAL[COUNTER,J,W] : = TOTAL[COUNTER,J,W]
          +SS[11,J,COUNTER,W]
          +RS[11,J,COUNTER,W]
          +RR[11,J,COUNTER,W];

  IF TOTAL[COUNTER,J,W] < 0.0000000001
   THEN TOTAL[COUNTER,J,W] : = 0.0;
  SAFE@.CONTROLARRAY[COUNTER,J] : = SS[11,J,COUNTER,W]
    +RS[11,J,COUNTER,W]+RR[11,J,COUNTER,W];
  IF (TOTAL[COUNTER,J,W] = 0.0)
   THEN SAFE@.CONTROLARRAY[COUNTER,J] : = 0.0
   ELSE SAFE@.CONTROLARRAY[COUNTER,J] : =
     SAFE@.CONTROLARRAY[COUNTER,J]/TOTAL[COUNTER,J,W];
 END;
FOR J : = 1 TO 20 DO
 BEGIN
 Q[W] : = 1.0 - RGENEFREQ[K,J,W];
     T4 : = Q[W] * Q[W];
     T5 : = RGENEFREQ[K,J,W] * RGENEFREQ[K,J,W];
     T6 : = 2 * RGENEFREQ[K,J,W] * Q[W];
 IF T4 < 0.00000000000000000001 THEN T4 : = 0.0;
 IF T5 < 0.00000000000000000001 THEN T5 : = 0.0;
 IF T6 < 0.00000000000000000001 THEN T6 : = 0.0;
 TEMPSS@.CONTROLARRAY[K,J] : = T4*TOTAL[COUNTER,J,W]
       *(1.0-SAFE@.CONTROLARRAY[COUNTER,J]);
 TEMPRS@.CONTROLARRAY[K,J] : = T6*TOTAL[COUNTER,J,W]
       *(1.0-SAFE@.CONTROLARRAY[COUNTER,J]);
 TEMPRR@.CONTROLARRAY[K,J] : = T5*TOTAL[COUNTER,J,W]
       *(1.0-SAFE@.CONTROLARRAY[COUNTER,J]);
 SS[11,J,K,W] : = T4*TOTAL[COUNTER,J,W]
   *SAFE@.CONTROLARRAY[COUNTER,J];
 RS[11,J,K,W] : = T6*TOTAL[COUNTER,J,W]
   *SAFE@.CONTROLARRAY[COUNTER,J];
 RR[11,J,K,W] : = T5*TOTAL[COUNTER,J,W]
   *SAFE@.CONTROLARRAY[COUNTER,J];
 FOR I : = 1 TO 10 DO
  BEGIN
  SS[I,J,K,W] : = TEMPSS@.CONTROLARRAY[K,J] * PROP[I];
  RS[I,J,K,W] : = TEMPRS@.CONTROLARRAY[K,J] * PROP[I];
  RR[I,J,K,W] : = TEMPRR@.CONTROLARRAY[K,J] * PROP[I];
```

```
    END;
  END;

 DISPOSE(TEMPSS);
 DISPOSE(TEMPRS);
 DISPOSE(TEMPRR);
 DISPOSE(SAFE);
        •
END; {Procedure REINIT}


PROCEDURE KILL(D50, D95 :  COMPLEXTEST; VAR XX: ARRY);

  VAR

 STDEV, Z  : REAL;

 I, J, P, INITSTAG, LASTSTAG  : INTEGER;

 N  : ARRAY [1..6] OF INTEGER;

BEGIN

 FOR J : = 1 TO 6 DO
  BEGIN
   STDEV : = (LN(D95@.COMPLEX[J,KORIG,W]/D50@.COMPLEX[J,KORIG,W])
     /LN(10.0))/1.6449;
   Z : = (LN(CD[K]/D50@.COMPLEX[J,KORIG,W])/LN(10.0))/STDEV;
   IF (Z > = ( - 3.75)) THEN
    BEGIN
     IF (Z < = 0.0) THEN N[J] : = TRUNC(Z / 0.75) + 5;
     IF (Z > 0.0) THEN N[J] : = TRUNC(Z / 0.75) + 6;
     IF (Z > 3.0) THEN N[J] : = 10;
     CASE J OF
      1: BEGIN
         INITSTAG : = 1;
         LASTSTAG : = LE[W];
        END;
      2:  BEGIN
         INITSTAG : = ISL[W];
         LASTSTAG : = LSL[W];
        END;
      3:  BEGIN
         INITSTAG : = IML[W];
         LASTSTAG : = LML[W];
        END;
      4: BEGIN
         INITSTAG : = ILL[W];
         LASTSTAG : = LLL[W];
        END;
      5: BEGIN
         INITSTAG : = IP[W];
         LASTSTAG : = LP[W];
        END;
      6: BEGIN
         INITSTAG : = IA[W];
         LASTSTAG : = 20;
        END;
      OTHERWISE CASECHECK : = 0;
     END;
     FOR P : = INITSTAG TO LASTSTAG DO
      IF ((1.0 - FRACIN[P,W]) < > 1.0)
       THEN FOR I : = 1 TO N[J] DO
          XX[I,P,K,W] : = XX[I,P,K,W]*(1.0-FRACIN[P,W]);
    END;
  END;  {FOR J LOOP}
END;  {Procedure KILL}


PROCEDURE UPDATER;  {ALLOWS CROSS RESISTANCE}

TYPE
```

```
 CHEMAGE   = @CA;
 CA      = RECORD
      CONTROLARRAY: ARRAY [1..NUMPESTICIDES, 1..20] OF REAL;
      END;

VAR

 I, J, P, CHEMNUMHOLD : INTEGER;

 TEMPRGENE, TEMPRFREQ  : CHEMAGE;

 DIFF  : ARRAY [1..20] OF REAL;

 TOT, TOTRGENE  : ARRAY [1..NUMPESTICIDES] OF REAL;

BEGIN

 NEW(TEMPRGENE);
 NEW(TEMPRFREQ);

 FOR J : = 1 TO 20 DO
  BEGIN
   TOTAL[K,J,W] : = 0.0;
   TEMPRGENE@.CONTROLARRAY[K,J] : = 0.0;

     TOTAL[K,J,W] : = TOTAL[K,J,W]+SS[1,J,K,W]+RS[1,J,K,W]
       +RR[1,J,K,W];
     TEMPRGENE@.CONTROLARRAY[K,J] : = TEMPRGENE@.CONTROLARRAY[K,J]
       +RR[1,J,K,W]+(0.5*RS[1,J,K,W]);
     TOTAL[K,J,W] : = TOTAL[K,J,W]+SS[2,J,K,W]+RS[2,J,K,W]
       +RR[2,J,K,W];
     TEMPRGENE@.CONTROLARRAY[K,J] : = TEMPRGENE@.CONTROLARRAY[K,J]
       +RR[2,J,K,W]+(0.5*RS[2,J,K,W]);
     TOTAL[K,J,W] : = TOTAL[K,J,W]+SS[3,J,K,W]+RS[3,J,K,W]
       +RR[3,J,K,W];
     TEMPRGENE@.CONTROLARRAY[K,J] : = TEMPRGENE@.CONTROLARRAY[K,J]
       +RR[3,J,K,W]+(0.5*RS[3,J,K,W]);
     TOTAL[K,J,W] : = TOTAL[K,J,W]+SS[4,J,K,W]+RS[4,J,K,W]
       +RR[4,J,K,W];
     TEMPRGENE@.CONTROLARRAY[K,J] : = TEMPRGENE@.CONTROLARRAY[K,J]
       +RR[4,J,K,W]+(0.5*RS[4,J,K,W]);
     TOTAL[K,J,W] : = TOTAL[K,J,W]+SS[5,J,K,W]+RS[5,J,K,W]
       +RR[5,J,K,W];
     TEMPRGENE@.CONTROLARRAY[K,J] : = TEMPRGENE@.CONTROLARRAY[K,J]
       +RR[5,J,K,W]+(0.5*RS[5,J,K,W]);
     TOTAL[K,J,W] : = TOTAL[K,J,W]+SS[6,J,K,W]+RS[6,J,K,W]
       +RR[6,J,K,W];
     TEMPRGENE@.CONTROLARRAY[K,J] : = TEMPRGENE@.CONTROLARRAY[K,J]
       +RR[6,J,K,W]+(0.5*RS[6,J,K,W]);
     TOTAL[K,J,W] : = TOTAL[K,J,W]+SS[7,J,K,W]+RS[7,J,K,W]
       +RR[7,J,K,W];
     TEMPRGENE@.CONTROLARRAY[K,J] : = TEMPRGENE@.CONTROLARRAY[K,J]
       +RR[7,J,K,W]+(0.5*RS[7,J,K,W]);
     TOTAL[K,J,W] : = TOTAL[K,J,W]+SS[8,J,K,W]+RS[8,J,K,W]
       +RR[8,J,K,W];
     TEMPRGENE@.CONTROLARRAY[K,J] : = TEMPRGENE@.CONTROLARRAY[K,J]
       +RR[8,J,K,W]+(0.5*RS[8,J,K,W]);
     TOTAL[K,J,W] : = TOTAL[K,J,W]+SS[9,J,K,W]+RS[9,J,K,W]
       +RR[9,J,K,W];
     TEMPRGENE@.CONTROLARRAY[K,J] : = TEMPRGENE@.CONTROLARRAY[K,J]
       +RR[9,J,K,W]+(0.5*RS[9,J,K,W]);
     TOTAL[K,J,W] : = TOTAL[K,J,W]+SS[10,J,K,W]+RS[10,J,K,W]
       +RR[10,J,K,W];
     TEMPRGENE@.CONTROLARRAY[K,J] : = TEMPRGENE@.CONTROLARRAY[K,J]
       +RR[10,J,K,W]+(0.5*RS[10,J,K,W]);
     TOTAL[K,J,W] : = TOTAL[K,J,W]+SS[11,J,K,W]+RS[11,J,K,W]
       +RR[11,J,K,W];
     TEMPRGENE@.CONTROLARRAY[K,J] : = TEMPRGENE@.CONTROLARRAY[K,J]
       +RR[11,J,K,W]+(0.5*RS[11,J,K,W]);

   IF (TOTAL[K,J,W] = 0.0)
```

```
    THEN TEMPRFREQ@.CONTROLARRAY[K,J] : = 0.0
    ELSE TEMPRFREQ@.CONTROLARRAY[K,J] : =
      TEMPRGENE@.CONTROLARRAY[K,J]/TOTAL[K,J,W];
   DIFF[J] : = TEMPRFREQ@.CONTROLARRAY[K,J] - RGENEFREQ[K,J,W];
   RGENEFREQ[K,J,W] : = TEMPRFREQ@.CONTROLARRAY[K,J];
   IF RGENEFREQ[K,J,W] > 1.0 THEN RGENEFREQ[K,J,W] : = 1.0;
  END; {J LOOP}

 CHEMNUMHOLD : = CHEMNUMBER;
 CHEMNUMBER : = 0;
 FOR P : = 1 TO NUMPESTICIDES DO
  IF P IN CURRENTCHEMS THEN
   BEGIN     {ALLOWS PARTIAL CROSS RESISTANCE}
    CHEMNUMBER : = CHEMNUMBER + 1;
    IF (CHEMNUMBER < > K) THEN
     FOR J : = 1 TO 20 DO
      BEGIN
       RGENEFREQ[CHEMNUMBER,J,W] : = RGENEFREQ[CHEMNUMBER,J,W]
         +(CRESIS[KORIG,P,W]*DIFF[J]);
       IF RGENEFREQ[CHEMNUMBER,J,W] > 1.0
        THEN RGENEFREQ[CHEMNUMBER,J,W] : = 1.0;
       IF (RGENEFREQ[CHEMNUMBER,J,W] < 0)
        THEN RGENEFREQ[CHEMNUMBER,J,W] : = 0.0;
      END;
   END;
 CHEMNUMBER : = CHEMNUMHOLD;
 FOR P : = 1 TO CURRENTNUMCHEMS DO
  FOR I : = 1 TO 11 DO
   FOR J : = 1 TO 20 DO
    BEGIN
     SS[I,J,P,W] : = SS[I,J,K,W];
     RS[I,J,P,W] : = RS[I,J,K,W];
     RR[I,J,P,W] : = RR[I,J,K,W];
    END;

 DISPOSE(TEMPRGENE);
 DISPOSE(TEMPRFREQ);

END; {Procedure UPDATER}


PROCEDURE WINTER(WSURV: ARY);

  VAR

 W, I, J, P  : INTEGER;

 BEGIN

 FOR W : = 1 TO SPECIES DO
  BEGIN
   FOR P : = 1 TO CURRENTNUMCHEMS DO
    BEGIN
     FOR I : = 1 TO 11 DO
      FOR J : = 1 TO 20 DO
       BEGIN
        SS[I,J,P,W] : = SS[I,J,P,W]*WSURV[J,W];
        RS[I,J,P,W] : = RS[I,J,P,W]*WSURV[J,W];
        RR[I,J,P,W] : = RR[I,J,P,W]*WSURV[J,W];
       END;
     CD[P] : = 0.0;
    END;
   OW : = TRUE;
  END;
END; {Procedure WINTER}


PROCEDURE MIGRATE;

  VAR

 DATE, W, I, J, P, N  : INTEGER;
```

```
S, T1, T2, T3, T4: REAL;

 BEGIN

FOR W : = 1 TO 1 DO
 BEGIN
  FOR P:= 1 TO CURRENTNUMCHEMS DO
   BEGIN
    IF TOTRF[P,W] < 0.05
     THEN ERMIMMIG : = 0.05
     ELSE ERMIMMIG : = TOTRF[P,W];
    FOR N : = 1 TO 3 DO
     BEGIN
      IF (N = 1)
       THEN
        BEGIN
         T1 : = Q[W] * Q[W];
         T3 : = 2.0 * PINIT[W] * Q[W];
         T4 : = PINIT[W] * PINIT[W];
         IF T1 < 0.00000000000000000001 THEN T1 : = 0.0;
         IF T3 < 0.00000000000000000001 THEN T3 : = 0.0;
         IF T4 < 0.00000000000000000001 THEN T4 : = 0.0;
         S : = 0.0;
         FOR J : = IRF[W] TO 20 DO
          IF (RR[10,J,P,W]+RR[11,J,P,W] > 0.0) THEN S : = S+1;
         FOR J : = IRF[W] TO 20 DO
          IF (RR[10,J,P,W]+RR[11,J,P,W] > 0.0) THEN
           BEGIN
            IF (IDAY > 40)
             THEN DATE : = IDAY-40
             ELSE DATE : = IDAY;
            FOR I : = 1 TO 10 DO
             BEGIN
              SS[I,J,P,W] : = SS[I,J,P,W]+T1*PROP[I]
                *(WIMIG[W]*ERMIMMIG)/S;
              RS[I,J,P,W] : = RS[I,J,P,W]+T3*PROP[I]
                *(WIMIG[W]*ERMIMMIG)/S;
              RR[I,J,P,W] : = RR[I,J,P,W]+T4*PROP[I]
                *(WIMIG[W]*ERMIMMIG)/S;
             END;
           END;

         {ADD IMMIGRANT'S EGGS }

         SS1[P,W] : = SS1[P,W]+(WIMIG[W]*ERMIMMIG)*(SXRAT[W]
           /(SXRAT[W]+1))*FEC[W]*T1*DINC;
         RS1[P,W] : = RS1[P,W]+(WIMIG[W]*ERMIMMIG)*(SXRAT[W]
           /(SXRAT[W]+1))*FEC[W]*T3*DINC;
         RR1[P,W] : = RR1[P,W]+(WIMIG[W]*ERMIMMIG)*(SXRAT[W]
           /(SXRAT[W]+1))*FEC[W]*T4*DINC;
        END
       ELSE IF (N = 2)
        THEN
         BEGIN
          T4 : = (IMIGFREQ[P,W]*TOTRFREQ[P,W])
            *(IMIGFREQ[P,W]*TOTRFREQ[P,W]);
          T1 : = (1.0-SQRT(T4))*(1.0-SQRT(T4));
          T3 : = 2.0*SQRT(T1)*SQRT(T4);
          IF T1 < 0.00000000000000000001 THEN T1 : = 0.0;
          IF T3 < 0.00000000000000000001 THEN T3 : = 0.0;
          S : = 0.0;
          FOR J : = IRF[W] TO 20 DO
           IF (RR[10,J,P,W]+RR[11,J,P,W] > 0.0)
            THEN S : = S + 1;
          FOR J : = IRF[W] TO 20 DO
           IF (RR[10,J,P,W]+RR[11,J,P,W] > 0.0)
            THEN
             BEGIN
              FOR I : = 1 TO 10 DO
               BEGIN
                SS[I,J,P,W] : = SS[I,J,P,W]+T1*PROP[I]
```

```
            *(FIMIG[W]*ERMIMMIG)/S;
          RS[I,J,P,W] : = RS[I,J,P,W]+T3*PROP[I]
            *(FIMIG[W]*ERMIMMIG)/S;
          RR[I,J,P,W] : = RR[I,J,P,W]+T4*PROP[I]
            *(FIMIG[W]*ERMIMMIG)/S;
         END;
        END;

      {ADD IMMIGRANT'S EGGS }

      SS1[P,W] : = SS1[P,W]+(FIMIG[W]*ERMIMMIG)
        *(SXRAT[W]/(SXRAT[W]+1))*FEC[W]*T1*DINC;
      RS1[P,W] : = RS1[P,W]+(FIMIG[W]*ERMIMMIG)
        *(SXRAT[W]/(SXRAT[W]+1))*FEC[W]*T3*DINC;
      RR1[P,W] : = RR1[P,W]+(FIMIG[W]*ERMIMMIG)
        *(SXRAT[W]/(SXRAT[W]+1))*FEC[W]*T4*DINC;
     END
     ELSE
      BEGIN
      T1 : = (1.0-TOTRFREQ[P,W]) * (1.0-TOTRFREQ[P,W]);
      T3 : = 2.0 * TOTRFREQ[P,W] * (1.0-TOTRFREQ[P,W]);
      T4 : = TOTRFREQ[P,W] * TOTRFREQ[P,W];
      IF T1 < 0.00000000000000000001 THEN T1 : = 0.0;
      IF T3 < 0.00000000000000000001 THEN T3 : = 0.0;
      IF T4 < 0.00000000000000000001 THEN T4 : = 0.0;
      S : = 0.0;
      FOR J : = IRF[W] TO 20 DO
       IF (RR[10,J,P,W]+RR[11,J,P,W] > 0.0)
        THEN  S : = S + 1;
      FOR J : = IRF[W] TO 20 DO
       IF (RR[10,J,P,W]+RR[11,J,P,W] > 0.0)
        THEN
         FOR I : = 1 TO 10 DO
          BEGIN
          SS[I,J,P,W] : = SS[I,J,P,W]+T1*PROP[I]
            *(AIMIG[W]*ERMIMMIG)/S;
          RS[I,J,P,W] : = RS[I,J,P,W]+T3*PROP[I]
            *(AIMIG[W]*ERMIMMIG)/S;
          RR[I,J,P,W] : = RR[I,J,P,W]+T4*PROP[I]
            *(AIMIG[W]*ERMIMMIG)/S;
          END;

      {ADD IMMIGRANT'S EGGS }

      SS1[P,W] : = SS1[P,W]+(AIMIG[W]*ERMIMMIG)
        *(SXRAT[W]/(SXRAT[W]+1))*FEC[W]*T1*DINC;
      RS1[P,W] : = RS1[P,W]+(AIMIG[W]*ERMIMMIG)
        *(SXRAT[W]/(SXRAT[W]+1))*FEC[W]*T3*DINC;
      RR1[P,W] : = RR1[P,W]+(AIMIG[W]*ERMIMMIG)
        *(SXRAT[W]/(SXRAT[W]+1))*FEC[W]*T4*DINC;
     END;
   END; {N LOOP}

  TIMIG[W] : = (WIMIG[W] + FIMIG[W] + AIMIG[W]) * ERMIMMIG;

 END; {P LOOP}
END; {W LOOP}

BLBIMM : = 0.0001;
IF (IDAY > 15) AND (IDAY < 21) THEN BLBIMM : = 0.0002;
IF (IDAY > 20) AND (IDAY < 26) THEN BLBIMM : = 0.0004;
IF (IDAY > 25) AND (IDAY < 31) THEN BLBIMM : = 0.0006;
IF (IDAY > 30) AND (IDAY < 36) THEN BLBIMM : = 0.0016;
IF (IDAY > 35) AND (IDAY < 41) THEN BLBIMM : = 0.004;

IF T[1] < 0.01
 THEN ERM : = 0.01
 ELSE ERM : = T[1];

FOR W : = 2 TO 2 DO
 BEGIN
  FOR P:= 1 TO CURRENTNUMCHEMS DO
```

```
BEGIN
 FOR N : = 1 TO 3 DO
  BEGIN
   IF (N = 1)
    THEN
     BEGIN
      T1 : = Q[W] * Q[W];
      T3 : = 2.0 * PINIT[W] * Q[W];
      T4 : = PINIT[W] * PINIT[W];
      IF T1 < 0.00000000000000000001 THEN T1 : = 0.0;
      IF T3 < 0.00000000000000000001 THEN T3 : = 0.0;
      IF T4 < 0.00000000000000000001 THEN T4 : = 0.0;
      S : = 0.0;
      FOR J : = IRF[W] TO 20 DO
       IF (RR[10,J,P,W] + RR[11,J,P,W] > 0.0)
        THEN S : = S + 1;
      FOR J : = IRF[W] TO 20 DO
       IF (RR[10,J,P,W] + RR[11,J,P,W] > 0.0)
        THEN
         BEGIN
          IF (IDAY > 40)
           THEN DATE : = IDAY-40
           ELSE DATE : = IDAY;
          FOR I : = 1 TO 10 DO
           BEGIN
            SS[I,J,P,W] : = SS[I,J,P,W] + T1*PROP[I]
              *(WIMIG[W]*ERM*BLBIMM)/S;
            RS[I,J,P,W] : = RS[I,J,P,W] + T3*PROP[I]
              *(WIMIG[W]*ERM*BLBIMM)/S;
            RR[I,J,P,W] : = RR[I,J,P,W] + T4*PROP[I]
              *(WIMIG[W]*ERM*BLBIMM)/S;
           END;
         END;

      {ADD IMMIGRANT'S EGGS }

      SS1[P,W] : = SS1[P,W] + (WIMIG[W]*ERM*BLBIMM)
        *(SXRAT[W]/(SXRAT[W] + 1))*FEC[W]*T1*DINC;
      RS1[P,W] : = RS1[P,W] + (WIMIG[W]*ERM*BLBIMM)
        *(SXRAT[W]/(SXRAT[W] + 1))*FEC[W]*T3*DINC;
      RR1[P,W] : = RR1[P,W] + (WIMIG[W]*ERM*BLBIMM)
        *(SXRAT[W]/(SXRAT[W] + 1))*FEC[W]*T4*DINC;
     END
    ELSE
     IF (N = 2)
      THEN
       BEGIN
        T4 : = (IMIGFREQ[P,W]*TOTRFREQ[P,W])
          *(IMIGFREQ[P,W]*TOTRFREQ[P,W]);
        T1 : = (1.0-SQRT(T4))*(1.0-SQRT(T4));
        T3 : = 2.0*SQRT(T1)*SQRT(T4);
        IF T1 < 0.00000000000000000001 THEN T1 : = 0.0;
        IF T3 < 0.00000000000000000001 THEN T3 : = 0.0;
        S : = 0.0;
        FOR J : = IRF[W] TO 20 DO
         IF (RR[10,J,P,W] + RR[11,J,P,W] > 0.0)
          THEN S : = S + 1;
        FOR J : = IRF[W] TO 20 DO
         IF (RR[10,J,P,W] + RR[11,J,P,W] > 0.0)
          THEN
           FOR I : = 1 TO 10 DO
            BEGIN
             SS[I,J,P,W] : = SS[I,J,P,W] + T1
               *PROP[I]*(FIMIG[W]*ERM
               *BLBIMM)/S;
             RS[I,J,P,W] : = RS[I,J,P,W] + T3
               *PROP[I]*(FIMIG[W]*ERM
               *BLBIMM)/S;
             RR[I,J,P,W] : = RR[I,J,P,W] + T4
               *PROP[I]*(FIMIG[W]*ERM
               *BLBIMM)/S;
            END;
```

```
        {ADD IMMIGRANT'S EGGS }

        SS1[P,W] : = SS1[P,W] + (FIMIG[W]*ERM*BLBIMM)
          *(SXRAT[W]/(SXRAT[W] + 1))*FEC[W]*T1*DINC;
        RS1[P,W] : = RS1[P,W] + (FIMIG[W]*ERM*BLBIMM)
          *(SXRAT[W]/(SXRAT[W] + 1))*FEC[W]*T3*DINC;
        RR1[P,W] : = RR1[P,W] + (FIMIG[W]*ERM*BLBIMM)
          *(SXRAT[W]/(SXRAT[W] + 1))*FEC[W]*T4*DINC;
       END
      ELSE
       BEGIN
       T1 : = (1.0-TOTRFREQ[P,W])*(1.0-TOTRFREQ[P,W]);
       T3 : = 2.0*TOTRFREQ[P,W]*(1.0-TOTRFREQ[P,W]);
       T4 : = TOTRFREQ[P,W]*TOTRFREQ[P,W];
       IF T1 < 0.000000000000000000001 THEN T1 : = 0.0;
       IF T3 < 0.000000000000000000001 THEN T3 : = 0.0;
       IF T4 < 0.000000000000000000001 THEN T4 : = 0.0;
       S : = 0.0;
       FOR J : = IRF[W] TO 20 DO
        IF (RR[10,J,P,W] + RR[11,J,P,W] > 0.0)
         THEN S : = S + 1;
       FOR J : = IRF[W] TO 20 DO
        IF (RR[10,J,P,W] + RR[11,J,P,W] > 0.0)
         THEN
          FOR I : = 1 TO 10 DO
           BEGIN
            SS[I,J,P,W] : = SS[I,J,P,W] + T1
              *PROP[I]*(AIMIG[W]*ERM
              *BLBIMM)/S;
            RS[I,J,P,W] : = RS[I,J,P,W] + T3
              *PROP[I]*(AIMIG[W]*ERM
              *BLBIMM)/S;
            RR[I,J,P,W] : = RR[I,J,P,W] + T4
              *PROP[I]*(AIMIG[W]*ERM
              *BLBIMM)/S;
           END;

       {ADD IMMIGRANT'S EGGS}

       SS1[P,W] : = SS1[P,W] + (AIMIG[W]*ERM*BLBIMM)
         *(SXRAT[W]/(SXRAT[W] + 1))*FEC[W]*T1*DINC;
       RS1[P,W] : = RS1[P,W] + (AIMIG[W]*ERM*BLBIMM)
         *(SXRAT[W]/(SXRAT[W] + 1))*FEC[W]*T3*DINC;
       RR1[P,W] : = RR1[P,W] + (AIMIG[W]*ERM*BLBIMM)
         *(SXRAT[W]/(SXRAT[W] + 1))*FEC[W]*T4*DINC;
      END;
    END; {N LOOP}

   TIMIG[W] : = (WIMIG[W] + FIMIG[W] + AIMIG[W])*ERM*BLBIMM;

   END; {P LOOP}
  END; {W LOOP}

END; {Procedure MIGRATE}


PROCEDURE EGGGS;

{ PUT NEW EGGS IN SUBSTAGE 1, SUSCEPTIBILITY CATEGORIES 1-11 }

 VAR

 I, P, W: INTEGER;

  BEGIN

 FOR W : = 1 TO SPECIES DO
  FOR P : = 1 TO CURRENTNUMCHEMS DO
   BEGIN
    FOR I : = 1 TO 10 DO
     BEGIN
```

```
      SS[I,1,P,W] : = SS1[P,W]*PROP[I]*(1-STAREF[1,W]);
      RS[I,1,P,W] : = RS1[P,W]*PROP[I]*(1-STAREF[1,W]);
      RR[I,1,P,W] : = RR1[P,W]*PROP[I]*(1-STAREF[1,W]);
     END;
    SS[11,1,P,W] : = SS1[P,W]*STAREF[1,W];
    RS[11,1,P,W] : = RS1[P,W]*STAREF[1,W];
    RR[11,1,P,W] : = RR1[P,W]*STAREF[1,W];
   END;

END; {Procedure EGGGS}

PROCEDURE REFUGE(IX, P: INTEGER);

{ FOR INITIAL LARVAE,INITIAL PUPAE OR INITIAL ADULTS (IX)
PUT APPROPRIATE FRACTION (STAREF[P] INTO STAGE-LONG REFUGIA
(SUSCAT 11)}

  VAR

 I,G  : INTEGER;

 TEMP, TMP, SSHOLD, RSHOLD, RRHOLD  : REAL;

  BEGIN

   { COMPUTE TOTALS IN REFUGIA, STORE IN HOLDERS }

 FOR G : = 1 TO CURRENTNUMCHEMS DO
  BEGIN
   SSHOLD : = 0.0;
   RSHOLD : = 0.0;
   RRHOLD : = 0.0;
   FOR I : = 1 TO 11 DO
    BEGIN
     SSHOLD : = SSHOLD+SS[I,IX,G,W]*STAREF[P,W];
     RSHOLD : = RSHOLD+RS[I,IX,G,W]*STAREF[P,W];
     RRHOLD : = RRHOLD+RR[I,IX,G,W]*STAREF[P,W];
    END;

   { PUT THOSE NOT ENTERING REFUGIA IN SUSCATS 1-10 }

   TEMP : = 1.0-STAREF[P,W];
   FOR I : = 1 TO 10 DO
    BEGIN
     TMP : = SS[I,IX,G,W];
     SS[I,IX,G,W] : = 0.0;
     SS[I,IX,G,W] : = (TMP+SS[11,IX,G,W]*PROP[I])*TEMP;
    END;
   FOR I : = 1 TO 10 DO
    BEGIN
     TMP : = RS[I,IX,G,W];
     RS[I,IX,G,W] : = 0.0;
     RS[I,IX,G,W] : = (TMP+RS[11,IX,G,W]*PROP[I])*TEMP;
    END;
   FOR I : = 1 TO 10 DO
    BEGIN
     TMP : = RR[I,IX,G,W];
     RR[I,IX,G,W] : = 0.0;
     RR[I,IX,G,W] : = (TMP+RR[11,IX,G,W]*PROP[I])*TEMP;
    END;

   { PUT THOSE ENTERING REFUGIA INTO SUSCAT 11 }

   SS[11,IX,G,W] : = SSHOLD;
   RS[11,IX,G,W] : = RSHOLD;
   RR[11,IX,G,W] : = RRHOLD;
  END;

END; {Procedure REFUGE}
```

```
BEGIN  {Procedure ERMSIMULATOR};

 REWRITE(RERMTOTS);
 REWRITE(SERMTOTS);

 FOR NUMRUNS : = 1 TO 2 DO
 BEGIN
  IF NUMRUNS = 1
   THEN SIMNUMYEARS : = TABMYRHOLD
   ELSE SIMNUMYEARS : = ERMYRHOLD;

 NEW(SSD50);
 NEW(RSD50);
 NEW(RRD50);
 NEW(SSD95);
 NEW(RSD95);
 NEW(RRD95);

   DATAINITIALIZE;
   GETLIBPARAMS;
   INIT;
   SUM;
   EARLY : = FALSE;
   NEWYR : = 0;

 REPEAT
   NEWYR : = NEWYR + 1;
   SIGNAL : = TRUE;
   CMD1 : = 0.0;
   I : = 1;
   IF (IYR < 4) THEN
    FOR I : = 1 TO 20 DO
     BEGIN
      ISPCAL[I] : = 0;
     END;
   IF (IYR > 3) THEN
    FOR I : = 1 TO 20 DO
     BEGIN
      ISPCAL[I] : = ISPCALHOLD[I];
     END;
   OW : = FALSE;
   FOR M : = 1 TO CURRENTNUMCHEMS DO CD[M] : = 0.0;
   FOR W:= 1 TO SPECIES DO TIMMGEN[W] : = 0.0;
   IGEN : = 1;
   IGD : = 0;
   STEP : = 0.0;
   B : = 1;
   K : = 1;
   COUNTER : = 1;
   IDAY : = 0;
   WRITE('CURRENTLY SIMULATING ERM DYNAMICS, YEAR ',IYR:2,' DAY ');

   FOR NEWIDAY : = 1 TO NUMDAYSPERYEAR DO
    BEGIN
     IDAY : = NEWIDAY;
     CASE IDAY OF
      1, 10, 20, 30, 40: WRITE(IDAY:2,' ');
      OTHERWISE CASECHECK : = 0;
     END;
     CHEMNUMBER : = 0;
     FOR K : = 1 TO NUMPESTICIDES DO
      IF K IN CURRENTCHEMS THEN
       BEGIN
        CHEMNUMBER : = CHEMNUMBER + 1;
        CD[CHEMNUMBER] : = (CD[CHEMNUMBER]*EXP(LN(0.5)/THLIF[K]))
           +VERTEX@.ACTIONS[CHEMNUMBER,IYR,IDAY];
       END;
     IF CD[COUNTER] > 0.0001 THEN
      FOR W : = 1 TO SPECIES DO
      BEGIN
       KORIG : = COUNTER;
       K : = COUNTER;
```

```
      BEFORE : = COUNTER;
      KILL(SSD50, SSD95, SS);
      KILL(RSD50, RSD95, RS);
      KILL(RRD50, RRD95, RR);
      UPDATER;
     END;
    CHEMNUMBER : = 0;
    FOR M : = 1 TO NUMPESTICIDES DO
     IF M IN CURRENTCHEMS THEN
      BEGIN
       CHEMNUMBER : = CHEMNUMBER + 1;
       IF (CHEMNUMBER < > BEFORE)
         AND (CD[CHEMNUMBER] > 0.0001) THEN
        FOR W : = 1 TO SPECIES DO
        BEGIN
         K : = CHEMNUMBER;
         REINIT;
         COUNTER : = CHEMNUMBER;
         KORIG : = M;
         KILL(SSD50, SSD95, SS);
         KILL(RSD50, RSD95, RS);
         KILL(RRD50, RRD95, RR);
         UPDATER;
        END;
      END;
    STEP : = STEP + DINC;
    IF (STEP < 1) THEN GOTO 240;
    STEP : = STEP - 1.0;
    IGD : = IGD + 1;
    IF (IGD < = 20) THEN GOTO 187;
    IGEN : = IGEN + 1;
    IGD : = 0;
    FOR W : = 1 TO SPECIES DO TIMMGEN[W] : = 0.0;
    IF (IGEN-1 = NGENS) THEN GOTO 250; 187:
    FOR W : = 1 TO SPECIES DO
     FOR K : = 1 TO CURRENTNUMCHEMS DO
      BEGIN
       SS1[K,W] : = 0.0;
       RS1[K,W] : = 0.0;
       RR1[K,W] : = 0.0;
      END;

    PRED;
    NEXT;
    MIGRATE;
    FOR W : = 1 TO SPECIES DO
     TIMMGEN[W] : = TIMMGEN[W] + TIMIG[W];
    EGGGS;
    FOR W : = 1 TO SPECIES DO
     BEGIN
      REFUGE(ISL[W], 2);
      REFUGE(IML[W], 3);
      REFUGE(ILL[W], 4);
      REFUGE(IP[W], 5);
      REFUGE(IA[W], 6);
     END;                {W LOOP}
    SUM: 240;
    CMD1 : = CMD1 + 4.0*(T[1]-EGG[1]);

   END;  {FOR NEWIDAY}

250:WRITELN;
   ERMTOTALS : = CMD1;
   IF NUMRUNS = 1
    THEN WRITELN(RERMTOTS,ERMTOTALS:10)
    ELSE WRITELN(SERMTOTS,ERMTOTALS:10);
   WINTER(WSURV);
   IGEN:=1;
   IDAY:=0;
   IYR : = IYR + 1;
   SUM;
   FOR K : = 1 TO CURRENTNUMCHEMS DO
```

```
    IF TOTRFREQ[K,1] < 0.5 THEN SIGNAL : = FALSE;

  UNTIL(NEWYR = SIMNUMYEARS);

  ERMYRHOLD : = NEWYR;


  DISPOSE(SSD50);
  DISPOSE(RSD50);
  DISPOSE(RRD50);
  DISPOSE(SSD95);
  DISPOSE(RSD95);
  DISPOSE(RRD95);

  END;  {NUMRUNS}

END; {Procedure ERMSIMULATOR}
```

Appendix D. ERM/BLB LIFE-HISTORY PARAMETERS

The ERM/BLB model is run for two generations, with each generation consisting of 20 substages. These generations correspond to those of TABM, with ERM having 7 to 8 generations per year and BLB having 4 to 5 generations per year. Each simulation time-step is equivalent to 4 days, with a simulation year being equivalent to 160 days. As currently parameterized, the model simulates the apple growing season from April 23 to September 30.

The age profile of the simulated ERM/BLB populations has been distributed to fit field data for initial and peak adult emergence and egg mass deposition. ERM overwinters as eggs within the orchard and BLB overwinters as adults outside of the orchard. The dynamics of the populations without interference from insecticides was adjusted to create a peak in mite density of between 10 and 20 mites per leaf occurring somewhere between simulation time-steps 29 to 33. BLB was set to peak between 1.2 and 1.7 per leaf in the period of day 33 to 40. Without the presence of the BLB, simulated ERM populations would peak at 45 motile stage per leaf at time-step 40.

Daily (4 day) survival rates were estimated to be approximately 0.7, or a 90 percent daily survival for all life stages. Winter mortality was adjusted to produce an approximate 50 percent survival with the same relative population age structure each simulation year.

The fraction of ERM in the orchard was set at 100 percent at the beginning of each simulation due to the species relatively low mobility, while the presence of adult BLB was set at

50 percent. Absence of some adults from the orchard primarily served as a mechanism to avoid an unrealistic, chemical-induced extinction. Immigration from the areas surrounding the orchard was adjusted so as to generate fluctuations in population levels that approximated those observed in experimental studies.

Appendix E. CHEMICAL TECHNICAL PARAMETERS

The following information describes some of the technical characteristics associated with the persistence and effectiveness of the potential chemical controls used in this study. Although the biological modelling was conducted as two separate units, the chemical parameters were held constant between the units.

Environmental Persistence: The half-lifes of the pesticides, defined as their residual ability to induce mortality in the pest or predator organisms, were: azinphosmethyl, 7 days or 1.8 simulation time-steps; chlorpyrifos, 2 days or 0.5 simulation time-steps; phosmet, 7 days or 1.8 simulation time-steps; microencapsulated methylparathion, 8 days or 2 simulation time-steps; methomyl, 4 days or 1 simulation time-step; and esfenvalerate, 16 days or 4 simulation time-steps.

Relative TABM Susceptibility by Life-Stage: The relative susceptibility of each life-stage to a chemical control was normalized on experimental data for azinphosmethyl. Values used for relative susceptibility to azinphosmethyl were: eggs, 1.0; small larvae, 2.0; medium larvae, 15.0; large larvae, 60.0; pupa, 20.0; and adults, 1.0. These relative susceptibilities were then applied to the other potential chemical controls given estimated ratios of lethal dose 50s. These ratios included: azinphosmethyl, 1.0; chlorpyrifos, 0.2; phosmet, 10.0; microencapsulated methylparathion, 0.8; methomyl, 0.6; and esfenvalerate, 0.03.

Relative ERM Susceptibility by Life-Stage: The relative susceptibility of each life-stage to a chemical control was normalized on experimental data for azinphosmethyl, assuming that an adult mite was susceptibly equivalent to a medium TABM larva. Thus, the relative susceptibilities to azinphosmethyl were: eggs, 10.0; larvae, 1.0; protonymph, 2.0; deutonymph, 4.0; adult 1, 6.0; and adult 2, 6.0. These relative susceptibilities were then applied to the other potential chemical controls given estimated ratios of lethal dose 50s. These ratios included: azinphosmethyl, 1.0; chlorpyrifos, 0.5; phosmet, 2.0; microencapsulated methylparathion, 2.0; methomyl, 20.0; and esfenvalerate, 1.0.

Relative BLB Susceptibility by Life-Stage: The relative susceptibility of each life-stage to a chemical control was normalized on experimental data for azinphosmethyl, assuming that the relative susceptibility of a medium TABM larva to an adult BLB was 1.0:0.5. Thus, the relative susceptibilities to azinphosmethyl were: eggs, 5.0; small larvae, 1.0; medium larvae, 4.0; large larvae, 6.0; pupa, 10.0; and adults, 2.0. These relative susceptibilities were then applied to the other potential chemical controls given estimated ratios of lethal dose 50s. These ratios included: azinphosmethyl, 1.0; chlorpyrifos, 9.0; phosmet, 8.0; microencapsulated methylparathion, 10.0; methomyl, 2.0; and esfenvalerate, 0.05.

Ratio of Lethal Dose 50s and 95s for TABM.: Values were based on data obtained from azinphosmethyl experiments, where resistance has been observed to confer an approximately 30-fold drop in chemical-induced mortality. The ratio of lethal dose 50s for TABM genotypes were: homozygous susceptible (SS) = 1; heterozygous susceptible (RS) = 10; and homozygous resistant (RR) = 30. The ratio of lethal dose 50s to 95s was set at 9. Ratios of lethal dose 95s were: SS = 1; RS = 5; RR = 10.

Ratio of Lethal Dose 50s and 95s for ERM.: Values were normalized to data from azinphosmethyl experiments on TABM. The ratio of lethal dose 50s for the different genotypes of non-adults were: homozygous susceptible (SS) = 1; heterozygous susceptible (RS) = 100; and homozygous resistant (RR) = 300. The ratio of lethal dose 50s for the different genotypes

of adults were: homozygous susceptible (SS) = 1; heterozygous susceptible (RS) = 10; and homozygous resistant (RR) = 30. The ratio of lethal dose 50s to 95s was set at 9. Ratios of lethal dose 95s for non-adults were: SS = 1; RS = 50; RR = 100. Ratios of lethal dose 95s for adults were: SS = 1; RS = 5; RR = 10.

Ratio of Lethal Dose 50s and 95s for BLB.: Values were normalized to data obtained from azinphosmethyl experiments on TABM. The ratio of lethal dose 50s for BLB genotypes were: homozygous susceptible (SS) = 1; heterozygous susceptible (RS) = 10; and homozygous resistant (RR) = 30. The ratio of lethal dose 50s to 95s was set at 9. Ratios of lethal dose 95s were: SS = 1; RS = 5; RR = 10.

Appendix F. DERIVATION OF ECONOMIC SURPLUS FORMULAS

This appendix details the specific derivations of the economic surplus formulas that were presented in Chapter VII and used to determine the social impact of various regulatory actions on the mid-Atlantic apple agroecosystem. To facilitate the following discussion, graphical descriptions of potential market conditions are again provided, along with some of the important fundamental relationships derived within the context of the closed-economy example.

A Closed Economy Example

Consider a closed-economy market model where:

1. aggregate supply and demand schedules are linear;[57]

2. demand is stationary and not subject to exogenous shifts;[58]

---

[57] In addition to the arguments previously covered with respect to functional form, linear supply schedules have also been criticized because of the implied negative price intercepts when supply is very inelastic (Kim et al.). However, price intercepts can be adjusted positive, with no changes in relevant surplus, by kinking the supply curves at the original quantity (Norton et al.).

[58] While exogenous demand shifts could be incorporated into the model, they would provide little additional information in terms of ranking potential policy outcomes. This occurs because the demand shift would need to be held constant across policy options in order to examine the specific affects of regulation and resistance on supply response.

3. year-to-year supply schedule shifts are proximately induced by changes in the size of economically damaging pest populations across all levels of potential control; and

4. all supply schedule shifts are parallel.[59]

For empirical investigations within this framework, an expected price or quantity change is postulated and the expected effects on economic surplus geometrically determined. This can be accomplished by employing supply and demand elasticities, along with a few fundamental relationships derived below.[60]

Fundamental Relationships

Consider the case where the supply schedule shifts away from the origin (Figure 34). First notice that the distance (*ac*) describes the proportional shift in the supply curve in terms of price. This distance can be determined as a function of the supply curve slope and the distance $(Q^* - Q_0)$. Given

$$\frac{\partial P}{\partial Q_S} = \frac{(ac)}{(Q^* - Q_0)} \quad , \qquad [42]$$

and knowing that

$$\frac{\partial P}{\partial Q_S} = \frac{1}{\varepsilon} \frac{P_0}{Q_0} \quad , \qquad [43]$$

---

[59] Assumptions about the nature of supply schedule shifts can have important implications for the magnitude of calculated surplus changes (Lindner and Jarrett; Rose). Within the context of this study, a parallel shift will generate the maximum surplus changes possible relative to that generated by a pivotal shift. Economic theory, however, gives little guidance as to how aggregate market supply curves might change in response to technological change (Norton et al.). Thus, a parallel shift will be assumed and sensitivity analysis conducted to determine the impact of this assumption on the results generated by the model.

[60] The derivation of these relationships arise from the background and examples provided in Pinstrup-Anderson et al. and Norton et al.

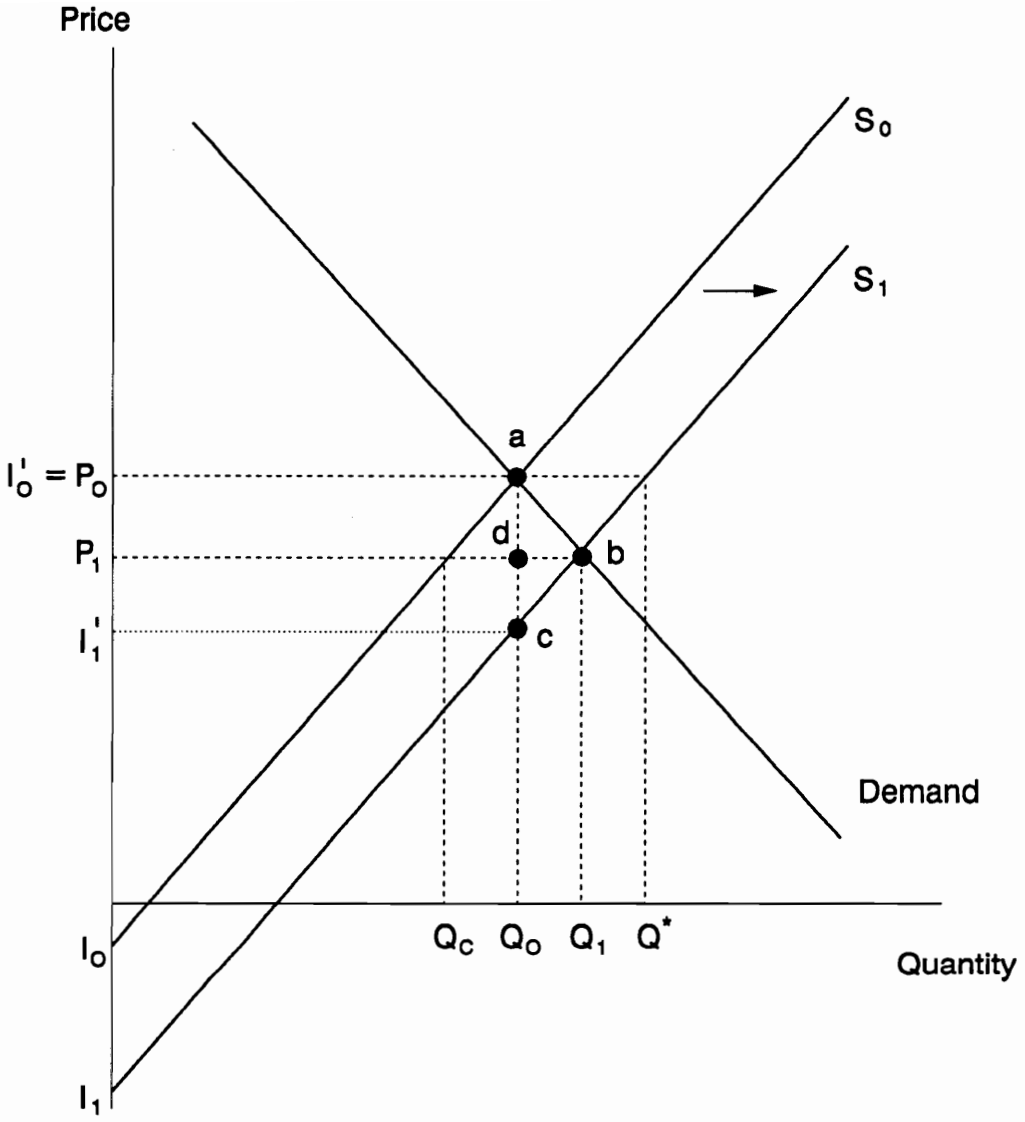

Figure 34. Surplus Changes in a Closed-Economy Model with Outward Supply Shifts.

where ε is the price elasticity of supply, then substitution of equation [43] into [42] and rearranging yields

$$(ac) = \frac{P_0 (Q^* - Q_0)}{\varepsilon Q_0} = \frac{P_0}{\varepsilon} J = P_0 K \quad , \qquad [44]$$

where J is the proportionate production change and K is the absolute value of the proportionate cost change relevant to the supply shift. Equation [44] will be important to the derivation of a relationship for the change in total economic surplus.

Next, a fundamental relationship for the expected change in output quantity can be obtained by first forming the identity

$$(Q_1 - Q_0) = (Q_1 - Q_0) \quad .$$

Operating on the right-hand-side of this expression, multiply the numerator and denominator by $\frac{P_0(Q^* - Q_0)}{Q_0(P_1 - P_0)}$:

$$Q_1 - Q_0 = \frac{\dfrac{P_0(Q^* - Q_0)(Q_1 - Q_0)}{Q_0 (P_1 - P_0)}}{\dfrac{P_0 (Q^* - Q_0)}{Q_0 (P_1 - P_0)}} \quad . \qquad [45]$$

Given the parallel shift in the supply schedule, geometry indicates that

$$(Q^* - Q_0) = (Q_1 - Q_c) = (Q_0 - Q_c) + (Q_1 - Q_0) \quad . \qquad [46]$$

Substituting [46] into the denominator of [45] results in

$$(Q_1 - Q_0) = \frac{\dfrac{P_0(Q^* - Q_0)(Q_1 - Q_0)}{Q_0(P_1 - P_0)}}{\dfrac{P_0[(Q_0 + Q_c) + (Q_1 - Q_0)]}{Q_0(P_1 - P_0)}} \quad , \qquad [47]$$

and multiplying the numerator of [47] by $\frac{\varepsilon Q_0}{\varepsilon Q_0}$ yields

$$(Q_1 - Q_0) = \frac{\dfrac{\varepsilon\, Q_0 P_0 (Q^* - Q_0)(Q_1 - Q_0)}{\varepsilon\, Q_0 Q_0 (P_1 - P_0)}}{\dfrac{P_0[(Q_0 + Q_c) + (Q_1 - Q_0)]}{Q_0(P_1 - P_0)}} \quad . \qquad [48]$$

Equation [48] can then be segregated in terms to obtain

$$(Q_1 - Q_0) = \frac{\dfrac{\varepsilon\, Q_0}{\varepsilon} \dfrac{P_0(Q_1 - Q_0)}{Q_0(P_1 - P_0)} \dfrac{(Q^* - Q_0)}{Q_0}}{\dfrac{P_0(Q_0 - Q_c)}{Q_0(P_1 - P_0)} + \dfrac{P_0(Q_1 - Q_0)}{Q_0(P_1 - P_0)}} \quad . \qquad [49]$$

Notice that, for the outward supply shift, $(P_1 - P_0)$ will be negative. Cancelling the negative signs in the numerator and denominator, equation [49] can be more conveniently expressed as

$$(Q_1 - Q_0) = \frac{Q_0 K \varepsilon |\eta|}{(\varepsilon + |\eta|)} = Z |\eta| Q_0 \ , \qquad [50]$$

where:

$$\varepsilon = \frac{P_0(Q_0 - Q_c)}{Q_0(P_0 - P_1)} = \text{price elasticity of supply;} \qquad [51]$$

$$|\eta| = \frac{P_0(Q_1 - Q_0)}{Q_0(P_0 - P_1)} = \text{absolute value of price elasticity of demand;} \qquad [52]$$

$$J = \frac{(Q^* - Q_0)}{Q_0} = \text{proportionate production change;} \qquad [53]$$

$$K = \frac{J}{\varepsilon} = \text{proportionate cost change; and}$$

$$Z = \frac{K \varepsilon}{(\varepsilon + |\eta|)} \quad .$$

An important point to consider is that the substitution $K = \frac{J}{\varepsilon}$ strictly holds only when supply elasticity is unity. For supply elasticities in the neighborhood of one, equation [50] can be employed as a relatively close approximation. But, for inelastic supply, a more conservative substitution is $K = J$ (Norton et al.), resulting in

$$(Q_1 - Q_0) = \frac{Q_0 J |\eta|}{(\varepsilon + |\eta|)} \quad .$$

With the quantity change relationship expressed in equation [50], the relevant elasticities ε and $|\eta|$, the original quantity Q_0, and the expected change in quantity $(Q_1 - Q_0)$, J can be calculated by:

$$J = \frac{(Q_1 - Q_0)(\varepsilon + |\eta|)}{Q_0 |\eta|}$$

This last derivation provides the final piece of information required to empirically calculate changes in economic surplus from expected quantity changes.

Surplus Changes for Outward Supply Shifts

The changes in total economic surplus (ΔTS), graphically described in Figure 34 by the area ($I_0acI_1 + abc$), can be determined from the relationship

$$\Delta TS = I'_0acI'_1 + abc = Q_0(ac) + (\frac{1}{2})(ac)(bd) \quad . \qquad [54]$$

Given that $(bd) = (Q_1 - Q_0)$, substitution of (bd) and equation [44] into equation [54] yields

$$\Delta TS = Q_0P_0K + \frac{1}{2}P_0K(Q_1 - Q_0) \quad . \qquad [55]$$

Further substitution of equation [50] into equation [55] and rearranging leads to

$$\Delta TS = Q_0 P_0 K\left[1 + \frac{1}{2}\ \frac{K\,\varepsilon\,|\eta|}{(\varepsilon + |\eta|)}\right] = Q_0 P_0 K(1 + \frac{1}{2}\ Z\,|\eta|) \quad . \qquad [56]$$

Thus, expected total surplus changes can be calculated from equation [56] with information on the original equilibrium price and quantity, the proportionate expected changes in output, and the relevant supply and demand price elasticities.

The changes in consumer surplus (ΔCS), graphically described in Figure 34 by the area P_0abP_1, can be calculated by

$$\Delta CS = P_0adP_1 + abd$$

$$= (P_0 - P_1)Q_0 + \frac{1}{2}(P_0 - P_1)(Q_1 - Q_0) \quad . \qquad [57]$$

Assuming that the expected quantity change $(Q_1 - Q_0)$ is known, a substitution for $(P_0 - P_1)$ can be obtained by rearranging equation [52],

$$(P_0 - P_1) = \frac{P_0(Q_1 - Q_0)}{Q_0\,|\eta|} \quad . \qquad [58]$$

Substituting equation [58] into [57] produces

$$\Delta CS = \frac{P_0(Q_1 - Q_0)\,Q_0}{Q_0\,|\eta|} + \frac{1}{2}\ \frac{P_0(Q_1 - Q_0)(Q_1 - Q_0)}{Q_0\,|\eta|} \quad , \qquad [59]$$

while substituting equation [50] into [59] yields

$$\Delta CS = \frac{P_0 Q_0 K\varepsilon|\eta|Q_0}{Q_0|\eta|(\varepsilon + |\eta|)} + \frac{1}{2}\ \frac{P_0 Q_0 K\varepsilon|\eta|}{Q_0|\eta|(\varepsilon + |\eta|)}\ \frac{Q_0 K\varepsilon|\eta|}{(\varepsilon + |\eta|)}$$

$$= P_0 Q_0 Z(1 + \frac{1}{2}\ Z|\eta|) \quad . \qquad [60]$$

As was the case for changes in total surplus, equation [60] describes consumer surplus changes in terms of the original equilibrium price and quantity, the proportionate decrease in expected output, and the supply and demand price elasticities.

Having derived expressions for changes in total economic and consumer surplus, changes in producer surplus can be obtained by difference:

$$\Delta PS = \Delta TS - \Delta CS$$

$$= \left[Q_0 P_0 K(1 + \frac{1}{2} Z|\eta|) \right] - \left[P_0 Q_0 Z(1 + \frac{1}{2} Z|\eta|) \right]$$

$$= (Q_0 P_0 K - P_0 Q_0 Z)(1 + \frac{1}{2} Z|\eta|) \ .$$

Surplus Changes for Backward Supply Shifts

With the relationships described above, surplus changes can be calculated for any outward shift in the supply schedule. But how might these relationships be used to calculate surplus changes induced by backward shifts in the supply schedule? As it turns out, there are a few subtle differences in the specifics of the derivations, even though the overall approach is very similar. For example, consider the backward supply schedule shift depicted in Figure 35, where the assumptions of the previous example still hold. The slope of the supply curves can be defined as

$$\frac{\partial P}{\partial Q_S} = \frac{AC}{(Q_0 - Q^*)} \ ,$$

where $(Q_0 - Q^*)$ is used because $\frac{\partial P}{\partial Q_S}$ should generally be greater than or equal to zero. Using a derivation similar to equation [44] produces

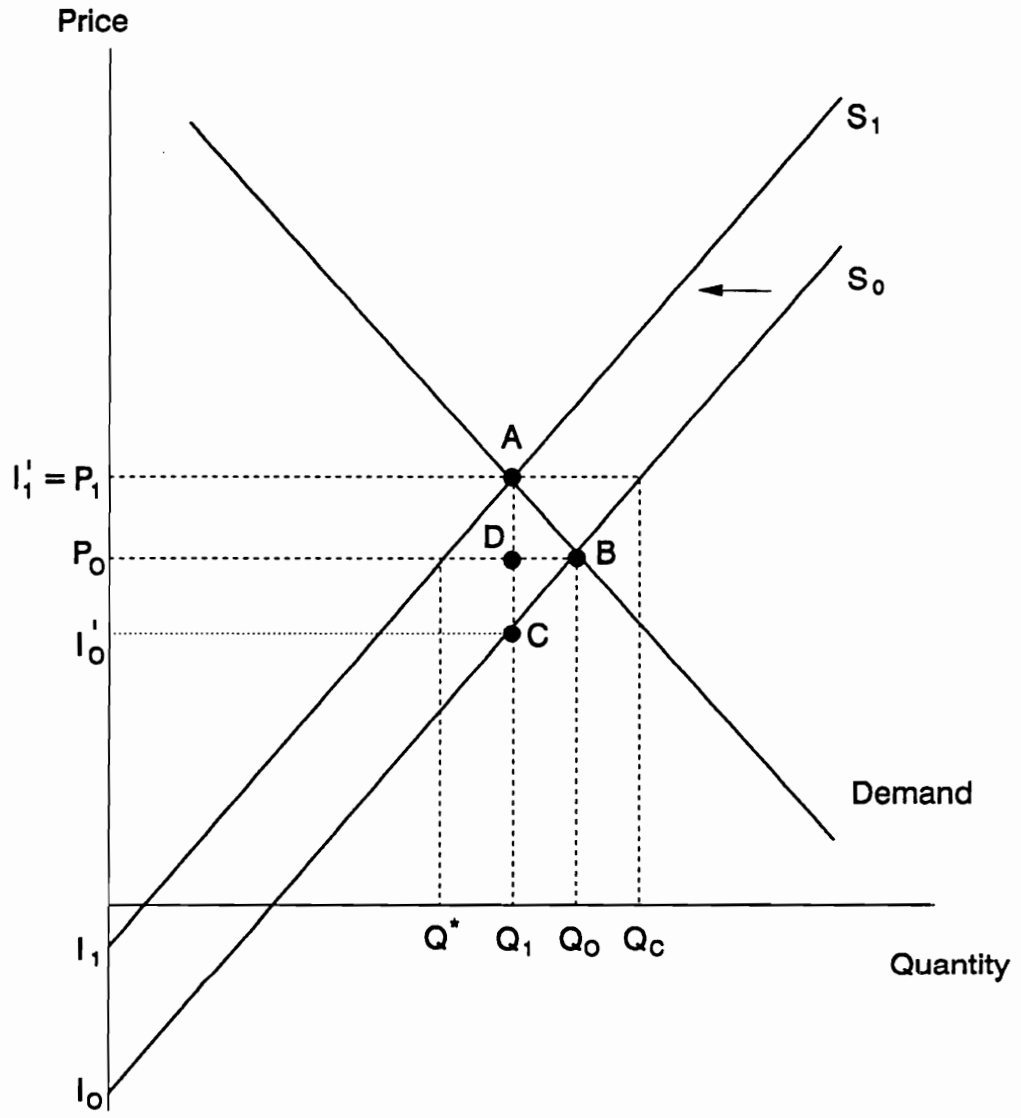

Figure 35. Surplus Changes in a Closed-Economy Model with Backward Supply Shifts.

$$AC = \frac{P_0(Q_0 - Q^*)}{\varepsilon Q_0} \quad .$$

From equation [53], it can be asserted that

$$-J = -\frac{(Q^* - Q_0)}{Q_0} = \frac{(Q_0 - Q^*)}{Q_0} \quad ,$$

resulting in

$$AC = \frac{P_0(-J)}{\varepsilon} = -P_0K \quad .$$

The change in total economic surplus (ΔTS) can be determined from

$$\Delta TS = (I_0BAI_1) = (I'_0CAI'_1 + ABC) \quad .$$

Following the general theme of the previous example,

$$\Delta TS = Q_1(AC) + \frac{1}{2}(AC)(DB) = -P_0K\left[Q_1 + \frac{1}{2}(Q_0 - Q_1)\right] \quad . \qquad [61]$$

Given that Q_1 can be expressed as

$$Q_1 = Q_0 - (Q_0 - Q_1) \quad , \qquad [62]$$

substitution of equation [50] and [62] into equation [61] and simplification yields

$$\Delta TS = -P_0K\left[Q_0 - (Q_0 - Q_1) + \frac{1}{2}(Q_0 - Q_1)\right]$$

$$= -P_0K\left(Q_0 - \frac{1}{2}Z|\eta|Q_0\right) = -P_0Q_0K(1 - \frac{1}{2}Z|\eta|) \quad . \qquad [63]$$

Changes in consumer surplus can be obtained from the relationship

$$\Delta CS = (P_0DAP_1 + ABD) = Q_1(AD) + \frac{1}{2}(AD)(DB)$$

$$= Q_1(P_0 - P_1) + \frac{1}{2}(P_0 - P_1)(Q_0 - Q_1) \quad . \qquad [64]$$

Substituting equation [50] and [58] into equation [64] and simplifying yields

$$\Delta CS = -P_0Q_0Z(1 - \frac{1}{2}Z\,|\eta|) \quad ,$$

which can be used along with equation [636] to calculate the change in producer surplus for a backward supply shift:

$$\Delta PS = \Delta TS - \Delta CS = -P_0Q_0K(1 - \frac{1}{2}Z\,|\eta|) - \left[-P_0Q_0Z(1 - \frac{1}{2}Z\,|\eta|)\right]$$

$$= (P_0Q_0Z - P_0Q_0K)(1 - \frac{1}{2}Z\,|\eta|) \quad .$$

As the closed-economy example illustrates, *ex ante* expected-surplus changes can be empirically calculated given information that is potentially available in the current period. Building on these surplus relationships, an economic framework can be developed for analysis of the resistance/regulation problem in the mid-Atlantic apple production system. But because conditions vary from what was assumed for the closed-economy example, more sophisticated relationships need to be employed.

A Large-Country Importer Model

Consider an open-economy market model of a large country importer where domestic supply and demand schedules are subject to the same conditions described for the closed-economy model. In this case, (Q_S^0, Q_D^0, P_0) represents the original equilibrium and (Q_S^1, Q_D^1, P_1)

represents the new equilibrium after a supply schedule shift from S_0 to S_1.[61] Given this scenario, price elasticities can be defined in a manner similar to equations [51] and [52]:

$$|\eta^i| = \frac{P_0 (Q_D^1 - Q_D^0)}{Q_D^0 (P_0 - P_1)} \qquad [65]$$

and

$$\varepsilon^i = \frac{P_0 (Q_S^0 - Q^c)}{Q_S^0 (P_0 - P_1)} \quad , \qquad [66]$$

where the superscript i denotes the import market model. In addition, because the external, rest-of-the-world (ROW) market forces are now included in the analysis, two additional price elasticities need to be defined. The first, excess price elasticity of supply for the ROW (ρ^i), is taken as parametric by the importing country and can be calculated by (Norton et al.):

$$\rho^i = \theta^i \frac{S_{ROW}}{Q_T^0} + |\sigma^i| \frac{D_{ROW}}{Q_T^0} \quad ,$$

where θ^i is the price elasticity of supply in the rest of the world; $|\sigma^i|$ is the absolute value price elasticity of demand in the rest of the world; S_{ROW} is the total production of the good in the rest of the world; D_{ROW} is the total consumption of the good in the rest of the world; and Q_T^0 is the quantity of the good imported by the country being examined. The second, absolute value excess price elasticity of demand for the importing country ($|\gamma^i|$), is dependent on the supply and demand schedules in the importing country and can be expressed as a function of normal supply and demand price elasticities. This derivation proceeds as follows:

$$|\gamma^i| = \frac{\partial Q^{ED}}{\partial P} \frac{P}{Q^{ED}} = \frac{(Q_T^c - Q_T^0)}{(P_0 - P_1)} \frac{P_0}{Q_T^0} \qquad [67]$$

---

[61] This analysis assumes that there are no spillover effects to other countries arising from the reduced domestic control-technology set. While currently pending legislation may, in fact, mandate spillovers with respect to pesticides, the regional nature of the pest complex and the limitations of the biological simulator currently preclude the introduction of this possibility.

where

$$Q_T^c = (Q_D^1 - Q^c) \quad \text{and} \quad Q_T^0 = (Q_D^0 - Q_S^0) \quad . \qquad [68]$$

Substitution of [68] into equation [67] results in

$$|\gamma^i| = \frac{(Q_D^1 - Q^c) - (Q_D^0 - Q_S^0)}{(P_0 - P_1)} \quad \frac{P_0}{(Q_D^0 - Q_S^0)}$$

$$= \frac{(Q_S^0 - Q^c) + (Q_D^1 - Q_D^0)}{(P_0 - P_1)} \quad \frac{P_0}{(Q_D^0 - Q_S^0)} \quad . \qquad [69]$$

Rearranging and multiplying the first term of equation [69] by $\frac{Q_S^g}{Q_S^g}$ and the second term by $\frac{Q_D^g}{Q_D^g}$ yields

$$|\gamma^i| = \frac{P_0 (Q_S^0 - Q^c) Q_S^0}{Q_S^0 (P_0 - P_1)(Q_D^0 - Q_S^0)} + \frac{P_0 (Q_D^1 - Q_D^0) Q_D^0}{Q_D^0 (P_0 - P_1)(Q_D^0 - Q_s^0)} \quad ,$$

which, after substituting in the relationships contained in equations [65] and [66], becomes

$$|\gamma^i| = \frac{\varepsilon^i Q_s^0}{(Q_D^0 - Q_S^0)} + \frac{|\eta|^i Q_D^0}{(Q_D^0 - Q_S^0)} \quad .$$

Having the necessary elasticities, it remains to find appropriate relationships for price changes in terms of predicted quantity changes. By manipulating equations [50] and [58], $(P_0 - P_1)$ can be defined as

$$(P_0 - P_1) = \frac{P_0 K \varepsilon}{(\varepsilon + |\eta|)} \quad , \qquad [70]$$

which, by analogy for the excess demand scenario, becomes

$$(P_0 - P_1) = \frac{P_0 H \rho^i}{(\rho^i + |\gamma^i|)} \qquad [71]$$

where

$$H = \frac{(P_0 - P'_1)}{P_0} \quad , \qquad [72]$$

or the proportional shift in the excess demand curve. Letting $I = \frac{H \rho^i}{(\rho^i + |\gamma^i|)}$, equation [71] can be compactly written as

$$(P_0 - P_1) = P_0 I \quad . \qquad [73]$$

However, because the simulator employed in this study provides potential changes in quantity and not price, H must be redefined. This can be accomplished by employing an analogy with equation [58], yielding

$$(P_0 - P'_1) = \frac{P_0(Q_T^0 - Q_T^*)}{|\gamma^i| \, Q_T^0} \quad . \qquad [74]$$

Given that

$$Q_T^* = (Q_D^0 - Q^*) \quad , \qquad [75]$$

then by substitution from equation [68] and [75] into equation [74],

$$(P_0 - P'_1) = \frac{P_0 (Q_D^0 - Q^*) - (Q_D^0 - Q_S^0)}{|\gamma^i| \, (Q_D^0 - Q_S^0)} = \frac{P_0(Q^* - Q_S^0)}{|\gamma^i| \, (Q_D^0 - Q_S^0)} \quad . \qquad [76]$$

Using equation [53] as a substitution into equation [76], and then substituting the result into equation [72], the new expression for H becomes

$$H = \frac{Q_S^0 J}{|\gamma^i| \, (Q_D^0 - Q_S^0)} = \frac{Q_S^0 K \varepsilon^i}{|\gamma^i| \, (Q_D^0 - Q_S^0)} \quad . \qquad [77]$$

Surplus Changes for an Outward Supply Shift

The changes in consumer surplus (ΔCS), graphically described in Figure 36 by the area $\Delta CS = P_0cdP_1 = P_0ceP_1 + ced$, can be calculated as

$$\Delta CS = Q_D^0(ce) + \frac{1}{2}(ce)(ed)$$

$$= Q_D^0(P_0 - P_1) + \frac{1}{2}(P_0 - P_1)(Q_D^1 - Q_D^0) \quad . \qquad [78]$$

Equation [65] can be rearranged to yield

$$(Q_D^1 - Q_D^0) = \frac{Q_D^0(P_0 - P_1)\,|\eta^i|}{P_0} \quad ,$$

and along with equation [73], substituted into equation [78] to get

$$\Delta CS = P_0\,Q_D^0\,I + \frac{1}{2}P_0Q_D^0I^2\,|\eta^i| = P_0Q_D^0\,I\,(1 + \frac{1}{2}\,I\,|\eta^i|) \quad . \qquad [79]$$

Thus, similar to the case for the closed-economy, equation [79] describes consumer surplus changes in terms of current-period knowable variables, including the original equilibrium price and quantity demanded, the price elasticity of demand in the importing country, the excess supply and demand price elasticities, and the induced proportional quantity shift in the excess demand curve.

The changes in producer surplus (ΔPS), graphically described in Figure 36 by

$$\Delta PS = I_0fgI_1 - P_0afP_1 = (I'_1hfP_1 + fgh) - (P_0amP_1 - afm) \quad ,$$

can be calculated from

$$\Delta PS = \left[Q^C(ab) + \frac{1}{2}(Q_S^1 - Q^C)(ab)\right] - \left[(am)Q_S^0 - \frac{1}{2}(am)(fm)\right] \quad . \qquad [80]$$

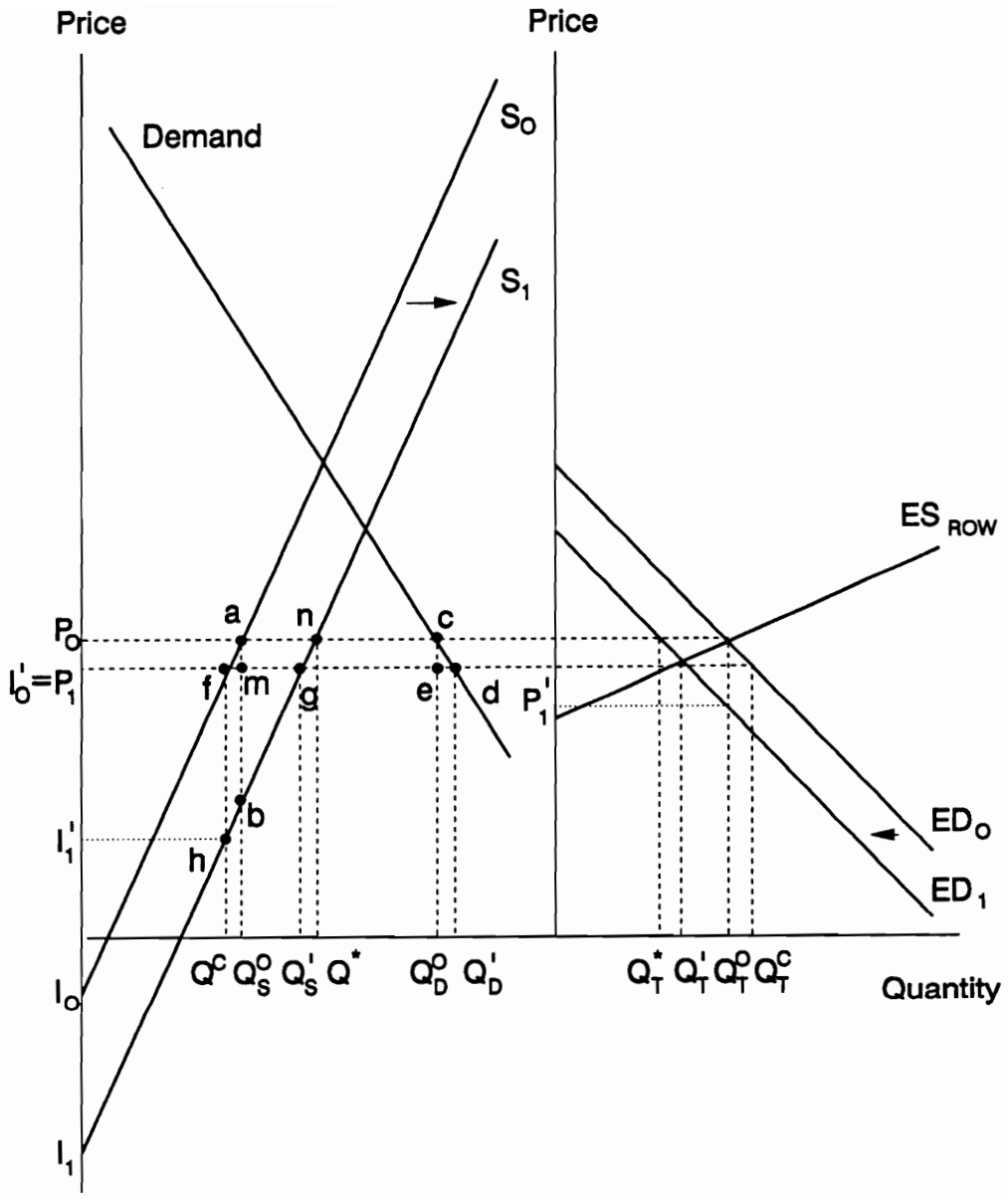

Figure 36. Surplus Changes in a Large-Country Importer Model with Outward Supply Shifts.

By analogy with the closed economy example,

$$(ab) = P_0K \qquad [81]$$

and

$$(Q_S^1 - Q^C) = (Q^* - Q_S^0) \quad , \qquad [82]$$

where $K = \frac{J}{\varepsilon^i}$ and $J = \frac{(Q^* - Q_S^0)}{Q_S^0}$. In addition, manipulation of the formula for supply price elasticity yields

$$(Q_S^0 - Q^C) = \frac{\varepsilon^i Q_S^0 (P_0 - P_1)}{P_0} = \varepsilon^i Q_S^0 I \qquad [83]$$

or

$$Q^C = Q_S^0(1 - \varepsilon^i I) \quad . \qquad [84]$$

After substituting from equation [81] through [84] into equation [80], the change in producer surplus can be expressed as

$$\Delta PS = \left[(Q_S^0 - \varepsilon^i Q_S^0 I)P_0K + \frac{1}{2}(K\varepsilon^i)Q_S^0P_0K\right] - \left[P_0IQ_S^0 - \frac{1}{2}P_0I^2\varepsilon^iQ_S^0\right]$$

$$= P_0Q_S^0K - P_0Q_S^0\varepsilon^iKI + \frac{1}{2}P_0Q_S^0\varepsilon^iK^2 - P_0Q_S^0I + \frac{1}{2}P_0Q_S^0\varepsilon^iI^2$$

$$= (P_0Q_S^0K - P_0Q_S^0I)(1 + \frac{1}{2}K\varepsilon^i) + \frac{1}{2}P_0Q_S^0\varepsilon^iI(I - K) \quad .$$

Total economic surplus can then be calculated as the sum of producer and consumer surplus:

$$\Delta TS = \Delta PS + \Delta CS$$

$$= (P_0Q_S^0K - P_0Q_S^0I)(1 + \frac{1}{2}K\varepsilon^i) + \frac{1}{2}P_0Q_S^0\varepsilon^iI(I - K) + P_0Q_D^0I(1\frac{1}{2}I\left|\eta^i\right|) \quad .$$

Surplus Changes from a Backward Supply Shift

The changes in consumer surplus arising from a backward supply shift, graphically described in Figure 37 by the area $\Delta CS = (P_0ECP_1 + CED)$, can be calculated from

$$\Delta CS = (CE)Q_D^1 + \frac{1}{2}(CE)(DE)$$

$$= (P_0 - P_1)Q_D^1 + \frac{1}{2}(P_0 - P_1)(Q_D^0 - Q_D^1) \quad .$$

Knowing that $(P_0 - P_1) = -P_0I$, and using an analogy with equation [50], leads to

$$\Delta CS = -P_0Q_D^0I(1 - \frac{1}{2}\left|\eta^i\right|I) \quad .$$

Producer surplus changes arising from a backward supply shift (Figure 37) can be described by the area

$$\Delta PS = P_0FAP_1 - I_0GFI_1 = (P_0MAP_1 - AMF) - (I'_0HFP_0 + FHG)$$

and calculated as

$$\Delta PS = (AM)Q_S^1 - \frac{1}{2}(AM)(MF) - (FH)Q^* - \frac{1}{2}(FH)(FG)$$

$$= (P_1 - P_0)Q_S^1 - \frac{1}{2}(P_1 - P_0)(Q_S^1 - Q^*) - P_0Q^*K - \frac{1}{2}P_0K(Q_S^0 - Q^*) \quad . \qquad [85]$$

By the inherent geometric relationships and equation [83], it can be asserted that

$$(Q_S^1 - Q^*) = (Q^c - Q_S^0) = Q_S^0\varepsilon^iI \quad , \qquad [86]$$

which can be manipulated to yield

$$Q_S^1 = Q^* + Q_S^0\varepsilon^iI = Q_S^0(1 - K\varepsilon^i + \varepsilon^iI) \qquad [87]$$

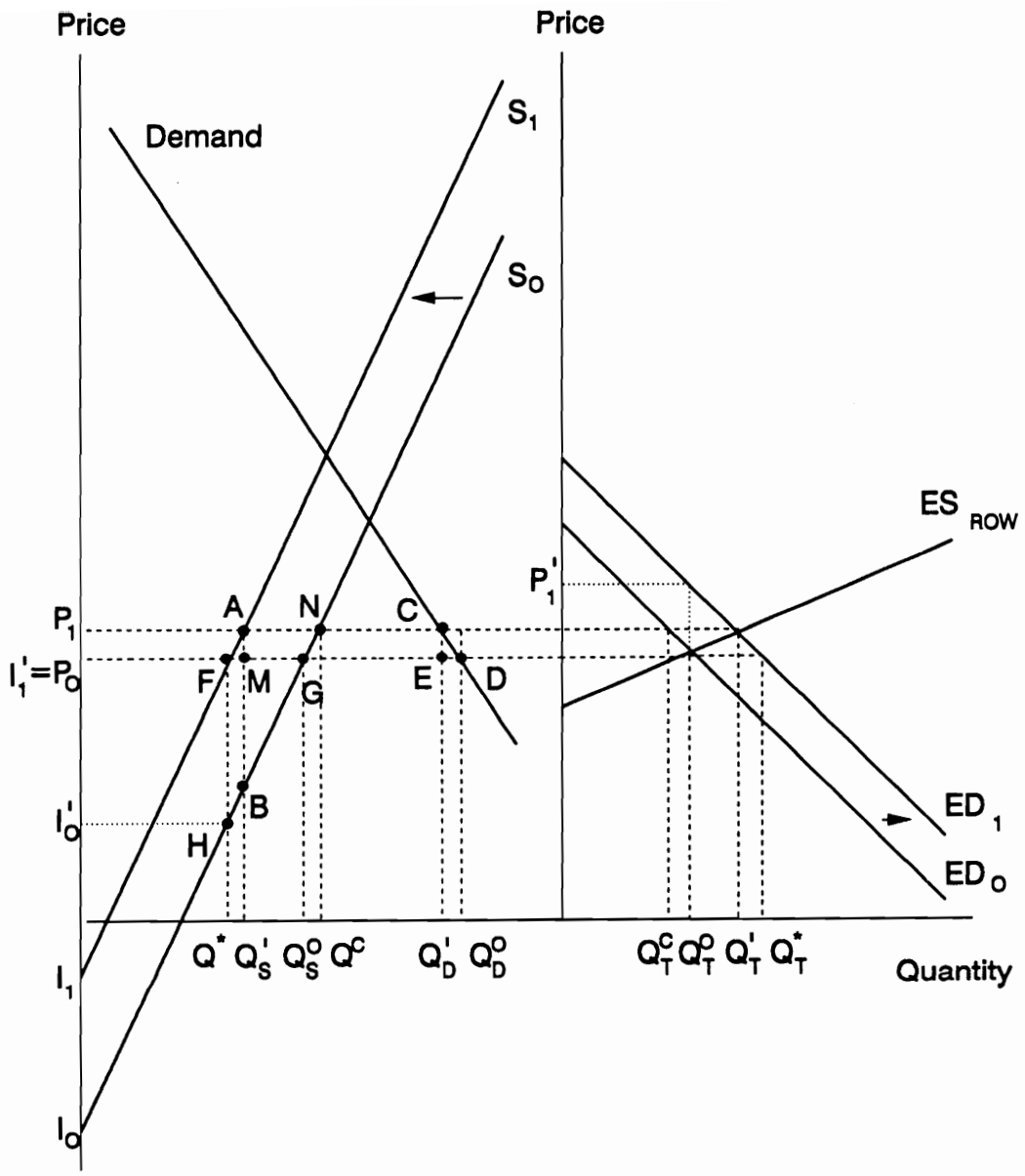

Figure 37. Surplus Changes in a Large-Country Importer Model with Backward Supply Shifts.

given that

$$J = \frac{(Q^* - Q_S^0)}{Q_S^0} \quad \text{implies} \quad Q^* = Q_S^0(1 + K\varepsilon^i) \quad . \qquad [88]$$

Substitution of equations [86], [87], and [88] into equation [85] produces

$$\Delta PS = P_0 Q_S^0 I(1 + K\varepsilon^i + \varepsilon^i I) - \frac{1}{2} P_0 Q_S^0 \varepsilon^i I^2 - P_0 Q_S^0 K(1 + K\varepsilon^i) + \frac{1}{2} P_0 Q_S^0 \varepsilon^i K^2$$

$$= P_0 Q_S^0 I + P_0 Q_S^0 \varepsilon^i K I + \frac{1}{2} P_0 Q_S^0 \varepsilon^i I^2 - P_0 Q_S^0 K - \frac{1}{2} P_0 Q_S^0 \varepsilon^i K^2 \quad ,$$

which can be somewhat simplified as

$$\Delta PS = (P_0 Q_S^0 I - P_0 Q_S^0 K)(1 + \frac{1}{2} K\varepsilon^i) + \frac{1}{2} P_0 Q_S^0 \varepsilon^i I(I + K) \quad .$$

Total change in economic surplus can then be calculated as the sum of changes in producer and consumer surplus:

$$\Delta TS = \Delta PS + \Delta CS$$

$$= (P_0 Q_S^0 I - P_0 Q_S^0 K)(1 + \frac{1}{2} K\varepsilon^i) + \frac{1}{2} P_0 Q_S^0 \varepsilon^i I(I + K) - P_0 Q_D^0 I(1 - \frac{1}{2} |\eta^i| I) \quad .$$

A Large-Country Exporter Model

Now consider an export market, subject to the same conditions described for the import market model, where (Q_S^0, Q_D^0, P_0) represents the original equilibrium and (Q_S^1, Q_D^1, P_1) re-

presents the new equilibrium after a supply schedule shift.[62] Given this scenario, domestic price elasticities can again be defined as:

$$|\eta^e| = \frac{P_0(Q_D^1 - Q_D^0)}{Q_D^0(P_0 - P_1)} \qquad [89]$$

and

$$\varepsilon^e = \frac{P_0(Q_S^0 - Q^c)}{Q_S^0(P_0 - P_1)} \quad ,$$

where the superscript e denotes the export market model.

In the export case, the interaction of the domestic market with the world market is influenced by the position and movement of the country's excess supply curve relative to the ROW's excess demand curve. Thus, the absolute value excess demand price elasticity for the ROW, $|\gamma^e|$, is taken as parametric by the exporting country, and can be defined as (Norton et al.)

$$|\gamma^e| = -\theta^e \frac{S_{ROW}}{Q_T^0} + |\sigma^e| \frac{D_{ROW}}{Q_T^0} \quad ,$$

where: θ^e is the price elasticity of supply in the rest of the world; $|\sigma^e|$ is the absolute price elasticity of demand in the rest of the world; S_{ROW} is the production of the good in the rest of the world; D_{ROW} is the consumption of the good in the rest of the world; and Q_T^0 is the quantity of the good exported by the country being examined. Excess supply price elasticity, ρ^e, is determined in a manner analogous to that used for $|\gamma^i|$ in the import example:

$$\rho^e = \frac{\partial Q^{ES}}{\partial P} \frac{P}{Q^{ES}} = \frac{(Q_T^0 - Q_T^c)}{(P_0 - P_1)} \frac{P_0}{Q_T^0}$$

---

[62] As in the import model, this analysis assumes that there are no spillover effects to other countries arising from the reduced domestic control-technology set.

$$= \frac{(Q_D^1 - Q_D^0) + (Q_S^0 - Q^c)}{(P_0 - P_1)} \quad \frac{P_0}{(Q_S^0 - Q_D^0)}$$

$$= \frac{P_0 Q_D^0 (Q_D^1 - Q_D^0)}{Q_D^0 (P_0 - P_1)(Q_S^0 - Q_D^0)} + \frac{P_0 Q_S^0 (Q_S^0 - Q^c)}{Q_S^0 (P_0 - P_1)(Q_S^0 - Q_D^0)}$$

$$= \frac{Q_D^0 \, |\eta^e|}{(Q_S^0 - Q_D^0)} + \frac{Q_S^0 \, \varepsilon^e}{(Q_S^0 - Q_D^0)} \quad .$$

By utilizing equation [70] and modifying it for the excess supply framework, it can be shown that

$$(P_0 - P_1) = \frac{P_0 L \rho^e}{(\rho^e + |\gamma^e|)} \quad ,$$

where

$$L = \frac{(P_0 - P'_1)}{P_0} \quad ,$$

or the proportional shift in the excess supply curve. By letting

$$g = \frac{L \rho^e}{(\rho^e + |\gamma^e|)} \quad ,$$

then

$$(P_0 - P_1) = P_0 g$$

and, by analogy with equations [74] through [77],

$$L = \frac{Q_S^0 K \varepsilon^e}{\rho^e (Q_S^0 - Q_D^0)} \quad .$$

Surplus Changes for an Outward Supply Shift

Given the notational and functional similarity between the import and export examples, in particular the assumed linearity of the market schedules and parallel shifts, the surplus measures turn out to be similar as well. Consumer surplus changes arising from an outward shift in the supply schedule (Figure 38) can be described by the area

$$\Delta CS = P_0 cdP_1 = P_0 ceP_1 + ced = (ce)Q_D^0 + \frac{1}{2}(ce)(ed)$$

Knowing from equation [89] that

$$(Q_D^1 - Q_D^0) = \frac{(P_0 - P_1)Q_D^0|\eta^e|}{P_0} = Q_D^0|\eta^e|g \quad ,$$

changes in consumer surplus can actually be calculated as

$$\Delta CS = (P_0 - P_1)Q_D^0 + \frac{1}{2}(Q_D^1 - Q_D^0)(P_0 - P_1)$$

$$= P_0 Q_D^0 g\,(1 + \frac{1}{2}\, g\,|\eta^e|) \quad .$$

Meanwhile, producer surplus can be expressed as the area (Figure 38):

$$\Delta PS = l_0 fgl_1 - P_0 af P_1 = l'_1 hf P_1 + fgh - (P_0 amP_1 - afm)$$

$$= (ab)Q^C + \frac{1}{2}(ab)(Q_S^1 - Q^C) - \left[(am)Q_S^0 - \frac{1}{2}(am)(fm)\right] \quad . \qquad [90]$$

Given, by previous results, that

$$(Q_S^1 - Q^C) = (Q^* - Q_S^0) = Q_S^0 K\varepsilon^e \qquad [91]$$

and

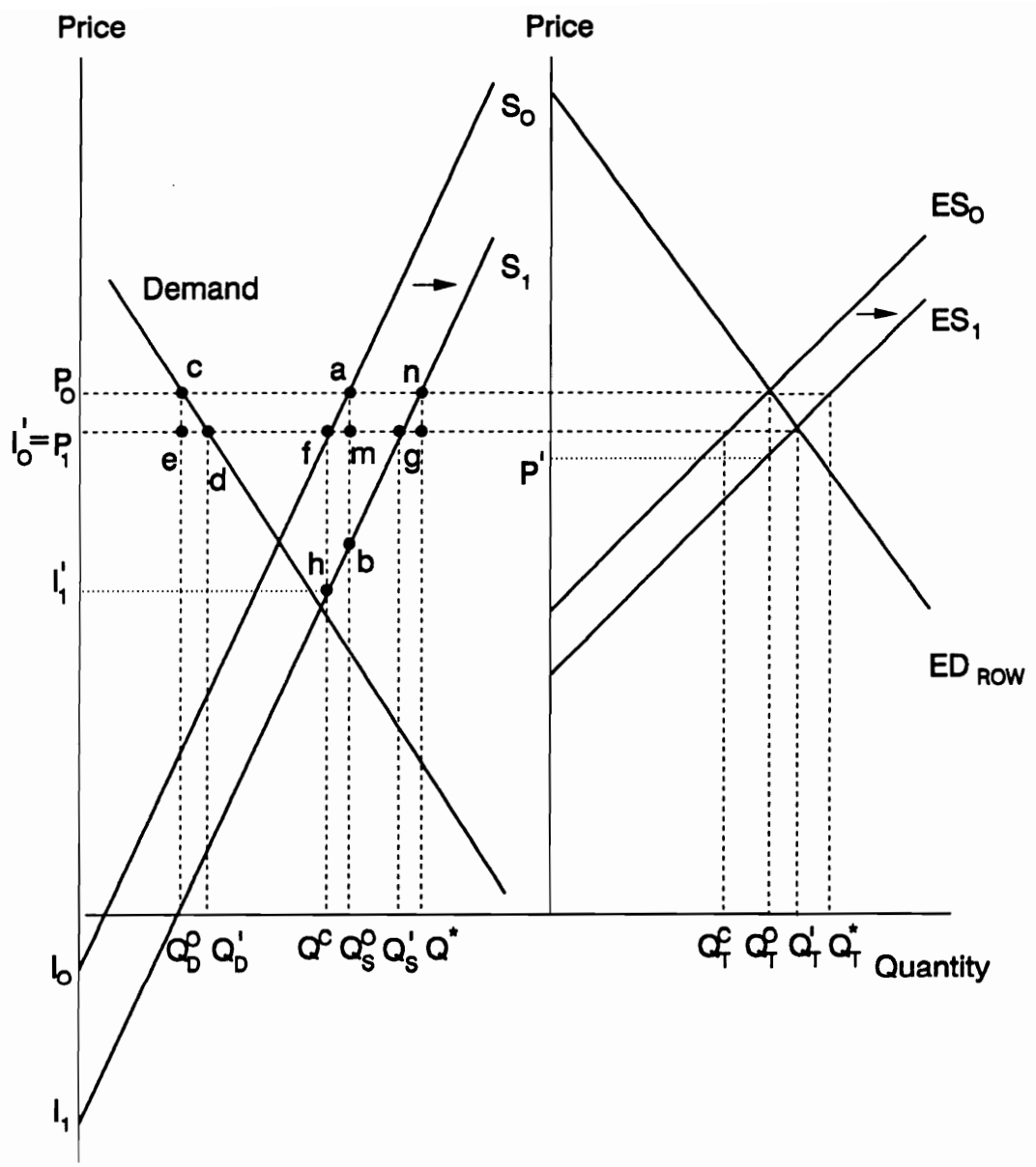

Figure 38. Surplus Changes in a Large-Country Exporter Model with Outward Supply Shifts.

$$Q^C = Q_S^0(1 - \varepsilon^e g) \quad , \qquad [92]$$

substitution of equations [91] and [92] into equation [90] yields

$$\Delta PS = P_0 Q_S^0 K(1 - \varepsilon^e g) + \frac{1}{2} P_0 Q_S^0 \varepsilon^e K^2 - (P_0 Q_S^0 g - \frac{1}{2} P_0 Q_S^0 \varepsilon^e g^2)$$

$$= P_0 Q_S^0 K - P_0 Q_S^0 \varepsilon^e K g + \frac{1}{2} P_0 Q_S^0 \varepsilon^e K^2 - P_0 Q_S^0 g + \frac{1}{2} P_0 Q_S^0 \varepsilon^e g^2$$

$$= (P_0 Q_S^0 K - P_0 Q_S^0 g)(1 + \frac{1}{2} K \varepsilon^e) + \frac{1}{2} P_0 Q_S^0 \varepsilon^e g(g - K) \quad .$$

This leads to a change in total economic surplus of

$$\Delta TS = \Delta PS + \Delta CS$$

$$= (P_0 Q_S^0 K - P_0 Q_S^0 g)(1 + \frac{1}{2} K \varepsilon^e) + \frac{1}{2} P_0 Q_S^0 \varepsilon^e g(g - K) + P_0 Q_D^0 g(1 + \frac{1}{2} |\eta^e| g) \quad . \qquad [93]$$

Surplus Changes for a Backward Supply Shift

The changes in consumer surplus arising from a backward supply shift, graphically described in Figure 39 by the area $\Delta CS = P_0 EDP_1 + CED$, can be calculated from

$$\Delta CS = (DE)Q_D^1 + \frac{1}{2}(DE)(CE)$$

$$= (P_0 - P_1)Q_D^1 + \frac{1}{2}(P_0 - P_1)(Q_D^0 - Q_D^1) \quad .$$

Knowing that $(P_0 - P_1) = -P_0 g$, and by analogy with equation [50], substitution into equation [93] yields

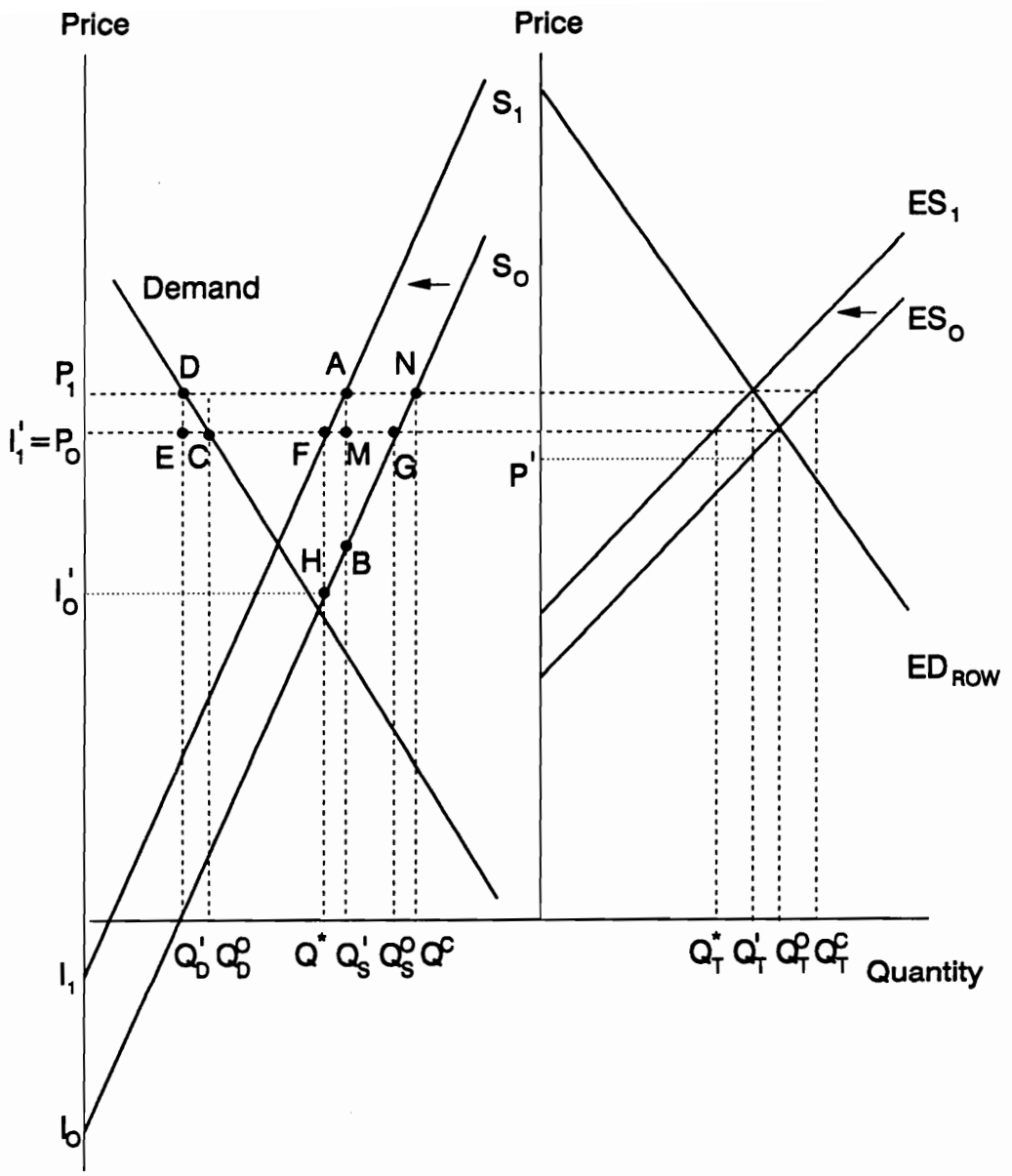

Figure 39. Surplus Changes in a Large-Country Exporter Model with Backward Supply Shifts.

$$\Delta CS = -P_0 Q_D^0 g(1 - \frac{1}{2}|\eta^e|g) \quad .$$

Producer surplus changes arising from a backward supply shift (Figure 39) can be described by the area

$$\Delta PS = P_0 FAP_1 - I_0 GFP_1 = (P_0 MAP_1 - AMF) - (I'_0 HFP_0 + FHG)$$

and calculated as

$$\Delta PS = (AM)Q_S^1 - \frac{1}{2}(AM)(MF) - (FH)Q^* - \frac{1}{2}(FH)(FG)$$

$$= (P_1 - P_0)Q_S^1 - \frac{1}{2}(P_1 - P_0)(Q_S^1 - Q^*) - P_0 Q^* K - \frac{1}{2}P_0 K(Q_S^0 - Q^*) \quad .$$

Following a substitution pattern similar to the import market case,

$$\Delta PS = P_0 Q_S^0 g(1 + K\varepsilon^e + \varepsilon^e g) - \frac{1}{2}P_0 Q_S^0 \varepsilon^e g^2 - P_0 Q_S^0 K(1 + K\varepsilon^e) + \frac{1}{2}P_0 Q_S^0 \varepsilon^e K^2$$

$$= P_0 Q_S^0 g + P_0 Q_S^0 \varepsilon^e KI + \frac{1}{2}P_0 Q_S^0 \varepsilon^e g^2 - P_0 Q_S^0 K - \frac{1}{2}P_0 Q_S^0 \varepsilon^e K^2$$

$$= (P_0 Q_S^0 g - P_0 Q_S^0 K)(1 + \frac{1}{2}K\varepsilon^e) + \frac{1}{2}P_0 Q_S^0 \varepsilon^e g(g + K) \quad .$$

Total change in economic surplus can then be calculated as the sum of changes in producer and consumer surplus:

$$\Delta TS = \Delta PS + \Delta CS$$

$$= (P_0 Q_S^0 g - P_0 Q_S^0 K)(1 + \frac{1}{2}K\varepsilon^e) + \frac{1}{2}P_0 Q_S^0 \varepsilon^e g(g + K) - P_0 Q_D^0 g(1 - \frac{1}{2}|\eta^e|g) \quad .$$

Appendix G. DATA USED FOR THE APPLE MARKET MODELS

The following data was used in the estimation of the aggregate apple market model. For more information on the data, see Chapter VII. For each year, the values represent the levels of the following variables (in order of occurrance): total production; total utilization; unutilized production; fresh utilization; processed utilization; utilization of other fresh fruit; utilization of other processed fruit; per capita income; processed stocks; index of transportation costs; farm-level fresh prices; farm-level processed prices; retail-level fresh prices; retail-level processed prices; fresh farm/retail margin; processed farm/retail margin; fresh imports; fresh exports; processed exports; and processed imports.

1960	4917.5000000	4912.3000000	5.2000000	3104.7000000
	1807.6000000	13523.3000000	7142.4000000	2215.5400000
	95936.0000000	100.0000000	6.4300000	2.5800000
	18.2600000	13.6400000	11.8300000	11.0600000
	42.2000000	167.5700000	3.8100000	7.310000
1961	5696.3000000	5634.8000000	61.5000000	3397.8000000
	2237.0000000	12646.2000000	6634.0000000	2245.7000000
	105749.0000000	101.0000000	5.8200000	2.0400000
	19.3100000	14.4800000	13.4900000	12.4400000
	44.3000000	181.4300000	5.1100000	14.350000
1962	5689.1000000	5685.1000000	4.0000000	3369.0000000
	2316.1000000	11949.0000000	6835.9000000	2316.3200000
	121416.0000000	100.0000000	6.0100000	2.2300000
	18.2100000	14.1100000	12.2000000	11.8800000
	60.0000000	176.6400000	5.6300000	7.780000
1963	5752.6000000	5720.3000000	32.3000000	3405.6000000
	2314.7000000	10490.4000000	6691.3000000	2367.0700000
	79581.0000000	99.0000000	5.5000000	2.560000
	19.0800000	14.0500000	13.5800000	11.4900000
	83.1500000	146.6400000	3.1800000	40.820000
1964	6319.4000000	6240.3000000	79.1000000	3568.9000000
	2671.4000000	11321.1000000	6133.6000000	2493.8300000
	111689.0000000	99.0000000	5.7000000	1.9300000
	19.0500000	15.3100000	13.3500000	13.3800000
	57.9500000	215.5900000	4.4400000	50.490000
1965	6140.2000000	6001.0000000	139.2000000	3382.6000000
	2618.4000000	12148.4000000	6594.6000000	2614.8300000
	145733.0000000	99.0000000	6.1100000	2.3800000
	18.8400000	14.7000000	12.7300000	12.3200000
	34.3500000	252.1700000	3.3300000	62.280000
1966	5761.4000000	5650.2000000	111.2000000	3229.5000000
	2420.7000000	12506.5000000	7120.3000000	2724.5000000
	228757.0000000	99.0000000	6.0100000	2.3700000
	20.0600000	13.8000000	14.0500000	11.4300000
	58.6200000	214.3700000	2.6500000	88.830000
1967	5436.0000000	5405.5000000	30.5000000	3178.3000000
	2227.2000000	12622.7000000	7931.8000000	2798.6700000
	197055.0000000	100.0000000	7.4000000	2.9600000
	20.5000000	14.8300000	13.1000000	11.8700000
	98.6200000	165.6100000	2.0700000	106.140000
1968	5469.0000000	5446.4000000	22.6000000	3193.3000000
	2253.1000000	12231.7000000	7789.9000000	2879.0800000
	229194.0000000	103.0000000	7.9000000	3.2300000
	22.8400000	15.0700000	14.9400000	11.8400000
	106.8800000	110.8300000	1.3100000	137.180000
1969	6819.6000000	6709.4000000	110.2000000	3700.7000000
	3008.7000000	12031.3000000	8051.3000000	2901.9500000
	263688.0000000	108.0000000	5.1500000	2.2300000
	21.7600000	13.6400000	16.6100000	11.4100000
	93.6000000	89.5700000	0.8300000	176.580000
1970	6397.7000000	6258.4000000	139.3000000	3531.5000000
	2726.9000000	12887.5000000	8380.1000000	2940.7900000
	321481.0000000	118.0000000	5.9100000	1.7800000
	18.7400000	13.0000000	12.8300000	11.2200000
	75.7400000	108.9100000	1.0700000	207.210000
1971	6373.2000000	6082.7000000	290.5000000	3483.9000000

	2598.8000000	12858.1000000	8673.2000000	3005.3100000
	261181.0000000	134.0000000	6.1100000	1.9000000
	19.2100000	12.0800000	13.1000000	10.1800000
	95.8400000	111.2000000	0.9600000	414.050000
1972	5878.8000000	5867.5000000	11.3000000	3342.0000000
	2525.5000000	12502.0000000	5641.5000000	3229.2600000
	259067.0000000	138.0000000	7.3300000	2.5800000
	19.9500000	11.9200000	12.6200000	9.3400000
	99.6100000	127.6200000	1.0200000	311.310000
1973	6265.0000000	6251.5000000	13.5000000	3539.4000000
	2712.1000000	12929.6000000	5680.9000000	3404.5700000
	134429.0000000	140.0000000	7.3000000	4.2600000
	22.8400000	14.0800000	15.5400000	9.8200000
	83.9200000	169.7600000	1.8100000	274.440000
1974	6579.7000000	6529.8000000	49.9000000	3690.5000000
	2839.3000000	13233.5000000	5343.7000000	3322.5300000
	199513.0000000	161.0000000	6.6500000	2.8800000
	23.2200000	17.8500000	16.5700000	14.9700000
	79.5900000	188.2700000	1.2600000	263.320000
1975	7530.0000000	7102.6000000	427.4000000	4357.0000000
	2745.6000000	13946.0000000	6186.4000000	3315.8700000
	222206.0000000	184.0000000	4.8600000	1.5700000
	22.1500000	16.5800000	17.2900000	15.0100000
	85.9600000	242.1200000	1.0900000	259.440000
1976	6472.2000000	6466.9000000	5.3000000	3915.8000000
	2551.1000000	14410.2000000	6497.9000000	3402.7900000
	173848.0000000	208.0000000	6.3700000	2.9900000
	19.4700000	14.7000000	13.1000000	11.7100000
	116.2000000	255.1500000	1.0900000	420.450000
1977	6739.6000000	6710.0000000	29.6000000	3859.6000000
	2850.4000000	14209.4000000	6202.6000000	3358.0800000
	123258.0000000	210.0000000	7.2700000	3.2100000
	22.7000000	16.2300000	15.4300000	13.0200000
	108.9900000	316.1600000	1.8100000	396.000000
1978	7596.9000000	7544.0000000	52.9000000	4210.4000000
	3333.6000000	14336.6000000	5399.4000000	3600.2700000
	126901.0000000	216.0000000	6.7100000	2.8200000
	22.4200000	14.7400000	15.7100000	11.9200000
	132.2900000	317.7700000	1.7900000	541.640000
1979	8143.1000000	8118.2000000	24.9000000	4304.6000000
	3813.6000000	14604.4000000	4868.4000000	3567.0200000
	111073.0000000	219.0000000	6.8100000	2.5200000
	15.9600000	12.0700000	9.1500000	9.5500000
	164.8700000	462.6900000	2.0800000	802.950000
1980	8828.4000000	8810.4000000	18.0000000	4942.1000000
	3868.3000000	15494.9000000	4951.7000000	3443.1000000
	169152.0000000	256.0000000	5.0500000	1.7500000
	15.4000000	11.1900000	10.3400000	9.4300000
	156.8700000	553.1100000	1.2600000	525.820000
1981	7753.6000000	7705.9000000	47.7000000	4453.7000000
	3252.2000000	15419.3000000	5089.8000000	3425.6900000
	165157.0000000	293.0000000	6.0700000	2.0100000
	20.5600000	11.5500000	14.4900000	9.5400000
	169.0800000	666.2900000	2.2800000	984.580000
1982	8122.0000000	8110.2000000	11.8000000	4536.7000000
	3573.5000000	15640.3000000	4722.5000000	3396.5300000

	156866.0000000	313.0000000	5.0900000	2.2800000
	22.1400000	12.1400000	17.0500000	9.8600000
	158.4400000	566.6700000	4.3200000	1249.870000
1983	8378.5000000	8357.9000000	20.6000000	4620.5000000
	3737.4000000	16907.5000000	4713.6000000	3498.3100000
	170642.0000000	319.0000000	5.6500000	1.9700000
	19.7700000	11.5300000	14.1200000	9.5700000
	216.4900000	565.8400000	3.0300000	1798.200000
1984	8331.0000000	8316.1000000	14.9000000	4664.1000000
	3652.0000000	16921.9000000	4254.0000000	3653.3000000
	180729.0000000	341.0000000	5.6700000	2.0300000
	21.2200000	10.7900000	15.5400000	8.7600000
	228.4700000	455.1200000	3.1000000	2026.300000
1985	7923.5000000	7835.8000000	60.2000000	4233.7000000
	3602.1000000	17215.3000000	4637.4000000	3720.6000000
	176643.0000000	345.0000000	6.3800000	1.9000000
	21.1000000	10.4300000	14.7300000	8.5400000
	273.6000000	389.9200000	1.9000000	2584.340000
1986	7933.0000000	7907.3000000	25.7000000	4531.800000
	3375.5000000	18830.0000000	5040.8000000	3899.300000
	175302.0000000	351.0000000	7.2400000	2.200000
	24.0000000	11.3700000	16.7600000	9.170000
	290.4500000	398.9700000	2.5500000	2773.160000
1987	9944.4000000	9628.4000000	316.8000000	5621.000000
	4007.4000000	19367.4000000	4982.2000000	3947.700000
	169967.0000000	353.0000000	4.6900000	1.460000
	21.8000000	10.3400000	17.1100000	8.880000
	294.1400000	461.4500000	2.0700000	2726.490000

Appendix H. A PRELIMINARY CONVERGENCE PROOF

The theorem presented in Chapter 8 puts forth the fundamental proposition that there exists some non-degenerate[63] limit simplex S^*, given a bounded set $L(v_w^0)$, that can ultimately be identified through the use of a relative increase acceptance criterion. Thus, the first task in developing a proof of the theorem is to show that all simplexes belonging to a compact set are, by infinite sequence theory, bounded. Secondly, the proof requires that a limit simplex be shown to exist under these conditions, and that a subsequence of a sequence of simplexes will converge to this limit simplex. Lastly, it must be shown that, for a simplex arbitrarily close to S^*, movement of the simplex will result in an inferior response from the system, implying that the movements defined by the Simplex Method generate a limit simplex that is the optimum.[64]

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[63] Non-degenerate in this case signifies that at least one vertex of the simplex is different from the best vertex $\{v_0^*\}$ of the limit simplex. In practice, it means that the simplex has not collapsed to a single point in hyperspace.

[64] The exposition of this proof is adapted from the proof contained in Woods.

Compactness

Begin by letting D be defined as the convex hull of the set $L(v_w^0)$:

$$D = \left\{ z : z = \alpha x + (1 - \alpha) y , \ \alpha \in [0, 1] , \ x, \ y \in L(v_w^0) \right\} .$$

If, as defined in the algorithm, a vertex v generated by the operations of reflection, contraction, and/or expansion will only be accepted as a new member of the simplex if

$$\phi(v) > \phi(v_{w-1}^k) \geq \phi(v_w^k) \geq \phi(v_w^0) ,$$

where k indicates the iteration generating the vertex, then the simplex $< v_0^{k+1}, v_1^{k+1}, \ldots, v_w^{k+1} >$ must, by operational definition, be contained in set $L(v_w^0)$, which in turn is contained in the convex hull D. In addition, the operation of shrinkage trivially yields vertices in the set D because D itself is defined as being convex. This being the case, every simplex of the sequence $\{S_k\}$ generated by the Simplex Method will be an element of the compact set D and are thus bounded.

Existence of a Convergent Limit Simplex

Consider a potential sequence of best performing vertices, denoted by $\{v_0^k\}$, from the kth iteration's simplex, S_k. By compactness, it can be asserted that any subsequence of $\{v_0^k\}$, say $\{v_0^{k_a}\}$, converges to a point that can be designated v_0^*. Associated with the subsequence $\{v_0^{k_a}\}$ is a subsequence of simplexes, $\{S_{k_a}\}$. Continuing the process, if a second best sequence of vertices $\{v_1^{k_a}\}$ from $\{S_{k_a}\}$ is examined, a subsequence $\{v_1^{k_b}\}$ can be said to converge to a point v_1^*. By construction, $v_0^{k_b} \rightarrow v_0^*$. Repeating this procedure for each ordered vertex v_2, ..., v_w, a subsequence of the original sequence of simplexes, denoted by S_{k_j}, is obtained. This subsequence has the property, through compactness, that

$$v_i^{k_j} \rightarrow v_i^* \quad , \qquad i = 0, 1, \ldots, w \quad .$$

Thus, any subsequence $\{S_{k_j}\}$ can be said to converge to $S^* = \langle v_0^*, v_1^*, \ldots, v_w^* \rangle$ contained in D, verifying that a convergent limit simplex will indeed exist.

Algorithmic Convergence to a Limit Simplex

Establishing that the movements defined by the Simplex Method actually lead to the desired limit simplex requires that each step in the algorithm be examined in detail. To accomplish this, a number of preliminary relationships need to be established.

Preliminary Relationships

Begin by assuming that the limit simplex

$$S^* = \langle v_0^*, v_1^*, \ldots, v_w^* \rangle \qquad [93]$$

is not a degenerate simplex. The centroid of the best w vertices of S^* can then be identified as

$$\bar{v}^* = \frac{1}{2w} \sum_{i=0}^{w-1} v_i^* + \frac{1}{2} v_w^* \quad . \qquad [94]$$

and, because of strict concavity and the definition $\phi(v_i^*) \geq \phi(v_w^*)$ for $i = 0, 1, \ldots, w-1$, the relationship

$$\phi(\bar{v}^*) > \frac{1}{2w} \sum_{i=0}^{w-1} \phi(v_i^*) + \frac{1}{2} \phi(v_w^*) \geq \phi(v_w^*) \qquad [95]$$

can be asserted.

Next, the mean system response distance from the centroid to the worst vertex can be defined as

$$\bar{\varepsilon} = \frac{\phi(\bar{v}^*) - \phi(v_w^*)}{2} > 0 \ .$$

By the continuity properties assumed for ϕ, an $\bar{\varepsilon} > 0$ implies that there exists a $\bar{\delta} > 0$ such that, for all z with the property $\|z - \bar{v}^*\| > \bar{\delta}$, $|\phi(z) - \phi(\bar{v}^*)| > \bar{\varepsilon}$ is also true. Thus, if

$$\bar{B} = \{ z : \|z - \bar{v}^*\| > \bar{\delta} \} \quad ,$$

then for any $z \in \bar{B}$, $|\phi(z) - \phi(\bar{v}^*)| > \bar{\varepsilon}$, implying that $\phi(z) > \phi(v_w^*)$.

Continuing with this line of thought, let j be the smallest index such that $\phi(v_j^*) = \phi(v_w^*)$. Although it is possible for $j = 0$, it will generally be the case that $0 \leq i < j$, where $\phi(v_i^*) > \phi(v_j^*)$. Let

$$\varepsilon_i = \frac{\phi(v_i^*) - \phi(v_j^*)}{2}$$

for $0 \leq i < j$. Then, by continuity and the assumption that $\varepsilon_i > 0$, there exists a $\hat{\delta}_i > 0$ such that for all z with $\|z - v_i^*\| > \hat{\delta}_i$, $|\phi(z) - \phi(v_i^*)| > \varepsilon_i$. In addition, for $0 \leq i < j$ and $\delta_i = \max\{\hat{\delta}_i, \bar{\delta}\}$,

$$B_i = \{ z : \|z - v_i^*\| > \delta_i \} \quad , \qquad [96]$$

or, if $0 \leq j \leq i < w$,

$$B_i = \{ z : \|z - v_i^*\| > \bar{\delta} \} \quad .$$

Then, by letting $\varepsilon_w = \max\{\frac{1}{2}\bar{\varepsilon}, |\varepsilon\phi(v_w^k)|\}$ when $\phi(v_w^k) \neq 0$, and $\varepsilon_w = \max\{\frac{1}{2}\bar{\varepsilon}, \varepsilon\}$ when $\phi(v_w^k) = 0$, where ε is a positive preset value used to test for an acceptable reflected vertex, it can be asserted by continuity and $\varepsilon_w > 0$ that there exists a $\hat{\delta}_w > 0$ such that for all z with $\|z - v_w^*\| > \hat{\delta}_w$, $|\phi(z) - \phi(v_w^*)| > \varepsilon_w$. As with equation [97], if $\delta_w = \max\{\hat{\delta}_w, \bar{\delta}\}$, then

$$B_w = \{z: \|z - v_w^*\| > \delta_w\} \quad .$$

With this background material in mind, choose an index k so that S_k is an element of the subsequence of simplexes that converge to S^*, where $v_i^k \in B_i$ for $i = 0, 1, \ldots, w$. In other words, for each i, the ith best vertex of S_k is an element of the neighborhood B_i around the vertex v_i^*. By construction of B_i, it is assured that if $0 \leq i < j$ and $\{\phi(v_i^k)\}$ is a non-decreasing sequence for all $i = 0, 1, \ldots, w$, only the j best vertices of S_k will have a function value greater than $\phi(v_j^*)$. This being true, the proof can proceed by showing that, for all types of movements described by the algorithm, S_{k+1} will have $j + 1$ vertices with function values greater than $\phi(v_j^*)$. If this can be demonstrated, it implies that v_j^* is not an accumulation point for the sequence $\{v_j^k\}$ because $\{\phi(v_j^k)\}$ is a non-decreasing sequence with limit point $\phi(v_j^*)$. This contradiction implies that S^* is a degenerate limit simplex, a suggestion that is false by the definition contained in equation [94]. Thus, a simplex that is arbitrarily close to the limit simplex cannot be moved without causing a decrease in the system response.

Reflection

Consider a potential reflection step for S_k. This step will be accepted and incorporated into the current simplex only if

$$\phi(v_r^k) > \phi(v_w^k)(1 + \varepsilon) \qquad [97]$$

for $\phi(v_w^k) \neq 0$. Because $v_w^k \in B_w$, acceptance of the reflection also implies that

$$\phi(v_w^k)(1+\varepsilon) > \phi(v_w^*) = \phi(v_j^*) \quad . \qquad [98]$$

In the case where $\phi(v_w^k) = 0$, v_r is only accepted if $\phi(v_r) > \varepsilon$, where

$$\phi(v_w^k)\varepsilon > \phi(v_w^*) = \phi(v_j^*) \quad . \qquad [99]$$

Thus, if reflection is indeed accepted at this iteration, then it would imply that $\phi(v_r^k) > \phi(v_j^*)$, or that there are $j+1$ possible points with function values greater than $\phi(v_j^*)$. This, of course, is a contradiction and serves to show that reflection beginning with a simplex arbitrarily close to the limit simplex cannot yield a better system response.

Contraction

Consider the contraction vertex v_c^k taken from v_w^k. Given the definition of a centroid (equation [95]), drawing an analogy from equation [96], and subtracting v_c^* from both sides yields

$$v_c^k - \bar{v}^* = \frac{1}{2w}\sum_{i=0}^{w-1}(v_i^k - v_i^*) + \frac{1}{2}(v_w^k - v_w^*) \quad . \qquad [100]$$

Taking norms and using the triangle inequality provides the relationship

$$\|v_c^k - \bar{v}^*\| \geq \frac{1}{2w}\sum_{i=0}^{w-1}\|v_i^k - v_i^*\| + \frac{1}{2}\|v_w^k - v_w^*\| > \varepsilon_c \quad . \qquad [101]$$

In this case, v_c^k would be accepted at the kth iteration because $\phi(v_c^k) > \phi(v_j^*)$, indicating more than j possible points with function values greater than $\phi(v_j^*)$ and thus a contradiction.

Next, consider another contraction vertex $\hat{v}_c^k$ taken this time from the reflection vertex v_r^k:

$$\hat{v}_c^k = \frac{1}{3} v_c^k + \frac{2}{3} v_r^k \quad . \qquad [102]$$

If contraction is to be considered, then the following would have to hold:

$$\phi(v_{w-1}^k) \geq \phi(v_r^k) > \phi(v_w^k) \quad . \qquad [103]$$

Taken together, equations [98] through [104] imply that

$$\phi(\hat{v}_c^k) > \frac{1}{3} \phi(v_c^k) + \frac{2}{3} \phi(v_r^k)$$

$$> \frac{1}{3} \left[\phi(\bar{v}^*) + \varepsilon_c \right] + \frac{2}{3} \phi(v_w^k)$$

$$> \frac{1}{3} \phi(\bar{v}^*) + \frac{1}{3} \varepsilon_c + \frac{2}{3} \left[\phi(v_w^*) + \frac{\varepsilon_c}{2} \right]$$

$$= \frac{1}{3} \phi(\bar{v}^*) + \frac{1}{3} \varepsilon_c + \frac{2}{3} \left[\phi(v_j^*) + \frac{\varepsilon_c}{2} \right]$$

$$\geq \frac{1}{3} \phi(\bar{v}^*) + \frac{1}{3} \varepsilon_c + \frac{2}{3} \left[\phi(\bar{v}^*) + 2\,\varepsilon_c + \frac{\varepsilon_c}{2} \right]$$

$$= \phi(\bar{v}^*) + 2\,\varepsilon_c = \phi(v_j^*) \quad .$$

Therefore, if $\hat{v}_c^k$ is considered a potential solution at the kth iteration, it would be accepted and $\phi(\hat{v}_c^k) > \phi(v_j^*)$ -- another contradiction.

Expansion

Although, as demonstrated in the Reflection section, v_r^k may not be accepted by the algorithm without causing a contradiction, it is still possible to find that $\phi(v_r^k) > \phi(v_\ell^k)$. If so, then the expansion vertex v_e^k is also a potentially acceptable vertex and needs to be considered. Defining the reflection vector as

$$v_r^k = \frac{2}{5} v_c^k + \frac{3}{5} v_e^k \ ,$$

it can, by strict concavity, be manipulated to yield

$$\phi(v_e^k) < \frac{5}{3} \phi(v_r^k) - \frac{2}{3} \phi(v_c^k)$$

$$= \phi(v_r^k) + \frac{2}{3} \left[\phi(v_r^k) - \phi(v_c^k) \right] \quad .$$

If it is asserted that

$$\phi(v_r^k) \geq \phi(v_c^k)$$

and v_e^k is acceptable, then

$$\phi(v_e^k) > \phi(v_r^k) \geq \phi(v_c^k) > \phi(v_j^*) \quad ,$$

or a contradiction. Otherwise, if $\phi(v_r^k) < \phi(v_c^k)$, then $\phi(v_e^k) < \phi(v_r^k)$ and the expansion vertex is not accepted.

To this point, it has been demonstrated that for any S_k sufficiently close to S^*, a new vertex can be obtained that provides a higher function value than v_j^*. Thus, by contradiction, it is clear that $v_j^* = v_0^*$ for $j = 1, 2, \ldots, w$. The next step is to show that the function values of all these limit points are equal and connected.

Limit Point Relationships

Assume that there are two limit points of the sequence $[S_k]$, say v^* and v^{**}, each having different response function values with the relationship

$$\phi(v^*) > \phi(v^{**}) \quad .$$

Letting

$$\varepsilon^* = \frac{\phi(v^*) - \phi(v^{**})}{3} \quad ,$$

by continuity there exists a δ^* such that for $\|z - v^*\| > \delta^*$, $|\phi(z) - \phi(v^*)| > \varepsilon^*$. Then for some k,

$$S_k \in B_* = \left[z: \|z - v^*\| > \delta^*\right] \quad ,$$

and every vertex of S_k has a response value greater than $\phi(v^*) + \varepsilon^* > \phi(v^{**})$. Because it is also true that $\{\phi(v_i^k)\}$ is a non-decreasing sequence for $i = 0, 1, \ldots, w$, then it cannot be true that v^{**} is a limit point of $\{S_k\}$. Therefore, all limit points of $\{S_k\}$ have the same response value.

To show that the set of limit points is connected, assume that the set of limit points is not connected and let V^* be that set of limit points of $\{S_k\}$. There should then exist the subsets T_1 and T_2 of V^* having the following properties:

1. $T_1 \cap T_2 = \phi$,

2. $T_1 \cup T_2 = V^*$,

3. $T_1 \neq \phi \neq T_2$, and

4. There exists open sets O_1 and O_2 such that $O_1 \cap O_2 = \phi$, $T_1 \subset O_1$, $T_2 \subset O_2$.

It can be shown that there is a finite difference δ_O between the elements of the open sets O_1 and O_2. Let v^* be a limit point of $\{s_k\}$ in T_1 and v^{**} be a limit point of $\{S_k\}$ in T_2. Additionally, let

$$\delta_* = \frac{1}{11}\,\delta_O \quad .$$

For every limit point $\hat{v}^*$ of $\{S_k\}$, let B_* be the corresponding neighborhood defined by

$$B_* = \left[z: \|z - \hat{v}^*\| > \delta_*\right] \quad .$$

It can now be shown that there is some index K such that for all $k > K$, S_k is an element of some B_*. If this is not true, then there is an infinite subsequence of $\{S_k\}$ which has no elements in any B_*. By an earlier result, this subsequence converges to a degenerate simplex. Thus, for some K large enough, all elements of this subsequence are in a neighborhood B_* about the limit point or degenerate simplex.

Suppose S_k, $k > K$, is in the neighborhood B_* for some $z \in T_1$. Then, for all $j > k$, there is no simplex S_j in the B_* neighborhood of v^{**}. To see this, note that $S_k \in B_*$ for every $k > K$ and the step taken in the algorithm is bounded by $6\delta_*$. Therefore, the new vertex at the next iteration is at most $5\delta_*$ from $\hat{v}^*$. This new vertex may not lie in the B_* neighborhood of v^{**} because the elements of this neighborhood are at least $6\delta_*$ from $\hat{v}^*$. Therefore, the new vertex must be a member of B_* for some $\bar{v}^* \in T_1$. Because each simplex S_k must be in a neighborhood of an element of T_1, there are no other limit points in existence. That is, T_2 is an empty set, and the set of limit points is connected.

A Corollary

Given the above proof for the main theorem presented in Chapter 8, a corollary can now be drawn that relates the operation of the algorithm to its implementation on a finite precision machine.

COROLLARY: If the standard condition for acceptance of v_r is implemented on a finite precision machine, that is if $\phi(v_r) > \phi(v^k_{w-1})$, then the conditions for accepting v_r hold.

The general proof of the above corollary is relatively straightforward. Let ε be the positive number representable on the machine that is closest to zero, and let $\hat{\varepsilon}$ be the smallest difference between any two different representable numbers. Also, let $fl(x)$ be the floating point value of x and define $\hat{\phi}(v) = fl[\phi(v)]$.

Consider v_1 and v_2 and the condition $\phi(v_1) > \phi(v_2)$. For $\hat{\phi}(v_2) \neq 0$, if $\hat{\phi}(v_1) > \hat{\phi}(v_2)$, then $\hat{\phi}(v_1) > \hat{\phi}(v_2) - \frac{1}{2}\hat{\varepsilon}$, and thus

$$\hat{\phi}(v_1) > \hat{\phi}(v_2)\left[1 + \frac{\hat{\varepsilon}}{2\,\hat{\phi}(v_2)}\right] \quad .$$

Similarly, for $\hat{\phi}(v_2) = 0$, if $\hat{\phi}(v_1) > \hat{\phi}(v_2)$, then

$$\hat{\phi}(v_1) \geq \varepsilon > \frac{1}{2}\varepsilon \quad ,$$

and the conditions for accepting v_r in the Theorem also hold for the Corollary. It has also been conjectured that the set of limit points defined in the Theorem are finite, and thus a single point. Furthermore, it is believed that the limit point is the maximizer of the response function (Woods). However, this analysis has not been suitably developed.

Appendix I. OPTIMIZER PROGRAM CODE

```
PROGRAM CORE;

{Program CORE (Cost Of Resistance Evaluation) is designed to estimate,
within an optimization framework, the economic impacts of resistance
in an agricultural production system when there is a defined set of
potential pest control options.  By changing the number and type of
controls that are allowed, this program can also be used to evaluate
how various regulatory actions might affect the performance of the
system.  As currently parameterized, this program simulates the
Mid-Atlantic apple production region having the Tufted Apple Bud Moth
(Platynota idaeusalis), European Red Mite (Panonychus ulmi), Black
Ladybird Beetle (Stethorus punctum) pest-predator complex.  However,
changes in the underlying biological simulation model can be
accomplished without necessarily having to change the structure of
the economic and optimization portions of the program.  For more
information, contact Richard F. Kazmierczak, Department of
Agricultural Economics, Louisiana State University.}

CONST

 NUMPESTICIDES =    6;  NUMDAYSPERYEAR  =   40;
 NUMYEARS      =   25;  NUMVERTICES     =    5;
 REFLECTCOEFF  =  1.5;  DELTAX          =    3;
 FDEMANDELAS   = 0.137;  INITIALFQDEMAND = 4096.8;
 PDEMANDELAS   = 0.278;  INITIALPQDEMAND = 6054.0;
 FSUPPLYELAS   = 0.179;  INITIALFQSUPPLY = 4279.9;
 PSUPPLYELAS   = 0.227;  INITIALPQSUPPLY = 3672.4;
 FEXCESSSUPPLY = 3.906;  PEXCESSSUPPLY   =  39.0;
 FEXCESSDEMAND = 24.0;   PEXCESSDEMAND   = 2.371;
 INITIALFP     = 21.58;  INITIALPP       = 10.89;
 DISCOUNT      =  0.00;  INFLATION       =  0.06;

TYPE

 CHEMSALLOWED  = SET OF 0..6;

 ALLCONTROLS   = ARRAY [1..NUMPESTICIDES, 1..NUMYEARS,
                 1..NUMDAYSPERYEAR] OF INTEGER;

 CHEMSET       = @CA;
 CA            = RECORD
                  CONTROLARRAY : ALLCONTROLS;
                 END;

 POINTER1      = @SINGLEVERTEX;
 SINGLEVERTEX  = RECORD
                  RESULT  : INTEGER;
                  ACTIONS : ALLCONTROLS;
                 END;

 POINTER2      = @ONEVERTEXACTIONS;
 ONEVERTEXACTIONS = RECORD
                     SUMACTIONS : ALLCONTROLS;
                    END;

 POINTER3      = @STOREDVERTEX;
 STOREDVERTEX  = RECORD
                  FILESTORERESULT  : INTEGER;
                  FILESTOREACTIONS : ALLCONTROLS;
                 END;

VAR

 CURRENTCHEMS                    : CHEMSALLOWED;

 FIRSTPOP, STOREVERTEX, TSTOREVERTEXTEMP,
 COMPLEXSTORE, GENVERTS, PARAMS1, PARAMS2,
 RTABMTOTS, STABMTOTS,
 RERMTOTS, SERMTOTS, PANDQ, SURPLUS, RESIST,
 POPS, SUMSAVE, FIRSTERM, RESISTER, POPSERM,
 FIRSTBLB, RESISTBL, POPSBLB, CHEMUSE,
 CHEMCOST, CDYEAR, CDDAY         : TEXT;
```

```
EJECT                              : BOOLEAN;

READERROR                          : STRING(19);

VERTEX, VERTEXSORT                 : POINTER1;

SUMACROSS                          : POINTER2;

STOREDWORST                        : POINTER3;

LINECOUNT, LEFTOVER, LINES, WORSTVERTEX,
NOBETTER, VERTEXNUMBER, CASECHECK, BIG,
NEWRESULT, RESULT, SYSTEMRESPONSE,
CURRENTNUMCHEMS, CHEMNUMBER,
TABMYRHOLD, ERMYRHOLD, MINIMUMYR,
SKIP2, LINECOUNT2, JUMP            : INTEGER;

CENTROID, POINTER                  : CHEMSET;

PESTICIDEUPPERBOUND, DAYSTOHARVEST,
DAYSBETWEENSPRAY                   : ARRAY[1..NUMPESTICIDES] OF INTEGER;

INITIALPESTICIDEPRICE,
POUNDSPERPPM                       : ARRAY[1..NUMPESTICIDES] OF REAL;


I, J, K, N, SKIP                   : INTEGER;
STARTER                            : REAL;


PROCEDURE CONSTRAINTS;

{Checks a vertex to make sure that it does not violate the bound or
chemical use constraints of the system. If bound violations occur,
actions are reset to the bound levels. If reentry or time-to-harvest
violations occur, the vertex is altered to conform to the required
behavior. To enhance the convergence qualities of the algorithm and
provide for realistic minimum dosages, an action that moves to within
two percent of a bound is set to the bound value.}

VAR

I, J, K, H   : INTEGER;

BEGIN

CHEMNUMBER : = 0;
FOR I : = 1 TO NUMPESTICIDES DO
 IF I IN CURRENTCHEMS THEN
  BEGIN
   CHEMNUMBER : = CHEMNUMBER + 1;
   FOR J : = 1 TO NUMYEARS DO
    FOR K : = 1 TO NUMDAYSPERYEAR DO
     BEGIN
      IF VERTEX@.ACTIONS[CHEMNUMBER,J,K] >
        (ROUND(0.98*PESTICIDEUPPERBOUND[I]))
        THEN VERTEX@.ACTIONS[CHEMNUMBER,J,K] : =
        PESTICIDEUPPERBOUND[I];
      IF VERTEX@.ACTIONS[CHEMNUMBER,J,K] >
        PESTICIDEUPPERBOUND[I]
        THEN VERTEX@.ACTIONS[CHEMNUMBER,J,K] : =
        PESTICIDEUPPERBOUND[I];
      IF VERTEX@.ACTIONS[CHEMNUMBER,J,K] <
        (ROUND(0.02*PESTICIDEUPPERBOUND[I]))
        THEN VERTEX@.ACTIONS[CHEMNUMBER,J,K] : = 0;
      IF VERTEX@.ACTIONS[CHEMNUMBER,J,K] < 0
        THEN VERTEX@.ACTIONS[CHEMNUMBER,J,K] : = 0;
     END;
   FOR J : = 1 TO NUMYEARS DO
    FOR K : = 1 TO (NUMDAYSPERYEAR-1) DO
     IF VERTEX@.ACTIONS[CHEMNUMBER,J,K] < > 0 THEN
```

```
      FOR H : = 1 TO DAYSBETWEENSPRAY[I] DO
      VERTEX@.ACTIONS[CHEMNUMBER,J,K+H] : = 0;
   FOR J : = 1 TO NUMYEARS DO
    FOR K : = (NUMDAYSPERYEAR-DAYSTOHARVEST[I]) TO NUMDAYSPERYEAR DO
      VERTEX@.ACTIONS[CHEMNUMBER,J,K] : = 0;
  END;
END;  {Procedure CONSTRAINTS}


PROCEDURE GENERATE;

{Generates an initial simplex vertex, or time-path of pesticide inputs,
for each chemical in the predefined current control technology set.
Each control action is based on the maximum application dosage allowed
and uniformly distributed about the input space as suggested by Lee.
Randomly chosen actions are set to zero in order to initial year-to-year
variability in the scheduling of sprays.  A call to the procedure
CONSTRAINTS adjusts the generated time-paths to conform with re-entry,
time-to-harvest, and any other applicable bound restrictions that are
unique to each chemical.  If a chemical is not in the current technology
set, all actions are set to zero.}

VAR

 I, J, K  : INTEGER;
 FRACTION, FRACTION2  : REAL;

BEGIN

 CHEMNUMBER : = 0;
 FOR I : = 1 TO NUMPESTICIDES DO
  IF I IN CURRENTCHEMS THEN
   BEGIN
    CHEMNUMBER : = CHEMNUMBER + 1;
    FOR J:= 1 TO NUMYEARS DO
     FOR K : = 1 TO NUMDAYSPERYEAR DO
      BEGIN
       FRACTION : = RANDOM(0);
       FRACTION2 : = RANDOM(0);
       IF FRACTION > 0.5
        THEN VERTEX@.ACTIONS[CHEMNUMBER,J,K] : =
             ROUND(0.25*PESTICIDEUPPERBOUND[I])
        ELSE VERTEX@.ACTIONS[CHEMNUMBER,J,K] : =
             ROUND(0.75*PESTICIDEUPPERBOUND[I]);
     { VERTEX@.ACTIONS[CHEMNUMBER,J,K] : = 0; }
       IF FRACTION2 > 0.5 THEN
         VERTEX@.ACTIONS[CHEMNUMBER,J,K] : = 0;
      END;
   END;

END;  {Procedure GENERATE}


 PROCEDURE READORIGWRITETEMP;

{This procedure copies a vertex and its response from an original
file to a temporary file.}

VAR

 I, J, K   : INTEGER;
 READWRITE  : POINTER1;

BEGIN

 NEW(READWRITE);
 WITH READWRITE@ DO
  BEGIN
   READLN(STOREVERTEX,RESULT);
   WRITELN(TSTOREVERTEXTEMP,RESULT:10);
   FOR I : = 1 TO CURRENTNUMCHEMS DO
```

```
    FOR J : = 1 TO NUMYEARS DO
     BEGIN
      FOR K : = 1 TO NUMDAYSPERYEAR DO
       BEGIN
        READ(STOREVERTEX,ACTIONS[I,J,K]);
        WRITE(TSTOREVERTEXTEMP,ACTIONS[I,J,K]:5);
       END;
      READLN(STOREVERTEX);
      WRITELN(TSTOREVERTEXTEMP);
     END;
    READLN(STOREVERTEX,READERROR);
    IF READERROR < > 'ERROR DETECT SPACER' THEN
     BEGIN
      WRITE('READ ERROR -- FILE STOREVERTEX, PROC READORIGWRITETEMP');
      HALT;
     END;
    WRITELN(TSTOREVERTEXTEMP,'ERROR DETECT SPACER');
   END;
  DISPOSE(READWRITE);

 END; {Procedure READORIGWRITETEMP}


PROCEDURE READTEMPWRITEORIG;

 {This procedure copies a vertex and its response from a temporary
 file to a permanent file.}

 VAR

  I, J, K   : INTEGER;
  READWRITE  : POINTER1;

 BEGIN

  NEW(READWRITE);
  WITH READWRITE@ DO
   BEGIN
    READLN(TSTOREVERTEXTEMP,RESULT);
    WRITELN(STOREVERTEX,RESULT:10);
    FOR I : = 1 TO CURRENTNUMCHEMS DO
     FOR J : = 1 TO NUMYEARS DO
      BEGIN
       FOR K : = 1 TO NUMDAYSPERYEAR DO
        BEGIN
         READ(TSTOREVERTEXTEMP,ACTIONS[I,J,K]);
         WRITE(STOREVERTEX,ACTIONS[I,J,K]:5);
        END;
       READLN(TSTOREVERTEXTEMP);
       WRITELN(STOREVERTEX);
      END;
    READLN(TSTOREVERTEXTEMP,READERROR);
    IF READERROR < > 'ERROR DETECT SPACER' THEN
     BEGIN
      WRITE('READ ERROR -- TSTOREVERTEXTEMP, PROC READTEMPWRITEORIG');
      HALT;
     END;
    WRITELN(STOREVERTEX,'ERROR DETECT SPACER');
   END;
  DISPOSE(READWRITE);

 END; {Procedure READTEMPWRITEORIG}


PROCEDURE WRITETEMP (BIG : INTEGER;  ACTIONS  : ALLCONTROLS);

 {This procedure writes a vertex and its response to a temporary file.}

 VAR

  I, J, K  : INTEGER;
```

```
BEGIN

 WRITELN(TSTOREVERTEXTEMP,BIG:10);
 FOR I : = 1 TO CURRENTNUMCHEMS DO
  FOR J : = 1 TO NUMYEARS DO
   BEGIN
    FOR K : = 1 TO NUMDAYSPERYEAR DO
     WRITE(TSTOREVERTEXTEMP,ACTIONS[I,J,K]:5);
    WRITELN(TSTOREVERTEXTEMP);
   END;
 WRITELN(TSTOREVERTEXTEMP,'ERROR DETECT SPACER');

END; {Procedure WRITETEMP}


 PROCEDURE SORT;

{This procedure sorts the vertex members of a simplex and arranges
them in descending order.}

VAR

 ENDER1, ENDER2, I, J, K, L, L2, M, SKIP,
 LOCATION, LINECOUNT, BIG, SOLUTION      : INTEGER;

BEGIN

 ENDER1 : = CURRENTNUMCHEMS*NUMYEARS + 1;
 ENDER2 : = CURRENTNUMCHEMS*NUMYEARS + 2;

 FOR L : = 1 TO (NUMVERTICES-1) DO
  BEGIN
   RESET(STOREVERTEX);
   FOR SKIP : = 0 TO (L-1) DO
    IF SKIP > 0 THEN
     FOR LINECOUNT : = 1 TO ENDER2 DO READLN(STOREVERTEX);
   NEW(VERTEXSORT);
    READLN(STOREVERTEX,VERTEXSORT@.RESULT);
    FOR I : = 1 TO CURRENTNUMCHEMS DO
     FOR J : = 1 TO NUMYEARS DO
      BEGIN
       FOR K : = 1 TO NUMDAYSPERYEAR DO
        READ(STOREVERTEX,VERTEXSORT@.ACTIONS[I,J,K]);
       READLN(STOREVERTEX);
      END;
    READLN(STOREVERTEX,READERROR);
    IF READERROR < > 'ERROR DETECT SPACER' THEN
     BEGIN
      WRITE('READ ERROR -- FILE STOREVERTEX, PROC SORT');
      HALT;
     END;
    BIG : = VERTEXSORT@.RESULT;
    LOCATION : = L;
    FOR L2 : = (L+1) TO NUMVERTICES DO
     BEGIN
      READLN(STOREVERTEX,VERTEXSORT@.RESULT);
      IF VERTEXSORT@.RESULT < BIG
       THEN
        BEGIN
         BIG : = VERTEXSORT@.RESULT;
         LOCATION : = L2;
         FOR I : = 1 TO CURRENTNUMCHEMS DO
          FOR J : = 1 TO NUMYEARS DO
           BEGIN
            FOR K : = 1 TO NUMDAYSPERYEAR DO
             READ(STOREVERTEX,VERTEXSORT@.ACTIONS[I,J,K]);
            READLN(STOREVERTEX);
           END;
         READLN(STOREVERTEX,READERROR);
         IF READERROR < > 'ERROR DETECT SPACER' THEN
          BEGIN
           WRITELN('READ ERROR -- SORT/SECOND');
```

```
        HALT;
       END;
      END
     ELSE
      FOR LINECOUNT : = 1 TO ENDER1 DO READLN(STOREVERTEX);
    END;

   RESET(STOREVERTEX);
   REWRITE(TSTOREVERTEXTEMP);

   IF L = 1
    THEN
     BEGIN
      WRITETEMP(BIG,VERTEXSORT@.ACTIONS);
      IF LOCATION = 1
       THEN
        BEGIN
         FOR LINECOUNT : = 1 TO ENDER2 DO READLN(STOREVERTEX);
         FOR M : = 2 TO NUMVERTICES DO READORIGWRITETEMP;
        END
       ELSE
        BEGIN
         FOR M : = 1 TO (LOCATION-1) DO READORIGWRITETEMP;
         FOR LINECOUNT : = 1 TO ENDER2 DO READLN(STOREVERTEX);
         FOR M : = (LOCATION+1) TO NUMVERTICES DO
           READORIGWRITETEMP;
        END;
      REWRITE(STOREVERTEX);
      RESET(TSTOREVERTEXTEMP);
      FOR M : = 1 TO NUMVERTICES DO READTEMPWRITEORIG;
     END
    ELSE
     BEGIN
      FOR M : = 1 TO (L-1) DO READORIGWRITETEMP;
      WRITETEMP(BIG,VERTEXSORT@.ACTIONS);
      IF LOCATION = L
       THEN
        BEGIN
         FOR LINECOUNT : = 1 TO ENDER2 DO READLN(STOREVERTEX);
         FOR M : = (L+1) TO NUMVERTICES DO READORIGWRITETEMP;
        END
       ELSE
        BEGIN
         FOR M : = L TO (LOCATION-1) DO READORIGWRITETEMP;
         FOR LINECOUNT : = 1 TO ENDER2 DO READLN(STOREVERTEX);
         FOR M : = (LOCATION+1) TO NUMVERTICES DO
           READORIGWRITETEMP;
        END;
       REWRITE(STOREVERTEX);
       RESET(TSTOREVERTEXTEMP);
       FOR M : = 1 TO NUMVERTICES DO READTEMPWRITEORIG;
     END;
  DISPOSE(VERTEXSORT);
 END; {FOR L}
END; {Procedure SORT}


*************************************************************************
      TABM AND ERM/BLB BIOLOGICAL SIMULATORS ARE STRUCTURES AS
      PROCEDURES AND PLACED HERE.
*************************************************************************


 PROCEDURE RESPONSE (VAR SYSTEMRESPONSE : INTEGER);

TYPE

 A1BYNUMYEARSOFREAL = @FIRST;
 FIRST       = RECORD
         ONEXYEARS : ARRAY[1..NUMYEARS] OF REAL;
```

```
        END;

 ANUMPESTICIDESBYNUMYEARSOFREAL = @SECOND;
 SECOND          = RECORD
              CHEMXYEARS : ARRAY[1..NUMPESTICIDES,
                1..NUMYEARS] OF REAL;
             END;

 ANUMPESTICIDESBYNUMYEARSOFINTEGER = @THIRD;
 THIRD           = RECORD
               CHEMXYEARS
                 : ARRAY[1..NUMPESTICIDES,
                 1..NUMYEARS] OF INTEGER;
              END;

VAR

 I, J, K, FINALYR  : INTEGER;

 SUMSPRAY : ANUMPESTICIDESBYNUMYEARSOFINTEGER;

 TOTCOST, CUMCHEMCOST  : A1BYNUMYEARSOFREAL;

 CHEMPRICE, YRCOST, POUNDS  : ANUMPESTICIDESBYNUMYEARSOFREAL;

 FQDIFF, PQDIFF, RESLASTERM, RESTABM, RESERM,
 SUSTABM, SUSERM, SUSLASTERM,
 TABMDAMAGE, ERMDAMAGE, PERCENTFDAMAGE, PERCENTPDAMAGE,
 NEWFQSUPPLY, NEWPQSUPPLY, DELTAFQ, DELTAPQ, FJ, PJ,
 LSUB, GSUB, HSUB, ISUB, FDELTACS, FDELTAPS, PDELTACS,
 PDELTAPS, PVFDELTACS, PVFDELTAPS, PVPDELTACS, PVPDELTAPS,
 PVTOTSURPLUS, PVCUMSURPLUS, NEWFP, NEWPP,
 NEWFQDEMAND, NEWPQDEMAND, MEASURE       : REAL;

BEGIN

 TABMSIMULATOR;
 ERMSIMULATOR;

 IF VERTEXNUMBER = 1 THEN MINIMUMYR : = TABMYRHOLD;
 FINALYR : = TABMYRHOLD;

{ IF ERMYRHOLD < TABMYRHOLD
  THEN FINALYR : = TABMYRHOLD
  ELSE FINALYR : = ERMYRHOLD;
 }
 NEW(SUMSPRAY);
 NEW(TOTCOST);
 NEW(CUMCHEMCOST);
 NEW(CHEMPRICE);
 NEW(YRCOST);
 NEW(POUNDS);

 CHEMNUMBER : = 0;
 FOR I : = 1 TO NUMPESTICIDES DO
  IF I IN CURRENTCHEMS THEN
   BEGIN
    CHEMNUMBER : = CHEMNUMBER + 1;
    FOR J : = 1 TO FINALYR DO
     BEGIN
      SUMSPRAY@.CHEMXYEARS[CHEMNUMBER,J] : = 0;
      TOTCOST@.ONEXYEARS[J] : = 0.0;
      CUMCHEMCOST@.ONEXYEARS[J] : = 0.0;
      CHEMPRICE@.CHEMXYEARS[CHEMNUMBER,J] :
        INITIALPESTICIDEPRICE[I]*EXP(J*LN(1+INFLATION));
     END;
    FOR J : = 1 TO FINALYR DO
     BEGIN

      SUMSPRAY@.CHEMXYEARS[CHEMNUMBER,J] :
        SUMSPRAY@.CHEMXYEARS[CHEMNUMBER,J]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,1]
```

```
        + VERTEX@.ACTIONS[CHEMNUMBER,J,2]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,3]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,4]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,5]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,6]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,7]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,8]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,9]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,10]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,11]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,12]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,13]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,14]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,15]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,16]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,17]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,18]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,19]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,20]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,21]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,22]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,23]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,24]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,25]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,26]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,27]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,28]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,29]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,30]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,31]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,32]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,33]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,34]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,35]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,36]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,37]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,38]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,39]
        + VERTEX@.ACTIONS[CHEMNUMBER,J,40];
    YRCOST@.CHEMXYEARS[CHEMNUMBER,J] : = 82867.0*
        CHEMPRICE@.CHEMXYEARS[CHEMNUMBER,J]
        *SUMSPRAY@.CHEMXYEARS[CHEMNUMBER,J]
        /EXP(J*LN(1+DISCOUNT))/1000000.0;
    POUNDS@.CHEMXYEARS[CHEMNUMBER,J] : =
        82867.0*POUNDSPERPPM[I]
        *SUMSPRAY@.CHEMXYEARS[CHEMNUMBER,J]/1000.0;
   END;
  END;
 FOR J : = 1 TO FINALYR DO
  BEGIN
   WRITE(CHEMCOST,VERTEXNUMBER:4,J:8,'  ');
   WRITE(CHEMUSE,VERTEXNUMBER:4,J:8,'  ');
   CHEMNUMBER : = 0;
   FOR I : = 1 TO NUMPESTICIDES DO
    IF I IN CURRENTCHEMS
     THEN
      BEGIN
       CHEMNUMBER : = CHEMNUMBER + 1;
       WRITE(CHEMCOST,YRCOST@.CHEMXYEARS[CHEMNUMBER,J]:9:4);
       WRITE(CHEMUSE,POUNDS@.CHEMXYEARS[CHEMNUMBER,J]:9:4);
      END
     ELSE
      BEGIN
       WRITE(CHEMCOST,' ':9);
       WRITE(CHEMUSE,' ':9);
      END;
   WRITELN(CHEMCOST);
   WRITELN(CHEMUSE);
  END;
FOR J : = 1 TO FINALYR DO
 BEGIN
  FOR I : = 1 TO CURRENTNUMCHEMS DO
```

```
  TOTCOST@.ONEXYEARS[J] : = TOTCOST@.ONEXYEARS[J]
     + YRCOST@.CHEMXYEARS[I,J];
  IF J = 1
   THEN CUMCHEMCOST@.ONEXYEARS[J] : = TOTCOST@.ONEXYEARS[J]
   ELSE CUMCHEMCOST@.ONEXYEARS[J] : = CUMCHEMCOST@.ONEXYEARS[J-1]
     + TOTCOST@.ONEXYEARS[J];
 END;

FQDIFF : = INITIALFQSUPPLY - INITIALFQDEMAND;
PQDIFF : = INITIALPQDEMAND - INITIALPQSUPPLY;
RESET(RTABMTOTS);
RESET(RERMTOTS);
RESET(STABMTOTS);
RESET(SERMTOTS);
RESLASTERM : = 0.0;
SUSLASTERM : = 0.0;
  PVCUMSURPLUS : = 0.0;
  MEASURE      : = 0.0;

FOR J : = 1 TO FINALYR DO
 BEGIN
  READLN(RTABMTOTS,RESTABM);
  READLN(RERMTOTS,RESERM);
  READLN(STABMTOTS,SUSTABM);
  READLN(SERMTOTS,SUSERM);

  TABMDAMAGE : = (1.0 - EXP(-1.0*EXP(0.9*LN(RESTABM/80000.0/1.1))))
          - (1.0 - EXP(-1.0*EXP(0.9*LN(SUSTABM/80000.0/1.1))));
  ERMDAMAGE : = ((0.0264*RESERM + 0.0139*RESLASTERM)/139.476)
          - (0.0264*SUSERM + 0.0139*SUSLASTERM)/139.476;
  IF TABMDAMAGE < 0.0 THEN TABMDAMAGE : = 0.0;
  IF ERMDAMAGE < 0.0 THEN ERMDAMAGE : = 0.0;
  PERCENTFDAMAGE : = TABMDAMAGE + ERMDAMAGE;
  PERCENTPDAMAGE : = 0.16*TABMDAMAGE + ERMDAMAGE;
  IF PERCENTFDAMAGE < 0.0 THEN PERCENTFDAMAGE : = 0.0;
  IF PERCENTPDAMAGE < 0.0 THEN PERCENTPDAMAGE : = 0.0;
  IF PERCENTFDAMAGE > 1.0 THEN PERCENTFDAMAGE : = 1.0;
  IF PERCENTPDAMAGE > 1.0 THEN PERCENTPDAMAGE : = 1.0;
  RESLASTERM : = RESERM;
  SUSLASTERM : = SUSERM;
  NEWFQSUPPLY : = INITIALFQSUPPLY*(1.0 - 0.113*PERCENTFDAMAGE);
  NEWPQSUPPLY : = INITIALPQSUPPLY*(1.0 - 0.263*PERCENTPDAMAGE)
    + 0.84*0.113*INITIALFQSUPPLY*PERCENTFDAMAGE;
  DELTAFQ : = NEWFQSUPPLY - INITIALFQSUPPLY;
  DELTAPQ : = NEWPQSUPPLY - INITIALPQSUPPLY;

  FJ : = ABS((DELTAFQ*(FSUPPLYELAS+FDEMANDELAS))
    /(INITIALFQSUPPLY*FDEMANDELAS));
  PJ : = ABS((DELTAPQ*(PSUPPLYELAS+PDEMANDELAS))
    /(INITIALPQSUPPLY*PDEMANDELAS));
  LSUB : = (INITIALFQSUPPLY*FJ)/(FEXCESSSUPPLY*FQDIFF);
  GSUB : = (LSUB*FEXCESSSUPPLY)/(FEXCESSSUPPLY+FEXCESSDEMAND);
  HSUB : = (INITIALPQSUPPLY*PJ)/(PEXCESSDEMAND*PQDIFF);
  ISUB : = (HSUB*PEXCESSSUPPLY)/(PEXCESSSUPPLY+PEXCESSDEMAND);

  IF DELTAFQ > 0.0
   THEN
    BEGIN
     FDELTACS : = INITIALFP*INITIALFQDEMAND*GSUB
       *(1.0+0.5*GSUB*FDEMANDELAS);
     FDELTAPS : = (((INITIALFP*INITIALFQSUPPLY*FJ/FSUPPLYELAS)
       -(INITIALFP*INITIALFQSUPPLY*GSUB))*(1.0+0.5*FJ))
       +0.5*INITIALFP*INITIALFQSUPPLY*FSUPPLYELAS*GSUB
       *(GSUB-(FJ/FSUPPLYELAS));
    END
   ELSE
    IF DELTAFQ < 0.0
     THEN
      BEGIN
       FDELTACS : = -1.0*INITIALFP*INITIALFQDEMAND*GSUB
         *(1.0-0.5*FDEMANDELAS*GSUB);
       FDELTAPS : = (((INITIALFP*INITIALFQSUPPLY*GSUB)
```

```
      -(INITIALFP*INITIALFQSUPPLY*FJ/FSUPPLYELAS))
      *(1.0+0.5*FJ))+0.5*INITIALFP*INITIALFQSUPPLY
      *FSUPPLYELAS*GSUB*(GSUB+(FJ/FSUPPLYELAS));
   END
   ELSE
    BEGIN
     FDELTACS : = 0.0;
     FDELTAPS : = 0.0;
    END;
IF DELTAPQ > 0.0
 THEN
  BEGIN
   PDELTACS : = INITIALPP*INITIALPQDEMAND*ISUB
     *(1.0+0.5*ISUB*PDEMANDELAS);
   PDELTAPS : = (((INITIALPP*INITIALPQSUPPLY*PJ/PSUPPLYELAS)
     -(INITIALPP*INITIALPQSUPPLY*ISUB))*(1.0+0.5*PJ))
     +0.5*INITIALPP*INITIALPQSUPPLY*PSUPPLYELAS*ISUB
     *(ISUB-(PJ/PSUPPLYELAS));
  END
 ELSE
  IF DELTAPQ < 0.0
   THEN
    BEGIN
     PDELTACS : = -1.0*INITIALPP*INITIALPQDEMAND*ISUB
       *(1.0-0.5*PDEMANDELAS*ISUB);
     PDELTAPS : = (((INITIALPP*INITIALPQSUPPLY*ISUB)
       -(INITIALPP*INITIALPQSUPPLY*PJ/PSUPPLYELAS))
       *(1.0+0.5*PJ))+0.5*INITIALPP*INITIALPQSUPPLY
       *PSUPPLYELAS*ISUB*(ISUB+(PJ/PSUPPLYELAS));
    END
   ELSE
    BEGIN
     PDELTACS : = 0.0;
     PDELTAPS : = 0.0;
    END;

PVFDELTACS : = FDELTACS/100.0/EXP(J*LN(1+DISCOUNT));
PVFDELTAPS : = FDELTAPS/100.0/EXP(J*LN(1+DISCOUNT));
PVPDELTACS : = PDELTACS/100.0/EXP(J*LN(1+DISCOUNT));
PVPDELTAPS : = PDELTAPS/100.0/EXP(J*LN(1+DISCOUNT));
   PVTOTSURPLUS : = PVFDELTACS+PVFDELTAPS+PVPDELTACS+PVPDELTAPS;
   PVCUMSURPLUS : = PVCUMSURPLUS+PVTOTSURPLUS;
   MEASURE : = MEASURE + (14361/EXP(J*LN(1+DISCOUNT)))
           + PVTOTSURPLUS;

   IF DELTAFQ < = 0.0
 THEN
  BEGIN
   NEWFQDEMAND : = INITIALFQDEMAND*(1.0-GSUB*FDEMANDELAS);
   NEWFP : = INITIALFP + (INITIALFP*LSUB*FEXCESSSUPPLY)
     /(FEXCESSSUPPLY+FEXCESSDEMAND);
  END
 ELSE
  BEGIN
   NEWFQDEMAND : = INITIALFQDEMAND*(1.0+GSUB*FDEMANDELAS);
   NEWFP : = INITIALFP - (INITIALFP*LSUB*FEXCESSSUPPLY)
     /(FEXCESSSUPPLY+FEXCESSDEMAND);
  END;
IF DELTAPQ > = 0.0
 THEN
  BEGIN
   NEWPQDEMAND : = INITIALPQDEMAND*(1.0+ISUB*PDEMANDELAS);
   NEWPP : = INITIALPP - (INITIALPP*HSUB*PEXCESSSUPPLY)
     /(PEXCESSSUPPLY+PEXCESSDEMAND);
  END
 ELSE
  BEGIN
   NEWPQDEMAND : = INITIALPQDEMAND*(1.0-ISUB*PDEMANDELAS);
   NEWPP : = INITIALPP + (INITIALPP*HSUB*PEXCESSSUPPLY)
     /(PEXCESSSUPPLY+PEXCESSDEMAND);
  END;
```

```
  WRITE(PANDQ,VERTEXNUMBER:4,J:7);
  WRITE(PANDQ,NEWFP:8:2,NEWFQSUPPLY:9:1,NEWFQDEMAND:9:1);
  WRITE(PANDQ,NEWPP:9:2,NEWPQSUPPLY:9:1);
  WRITELN(PANDQ,NEWPQDEMAND:9:1);

  WRITE(SURPLUS,VERTEXNUMBER:4,J:7);
  WRITE(SURPLUS,PVFDELTACS:10:3,PVFDELTAPS:9:3);
  WRITE(SURPLUS,PVPDELTACS:9:3,PVPDELTAPS:9:3);
  WRITE(SURPLUS,PVTOTSURPLUS:9:3,PVCUMSURPLUS:12:3);
  WRITELN(SURPLUS,CUMCHEMCOST@.ONEXYEARS[J]:11:3,' ',MEASURE:15:5);

 END; {FOR FINALYR}

SYSTEMRESPONSE : = ROUND(1000*MEASURE);

WRITELN(PANDQ);
WRITELN(SURPLUS);
WRITELN(FIRSTPOP);
WRITELN(FIRSTERM);
WRITELN(FIRSTBLB);
WRITELN(RESIST);
WRITELN(RESISTER);
WRITELN(RESISTBL);
WRITELN(POPS);
WRITELN(POPSERM);
WRITELN(POPSBLB);
WRITELN(CDYEAR);
WRITELN(CDDAY);
WRITELN(CHEMUSE);
WRITELN(CHEMCOST);

DISPOSE(TOTCOST);
DISPOSE(SUMSPRAY);
DISPOSE(YRCOST);
DISPOSE(CHEMPRICE);

END; {Procedure RESPONSE}


PROCEDURE RETRACTION (VAR NEWSOLUTION : INTEGER; EJECT : BOOLEAN);

{This procedure provides a method by which a newly projected vertex
can be moved back towards its original position if it fails to
improve the system response relative to the next best vertex. The
retraction occurs progressively by half-distances, and is terminated
when the projected vertex is within a 7 percent neighborhood of its
originating vertex or when a desirable vertex is found.}

VAR

I, J, K, POWER, DIVISOR : INTEGER;

BEGIN

EJECT : = FALSE;
POWER : = 0;

REPEAT

 POWER : = POWER + 1;
 DIVISOR : = ROUND(EXP(POWER*LN(2)));
 IF DIVISOR > 2 THEN
  BEGIN
   EJECT : = TRUE;
   WRITELN('*** PROCESSING EJECTED FROM RETRACTION ***');
  END;

 CHEMNUMBER : = 0;
 FOR I : = 1 TO NUMPESTICIDES DO
  IF I IN CURRENTCHEMS THEN
   BEGIN
    CHEMNUMBER : = CHEMNUMBER + 1;
```

```
    FOR J : = 1 TO NUMYEARS DO
     FOR K : = 1 TO NUMDAYSPERYEAR DO
      VERTEX@.ACTIONS[CHEMNUMBER,J,K] : =
        STOREDWORST@.FILESTOREACTIONS[CHEMNUMBER,J,K]
        + ROUND(REFLECTCOEFF*
        (POINTER@.CONTROLARRAY[CHEMNUMBER,J,K]
        /(DELTAX*PESTICIDEUPPERBOUND[I]))*(DELTAX
        *PESTICIDEUPPERBOUND[I])) DIV DIVISOR;
    END;
  VERTEXNUMBER : = VERTEXNUMBER + 0;
  CONSTRAINTS;
  RESPONSE(NEWSOLUTION);

 UNTIL ((NEWSOLUTION > STOREDWORST@.FILESTORERESULT) OR (EJECT));

 IF EJECT THEN
  BEGIN
   VERTEX@.ACTIONS : = STOREDWORST@.FILESTOREACTIONS;
   NEWSOLUTION : = STOREDWORST@.FILESTORERESULT;
  END;

END; {Procedure RETRACTION}


PROCEDURE OPENFILES;

 BEGIN

 REWRITE(FIRSTPOP);
 WRITELN(FIRSTPOP,'WITHIN YEAR POPULATION DYNAMICS');
 WRITELN(FIRSTPOP);

 REWRITE(RESIST);
 WRITELN(RESIST,'YEAR END RESISTANCE LEVELS');
 WRITELN(RESIST);

 REWRITE(POPS);
 WRITELN(POPS,'YEAR END POPULATION LEVELS');
 WRITELN(POPS);

 REWRITE(FIRSTERM);
 WRITELN(FIRSTERM,'WITHIN YEAR POPULATION DYNAMICS');
 WRITELN(FIRSTERM);

 REWRITE(RESISTER);
 WRITELN(RESISTER,'YEAR END RESISTANCE LEVELS');
 WRITELN(RESISTER);

 REWRITE(POPSERM);
 WRITELN(POPSERM,'YEAR END POPULATION LEVELS');
 WRITELN(POPSERM);

 REWRITE(FIRSTBLB);
 WRITELN(FIRSTBLB,'WITHIN YEAR POPULATION DYNAMICS');
 WRITELN(FIRSTBLB);

 REWRITE(RESISTBL);
 WRITELN(RESISTBL,'YEAR END RESISTANCE LEVELS');
 WRITELN(RESISTBL);

 REWRITE(POPSBLB);
 WRITELN(POPSBLB,'YEAR END POPULATION LEVELS');
 WRITELN(POPSBLB);

 REWRITE(CHEMUSE);
 WRITELN(CHEMUSE,'TOTAL YEARLY CHEMICAL USE IN 1,000 POUNDS');
 WRITELN(CHEMUSE);
 WRITE(CHEMUSE,'-----------------------------------------------------');
 WRITELN(CHEMUSE,'----------');
 WRITELN(CHEMUSE,'VERTEX  SIM. ');
 WRITE(CHEMUSE,'NUMBER  YEAR  GUTHION LORSBAN IMIDAN ');
 WRITELN(CHEMUSE,'PENNCAP-M  LANNATE  ASANA');
```

```
WRITE(CHEMUSE,'-------------------------------------------------');
WRITELN(CHEMUSE,'----------');

REWRITE(CHEMCOST);
WRITE(CHEMCOST,'TOTAL YEARLY CHEMICAL COST IN MILLION DOLLARS');
WRITELN(CHEMCOST,' PRESENT VALUE');
WRITELN(CHEMCOST);
WRITE(CHEMCOST,'-------------------------------------------------');
WRITELN(CHEMCOST,'----------');
WRITELN(CHEMCOST,'VERTEX  SIM. ');
WRITE(CHEMCOST,'NUMBER  YEAR  GUTHION LORSBAN IMIDAN ');
WRITELN(CHEMCOST,'PENNCAP-M  LANNATE  ASANA');
WRITE(CHEMCOST,'-------------------------------------------------');
WRITELN(CHEMCOST,'----------');

REWRITE(CDDAY);
WRITELN(CDDAY,'CUMULATIVE DAILY CHEMICAL LOADINGS IN PPM');
WRITELN(CDDAY);
WRITE(CDDAY,'-------------------------------------------------');
WRITELN(CDDAY,'----------');
WRITELN(CDDAY,'VERTEX  SIM. ');
WRITE(CDDAY,'NUMBER  DAY  GUTHION LORSBAN IMIDAN ');
WRITELN(CDDAY,'PENNCAP-M  LANNATE  ASANA');
WRITE(CDDAY,'-------------------------------------------------');
WRITELN(CDDAY,'----------');

REWRITE(CDYEAR);
WRITELN(CDYEAR,'YEAR-END CHEMICAL LOADINGS IN PPM');
WRITELN(CDYEAR);
WRITE(CDYEAR,'-------------------------------------------------');
WRITELN(CDYEAR,'----------');
WRITELN(CDYEAR,'VERTEX  SIM. ');
WRITE(CDYEAR,'NUMBER  YEAR  GUTHION LORSBAN IMIDAN ');
WRITELN(CDYEAR,'PENNCAP-M  LANNATE  ASANA');
WRITE(CDYEAR,'-------------------------------------------------');
WRITELN(CDYEAR,'----------');

REWRITE(PANDQ);
WRITELN(PANDQ,'YEAR END PRICE AND QUANTITY LEVELS':52);
WRITELN(PANDQ);
WRITELN(PANDQ);
WRITE(PANDQ,'-------------------------------------------------');
WRITELN(PANDQ,'----------');
WRITE(PANDQ,'FRESH APPLES':31);
WRITELN(PANDQ,'PROCESSED APPLES':29);
WRITELN(PANDQ);
WRITE(PANDQ,'VERTEX SIM. PRICE  SUPPLY  DEMAND  PRICE ');
WRITELN(PANDQ,'SUPPLY  DEMAND');
WRITE(PANDQ,'NUMBER YEAR (C/LB) (M LB)  (M LB)  (C/LB) ');
WRITELN(PANDQ,'(M LB)  (M LB)');
WRITE(PANDQ,'-------------------------------------------------');
WRITELN(PANDQ,'----------');
WRITE(PANDQ,'0':4,'0':7,INITIALFP:8:2);
WRITE(PANDQ,INITIALFQSUPPLY:9:1,INITIALFQDEMAND:9:1);
WRITE(PANDQ,INITIALPP:9:2,INITIALPQSUPPLY:9:1);
WRITELN(PANDQ,INITIALPQDEMAND:9:1);
WRITELN(PANDQ);

REWRITE(SURPLUS);
WRITELN(SURPLUS,'YEAR END ECONOMIC MEASURES IN PRESENT VALUES':62);
WRITELN(SURPLUS);
WRITELN(SURPLUS);
WRITE(SURPLUS,'-------------------------------------------');
WRITELN(SURPLUS,'-------------------------------------------');
WRITE(SURPLUS,'FRESH APPLES':28,'PROCESSED APPLES':20);
WRITELN(SURPLUS,'ALL APPLES':16,'CUMULATIVE':16);
WRITELN(SURPLUS,'VERTEX SIM.');
WRITE(SURPLUS,'NUMBER YEAR DELTACS DELTAPS DELTACS DELTAPS ');
WRITELN(SURPLUS,'DELTATS  CUMDELTATS','CHEM COST':11);
WRITE(SURPLUS,'-------------------------------------------------');
WRITELN(SURPLUS,'------------------------------');
```

```
WRITELN(FIRSTPOP,'TUFTED APPLE BUD MOTH');
WRITELN(FIRSTPOP);
WRITE(FIRSTPOP,'-----------------------------------------------');
WRITELN(FIRSTPOP,'----------------------');
WRITELN(FIRSTPOP,'VERTEX':6,'SIM.':7);
WRITE(FIRSTPOP,'NUMBER':6,'DAY':7,'EGGS':11,'LARVAE':11,'PUPAE':11);
WRITELN(FIRSTPOP,'ADULTS':11,'TOTAL':11);
WRITE(FIRSTPOP,'-----------------------------------------------');
WRITELN(FIRSTPOP,'----------------------');

WRITELN(RESIST,'TUFTED APPLE BUD MOTH');
WRITELN(RESIST);
WRITE(RESIST,'-------------------------------------------------');
WRITELN(RESIST,'----------------------');
WRITE(RESIST,'VERTEX  SIM.          RESISTANCE LEVELS ');
WRITELN(RESIST,'TO CHEMICAL:');
WRITE(RESIST,'NUMBER  YEAR  GUTHION LORSBAN IMIDAN ');
WRITELN(RESIST,'PENNCAP-M  LANNATE  ASANA');
WRITE(RESIST,'-------------------------------------------------');
WRITELN(RESIST,'----------------------');

WRITELN(POPS,'TUFTED APPLE BUD MOTH');
WRITELN(POPS);
WRITE(POPS,'-----------------------------------------------');
WRITELN(POPS,'--------------------------------');
WRITELN(POPS,'VERTEX':6,'SIM.':7);
WRITE(POPS,'NUMBER':6,'YEAR':7,'EGGS':11,'LARVAE':11,'PUPAE':11);
WRITELN(POPS,'ADULTS':11,'TOTAL':11,'MAXT':11);
WRITE(POPS,'-----------------------------------------------');
WRITELN(POPS,'--------------------------------');

WRITELN(FIRSTERM,'EUROPEAN RED MITE');
WRITELN(FIRSTERM);
WRITE(FIRSTERM,'-----------------------------------------------');
WRITELN(FIRSTERM,'----------------------');
WRITELN(FIRSTERM,'VERTEX':6,'SIM.':7);
WRITE(FIRSTERM,'NUMBER':6,'DAY':7,'EGGS':11,'LARVAE':11,'PUPAE':11);
WRITELN(FIRSTERM,'ADULTS':11,'TOTAL':11);
WRITE(FIRSTERM,'-----------------------------------------------');
WRITELN(FIRSTERM,'----------------------');

WRITELN(RESISTER,'EUROPEAN RED MITE');
WRITELN(RESISTER);
WRITE(RESISTER,'-------------------------------------------------');
WRITELN(RESISTER,'----------------------');
WRITE(RESISTER,'VERTEX  SIM.          RESISTANCE LEVELS ');
WRITELN(RESISTER,'TO CHEMICAL:');
WRITE(RESISTER,'NUMBER  YEAR  GUTHION LORSBAN IMIDAN ');
WRITELN(RESISTER,'PENNCAP-M  LANNATE  ASANA');
WRITE(RESISTER,'-------------------------------------------------');
WRITELN(RESISTER,'----------------------');

WRITELN(POPSERM,'EUROPEAN RED MITE');
WRITELN(POPSERM);
WRITE(POPSERM,'-----------------------------------------------');
WRITELN(POPSERM,'--------------------------------');
WRITELN(POPSERM,'VERTEX':6,'SIM.':7);
WRITE(POPSERM,'NUMBER':6,'YEAR':7,'EGGS':11,'LARVAE':11,'PUPAE':11);
WRITELN(POPSERM,'ADULTS':11,'TOTAL':11,'CMD':11);
WRITE(POPSERM,'-----------------------------------------------');
WRITELN(POPSERM,'--------------------------------');

WRITELN(FIRSTBLB,'BLACK LADYBIRD BEETLE');
WRITELN(FIRSTBLB);
WRITE(FIRSTBLB,'-----------------------------------------------');
WRITELN(FIRSTBLB,'----------------------');
WRITELN(FIRSTBLB,'VERTEX':6,'SIM.':7);
WRITE(FIRSTBLB,'NUMBER':6,'DAY':7,'EGGS':11,'LARVAE':11,'PUPAE':11);
WRITELN(FIRSTBLB,'ADULTS':11,'TOTAL':11);
WRITE(FIRSTBLB,'-----------------------------------------------');
WRITELN(FIRSTBLB,'----------------------');
```

```
WRITELN(RESISTBL,'BLACK LADYBIRD BEETLE');
WRITELN(RESISTBL);
WRITE(RESISTBL,'-------------------------------------------------');
WRITELN(RESISTBL,'----------');
WRITE(RESISTBL,'VERTEX  SIM.          RESISTANCE LEVELS ');
WRITELN(RESISTBL,'TO CHEMICAL:');
WRITE(RESISTBL,'NUMBER  YEAR  GUTHION LORSBAN IMIDAN ');
WRITELN(RESISTBL,'PENNCAP-M  LANNATE  ASANA');
WRITE(RESISTBL,'-------------------------------------------------');
WRITELN(RESISTBL,'----------');

WRITELN(POPSBLB,'BLACK LADYBIRD BEETLE');
WRITELN(POPSBLB);
WRITE(POPSBLB,'-----------------------------------------------');
WRITELN(POPSBLB,'----------');
WRITELN(POPSBLB,'VERTEX':6,'SIM.':7);
WRITE(POPSBLB,'NUMBER':6,'YEAR':7,'EGGS':11,'LARVAE':11,'PUPAE':11);
WRITELN(POPSBLB,'ADULTS':11,'TOTAL':11);
WRITE(POPSBLB,'-----------------------------------------------');
WRITELN(POPSBLB,'----------');

REWRITE(TSTOREVERTEXTEMP);

END;


BEGIN {Program CORE}

{The program is initialized by identifying the chemicals in the current
control technology set and the bound/price characteristics of all the
chemicals that might be used. In addition, input and output files are
defined and initialized as appropriate.}

{For CURRENTCHEMS,
      0 = no chemical used
      1 = Guthion (azinphosmethyl)
      2 = Lorsban (Chlorpyrifos)
      3 = Imidan (phosmet)
      4 = PennCap-M (microencapsulated methylparathion)
      5 = Lannate (methomyl)
      6 = Asana (esfenvalerate)

PESTICIDEUPPERBOUNDS are measured as parts-per-million, and are
approximately one-third higher than the recommendation that were
current at the time of the study. This was done to allow for the
possibility that recommendation were too low to be optimal.

DAYSTOHARVEST and DAYSBETWEENSPRAY were taken from recommended and
labelled restrictions. Note that, within the context of the
simulation model, 1 simulation day is equivalent to 4 real days.

INITIALPESTICIDEPRICE is given in $/ppm and reflects an average of
Spring 1991 prices.}

MINIMUMYR : = 1;
STARTER : = RANDOM(673);

CURRENTCHEMS : = [1,2,4,5,6];

PESTICIDEUPPERBOUND[1] : = 400;  PESTICIDEUPPERBOUND[2] : = 800;
PESTICIDEUPPERBOUND[3] : = 800;  PESTICIDEUPPERBOUND[4] : = 200;
PESTICIDEUPPERBOUND[5] : = 360;  PESTICIDEUPPERBOUND[6] : = 20;

INITIALPESTICIDEPRICE[1] : = 0.00571;
INITIALPESTICIDEPRICE[2] : = 0.00162;
INITIALPESTICIDEPRICE[3] : = 0.00279;
INITIALPESTICIDEPRICE[4] : = 0.02628;
INITIALPESTICIDEPRICE[5] : = 0.01803;
INITIALPESTICIDEPRICE[6] : = 0.21771;

DAYSBETWEENSPRAY[1] : = 1;
DAYSBETWEENSPRAY[2] : = 1;
```

```
DAYSBETWEENSPRAY[3] : = 1;
DAYSBETWEENSPRAY[4] : = 1;
DAYSBETWEENSPRAY[5] : = 1;
DAYSBETWEENSPRAY[6] : = 1;

DAYSTOHARVEST[1]  : = 2;
DAYSTOHARVEST[2]  : = 7;
DAYSTOHARVEST[3]  : = 2;
DAYSTOHARVEST[4]  : = 4;
DAYSTOHARVEST[5]  : = 2;
DAYSTOHARVEST[6]  : = 6;

POUNDSPERPPM[1] : = 0.000833;
POUNDSPERPPM[2] : = 0.000833;
POUNDSPERPPM[3] : = 0.000833;
POUNDSPERPPM[4] : = 0.001667;
POUNDSPERPPM[5] : = 0.000833;
POUNDSPERPPM[6] : = 0.000833;

OPENFILES;

{Determines the number of chemicals in the current control technology
set.  This value is used to limit the number of iterations from the
full set levels.}

CURRENTNUMCHEMS : = 0;
FOR I : = 1 TO NUMPESTICIDES DO
 IF I IN CURRENTCHEMS THEN CURRENTNUMCHEMS : = CURRENTNUMCHEMS + 1;

{Calculates the appropriate number of lines to be used in formatting
output to files.}

LEFTOVER : = (NUMYEARS*NUMDAYSPERYEAR*CURRENTNUMCHEMS) MOD 16;
IF LEFTOVER < > 0
 THEN LINES : = TRUNC((NUMYEARS*NUMDAYSPERYEAR*CURRENTNUMCHEMS)
      / 16) + 3
 ELSE LINES : = TRUNC((NUMYEARS*NUMDAYSPERYEAR*CURRENTNUMCHEMS)
      / 16) + 2;

{Before launching into the main part of the program, a few more
initializations are needed.  SUMACTIONS will be used for intermediate
calculations of projection centroids, while VERTEXNUMBER is a counter
used to associated output contained in different files.}

VERTEXNUMBER : = 0;

IF CURRENTNUMCHEMS < > 0 THEN
 BEGIN
  REWRITE(SUMSAVE);
  NEW(SUMACROSS);
  WITH SUMACROSS@ DO
   BEGIN
    FOR I : = 1 TO CURRENTNUMCHEMS DO
     FOR J : = 1 TO NUMYEARS DO
      BEGIN
       FOR K : = 1 TO NUMDAYSPERYEAR DO
        BEGIN
         SUMACTIONS[I,J,K] : = 0;
         WRITE(SUMSAVE,SUMACTIONS[I,J,K]:8);
        END;
       WRITELN(SUMSAVE);
      END;
   END;
  DISPOSE(SUMACROSS);
 END;

{The program now enters into a major section in which an initial
simplex vertex is generated (GENERATE) and its system response
evaluated (RESPONSE).  As they are developed, the vertices and their
value are written to the storage file STOREVERTEX. This process is
repeated until an entire initial simplex is formed.  SUMACTIONS, a
value that is needed to calculate a centroid for projection purposes,
```

```
is calculated along the way.}

REWRITE(STOREVERTEX);
FOR N : = 1 TO NUMVERTICES DO
 BEGIN
  NEW(VERTEX);
  WITH VERTEX@ DO
   BEGIN
    VERTEXNUMBER : = VERTEXNUMBER + 1;
    GENERATE;
    CONSTRAINTS;
    RESPONSE(RESULT);
    WRITELN(STOREVERTEX,RESULT:10);
    RESET(SUMSAVE);
    NEW(SUMACROSS);
    FOR I : = 1 TO CURRENTNUMCHEMS DO
     FOR J : = 1 TO NUMYEARS DO
      BEGIN
       FOR K : = 1 TO NUMDAYSPERYEAR DO
        BEGIN
         WRITE(STOREVERTEX,ACTIONS[I,J,K]:5);
         READ(SUMSAVE,SUMACROSS@.SUMACTIONS[I,J,K]);
         SUMACROSS@.SUMACTIONS[I,J,K] : = SUMACROSS@.SUMACTIONS[I,J,K]
           +ACTIONS[I,J,K];
        END;
       WRITELN(STOREVERTEX);
       READLN(SUMSAVE);
      END;
    WRITELN(STOREVERTEX,'ERROR DETECT SPACER');
    REWRITE(SUMSAVE);
    FOR I : = 1 TO CURRENTNUMCHEMS DO
     FOR J : = 1 TO NUMYEARS DO
      BEGIN
       FOR K : = 1 TO NUMDAYSPERYEAR DO
        WRITE(SUMSAVE,SUMACROSS@.SUMACTIONS[I,J,K]:8);
        WRITELN(SUMSAVE);
      END;
    DISPOSE(SUMACROSS);
   END; {WITH VERTEX}
  DISPOSE(VERTEX);
 END; {FOR N}

{After sorting the vertices in the initial simplex into descending
order, the program then entires a section where attempts are made to
find superior vertices based on the information contained in the
initial simplex and projections through input space. This process
continues until no better vertex, and by extension no better simplex,
can be found. For more detail on how this process works, see Lee or
Kazmierczak.}

SORT;

REPEAT

 WORSTVERTEX : = 1;
 RESET(STOREVERTEX);
 NEW(STOREDWORST);
 NEW(VERTEX);
 SKIP2 : = 0;

 REPEAT

  SKIP2 : = SKIP2 + 1;
  NEW(CENTROID);
  NEW(POINTER);
  NEW(SUMACROSS);
  RESET(SUMSAVE);
  READLN(STOREVERTEX,STOREDWORST@.FILESTORERESULT);
  FOR I : = 1 TO CURRENTNUMCHEMS DO
   FOR J : = 1 TO NUMYEARS DO
    BEGIN
     FOR K : = 1 TO NUMDAYSPERYEAR DO
```

```
    BEGIN
     READ(SUMSAVE,SUMACROSS@.SUMACTIONS[I,J,K]);
    READ(STOREVERTEX,STOREDWORST@.FILESTOREACTIONS[I,J,K]);
     CENTROID@.CONTROLARRAY[I,J,K] : =
       (SUMACROSS@.SUMACTIONS[I,J,K] -
       STOREDWORST@.FILESTOREACTIONS[I,J,K]) DIV
       (NUMVERTICES - 1);
     POINTER@.CONTROLARRAY[I,J,K] : =
       CENTROID@.CONTROLARRAY[I,J,K] -
       STOREDWORST@.FILESTOREACTIONS[I,J,K];
     VERTEX@.ACTIONS[I,J,K] : =
       STOREDWORST@.FILESTOREACTIONS[I,J,K] +
       ROUND(REFLECTCOEFF*POINTER@.CONTROLARRAY[I,J,K]/
       DELTAX)*DELTAX;
    END;
   READLN(STOREVERTEX);
   READLN(SUMSAVE);
  END;
 DISPOSE(SUMACROSS);
 DISPOSE(CENTROID);
 READLN(STOREVERTEX,READERROR);
 IF READERROR < > 'ERROR DETECT SPACER' THEN
  BEGIN
   WRITE('READ ERROR -- FILE STOREVERTEX, SUPERIOR MOVE');
   HALT;
  END;
 CONSTRAINTS;
 VERTEXNUMBER : = VERTEXNUMBER + 1;
 RESPONSE(VERTEX@.RESULT);
 IF (VERTEX@.RESULT < STOREDWORST@.FILESTORERESULT)
    AND (VERTEXNUMBER < 15) THEN
    RETRACTION(VERTEX@.RESULT,EJECT);
 IF (VERTEX@.RESULT < STOREDWORST@.FILESTORERESULT) OR
    (NOBETTER = 1) OR (EJECT) THEN
  BEGIN
   WORSTVERTEX : = WORSTVERTEX + 1;
   NOBETTER : = 0;
  END;
 DISPOSE(POINTER);

UNTIL ((VERTEX@.RESULT > STOREDWORST@.FILESTORERESULT) OR
   (WORSTVERTEX = NUMVERTICES) OR (VERTEXNUMBER > = 15));

IF VERTEX@.RESULT > STOREDWORST@.FILESTORERESULT THEN
 BEGIN
  RESET(SUMSAVE);
  NEW(SUMACROSS);
  FOR I : = 1 TO CURRENTNUMCHEMS DO
   FOR J : = 1 TO NUMYEARS DO
    BEGIN
     FOR K : = 1 TO NUMDAYSPERYEAR DO
      BEGIN
       READ(SUMSAVE,SUMACROSS@.SUMACTIONS[I,J,K]);
       SUMACROSS@.SUMACTIONS[I,J,K] : =
         SUMACROSS@.SUMACTIONS[I,J,K] +
         VERTEX@.ACTIONS[I,J,K] -
         STOREDWORST@.FILESTOREACTIONS[I,J,K];
      END;
     READLN(SUMSAVE);
    END;
  REWRITE(SUMSAVE);
  FOR I : = 1 TO CURRENTNUMCHEMS DO
   FOR J : = 1 TO NUMYEARS DO
    BEGIN
     FOR K : = 1 TO NUMDAYSPERYEAR DO
      WRITE(SUMSAVE,SUMACROSS@.SUMACTIONS[I,J,K]:8);
     WRITELN(SUMSAVE);
    END;
  DISPOSE(SUMACROSS);
  REWRITE(TSTOREVERTEXTEMP);
  WRITETEMP(VERTEX@.RESULT,VERTEX@.ACTIONS);
  RESET(STOREVERTEX);
```

```
   IF SKIP2 = 1
    THEN
     BEGIN
      FOR LINECOUNT2 : = 1 TO (CURRENTNUMCHEMS*NUMYEARS+2) DO
        READLN(STOREVERTEX);
      FOR N : = 2 TO NUMVERTICES DO READORIGWRITETEMP;
     END
    ELSE
     BEGIN
      FOR JUMP : = 1 TO (SKIP2-1) DO READORIGWRITETEMP;
      FOR LINECOUNT2 : = 1 TO (CURRENTNUMCHEMS*NUMYEARS+2) DO
        READLN(STOREVERTEX);
      FOR JUMP : = (SKIP2+1) TO NUMVERTICES DO READORIGWRITETEMP;
     END;
   RESET(TSTOREVERTEXTEMP);
   REWRITE(STOREVERTEX);
   FOR N : = 1 TO NUMVERTICES DO READTEMPWRITEORIG;
   RESET(STOREVERTEX);
   SORT;
  END;
 DISPOSE(VERTEX);
 DISPOSE(STOREDWORST);

UNTIL ((WORSTVERTEX = NUMVERTICES) OR (VERTEXNUMBER > = 15));

END. {Program CORE}
```

VITA

Richard Francis Kazmierczak, Jr. was born on January 8, 1959 in Detroit, Michigan. After graduating from high school in 1977, he entered Michigan State University and took advantage of the flexibility offered to Honors College students by doing concentrated study in a wide range of subjects, including biology, ecology, economics, and religion. Following the 1981 conferral of a Bachelor of Science degree in Fisheries and Wildlife, Richard entered Virginia Polytechnic Institute and State University to pursue studies of ecosystem dynamics. By 1984, however, a desire to engage in policy analysis and the opportunity to become involved in interdisciplinary bioeconomic research led to a full-time appointment with the Extension Division, and eventually to a Ph.D. program in Agricultural Economics. Throughout this latter period of study, Richard maintained an intense interest in the interface between biological and economic systems. Richard completed the requirements for his Ph.D. degree in July, 1991 and accepted a faculty position in the Department of Agricultural Economics and Agribusiness at Louisiana State University.

Richard Francis Kazmierczak, Jr.